2019 AVA Annual Conference

AVA
AUSTRALIAN VETERINARY ASSOCIATION

Proceedings of the Australian Veterinary Association Ltd Annual Conference, Perth 2019

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Proceedings of the Australian Veterinary Association Ltd, Annual Conference, Perth 2019

Held at the Perth Convention and Exhibition Centre, Perth, 5-10 May 2019

Thank you to the AVA Conference Program Committee

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AVA President's Welcome

Each year the AVA conference is the highlight of my calendar. The 2019 AVA Annual Conference is a credit to the dedicated Scientific Committee, the team of AVA staff, all of the AVA Groups, members, volunteers and industry supporters who have worked tirelessly to make this event a success.

This team has engaged a stellar line up of speakers, both local and international. After an outstanding debut in 2018, we have built on the VetED talks for 2019. To ensure no one misses out on this showcase of inspirational stories and thought provoking ideas from members of our profession, we have moved this session to be one of our plenaries.

I am particularly looking forward to our Food Security Day on Thursday 9 May. This day is themed "Rising to the Challenge - Food Security, Biodiversity and Welfare", and is relevant to veterinarians whether they are involved in animal production, animal welfare, conservation biology or just want to be well informed of the scientific debate.

Our fantastic social program will not go unnoticed, as well as the opportunity to extend your stay and explore some of the wonders of Perth and the greater West.

I can't wait to see you at the conference!

A note from the Convenor

On behalf of the Conference Scientific Committee and the AVA - I hope you have a great conference experience this year. We have tried very hard to create a conference program which will appeal to all facets of our profession and help all vets to keep abreast with the changing science and structure of our profession.

The core conference program contains - the latest in Small Animal soft tissue surgery with Catherine Sturgeon from the UK, we have Gayle Hallowell speaking on the latest advances in equine fluid therapy, clinical pathology and management of the sick foal. The Dental and Behaviour streams have also invited world eminent speakers and all international speakers are supported by a full and varied cast of talented Australian speakers.

This year we have included a number of sessions to help us all to understand and appreciate and manage the world we are living and working in today. These sessions include subjects such as unconscious gender bias, the science behind veterinary communication, a change management workshop, career pathways session and a plenary on the nuances of "Work Life Balance".

Animal Welfare and sessions looking at the future of the profession also feature strongly in the program. For those who are interested in the bigger picture - we have a full day looking at Food Security into the future. World renowned speakers will consider options for the future and the effects of food production on the environment, biodiversity, animal welfare, biosecurity and role of the veterinary profession.

The scientific committee have made a huge effort to develop a program which will engage all the profession and make sure all delegates go home with new skills, new perspectives and hopefully new friends and alliances.

Putting this program together with the beauty of Perth and Western Australia, the wonderful array of social events and the excellent workshops scheduled before and immediately after the conference - has been both exciting and a pleasure and we look forward to catching up with you all during the conference.

Dr Sue Beetson

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2019 AVA Annual Conference

Conference Program

Perth Convention and Exhibition Centre
5-10 May 2019

Sunday 5 May 2019	
1:00-5:00	The AVA Innovation, Research and Development Symposium - Meeting Room 8
6:00-8:00	Welcome Reception and Exhibition Opening - Exhibition Hall

Monday 6 May 2019	Riverside Theatre	River View Room 4	River View Room 5	Meeting Room 1
Stream	Small Animal	Behaviour sponsored by Vetoquinol	Business, Industry	Equine
8:15-9:15	**Feline urethral obstruction and rupture** A practical step by step approach to assisting the blocked cat and to revise the options for managing urethral disruption Catherine Sturgeon	**Environmental enrichment: can living conditions lead to cat's behavioural problems?** Environmental Enrichment can be both a way to prevent behavioral problems and treat the existing ones. Gonçalo da Graça Pereira	State of the market panel - An update on key changes in the boarder business environment and the Australian veterinary business landscape Panel	**Practical fluid therapy for use in the field and clinic** Which fluid types and when to use them in a variety of case based scenarios Gayle Hallowell
9:15-10:15	**Feline tail pull injury** Urinary dyssynergia is a tragic consequence of a sudden traction force to the tail. We will review how best to manage these cats. Catherine Sturgeon	**Approaching main causes of elimination problems in cats** Is it simply a toilet or do details make the difference? Gonçalo da Graça Pereira		**Handling horse emergencies at an event** How to handle horse emergency scenarios at horse sporting events Judith Medd
10:15-10:45	Morning Tea - Exhibition Hall			
10:45-12:15	President's Welcome Plenary - Work-Life balance; the Myths, the Lessons and the Responsibilities Nigel Marsh			
12:15-1:45	Lunch - Exhibition Hall			
Stream	Small Animal	Behaviour sponsored by Vetoquinol	Business, Industry	Equine
1:45-2:45	**He ate what?** An exploration of the management of a range of ingested toxins and foreign bodies Katrin Swindells	Understanding emotional development in dogs and why it matters Kersti Seksel	**Keeping vets happy at work:** Results of BVA project on workplace satisfaction, unconscious bias and gender discrimination in the veterinary profession Gudrun Ravetz	**Getting the most from clinical pathology - what's useful and what's not** What is the value of clinical pathology, above physical examination, in critically ill equine cases Gayle Hallowell
2:45-3:30	Afternoon Tea - Exhibition Hall			
3:30-5:00	AVA Awards Ceremony and Annual General Meeting			
5:00-6:00	AFAST and TFAST: How can I get the best from these tests? Learn more about these tests and their application in everyday practice Zoe Lenard	Trauma and its effect on the brain What do patients need to process and overcome traumatic events Jacqui Ley	What do Australian customers say about vets? Find out what the research says about what your clients think and how they make decisions Alison Lambert	How to manage the non-weightbearing horse Foot abscess or fracture? How to diagnose and treat horses that present with severe lameness. Josie Hardwick
6:00-7:00	Happy Hour - Exhibition Hall			

** This program was correct at the time of printing but is subject to change. For the latest program visit http://conference.ava.com.au

This session is to be recorded as a podcast

Sunday 5 May 2019	
9:30-4:45	The AVA Innovation, Research and Development Symposium
6:00-8:00	Welcome Reception and Exhibition Opening - Exhibition Hall

Monday 6 May 2019				
	Meeting Room 3	Meeting Room 6	Meeting Room 7	Meeting Room 8
Stream	Cattle	Dentistry, Unusual Pets	Sheep, Camelids, Goats	Public Health
8:15-9:15	**Insurance or bull** Attention to detail in both an insurance proposal and a future claim can help avoid undue pressure and ethical dilemmas following the breakdown of a bull Alan Guilfoyle	**Examination and diagnosis of oral diseases in rabbits and rodents** Good anatomic knowledge, clinical assessment and radiographs are the keys to diagnosis and treatment Jerzy Gawor	How To: **(a) Overview of disease diagnostic approaches -** How to systematically investigate diseases of small ruminants Peter Windsor **(b) Decision making and problem solving practise in practice -** Examining the key attributes of a skilled, experienced, problem solver veterinarian Helen McGregor	**(a) What makes an effective One Health practitioner? Opinions of Australian OH experts** Sandra Steele **(b) One Health for Systems Strengthening** A presentation on One Health research currently being conducted in the Asia-Pacific region and how this work benefits Australia Francette Geraghty-Dusan
9:15-10:15	**Slings & surgery: A clinical perspective on bull sheaths** The applied anatomy, pathophysiology and clinical management of conformational problems of the bovine preputium Peter Irons	**Rabbit dentistry - common treatments** Oral and dental pathology is one of the most common disorders encountered in rabbits. The general practitioners must know the basics Jerzy Gawor	How To: **(a) Animal Welfare and Farmer Depression: How to broach the elephant in the room?** When a farm visit discloses an impending disaster. How do we get farmers to have those difficult discussions? Susan Swaney **(b) Faecal worm egg count reduction testing:** best practice protocols to ensure optimum efficiency and accuracy Brown Besier	**International perspectives on One Health** One Health in livestock research in Africa and Asia Delia Grace
10:15-10:45	Morning Tea - Exhibition Hall			
10:45-12:15	President's Welcome Plenary - Work-Life balance; the Myths, the Lessons and the Responsibilities Nigel Marsh			
12:15-1:45	Lunch - Exhibition Hall			
Stream	Cattle, Industry, Public Health	Dentistry	Sheep, Camelids, Goats	Welfare, Industry
1:45-2:45	**Syndromic surveillance – a biosecurity frontline for northern Australia** An overview of the unique animal biosecurity risks to northern Australia, and the systems in place to address them. Cassandra Wittwer	**Use of local anaesthesia in dentistry** Intra-operative oral pain can be managed very effectively with the use of local anaesthetic agents Anthony Caiafa	Research Update: **(a) Alternative engagement methods for improved peri-urban smallholder livestock health and farm biosecurity.** Nic Schembri **(b) Aphid associated photosensitisation** Outbreak of photosensitisation in cattle and horses following the ingestion of aphids Colin Trengove	**Welfare sentience and law: an international perspective** International public opinion and law is changing animal use practices Don Broom
2:45-3:30	Afternoon Tea - Exhibition Hall			
3:30-5:00	AVA Awards Ceremony and Annual General Meeting			
5:00-6:00	**FMD Training - Nepal 2018** Summary of FMD Training in Nepal. What the disease looks like in Nepal and lessons for Australia. Stuart Skirving	**Common dental problems in paediatric patients** The importance of the early identification and treatment of oral pathology in young patients Tara Cashman	Research Update: **(a) Does small lungworm reduce production in heavily infected sheep?** Small lungworm infections are often detected at abattoirs, whilst few obvious clinical signs are seen. Heavy infections may cause production loss Jenny Hanks **(b) Application of virtual herding technology on individual and groups sheep movements and assessing the systems impact on animal welfare** Danila Marini	**Assessment and treatment of chronic pain in small animals** Tools available to assist clinicians in recognise chronic pain in small animal patients and explore treatment options Leon Warne
6:00-7:00	Happy Hour - Exhibition Hall			

Tuesday 7 May 2019					
	Riverside Theatre	River View Room 4	River View Room 5	Meeting Room 1	Meeting Room 3
Stream	Small Animal	Behaviour sponsored by Vetoquinol	Business, Industry	Equine	Cattle, Welfare
8:00-9:00	**Dog attack – Decision making in the critical trauma patient** Initial assessment, diagnostics and treatment of big dog versus little dog/cat interactions and the common sequelae Katrin Swindells	**Who is guilty? Mind? Body? Or both?** The relation between body and mind is well studied in humans. Is it the same in our companion animals? Gonçalo da Graça Pereira	**State of human resources** Panel	**Case discussion on the management of the sick foal** Case examples of some common and less common scenarios encountered in foals and options for management in the field if referral is not an option. Gayle Hallowell	**Aspects of assessing pain during disease and operations in farm animals** Research information indicates that pain can be minimised on farms and the public demand that this be done Don Broom
9:00-10:00	**Supporting the trauma patient** Review the use of thoracic drains, oesophagostomy tubes and confidently plan and action open wound management Catherine Sturgeon	**You know you want it - medication!** Psychotropic medications. What to use, when to use it and how to use it Jacqui Ley		**Rectal tears** Diagnosis, Immediate management and Guiding owner's decision making Pete Harding	**UPDATE: Pain Relief in Cattle** How to systematically investigate diseases of small ruminants Peter Windsor
10:00-10:45	Morning Tea - Exhibition Hall				
Stream	Small Animal	Behaviour sponsored by Vetoquinol	Business, Industry	Equine	Cattle, Industry, Public Health
10:45-11:45	**Surgical haemorrhage: arrest the bleed** Anticipate bleeding, recognise risk and predisposing factors and revise what to do in the event of a intra operative bleed. Catherine Sturgeon	**Noise sensitivity or noise phobia? What is the difference and why it matters** Kersti Seksel	**State of the international market** Panel	**Examination of the 'sore-backed' horse – a practical approach** Physical examination and diagnostic approach on a horse with a sore back and the clinical significance of 'abnormal' findings. Rachael Smith	**Anti microbial stewardship - What's the resistance -** Practical information for discussing antimicrobial stewardship in your practice. Michael Dhar
12:00-1:15	Plenary - The science of vet talk Cindy Adams Gilruth Award				
1:15-2:30	Lunch - Exhibition Hall				
Stream	Small Animal	Behaviour sponsored by Vetoquinol	Welfare, Sheep, Camelids, Goats, Industry	Equine	Cattle
2:30-3:30	**Dealing with dyspnoea: a sensible approach to imaging dogs and cats** A logical and orderly assessment to which imaging test to use in the dyspnoeic patient Zoe Lenard	**Chronic stress and renal disease in cats** The impact in the kidney of environmental stressors existing in the current living conditions of cats will be presented Gonçalo da Graça Pereira	**(a) Novel indicators of pain including facial action units in lambs** Lambs were administered pain relief and assessed for pain post mulesing using behaviour, facial action units and activity. Serina Hancock	**Foal resuscitation: What is possible in the field?** How and when to make a significant difference in the outcomes of critically ill foals in the field. David Byrne	**The future of milking management in Australasia** Based on relevant, and recent research, we have a pathway to optimising milking management for Australian and NZ cows. John Penry
3:30-4:00	Afternoon Tea - Exhibition Hall				
Stream	Small Animal, Business	Behaviour sponsored by Vetoquinol	Sheep, Camelids, Goats, Public Health, Industry	Equine	Cattle
4:00-5:00	**Introduction** Steven Metcalfe **Non-neoplastic and neoplastic disorders linked to desexing in dogs** Activation of luteinizing hormone receptors in non-reproductive tissues may explain the higher incidence of some long-term health problems following desexing Michelle Kutzler	**The dog play study** Dr Jacqui Ley has been surveying Australian dog owners about how they play with their pets. Come and hear the results Jacqui Ley	**Antimicrobial stewardship: Science and regulation** Key elements of AMS, principally for grazing ruminants, and the respective roles of clinical risk assessment and regulation Ray Batey and Helen	**Snake envenomation in the horse** Diagnosis and treatment of elapid snake envenomation in Australian horses. Annemarie Cullimore	**Mitigating eye disease in sheep and cattle destined for live export** The investigation of the causes, treatment and prevention of severe eye disease in sheep and cattle in the live-export supply chain. Michael Laurence
5:00-6:00		**Things you should consider when anaesthetising older patients** Kirsti Seksel	**Antimicrobial stewardship: Science and regulation** Discussion panel	**What is equine chiropractic treatment and how can it help** A detailed description of what equine chiropractic is, who should be performing it, and how it can help to assess and treat abnormal function or pain of the axial musculoskeletal system. Rachael Smith	**(a) Antimicrobial Use and Resistance in Western Australia Dairy Herds: A pilot study** Herb Rovay **(b) Qualitative behavioural assessment of cattle** Can we use assessment of behaviour to indicate welfare of cattle? Anne Barnes
6:00-7:00	Happy Hour - Exhibition Hall				

Tuesday 7 May 2019

	Meeting Room 6	Meeting Room 7	Meeting Room 8	Meeting Room 2
Stream	Dentistry	Unusual Pets	Conservation Biology	Education, Public Health
8:00-9:00	**Understanding tooth resorption and how to manage cases** Understanding the different stages and types of tooth resorption their clinical importance Rebecca Nilsen	**Top ten tips for avian surgery** Avian surgery is often regarded as difficult. The author offers 10 tips to make it easier and successful Bob Doneley	**Science communication to enhance community engagement in wildlife conservation** An overview of the science communication toolbox, to assist those working in wildlife conservation to communicate effectively with the community Phil Tucak	**(a) Why do veterinarians leave clinical practice?** Influences and reasons for veterinary clinicians to stop working in clinical practice Alejandra Arbe-Montoya **(b) Altruism - professional or pathological and fit for purpose in the veterinary profession?** What is altruism, and is it an appropriate trait for veterinary health care providers? Adele Feakes
9:00-10:00	**Treatment options for linguoverted canine teeth in dogs** This malocclusion is associated with pain and thus requires treatment. Different treatment options are available Jerzy Gawor	**(a) Reproductive disease in pet birds: a problem of the chicken or the egg?** Courtney Dunne **(b) Tips on sunshinevirus in phythons** How to tell if your python is infected, and what to do about it Jane Wesson	**Faunal crisis - a time like no other** Managing habitat degradation is a key strategy to prevent threatened species extinctions. This talk will investigate issues and solutions Michael Banyard	**(a) Veterinary education in Australia - a student's perspective** Cultural trends that motivate or dissuade young veterinarians Sam Kiernan **(b) Knights or knaves – are veterinary graduands different to others?** Altruism is 'revealed' using a choice experiment with engineering, science, non-STEMM, nursing and veterinary science students. Adele Feakes
10:00-10:45	Morning Tea - Exhibition Hall			
Stream	Dentistry	Unusual Pets	Conservation Biology	Education
10:45-11:45	**Malignant oral tumours - preparing for the worst** Each proliferative or ulcerative lesion should be considered a potential tumor Jerzy Gawor	**(a) How to approach the neurological Rabbit** How to identify, stabilise and treat the most common neuropathies of Australian rabbits Kelly Giles **(b) Distinguishing between hypomotility and obstruction in the inappetant rabbit** Nicole Su	**(a) Australian wildlife - gaining experience in the field** Phil Tucak **(b) Surviving wildlife admissions in general practice** - Management of wildlife that are likely to be presented to the GP vet practice. How to care for the cases, the environment and yourself Gary Beilby	**(a) Wellbeing in veterinary students: a student led study** The understanding of wellbeing among veterinary students Evangeline Beech **(b) Program development of day one competencies in Small Animal Surgery** Frans Venter
12:00-1:15	Plenary - The science of vet talk Cindy Adams Gilruth Award			
1:15-2:30	Lunch - Exhibition Hall			
Stream	Dentistry	Unusual Pets	Conservation Biology	Education
2:30-3:30	**Oral anatomy and dental charting for practitioners** This presentation outlines clinically important oral anatomy and a practical way to record clinical findings Michael Lawley	**Abnormal faeces in the adult rabbit** True diarrhoea in adult rabbits is uncommon. This talk investigates the possible causes of diarrhoea in rabbits and treatment options Gerry Skinner	**(a) Interactions between land use change, flying-fox ecology and Hendra virus dynamics** Alison Peel **(b) Before it's too late - An overview of conservation and wildlife medicine research projects undertaken at Murdoch University** Kristin Warren	**(a) Do skills translate from project to practice?** How does completion of a research-based project compared to other content within a veterinary degree. Peter Irons **(b) Teaching excellence award presentation**
3:30-4:00	Afternoon Tea - Exhibition Hall			
Stream	**Dentistry**	**Unusual Pets**	**Welfare**	**Education**
4:00-5:00	**Managing oral fractures - the dentists' way!** In head trauma, multiple injuries of maxillofacial structures may occur so the patient requires thorough assessment Jerzy Gawor	**Water quality impacts on the health of freshwater turtles** Poor water quality parameters, such as high nitrate and ammonia, cause anorexia and skin lesions in freshwater turtles Anne Fowler	**Pets in the park** How charitable actions or involvement can result in innumerable benefits, not only for recipients but also givers, companies, community Mark Kelman	**Experience in communication cirricula for veterinary education** Essential education for career success and satisfaction Cindy Adams
5:00-6:00	**Surgical extractions and avoiding complications** This lecture will look at the steps involved in performing a successful and uncomplicated surgical extraction Anthony Caiafa	**Common avian presentations in veterinary practice** This presentation is aimed at veterinary practitioners who do not regularly deal with birds. Mike Cannon	**Assessing and optimising the quality of life of zoo animals** An overview of the unique challenges and complexities of evaluating quality of life in zoo animals Simone Vitali	**(a) When are non-technical skills important? Earlier or later** What influences veterinary students' perceived importance of non-technical skills Madison Helms **(b) Exploring the gap between anaesthesia skill confidence and competence** - Student self-evaluations are often misaligned with supervisor assessments Jennifer Carter
6:00-7:00	Happy Hour - Exhibition Hall			

Wednesday 8 May 2019					
	Riverside Theatre	River View Room 4	River View Room 5	Meeting Room 1	Meeting Room 3
Stream	Small Animal	Behaviour sponsored by Vetoquinol	Business	Equine - Research Session	Cattle
8:00-9:00	**Caesarean section: optimise your approach** Revise your pre, intra and post operative plans and techniques for the C-section Catherine Sturgeon	**What to do when training does not help** Kersti Seksel	**Change Management Workshop** Prepare, manage and reinforce: A practical approach to implementing effective practice changes with your team Sue Crampton and Tania Gover	**(a) Significance and outcome of tarsal fractures on pre-sale yearling radiographs** Fractures in juvenile horses before entering race training Cate Steel **(b) Minimising microbial growth during liquid storage of stallion spermatozoa** A development which allows stallion spermatozoa to be stored at room temperature for up to 7 days Jen Clulow	**Practical aspects of sperm morphology and motility** How to achieve accurate sperm morphology and motility results and their application Tracy Sullivan
9:00-10:00	**(a) New ASAV Standards of Care: Anaesthesia guidelines for dogs and cats** Introduction and discussion Leon Warne **(b) The value of surgical checklists** Surgical checklists have gained popularity, however are they worth the additional time? Carla Appelgrein	**Old cats: behaviour problems and treatment** Relation between aging processes and behavioural changes. Treatment will include 4 different approaches, which will not stop aging, but will delay it. Gonçalo da Graça Pereira		**(a) TIVA in horses: Guaifenesin compared with midazolam** Investigating total intravenous anaesthesia of ketamine, medetomidine, and guaifenesin or midazolam Alexandra Cunneen **(b) Clearance of dexamethasone sodium phosphate following nebulisation of clinical doses in adult horses** Time of dexamethasone clearance from the urine and plasma after nebulisation Nicolle Symonds	**Livestock export - the role of veterinarians** What do livestock export veterinarians do? Balancing animal welfare with regulatory frameworks and commercial realities across the globe. Holly Ludeman and Renee Willis
10:00-10:30	Morning Tea - Exhibition Hall				
Stream	Small Animal	Behaviour sponsored by Vetoquinol	Business, Industry, Education	Equine	Cattle
10:30-11:30	**GDV - great surgery is only half the battle** Optimal outcomes for GDV's require a holistic approach Katrin Swindells	**Do you know what the dog is trying to tell you? Reading body language 101** Kersti Seksel	**Communication in veterinary medicine: mainstream activity or minority sport?** Maintaining communication teaching and learning in the practice setting Cindy Adam	**When gelding goes wrong** Where are we at? Complication management and options to reduce them Pete Harding	**Investigating congenital abnormalities in cattle** Is it due to an inherited abnormality? Peter Windsor
11:30-1:30	Plenary - VetEd Talks				
1:30-2:30	Lunch - Exhibition Hall				
Stream	Small Animal	Behaviour sponsored by Vetoquinol	Business, Industry, Education	Equine	Cattle, Public Health
2:30-3:30	**Acute phase proteins in small animals** Diagnostic, prognostic and monitoring role of the major acute phase proteins Gabriele Rossi	**Life skills for kittens: what can owners do to help kittens grow up into confident cats?** Tips and tasks for owners to work with their kittens in order that they become confident adults Gonçalo da Graça Pereira	**Practical in practice communication** A primer on communication for all team members - the do's and don't of talking to clients and one another Cindy Adams	**Case discussion of when a colic is not a colic** This session, using cases, will discuss the wide variety of cases that mimic abdominal pain in the horse Gayle Hallowell	**Sudden death in cattle and sheep** Investigating sudden death in cattle and sheep in central NSW Bruce Watt
3:30-4:00	**Afternoon Tea** - Exhibition Hall				
Stream	Small Animal	Behaviour sponsored by Vetoquinol	Business, Industry, Education	Equine	Cattle
4:00-5:00	**Should I give maropitant or meloxicam to the vomiting dog?** Inappropriate medical decisions could result in adverse outcomes for patients Katrin Swindells	**Canine aggression: when dogs don't like anyone (part 1)** Why dogs bite and how to treat aggression due to mental illness in dogs Jacqui Ley	**Core skills of veterinary communication** Identify and practice communication skills that have a profound impact on building relationships, improving efficiency and accuracy in the consultation Cindy Adams	**Cardiovascular derangement in the critically ill horse** Underlying pathophysiology of the cardiac changes seen in animals with critical illness Gayle Hallowell	**Automated mastitis diagnostics during milking – technology, performance and challenges** John Penry
5:00-6:00	**Brachycephalics and their comorbidities** With the ever-increasing popularity of brachycephalics our role in their survival is mounting. Carla Appelgrein	**Canine aggression: when dogs don't like anyone (part 2)** Understanding why dogs bite and how to treat aggression due to mental illness in dogs Jacqui Ley	**(a) Ten ways to improve your veterinary mentation** Brian Mc Erlean **(b) Why the long face? Sucking the marrow from your veterinary career** Paul Davey	**Abdominal ultrasound of the colicky horse** How to perform and interpret transcutaneous abdominal ultrasound in a horse with colic Josie Hardwick	**Immune responsiveness as a predictor of health and welfare of Australian dairy cattle** An immune testing protocol for Australian dairy cattle Josh Aleri
6:00-7:00	Happy Hour - Exhibition Hall				

Wednesday 8 May 2019

	Meeting Room 6	Meeting Room 7	Meeting Room 8	Meeting Room 2
Stream	Sheep, Camelids, Goats	Welfare, Industry	Acupuncture	Public Health, Industry
8:00-9:00	**From over the fence: What is the future role of vets in extensive livestock systems** A facilitated workshop; including findings from the last workshop in Melbourne (ASAV/SCGV) to identify the challenges and possible approaches to resolve this complex issue Helen McGregor	**Sentience, welfare, ethics and human entertainment** How should we value other animals and should we allow harms to sentient beings for our amusement? Don Broom	**Fascia - the forgotten tissue (part 1)** The connective tissue as fascia has for long been neglected, new research show that it is important - but why? Rikke Schultz	**Bites, scratches, scrubs - infection control in companion animal practices** Personal and environmental infection control practices are critical to prevent nosocomial and zoonotic infections in veterinary practice. Angela Willemsen
9:00-10:00	**(a) Reproductive wastage in young ewes** Update on the causes of reproductive wastage in young ewes Caroline Jacobson **(b) Diagnosis and management of respiratory diseases in small ruminants** Preparing for the 'cost of respiratory diseases from intensification' requires greater awareness of both the endemic and transboundary respiratory disease risks Peter Windsor	**Tourism and marine mammal welfare** Tourism can lead to poor welfare of captive and wild marine mammals but good practice can minimise this Don Broom	**Fascia - the forgotten tissue (part 2)** The connective tissue as fascia has for long been neglected, new research show that it is important - but why? Rikke Schultz	**(a) AMR in companion animal practice: A tragedy of the commons** Why small animal practitioners should care about AMR and create a culture of stewardship in their clinics. Guy Weerasinghe **(b) Reporting AMR usage at the point of administration** Taking the guesswork out of anti-microbial usage by recording all treatments at the point of administration Ben Madin
10:00-10:30	Morning Tea - Exhibition Hall			
Stream	Sheep, Camelids, Goats	Welfare, Conservation Biology	Acupuncture	Public Health, Industry
10:30-11:30	**(a) Local research update** Update on R&D projects relevant to sheep health and production Caroline Jacobson **(b) The challenge to improving lamb survival that won't go away** Snap shot of current R&D undertaken by Murdoch University and its collaborators. Andrew Thompson	**(a) Legal frameworks for wild animal welfare** Jurisdictions vary in statutory protections provided to wild animals and the regulation of harmful activities like land clearing Hugh Finn **(b) Land clearing: the costs borne by wildlife** Physical injuries and pathological conditions seen in wildlife Nahiid Stephens	**Acupuncture and rehabilitation for polyradiculoneuritis (coonhound paralysis) - useful tips and tricks** Acupuncture combined with biomechanical medicine and rehabilitation techniques can improve the recovery rate of Polyradiculoneuritis Kim Lim	**(a) Innovative ways to enhance early detection of significant diseases** Innovative ways to incentivise and normalise reporting for early detection of pests and diseases are discussed Michelle Rodan **(b) Global burden of animal diseases: problem, solution and legacy** The Global Burden of Animal Diseases will develop a system that provides information on the economic impact of livestock diseases Mieghan Bruce
11:30-1:30	Plenary - VetEd Talks			
1:30-2:30	Lunch - Exhibition Hall			
Stream	Sheep, Camelids, Goats	Welfare, Industry	Acupuncture	Public Health
2:30-3:30	**(a) NUMNUTS - providing pain relief for lamb marking** The research underpinning a new tool for delivering local anaesthetic to lambs at the time of ring application Alison Small **(b) Protozoal infections in sheep** Update on current understanding Caroline Jacobson	**The welfare of animals during transport** Scientific evidence indicates the factors affecting animal welfare during and after transport so some practices should be banned Don Broom	**Acupuncture points on the head with regard to cranio-sacral therapy** Acupuncture points of the head is related to the underlying anatomy of the cranial nerves and to the cranium. Rikke Schultz	**(a) Coxiella burnetii seroprevalence in Australian veterinary workers** Results of a cross-sectional study Emily Sellens **(b) Methods to detect C. burnetii from the environment** A systematic review of sampling methods to detect C. burnetii in the environment Hasanthi Abeykoon
3:30-4:00	Afternoon Tea - Exhibition Hall			
Stream	Dentistry	Welfare, Industry	Acupuncture	Public Health
4:00-5:00	**Introducing endodontics: pulp capping in practice** Preserving a vital immature or mature tooth within the periodontium is the goal of the veterinary dentist whenever possible Jerzy Gawor	**Emerging animal welfare issues for Australian abattoirs** The impact of animal welfare standards and slaughter techniques on welfare in red meat and poultry abattoirs Leisha Hewitt	**Myofascial kinetic lines in horses and dogs (part 1)** The kinematics of the body can be described by continues lines of muscles, fascia and other connective tissues. Rikke Schultz	**(a) Foot and mouth disease: are we prepared for an outbreak?** Australia needs to be prepared for an emergency animal outbreak focusing on various different pre and post outbreak aspects Wilna Vosloo **(b) How to catch an exotic disease** Megan Curnow
5:00-6:00	**Never too old: Geriatric feline anaesthesia for the general practitioner** Tips and essential tools on how to safely and confidently anaesthetise the geriatric cat in general practice Martine van Boeijen	**Mobile abattoirs - One more day on the farm** From March 2019, licensed on-farm mobile slaughter units offer a robust alternative to current processing options Phil Larwill	**Myofascial kinetic lines in horses and dogs (part 2)** The kinematics of the body can be described by continues lines of muscles, fascia and other connective tissues. Rikke Schultz	**(a) Improving detection, investigation and management of emergency animal diseases** Designing a training package to educate veterinarians Tabita Tan **(b) Reconstructing FMD outbreak transmission networks** Evaluation of fitness for purpose of FMD outbreak transmission network models, further model development and application Simon Firestone
6:00-7:00	Happy Hour - Exhibition Hall			

Thursday 9 May 2019				
	Riverside Theatre	River View Room 4	Meeting Room 3	Meeting Room 1
Stream	Small Animal	Business	Equine	Dentistry
8:00-9:00	**Bilary Tract Surgery** We ask ourselves : Why does the bile stop flowing? What are the options and is surgery always appropriate? Catherine Sturgeon	The Hub Topical issue - small group discussion with the experts	**Case discussions on equine emergency and trauma cases** This session will present a wide variety of equine cases and the management using gold standard principles of emergency and critical care in order to obtain an optimal outcome. Gayle Hallowell	**Setting up a dental service** Why is a comprehensive dental service important for your practice? A step-be-step guide to improving your dental service is discussed Michael Lawley
9:00-10:00	**Adrenal surgery** Review the fascinating physiology of these endocrine glands, the havoc they cause when they malfunction and the considerations given to adrenal surgery Catherine Sturgeon		**Medical management of colic in practice** Focus on the conservative management of large intestinal impactions and sand enteropathy. Emma McConnell	**Building the equine dental side of your mixed or equine practice** Join us to discuss some tips when it comes to trying to build the equine dental side of your practice. Oliver Liyou
10:00-10:30	Morning Tea - Exhibition Hall			
Stream	Small Animal, Business	Business, Education	Equine	Dentistry
10:30-11:30	**Take my breath away... a highway to the danger-zone** With an increase in the popularity of our brachycephalic dogs, an understanding of upper airway pathophysiology, methods of assessment and prophylactic management is necessary in order to be the best of the best Aaron Moles	**Veterinary communication: why and how does it go wrong** From paternalism to partnership – building strong client relationships Cindy Adams	**Performing a post race veterinary exam and common findings** Performing a post race veterinary examination at a race course and the use of the Alivecor ECG monitor in post race examinations Judith Medd	**Challenging periodontal disease case management** Periodontal disease is generally described in two stages, gingivitis and periodontitis. Both are manageable although often require very complex approach Jerzy Gawor
11:30-12:30	Ben Cunneen Memorial Plenary - No food security without food safety: lessons from low and middle income countries Delia Grace			
12:30-2:00	Lunch - Exhibition Hall			
Stream	Small Animal	Business	Equine	Dentistry
2:00-3:00	**Oesophageal FB: to scope or operate** Bones, hooks, hoof pairings and chew sticks are amongst the objects we may encounter. We take a look at how to successfully remove Catherine Sturgeon	**How to take the message home to your practice and staff** High performance veterinary practice culture: How to use a relational approach for achieving quality, efficiency and resilience in the work place Cindy Adams	**Strategic deworming in horses: the current thinking** Overview of the current thinking regarding parasite management strategies and anthelmintic resistance, relevant to Australian equine practitioners Emma McConnell	**What went wrong? Mistakes and malpractice in small animal dentistry** In medicine, errors occur but they may occur after intentional or unintentional actions. The first group is classified as malpractice the second is medical error Jerzy Gawor
3:00-3:30	Afternoon Tea			
3:30-4:30	**Enteropathogens in cats and dogs** Common enteric infectious agents causing diarrhoea in cats and dogs and diagnostic tests used to detect these will be discussed Jason Stayt	**Pack your tool kit - big ideas and changes from the week** In this final session, Paula will recap the key learnings from the week and make sure we can all have our tools at the ready Paula Parker	**Dealing with diarrhoea in the adult horse** The systematic approach to diagnosis and treatment of acute diarrhoea in the adult horse Annemarie Cullimore	**Homecare advice - what should we be telling clients?** A detailed look at homecare options to reduce the accumulation of dental plaque and ultimately prevent or control periodontal disease Rebecca Nilsen
4:30-6:00	AVA Member Forum			
7:00-Midnight	AVA Conference Dinner			

Thursday 9 May 2019			
	River View Room 5	Meeting Room 6	Meeting Room 2
Stream	Food Security Day	Career Development	Vets in the Community
8:00-9:00	**Opening and chair** Mark Schipp **Food security internationally and Australia's role: aligning nutrition, biodiversity, welfare** Achieving sustainable food and nutrition security for all by 2050: synopsis, challenges and opportunities Robyn Alders	**(a) Internships and residencies - Australia and beyond** Guy Bird **(b) ASAV GP training** Alistair Webb **(c) Why do your membership** Zoe Lenard **(d) Should I do a PhD** Meredith Flash **(e) A career in the government**	**VetTrain - Landmark veterinary training** Addressing the global challenges to the health and welfare of domestic pets, livestock, wildlife, humans and the environment Maryann Dalton
9:00-10:00	**Conserving Australia's biodiversity, while doubling food production** This talk reviews the current and projected ecological state of Australia's native animal and plants populations, and proposes a bold and budgeted plan to balance increased food production with biodiversity conservation Adrian Ward		**EDVOS pet safety program – The link between animal abuse and family violence** EDVOS has been working to address the issue of pets in families facing violence – recognising the significant role they play in family dynamics and the importance of supporting them too when women and children are fleeing family violence Jenny Jackson
10:00-10:30	Morning Tea - Exhibition Hall		
Stream	Food Security Day	Career Development	Acupuncture
10:30-11:30	**Some animal production methods are unsustainable: factors include poor welfare** The future of animal production depends on consumer attitudes to animal welfare and other aspects of sustainability: be proactive Don Broom	**(a) Tips on how to write a paper for publication** Using illustrations and anecdotes, Anne will give us some of her personal tips and tricks for putting together a readable and publishable scientific paper Anne Jackson (b) Tips on what makes a winning poster	**Fascia planes, acupuncture meridians and points** Effort has been put in the search of the anatomical foundation for acupuncture points. Is it fascia? Rikke Schultz
11:30-12:30	Ben Cunneen Memorial Plenary - No food security without food safety: lessons from low and middle income countries Delia Grace		
12:30-2:00	Lunch - Exhibition Hall		
Stream	Food Security Day	Behaviour sponsored by Vetoquinol	Acupuncture
2:00-3:00	**Wildlife conservation programs in pastoral landscapes in Northern Australia** Pastoralism is a dominant land-use in Northern Australia. AWC is working with pastoralists to integrate biodiversity conservation with cattle production John Kanowski	**I'm getting bald! Is it stress?: feline psychogenic alopecia** How to diagnose and approach Feline Psychogenic Alopecia? Which are the steps to achieve the final diagnose? Which are the main possible treatments to support these animals? Gonçalo da Graça Pereira	**Myofascial kinetic lines and acupuncture meridians (primary and 8 extras)** Most of the Myofascial Kinetic Lines follow acupuncture meridians very well and the ancient Chinese literature actually describe fascia, hear how Rikke Schultz
3:00-3:30	Afternoon Tea		
3:30-4:30	**Australia and the global context** The global context, and Australia's position, regarding one health, food security and the environment from the Australian Chief Veterinary Officer Mark Schipp	**Noise reactivity: diagnose and treatment** Diagnose of Noise Reactivity is based in the behavioural signs. The treatment will depend on these signs, but it can be from a environmental adaptation, to behavioural modification plan, including pharmacologic approach Gonçalo da Graça Pereira	**Test and treatment of the lines with specific acupuncture points** Along site the dissections our colleague Dr. Tove Due has developed diagnostic tests and treatment points for each Line Rikke Schultz
4:30-6:00	AVA Member Forum		
7:00-Midnight	AVA Conference Dinner		

A systematic review and critical appraisal of sampling methods to detect *Coxiella burnetii* in the environment

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Introduction

Q fever is a zoonotic disease caused by the intracellular bacterium, *Coxiella burnetii*. The primary mode of transmission of *C. burnetii* is by inhalation of aerosols originating from infected animals and contaminated environments. The organism has a very low infective dose, can persist in the environment for long periods and can become wind-borne. Detection of *C. burnetii* in the environment is therefore important during human and animal outbreak investigations and to inform recommendations for the control and prevention of Q fever. In this study, we systematically reviewed and critically appraised published literature on the methods used to detect *C. burnetii* in different environmental samples.

Methods

A search of four electronic data bases (Web of Science [all databases], CAB Abstracts, Scopus and MEDLINE), with subsequent hand searching, identified 46 eligible records published in English since 1950.

Results

These studies described the sampling of mainly dust, air, soil and water to determine the presence of *C. burnetii* during outbreak (37%) and in non-outbreak (63%) situations from around the world. A larger portion of studies were published between 2010 and 2015. The most commonly tested sample type was dust, which was collected mostly by wiping surfaces with swabs, cloths or sponges. The majority demonstrated the presence of *C. burnetii* by detection of DNA using PCR based techniques, while rRNA pyrosequencing and microarray analysis were used infrequently. Eight studies attempted and succeeded in demonstrating viability of the organism in these environmental samples. Although many studies investigated *C. burnetii* at repeated time points, the extent of research on geographical contamination and spread was low.

Discussion and conclusions

Identifying a single best environmental sampling method to detect *C. burnetii* is challenging to due to the variability in methods used among reviewed studies. The utility of these studies for managing or preventing outbreaks is questionable as they typically only focus on single samples. Targeting multiple environmental sample types is recommended to avoid misdetection during endemic or outbreak situations. Future research should focus on validating and standardising sampling methods.

Practical In Practice Communication: How to Maintain and Enhance Communication Skills for Success

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Communication is a core clinical skill that needs to be taught, learned and implemented in practice settings with intention.[1,2] (Adams and Kurtz 2006) Veterinary schools across Australia, Caribbean, Europe, Japan, North America, United Kingdom and more recently Latin America have developed or commenced development of communication skills programs and assessment strategies. These initiatives have been fuelled by overwhelming evidence that points to the need for communication skills training critical for practice success. There is 40 years of research in human medicine linking communication to outcomes including diagnostic accuracy, satisfaction, understanding and recall, compliance, and reduction in complaints. In the area of veterinary medicine there is close to 15 years of research building on evidence from human medicine but within the veterinary context. Communication is closely linked to clinical reasoning, medical problem solving and significant outcomes of care such as accuracy, efficiency, supportiveness, adherence to treatment recommendations, and client and veterinarian satisfaction.[3,4,5,6,7] Over the last 10 or so years communication skills education and training is taking place on site in veterinary practices resulting in improved relationship-centred communication characterized by a balanced distribution of talk between the veterinarian and the client, stronger partnerships and rapport between practice team members and clients and attainment of the client's perspective (needs, concerns, feelings, expectations) which is critical when giving information to clients and helping them make decisions in the best interest of the patient(s). Development of communication to a necessary level of competence is no longer optional if a practice wants to achieve important outcomes. The practice of excellent veterinary medicine is inextricably linked with skilled communication – you cannot have one without the other. This is true no matter whether you work in rural or urban settings primary or specialty care, small animal, equine, production animals, exotics or public health.

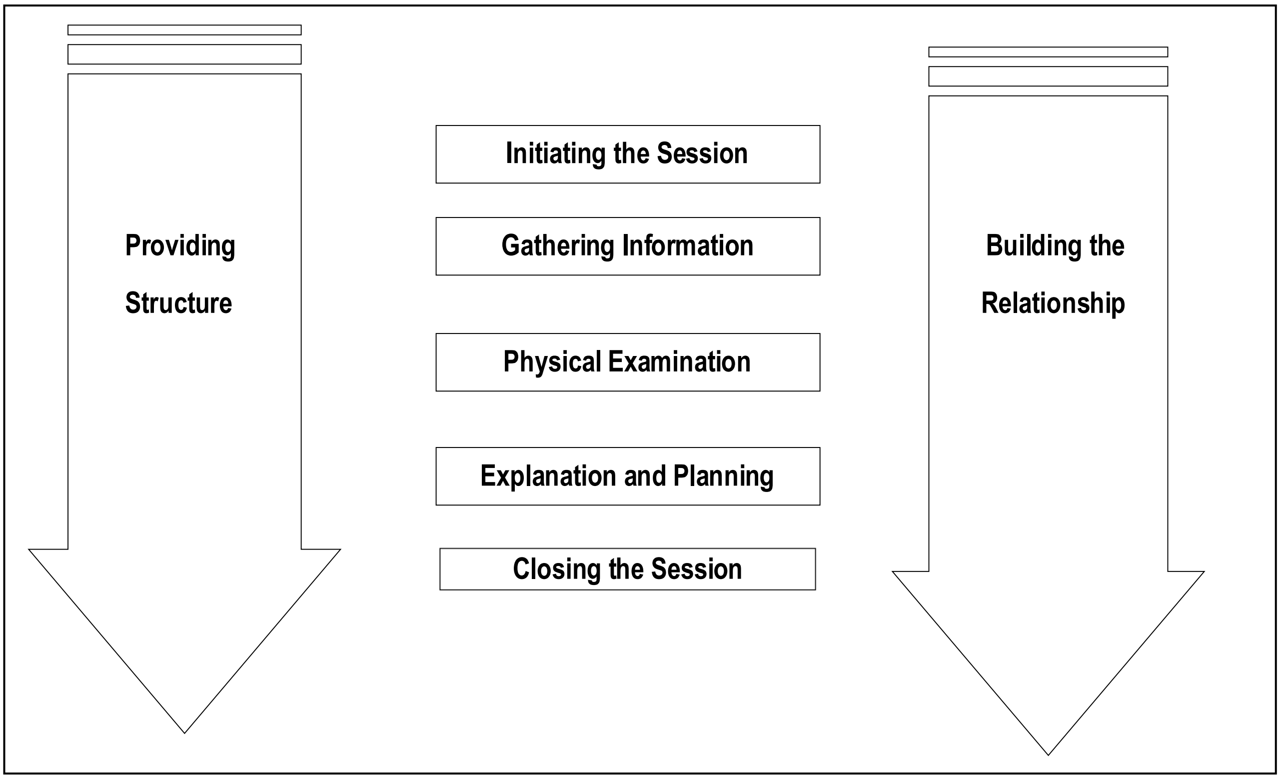
Communication sills are a vital aspect of interactions between veterinarians, nurses, receptionists, managers and there are three broad types of communication skills that need to be taught and learned: content, process and perceptual skills. Content skills are what the animal healthcare professionals communicate – the substance of their questions and responses, the information that they gather and give, the treatments they discuss. Process skills are the ways that the professionals communicate with clients and one another, how they go about getting the history or providing information, the verbal and non-verbal skills they use, how they develop the relationship with the client and the way they organize and structure the conversation. Perceptual skills have to do with what the healthcare professional is thinking and feeling including their clinical reasoning and problem solving skills; their attitudes, values and beliefs; their awareness of feelings and thoughts about the client and patient; their awareness about their own confidence, biases and values. It's important to note that all three skills are highly integrated and cannot be considered in isolation. Attention to one skill without consideration of the others has a significant impact on the communication and its impact on animal, client and health of the practice.

So what does it take to teach and learn communication? The teaching and learning of clinical communication skills requires the same level of attention and intentionality as the teaching and learning of any other clinical skill. Whether simply watching seasoned people interact with clients works to advantage in practice settings (or not) depends on the level of awareness and the modelling and coaching skills of those who take on the role of 'teaching' communication. Reading articles, attending lectures, and watching demonstrations of people communicating (live or recorded) during staff meetings or rounds can be useful ways to raise awareness and increase knowledge about communication but does not necessarily enhance communication skills and competence for practice. For the purpose of this paper we will discuss the top 3 elements that are necessary for moving communication competence forward in the practice environment and the preconditions necessary to develop and maintain communication teaching and learning in veterinary practice settings. First, the top 3 elements necessary for teaching and learning communication include the need for a:[8]

1. Detailed and clearly defined skills to be learned
2. Way to observe or record communication skills in action followed by discussion
3. Conversation that includes well-intentioned detailed feedback about what the observed is doing that is effective and what would make the communication more effective

Detailed and clearly defined skills to be learned:

The most comprehensive, applicable and utilized framework for communicating in veterinary medicine is the Calgary-Cambridge Communication Process Guide. The guides were originally developed for human medicine and later adapted for veterinary medicine.[9,10] The guides consist of 73 communication skills organized around an framework including: initiating the interaction, gathering information, physical exam (if it is part of the interaction), building a relationship, structuring the conversation, explaining and planning with the client(s), closing and making plans for follow up with the practice. Fifty-eight of the evidence based skills are process skills plus an additional 15 process skills related specifically to giving information to clients and making plans for follow up. While the categories inherent to this framework seem logical, use of essential communication skills that are identified are often overlooked including discovering the client's views and expectations, negotiating, encouraging questions, repetition of advice, checking for understanding and categorizing information. The guides are the backbone of communication teaching and learning in veterinary education, continuing education and practice around the world. Figure 1 shows the organizational framework for the Calgary-Cambridge process guide.



Way to observe or record communication skills in action followed by discussion :

The second essential for maintaining and enhancing effective communication in practice is observation of animal heath care workers while they communicate with clients. This may sound to be too difficult or intrusive but it is critical to be able to obtain a baseline of understanding of the other person's skills and areas that would benefit from enhancement. One strategy might involve observing the team member in real time intervening if useful, by modelling relevant communication skills followed by a debrief after the client interaction. During the debriefing and if time is available it is useful to discuss and practice impromptu, alternate ways of communicating. This is where the list of 73 skills talked about above is helpful in focusing on specific areas of the interaction – initiating the interaction for example – that can be enhanced with the opportunity to rehearse. Lets assume for example that the observer noticed that when the nurse brought a new client into the exam room she did not clarify her role and the client assumed that the nurse was the veterinarian. A simple rewind of the initiation portion helps solidify the skills of clarifying your role when you first meet a client. A second approach for enhancing communication competence in the practice involves installing video or at least audio recording devices in 1 or more exam rooms to record any and all animal health care providers during their consultations. Informed consent needs to be obtained for the client and anyone else who will be on such recordings. The video or audio recordings can then be replayed and discussed with the person interacting with the client. A third option is role-play using practice based vignettes. Focusing attention using role play can be an agenda item for staff meetings or other collaborative activities.

Conversations that includes well-intentioned detailed feedback about what the observed is doing that is effective and what would make the communication more effective:

After observation or recording consultations it is time for well-intentioned feedback for the individual that interacted with the client(s). In order to support the ongoing development of communication at the practice level we must work toward supportive relationships across our staff and doctor compliment. For many, giving and receiving feedback has been a negative experience leaving people feeling belittled, embarrassed or ashamed. Feedback has often come off as telling another person one good thing that took place followed by a litany of faults, omissions or inaccuracies, and then to point out how to do things differently. This kind of feedback can create defensiveness rather than learning and forward movement. For the most part, attention to the importance of a supportive relationship for giving feedback has been overlooked. For feedback to be effective and useful it must be well-intentioned, detailed and descriptive. The person offering the feedback is basically holding a mirror up to reflect back to the other person what they saw and its impact on the interaction with the client. A detailed set of strategies for analysing interactions and giving feedback is referred to as agenda-led outcome based analysis (ALOBA)[8]. ALOBA is a much used and well-recognized framework for organizing and leading feedback sessions in communication program in human and veterinary education as well as veterinary practice settings.

How to get started with a 'program' to enhance communication teaching and learning in practice?

Dedicated or explicit communication teaching and learning at the practice level by practice team members that have the necessary training themselves is not widespread but it's catching on. Success of internal teaching and learning effective communication begins with a desire for excellent communication in practice alongside excellence in all other clinical domains. Success also comes from the creation or maintenance of a supportive workplace that strives for the same level of partnership that we are trying to achieve within the veterinarian/nurse, receptionist-client relationship. Mastery of appropriate communication coaching and teaching techniques must also be obtained and maintained by designated people in the practice. Hiring practices must consider, in addition to clinical skills and knowledge, the ability and desire to work well in a team including learning and implementing effective communication. Another contributor to the success of communication teaching and learning is the creation of space in staff meetings for discussion about communication and opportunities for external continuing education. Challenges abound, the relationship between communication and outcomes important to practice success is too compelling to be left unattended.

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Food security internationally and Australia's role: nutrition, biodiversity, welfare

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Key Messages

As veterinarians, we have a responsibility to encourage:

(1) Evidence-based debates on sustainable human and animal nutrition and appropriate welfare;
(2) Use of food for people and feed for animals that are ecologically, economically and socially sustainable;
(3) The keeping of appropriately-nourished companion animals with a low carbon footprint; and
(4) Land management practices involving animals that enhance soil health and biodiversity.

Introduction

Defining and facilitating sustainable and ethical food systems in support of human, animal and planetary health is among the greatest challenges facing humanity today.[1] As the global population grows from 7 billion in 2010 to a projected 9.8 billion in 2050, and incomes grow across the developing world, overall food demand is on course to increase by more than 50 percent, and demand for animal-based foods by nearly 70 percent .[2] Even with our current population, over 10 per cent of people globally are undernourished and approximately 30 per cent are deficient in key micronutrients.[3] The overwhelming burden of child undernutrition exists in low- to middle-income countries (LMICs).[4] Approximately 50 per cent of pregnant women in these same countries suffer from anaemia. In high-income countries, unacceptable levels of obesity and micronutrient deficiencies occur more frequently in vulnerable groups of people and animals.

Sustainable Development Goals: veterinarians, biodiversity and food and nutrition security

As veterinarians, we have a complex set of responsibilities as we serve both animals and people. How do we balance the need for increased quantity and improved quality of food for people while also ensuring the survival of other animal species? Sustainable food and nutrition security lies at the heart of the United Nations Sustainable Development Goals (SDGs).[5] Interlinkages between the SDGs highlight the fundamental role of a healthy, diverse environment as a key factor in achieving sustainable human health and wellbeing.

In making decisions about how to meet food needs and preferences, it is essential to take local agro-ecological conditions into account.[6] In LMICs, efficient and appropriate consumption of animal-source food (ASF) can provide proteins with an optimal balance of amino acids to meet human requirements, as well as bioavailable micronutrients such as haem iron and preformed vitamin A that can significantly enrich cereal-based and energy-rich diets.[7,8] These foods are infrequently consumed in many resource-limited settings in LMICs, particularly when the risk of livestock losses to disease is high, and where domestic animals also offer a means of storing wealth and meeting financial and cultural needs. In high-income countries, inadequate access to nutritious and affordable ASF and uninformed food choices due to insufficient knowledge and negative perceptions of cheaper ASF options (such as offal) contribute to the consumption of energy-rich, micronutrient-poor diets by vulnerable groups.

Defining sustainable diets for humans, companion animals, livestock and wildlife is finally becoming a more mainstream discussion. With increasing human population has come: increasing urbanisation; increasing globalisation of food systems; increasing numbers of malnourished people and animals (i.e. under- and over-nutrition and micronutrient deficiency); expansion of agricultural lands; decreasing biodiversity at genetic, species and ecosystem levels and for food and agriculture; and increased global movement accompanied by spread of diseases and pest species.

Australian and global food systems: veterinarians, food, feed and welfare

Population dynamics for both humans and free-living animals are closely linked to food availability. Over the past 10,000 years, agricultural production has fed an ever increasing and, now, increasingly urban human population. Following the end of the Second World War, agriculture has become increasingly industrialised, focussing on the production of increased quantity of food. This increase has been driven by intensification and through expansion of agricultural activities into new areas. Over the past 200 years in Australia, agricultural land use has increased significantly and has been associated with a loss of half the soil organic carbon, increased clearing of land, decreased biodiversity, an overall decrease in rainfall in cleared areas and declining nutritional profile of food. Clearly these trajectories are not sustainable. It's interesting to note that while Australia's agricultural production has been hailed an economic success story with an important role in contributing to regional food security, our own national health statistics show an increasing number of households that are food insecure and individuals who are malnourished. The growing rates of obesity and increasing micronutrient deficiency in Australians (and our companion animals) are alarming, especially in vulnerable groups such as women of reproductive age and the elderly. To our immediate north, approximately half of the children in Timor-Leste are stunted, limiting their ability to reach their genetic potential in terms of cognition, physical strength and health. Poor diet plays a large part in the malnutrition seen in both countries. For many Australians, 40 per cent of our diet consists of junk food. In Timor-Leste and many other LMICs, diets, especially for infants and young children, are monotonously cereal-based. The addition of even a small quantity of animal-source food would transform the nutritional quality of their diets.[7] More broadly, the German Nutrition Society states that it is difficult or impossible to attain an adequate supply of some nutrients with a pure plant-based diet.[9] They do not recommend a pure plant-based diet for pregnant women, lactating women, infants, children or adolescents. In sustainability terms, a pure plant-based diets supports a lower human carrying capacity than diets such as ovo-lacto vegetarian.[10]

How do we respond to this multifaceted situation? The latest strategy to develop the north of Australia emphasises the opportunity to increase food production to meet the growing demand in our region. How can we employ the principles of regenerative agriculture[11] to prevent further loss of soil health, water resources and biodiversity, control invasive diseases and pests and still make enterprises financially viable for generations to come? The sustainable use and conservation of biodiversity for food and agriculture call for approaches in which genetic resources, species and ecosystems are managed in an integrated way in the context of production systems and their surroundings.[12] In particular, for many types of associated biodiversity and wild foods, sustainable use and conservation require in situ or on-farm management integrated into strategies at ecosystem or landscape levels with ex situ conservation serving as a complementary strategy. How do we care for the soil and wider environment, as well as encouraging debate around what type of animal-source food is produced and for whom? Australia's arable land, that is land used for cropping, has dropped to around 6 per cent of land mass. Does it make sense to use this precious resource to produce crops that are fed animals that are then eaten by people? Nutritional modelling suggests that a more sustainable diet would be achieved by ensuring that human-edible food (e.g. cereals, pulses, etc) is eaten directly by people.[13] The environmental impact of animal production could be significantly reduced in Australia if we consumed more of the carcass and producers were paid for all edible parts of the carcass. In many cases, the highest concentration of micronutrient-rich food is lost to the human food chain in Australia partly due to changing dietary preferences.[14] The scarce nutrients in offal are used for dog and cat food, for fertilizer or are exported to countries that still value them. In terms of carrying capacity, our livestock herds could be reduced and still provide a similar quantity of nutrients if more of us adopted nose-to-tail eating habits. In terms of the growing demand for high-quality dog and cat food, should we be recommending that owners consider switching to food containing insect protein sources or keeping herbivorous companion animals?

The future of food and feed: veterinarians' role in global change

Our veterinary training in comparative physiology, nutrition and health makes us invaluable members of the team effort that is required to achieve sustainable development in Australia and globally. Veterinarians have and continue to play a crucial role in the prevention and control of infectious disease, nutrition science, conservation biology and food and nutrition security across a wide range of species. Our profession is well-placed to contribute to achieving many of the SDGs, from SDG2 (zero hunger and sustainable agriculture) to SDG3 (ensuring healthy lives and promoting wellbeing for all) to SDG12 (responsible consumption and production). In the 21st century, veterinarians must hold as a key concern, not only the health, welfare and respectful treatment of animals, but also the ecological systems that sustain them and us.

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Immune responsiveness as a predictor of health and disease in Australian dairy cattle

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Abstract
This paper describes our current approach on the assessment of immune responsiveness as a predictor of dairy cattle health and disease under the Australian pasture-based production system. Furthermore, the relationships between antibody and cellular immune responses and health and performance traits (stress responsiveness, internal parasite burden and milk production and quality) were investigated. The immune testing protocol developed herein was reliable for inducing and measuring immune responsiveness in Australian dairy cattle. Results suggested that animals with superior immune competence are better able to cope with management-induced stress. No significant correlations between immune competence and productivity were observed.

Introduction
The welfare of dairy cattle may be compromised during the periparturient period, due to the increased incidence of health disorders such as mastitis, milk fever, retained fetal membranes and gastrointestinal conditions observed during this period (Uribe et al., 1995, Berry et al., 2003, Cummins et al., 2012). Furthermore, increased incidence of these disorders during the periparturient period has a significant negative impact on herd profitability (Kossaibati and Esslemont, 1997). Previous selection for production traits, with limited emphasis on survival and longevity traits, is thought to have contributed to the increased incidence of periparturient health problems observed in modern dairy cows (Miglior et al., 2005, Oltenacu and Broom, 2010). It has also been shown that modern dairy cattle can experience a prolonged state of periparturient immunosuppression that can result in increased periparturient health disorders (Aleri et al., 2016). These observations were a major driving force for developing strategies aimed at predicting dairy cattle health and welfare using immune function testing. This holistic health and production improvement strategy has been referred to as immune competence testing or the High immune technology (HIR). The technique involves ranking of animals based on Estimated Breeding Values (EBVs) of their adaptive immune response phenotypes which include antibody and cell-mediated immune responses that also capture key innate mechanisms required to make robust adaptive responses. Thereafter these responses are correlated with health and production performance. This paper describes our current approach on the assessment of immune responsiveness as a predictor of dairy cattle health and disease under the Australian pasture-based production system as well as assessing the relationships between antibody and cellular immune responses and health and performance traits (stress responsiveness,

internal parasite burden and milk production and quality) in Holstein-Friesian and Holstein-Friesian x Jersey crossbred heifers raised in a pasture-based production system.

Materials and Methods

Part 1: Studies on the development of the immune testing protocol

Experiment 1- In these studies immune competence testing was undertaken to assess antibody and cell-mediated immune responses, using a modified protocol to that used in previous studies (Heriazon et al., 2009; Hernandez et al., 2005). Modifications included the use of different test antigens. In the current study, a commercial clostridial / letosporosis vaccine (Ultravac® 7 in 1, Zoetis, West Ryde, NSW, Australia) was used to stimulate measurable immune responses using the protocol shown in **(Table 1).** Combined immune responses for each animal were generated using their antibody and cell-mediated immune responses. We assessed the repeatability of secondary and tertiary immune responses measured in 100 calves (5-6 months, secondary response) and again as yearlings (12-13 months of age, tertiary response).

Experiment 2 - In another experiment, we compared the ranking of dairy calves for general immune competence using two different testing protocols. The protocols used were identical with the exception of employing different antigens to induce measurable immune responses. In the first protocol specific test antigens, as have been extensively used in the North American dairy industry to assess immune competence, were used (protocol 1) and in the other protocol a commercial 7 in 1 vaccine was used (protocol 2) to induce immune responses. In the study, antibody-mediated immune responses and cell-mediated immune responses were assessed in 40 male dairy calves managed at pasture using both protocols in a cross-over design.

Table 1: Immune testing protocol that was used in the study to assess immune function as a predictor of dairy cattle health and function

Visits	Date / time	Activities / procedures during the visits
Visit 1	Day 0	Bleeding (serum baseline antibody and stress response indicator levels), Immunization
Visit 2	Day 8 or 9	Bleeding (to assess AMIR and stress response), Skin measures, Inject for skin test
Visit 3	Day 10 or11	Bleeding (to assess AMIR and stress response), Skin test reading (to assess CMIR)

Part 2: **Associations between immune competence, stress responsiveness and production in Holstein-Friesian and Holstein-Friesian x Jersey heifers reared in a pasture-based production system in Australia.**

We assessed antibody and cellular immune responses in first lactation dairy heifers reared under a pasture-based production system and to investigate associations between immune competence and stress responsiveness, health and productivity.

Results and Discussion

Part 1, Experiment 1 - In this experiment we further investigated the correlation between immune responsiveness and average daily weight gain. A total of 100 Holstein-Friesian heifer calves were enrolled into the study when 5-6 months of age and followed up as yearlings at 12-13 months of age. The analysis of correlations for immune responses in calves and yearlings showed that the secondary and tertiary antibody-mediated immune responses were well correlated (r=0.651, Adjusted R2=0.418, P<0.0001), whereas correlations between secondary and tertiary cell-mediated immune responses were poor (r=0.078, Adjusted R2 = -0.004, P= 0.450). Based on test results heifers were ranked for AMIR, CMIR or combined AMIR/CMIR. The results on the McNemar's test provided evidence that the proportions of High and low immune responders within the immune ranking groups were not disproportionate, permitting a repeatability test to be performed. The Cohen Kappa (κ) test of agreement was used to test the consistency of immune response category ranking of an individual. The level of agreement for an animal to be identified as either a High or low responder across the two test periods was moderate to high (κ=0.445) for antibody-mediated immune responses, poor for cell-mediated immune responses (κ=-0.055), and moderate for combined immune responses (κ=0.395). The high consistency of secondary and tertiary AMIR suggests that potential benefits could be gained by early ranking of animals based on their immune response. (Mallard et al., 1992; Mallard et al., 2011; Mallard et al., 1998).

In experiment 2, our primary focus was to compare the ranking of calves for immune competence when assessed using two different testing protocols which only differed in the antigens used to induce measurable immune responses. We hypothesized that animals would rank similarly for each immune competence trait when using either test protocol. Results of the study largely supported our hypothesis with rankings for AMIR, CMIR or CIR not differing significantly when assessed using either test protocol. The present findings are not surprising based on the role of genetics on the influence of immune responses. Various studies have suggested AMIR and CMIR are moderately heritable (20-40%) in dairy cattle (Wagter et al., 2000; Abdel-Azim et al., 2005; Hernandez et al., 2006; Thompson-Crispi et al., 2012b). This suggests that moderate genetic gains could be expected from selecting animals based on their inherent general immune competence which is expected to improve their ability to resist a variety of diseases. Selection strategies aimed at improving immune competence are expected to provide a strong platform for breeding animals that are better able to cope with disease challenges encountered in their production environments.

Part 2 - The objectives of this study were to assess the antibody and cellular immune responses of dairy yearling heifers aged 12 – 15 months which were reared under a pasture-based production system and to determine associations between immune competence and stress responsiveness, internal parasite burden, milk production, SCC, milk protein and milk fat yield. Heifers identified as below average for both their ability to mount antibody and cellular immune responses had higher cortisol concentrations following mustering and handling when compared to their counterparts. Therefore, findings of this study suggest that ability to mount antibody responses was (favourably) correlated with ability to cope with management-induced stress; however, the relationship was not strong (r = -0.220, P <0.01). This association can be considered "favourable" as enhanced immune competence was correlated with improved ability to cope with stress (lower cortisol concentrations following handling). Antibody immune responses were also favourably correlated with internal parasite burden as assessed by WEC. No significant differences were observed between immune competence phenotype groupings for cellular, antibody and OIC immune responses and milk yield, SCC, fat and protein content. Phenotypic correlations between immune competence traits and milk production traits were generally weak and non-significant with the exception of cellular responses and milk yield which was favourable and approached significance.

Conclusion

The use of immune function as a holistic health approach has potential to significantly improve of animal health and welfare and improve productivity. Data from these studies confirm that the immune testing protocol developed in the Australian context was a reliable method for inducing and measuring immune responsiveness in dairy cattle. Animals with below average antibody and cellular immune competence had higher serum cortisol concentrations compared with their counterparts following handling, suggesting they had a reduced ability to cope with management-induced stress. Furthermore, a significant negative (favourable) correlation was observed between antibody responses and stress responsiveness. Similarly, correlations between antibody responses and internal parasite burden were significant and negative (favourable). No correlations were observed between overall immune competence and milk yield, milk fat and milk protein content.

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Brachycephalic dogs and comorbidities

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With the ever-increasing popularity of brachycephalic breeds, our role in their survival is mounting.

Upper respiratory tract disease is well described in brachycephalic dogs and recent descriptions further outline the abnormalities in conformation based on CT findings in several brachycephalic breeds (Auger *et al*. 2016). Clinical signs include snoring, inspiratory dyspnoea, exercise intolerance, cyanosis and, in the most severe cases, episodes of syncope (Bright *et al*. 1997). These clinical signs will progress with age as obstructive upper airway disease results in constant negative pressure in the larynx (Torrez *et al.* 2006) resulting in weakening of the laryngeal cartilage and eventual laryngeal collapse. Early intervention is crucial to delay the progression of laryngeal collapse, even in young brachycephalic dogs that may not display severe clinical signs. All brachycephalic dogs will benefit from surgical intervention. The point of early intervention is to slow down the progression of laryngeal collapse. Evaluating the larynx for laryngeal collapse is important for staging, making treatment recommendations and advising prognosis. Furthermore, it is crucial to evaluate the tracheal diameter as this is useful prognostic information to facilitate ongoing management and expectations.

Brachycephalic dogs often display gastro-intestinal signs which increases their anaesthetic risk (Lecoindre *et al,* 2004; Poncet *et al.* 2006). Clinical signs may include unprovoked gastroesophageal reflux or vomiting, which places them at a significant risk for aspiration pneumonia and oesophageal inflammation. They may also display intermittent diarrhoea without dietary change, be underweight despite a healthy appetite, display choking or gagging, and nasal discharge. Reasons for gastro-intestinal signs may include inflammatory bowel disease, delayed gastric emptying, sliding hiatal hernias and oesophageal redundancy. Many owners describe signs of regurgitation when brachycephalic dogs are excited or during episodes of respiratory distress, but this is easily missed or accepted as normal.

Hiatal hernias has been recognized with increased frequency at Murdoch University in brachycephalic dogs. These dogs have failure of coaxial alignment of the dorso-lateral oesophageal rim, creating a laterally widened hiatus with no dorsal margin. This malformation allows the stomach to slide into the chest cavity, termed a type I sliding hiatal hernia. A hiatal hernia will be associated with chronic gastro-oesophageal reflux, which allows either ingesta or fluid from the stomach to pass retrograde into the oesophagus. Oesophagitis is a common sequela because of exposure to gastric acid, pepsin, trypsin, bile salts and duodenal bicarbonate. The severity of the oesophagitis is relative to the frequency and content of the reflux (Washabau *et al*. 2012) and oesophagitis has been documented in brachycephalic dogs (Poncet *et al.* 2005). Severe oesophagitis will also exacerbate regurgitation resulting in a continuous vicious cycle. It is therefore advantageous to diagnose and manage cases as early as possible.

Obtaining a pre-operative diagnosis of a hiatal hernia is challenging. The dynamic nature of the herniation makes it difficult to capture the event with video-fluoroscopic swallowing studies, thoracic radiographs or endoscopy, which then leads to an incorrect diagnosis (Broux et al., 2018; Mayhew *et al.* 2017; Reeve *et al.* 2017). The gold standard diagnosis of a hiatal hernia is at surgery or post mortem examination (Reeve et al., 2017). Surgical exploration also allows for surgical correction of the hernia and an opportunity to obtain intestinal biopsies to diagnose inflammatory bowel disease.

Brachycephalic obstructive airway syndrome has been shown to exacerbate clinical signs associated with a hiatal hernia (Boucher *et al.* 2016). This is due to the development of decreased intrathoracic pressure during inspiration, further contributing to hiatal herniation (Poncet et al. 2006, 2005). Therefore with management of brachycephalic obstructive upper airway disease, gastro-intestinal signs may improve. A factor that should not be forgotten is that laryngeal collapse will be progressive, despite surgical correction of upper airway components. As the laryngeal collapse worsens, this will result in return of increased intra-thoracic pressure and the gastro-intestinal signs will return or become exacerbated as brachycephalic dogs age. We recommend surgical correction of hiatal hernias with upper airway surgery in a dog with a history or regurgitation.

Brachycephalic dogs are complex to manage pre-operatively, intra-operatively and post-operatively. It is crucial to obtain prognostic information such as the stage of laryngeal collapse, tracheal diameter or presence of aspiration pneumonia pre-operatively. It is crucial that gastro-intestinal signs are discussed and aggressively managed. Intensive monitoring and management post-operatively is vital to address the complex interaction of all the disease processes.

This talk will focus on the comorbidities identified in brachycephalic breeds including the following:

- client education
- how to perform an upper airway examination and why this is important
- when to recommend surgical intervention
- the purpose of pre-operative thoracic radiographs
- concurrent gastro-intestinal problems that require surgical considerations such as inflammatory bowel disease and hiatal hernias
- post-operative care considerations

We will explore the current literature and devise a surgical plan for brachycephalic dogs.

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The value of surgical checklists

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Human error is always possible and can be devastating. Surgical checklists have become a topic of discussion but are they worth the additional time to formulate and implement? The volume and complexity of what we do on a daily basis requires input from many, since it has exceeded our individual ability to deliver its benefits correctly, safely, and reliably. Knowledge has both saved us and burdened us, but are we able to create and follow checklists for every possible situation? Is a checklist just an increase in bureaucracy or is it the science of averting human error?

The unreliability of human memory and attentiveness, especially when it comes to mundane matters, increases under the strain of more pressing events such as emergency surgery or less routinely performed procedures (Rodriguez *et al*. 2018). Factors associated with an increased risk of complications in people have been identified and include emergency operations and unplanned changes in the surgical procedure (Gawande *et al*. 2003, Lata *et al*. 2011). Stress increases the risk of complications and checklists may help us reduce the impact of human error. In a stressful situation autonomy can have disastrous consequences. Man is fallible but maybe men are less so (Gawande, 2010).

The surgical safety checklist consists of a series of inquiries or assurances designed to confirm everything is ready. It is usually divided into three stages where the team allows time for communication such as:

Prior to anaesthetic induction:

- Is the anaesthetic machine set-up?
- Has all the required pre-anaesthetic patient parameters been checked?
- Is the surgical site confirmed?
- Are any additional procedures planned under the same anaesthetic?
- Is any excessive blood loss expected?
- Has the nurse confirmed the patient positioning and the equipment required?

Before skin incision:

- Are the patient identity, procedure and surgical site confirmed?
- Is the necessary imaging displayed?
- Is the sterility of the surgical instruments confirmed?
- What is the anticipated surgical time?
- Have antibiotics been administered within 30 minutes prior to incision?

Before the patient leaves the theatre:

- Is the swab count reported and correct?
- Has sterility been compromised?
- Is there any equipment concerns?
- Who will submit the samples collected?
- Who will perform the client communication?
- Is post-operative imaging required?
- Has the post-operative care plan been discussed, including any anaesthetic concerns, surgical concerns, analgesia, antibiotic or other medication requirements.

A checklist can be modified for certain procedures. For example we have created a checklist for complicated cases such as patients requiring an adrenalectomy or brachycephalic upper airway surgery. If an uncommon procedure is performed a team briefing is implemented to ensure all the instruments required are available and the samples required are taken.

The checklist is an insistence that people talk to one another, it is a strategy to foster teamwork. The implementation of surgical checklists can improve the detection of safety hazards, decrease overall surgical complication rates and in-hospital mortality. The report of one of the original prospective studies on implementing surgical checklists across eight human hospitals worldwide concluded that the rate of surgical complications at all sites dropped from 11.0% to 7.0% after introduction of the checklist, and the total in-hospital rate of death dropped from 1.5% to 0.8% (Semel *et al*. 2010).

These findings have been emulated in veterinary medicine. In one study all surgeries performed at the University of Illinois were enrolled and compared before and after the implementation of a surgical checklist. Complications were prospectively recorded when witnessed by an observer, and all other perioperative complications were retrospectively recorded from veterinary records. There were more perioperative and postoperative complications when surgeries were performed without a surgical checklist at 40.9% than there were when surgeries were performed with a surgical checklist at 29.3% (Cray et al. 2018). In another study two similar populations of dogs were evaluated for 2 weeks after gastrointestinal foreign body surgery to determine the impact of a surgical checklist on the surgical site infection rate. The surgical site infection rate had a significant decrease from 19.9% to 11.9% with the use of the surgical checklist (Launcelott *et al*. 2019). A checklist will only help if you measure the actual complication rates, deaths and system failures to identify where things went wrong and what needs to change to fix it. Don't assume complications are normal or to be expected.

During this time we will discuss how to construct a checklist, personalised to suit your situation. Every practice has unique requirements. It should empower you to think about the unforeseen – especially for non-routine or emergency situations. Just ticking boxes is not the ultimate goal, it is to embrace a culture of teamwork and discipline.

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Why veterinarians leave clinical practice?

Reasons for leaving clinical practice by former veterinary clinicians

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Introduction

Veterinary clinicians have an important role in caring for household pets as well as food production animals. Australia is one of the countries with the highest pet ownership incidence in the world and the veterinary industry is central in pet ownership expenditure [1]. Furthermore, rural veterinarians are key for the upkeep of the livestock industry. Therefore having an adequate number of veterinarians to meet the needs of the communities is crucial [2].

Lately there is the perception of a veterinary shortage particularly in rural veterinary practice [3-5] and an anecdotal increase of employment demand for veterinarians with two or more years of experience in general clinical practice (Eagleton, pers. comm.) However, economic models in Australia and overseas indicate low income and stagnant market growth in the veterinary industry as well as an expected oversupply of veterinarians leading to potential underemployment [2, 6-8]. In 2003 a report in Australian rural veterinary services established that, at the time, there was no crisis in veterinary availability but future local and chronic shortages could be possible depending on factors affecting the willingness of veterinarians to work in rural practice [9]. It has been suggested that attrition rates of veterinary clinicians may be an important contributor to the increased need of employees by private veterinary practice [10]. Intention to leave rates up to 37% have been reported in Australia and New Zealand [10] and in 2017 the British Veterinary Association reported that a quarter of surveyed veterinarians wanted to leave the profession and 37% often thought about leaving the profession [11]. This paradox requires further investigation. Understanding why clinicians leave practice and addressing those issues is vital to the veterinary industry.

This study aims to understand the key reasons for veterinary clinicians to leave clinical practice. This qualitative paper will present the reasons provided by former veterinary clinicians during in depth interviews as of why they left clinical practice.

Methodology

The study uses thematic analysis to explore the responses of 26 former veterinary clinicians on their experiences as veterinarians and reasons surrounding their decision to leave clinical practice. Recruitment was achieved through several social medial platforms and alumni offices of four out of seven Australian universities that provide a veterinary degree. The interviews were conducted between February and April 2018. Interviews were semi-structured and between 30-120 minutes long. Inclusion criteria for participants included having graduated as a veterinarian, worked in veterinary clinical practice for a minimum of six month and have left clinical practice within the last ten years. Individuals interviewed described their experiences as veterinary clinicians as well as the factors surrounding their decision-making process into leaving veterinary practice. The interviews were transcribed by independent transcription companies prior to coding. Coding was then done by Thematic Analysis using Nvivo 11 and 12. This project was approved by the Human Ethics Committee in December 2017.

Reasons for leaving clinical practice

Individuals interviewed described their experiences as veterinary clinicians as well as the factors surrounding their process into leaving veterinary practice. The results show a complex decision-making process into leaving which were separated in three major themes: Influential factors, work experiences and impact on the individual. However, it is the combination of these factors that ultimately makes participants leave practice.

Reasons for participants to leave clinical practice can be described as the combination of the following themes:

- Influential factors: Circumstances in the participant's lives outside of work. It can be seen as the way the participant's personal relationships, their motivations to become veterinarians, their mentality and overall health will influence how they approach their work life and how they will perceive their work experiences. For example: Some of the participant's perspectives around work change as their family responsibilities evolve through their working life as shown in the following quote: *'the big calling was for me was the ability to spend time with my children when they wanted to spend time with me. Because I know they'll grow up.'* (P14). Their perceptions of themselves and their work are also shown to be important influential factors as seen in the following example: *'I am very an all or nothing person (...) I like to give it 100% and if I can't give it 100% I'm kind of not prepared to give it 50%.'* (P15). Influential factors become crucial in how work experiences impact the participants and in how they make the decision into leaving clinical practice.
- Work experiences: Situations participants experienced during clinical practice. These were associated with: clinical work itself such as adverse clinical outcome in a patient (e.g. '*I had one anaesthetic death, very traumatic*' (P21)), their employment conditions such as perceived low remuneration (e.g. '*Wages. [laughs] Money. I think veterinarians are not paid anywhere near what they should be paid (...) it's appalling*' (P10)) or their workplace relationships such as workplace bullying (e.g. *'I'm going to be quite honest, there was workplace bullying and sexual discrimination going on, female in the cattle industry and that kind of stuff.'* (P4)).
- Impact: The personal and professional consequences of the combination of influential factors and work experience will have on the participant. Sub themes included: impact on mental health (e.g. *I took what I felt was missing from them emotionally, and I took that on myself. I took on the emotional burden of feeling that sadness and care for the animal* (P6)); relationships (e.g. *'...you never had a break and down times. So all of that impacts on your relationship...'* (P1)) and professional development (e.g. *On a clinical level, I felt that my learning curve have plateaued* (P5)). Once the impact becomes too big to bear, they leave clinical practice.

Summary:

The research suggests that a combination between influential factors and work experiences create an impact in the participants that drive their decision-making process into leaving practice. Targeting some of these elements could potentially increase retention. For example, understanding and acknowledging the professional mentality of veterinarians and their perception of the veterinary industry and adjusting it with education and support should be of benefit. At the same time, improving the work experiences and employment conditions for clinicians by, for instance, increasing wages or implementing anti-bullying strategies in practice may mitigate a negative impact. The combination of both, support and improvement of employment conditions can lead to positive and more pleasant work experiences which may improve retention. However, it is important to acknowledge that intervention and change will be challenging and further research in this area is crucial to develop appropriate retention strategies.

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Faunal crisis – a time like no other

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Introduction

In June 2018 the Australian Government Senate referred the matter of "Australia's faunal extinction crisis" to the Environment and Communications Reference Committee (1) to investigate, amongst other things, the ongoing decline in the population and conservation status of Australia's nearly 500 threatened fauna species. Submissions were received from 409 institutions and individuals including the Australian Veterinary Association. The faunal extinction crisis is a matter of significant public concern.

Veterinarians in Conservation

Veterinarians are involved in many aspects of conservation, conveniently grouped into: conservation medicine; conservation of endangered species; off-reserve conservation; sustainable use of wildlife; import and export control of wildlife trade including quarantine and border control; education and advocacy. These categories cover most of the important work vets do including wildlife disease surveillance, zoo medicine, captive breeding, feral and invasive species control and conservation policy development.

The in-clinic treatment of injured wildlife by vets across Australia amounts to approximately 500,000 cases annually. The motivation for treatment is both welfare and conservation. Unfortunately a significant proportion of animals triaged are euthanased or subsequently die if returned to the wild. Non-threatened species are seen commonly. (2) Most of these animals require rescue because of previous direct or indirect human interaction.

How does the mortality of wildlife seen at wildlife rescue compare with mortality of all causes? Habitat loss, in total area and quality, is the factor most responsible for morbidity, mortality and declining wildlife populations. (3) Land clearing for agricultural purposes and to a lesser extent for urban and infrastructure development is the main cause of habitat loss. (4) Between 2013-2015 it is estimated that 900,000 mammals and 2.6 million birds and 30.6 million reptiles died annually as a result of land clearing in Queensland. (5) Queensland has been identified as a significant 'front' of land clearing (currently active), largely as an historic consequence that land clearing in other productive agricultural areas of SE and SW Australia took place decades or centuries earlier. Using mortality and species data from wildlife rescue in SE Queensland (6) and mortality estimates from land clearing (5) it is clear that the number of animals killed or injured by land clearing exceeds by a factor of 30 the number of animals receiving care in wildlife rescue (Fig 1, Wildlife Mortality: Seen at Animal Rescue v's Land Clearing). These conservation and welfare issues are still largely being ignored by state governments, policy makers and some welfare advocates yet they have been on the radar of conservationists for decades.

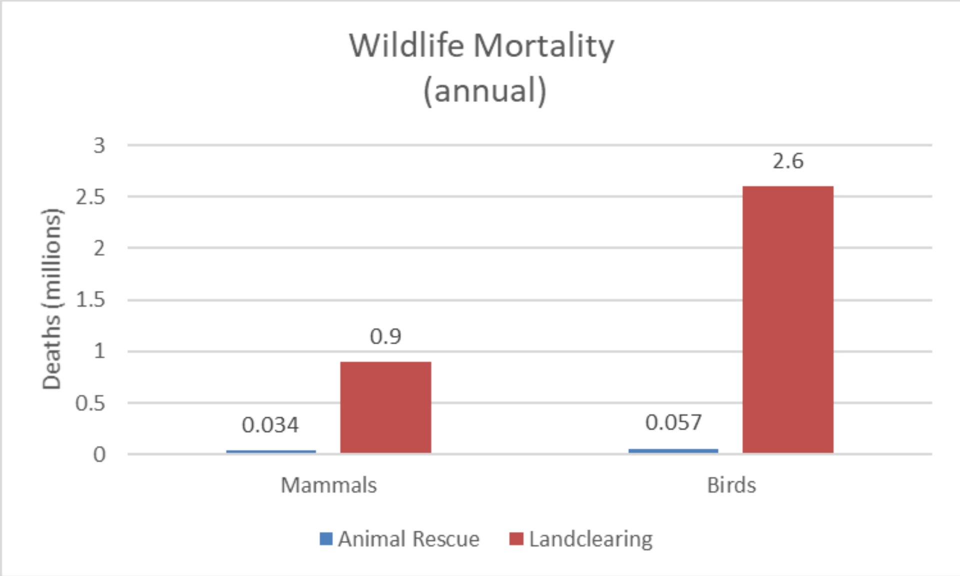

Figure 1. Wildlife Mortality Comparison. Seen at Animal Rescue v's Land Clearing.

This rather distressing picture begs the question, "In what additional ways could the veterinary profession contribute to better outcomes for these un-treated animals?" To consider this question we need to look at the primary drivers and thereby reconsider where the potential intervention points are.

Biodiversity and Food Security

The diversity of veterinary roles in overlapping areas like food production, public health and conservation means that we, as a professional group, have a broad focus and should be in a position to contribute a balanced perspective. Meeting projected human food demand to 2050 in a sustainable manner will be challenging. Without a suite of improvements in productivity and decreased per capita demand in some sectors it has been estimated that an additional area of 593 MHa of agricultural land world wide will be required (7), an area twice the size of India. This is clearly impossible without catastrophic consequences. Food security implicitly and by international agreement operates within the framework of 'sustainable' food security, that is without further loss of biodiversity and additional net green house gas emissions.

Drivers

The key drivers in biodiversity loss in Australia have been identified (3) and can be linked together to show their cause and effect. The three main drivers are: Climate Change, Population Growth and Economic Growth. These 'drivers' are international and their management requires political and global cooperation.

There is an excellent amalgam of international bodies who have recognised and identified the issues. The scientific output and policy options are prodigious, indeed, daunting at times. But without international agreement there can be no way forward. They include the International Panel of Climate Change (IPCC), Intergovernmental Science-Policy Platform on Biodiversity and Ecosystem Services (IPBES), International Union for Conservation of Nature (IUCN), United National Environment Program (UNEP), World Wildlife Fund for Nature (WFF). Conference of Parties (COP) is a governing body of an international convention which has its structure recognised under international law. These bodies, and others not mentioned, have programmes developed over many sessions (COPs) and iterations which are, international, multilateral, comprehensive and actionable. They include: Sustainable Development Goals, Archi Biodiversity Targets and the Agreement on Climate Change.

The point of labouring this is to highlight that Australia doesn't stand alone; our markets, our ethics, our responses have international consequences and this extends from clearing a block of country in the Barrier Reef catchment to maintaining wetlands under the RAMSAR agreement for migratory birds. Just putting a road through a swamp, under the legislative control of a local government, does not operate in isolation and has biodiversity consequences however small it might seem in isolation. However this point is frequently not acknowledged by federal politicians let alone regional councils. Was the development of the Gold Coast preceded by any consideration of its impact on the mangroves affected? Was the push to open up the US West with railroads and farms thought of in any context besides development?

Human Population Numbers

There are broadly three phases to the change in growth rate of human populations. The slow phase up to industrialisation, then faster until approximately 1950 and now a phase referred to as the "Great Acceleration". The Great Acceleration is unprecedented and as such there is no living memory of such times nor general understanding of the issues it has brought. We need to reassess and tackle this problem from first principles because the text books and current knowledge base does not have a solution for the problem. The normal response is to say, well this worked in the past, let's apply the same solution.

Time Scales

Understanding the implications of these different time scales is important. During the pre-industrial times the change was very slow and the centres at which population growth occurred were isolated. There were huge expanses of undisturbed nature which could buffer these isolated changes. The post-industrial phase changed that, some global effects were beginning, the increases in atmospheric CO_2 concentration, the reach of railways, carving up forests and global trade on a scale previously not possible. These first two phases brought relatively slow change giving some opportunity for adaptation. The Great Acceleration has changed that. The speed of change, the shorter time frame and the prospect that switch points have been reached, brings urgency for action and the peril of inaction.

The Great Acceleration

We now have an interlocked network moving in sync. No longer are actions independent and of little global importance. Also because of "legacy effects" the impact of each incremental, say, 10m people added to the population is greater than the previous 10m people. The reserve buffering capacity is diminished as the natural world is placed under increasing and eventually unsustainable pressure. Legacy effects operate on a much smaller scale also and are of great importance in understanding changes in wildlife abundance. The decreasing abundance of koalas in SQ Queensland has been noted as an example. While the land clearing in the Pine Rivers Shire largely stopped many years ago, the number of koalas continues to fall. The same legacy effects have seen the population of koalas on the Koala Coast (Redlands Shire) decrease by 80% over the period 1996-2015. (5)

Wildlife Numbers and Ecosystem Pressured

Historic accounts available in the writings of explorers in the 18th and 19th Century and landscape paintings of the period present a wonderful and visceral account of how things were. Captain Cook's diary 29th April 1770 records, "After I had returned from sounding the Bay [Botany] I went over to a Cove on the North side of the Bay, where, in 3 or 4 Hauls of the [net], we caught about 300 pounds weight of Fish, …". (8) Alexander von Humboldt in his travels in South America in 1800 described flocks of migratory geese darkened the sky for days as they passed. (9)These accounts are interesting as establishing a true base line cannot be reconstructed and we now present data in terms like, "Koala numbers have declined 80% since or over the period examined" without appreciating what they were before human impacts.

Terrestrial Animals

Assessing the changes in the abundance of fauna requires understanding of the way the estimates are made and the areas to which they apply. Further examination of multiple sources over multiple time frames provides information on trends. Looking at declines in some species doesn't establish much as other species may thrive in the altered environment. At the end of the day, it is usually the trends and the ecosystem scale measures which are most important for policy decisions. The Living Planet Index which has been prepared by the World Wildlife Fund (WWF) since 1970 is such an index. It assesses the health of 16,704 populations of 4,005 species. The trend shows a decline in 60% between 1970 and 2014. (10) Over half the fauna assessed in 44 years has been lost. What has been lost since pre-industrial times would be much higher but estimates are not available.

Marine Animals

Average abundance of freshwater populations assessed by the LPI have declined by 83% between 1970 and 2014. (10)

Solutions

Science and technology are improving; policy and politics are not. All scientists know that without asking the right question you cannot get the right answer. This is a simple fact, all too often ignored by policy makers and more alarmingly by science base groups and those with strong commercial interests. The imperative of doing 'something' can be an excuse to ignore the science when it is far from clear what action should be taken, for example the role of badgers in the spread of TB in dairy cattle in UK. Alternatively the case for not-doing anything because of false information or deliberately not wanting to address the facts because of the influence of narrow vested interests. For example, the policy issues facing the regulation of water use in the Murray Darling Basis or coal mining. It is common, in fact, almost universal, that researchers focus their studies on the measurable variables which are the subject of their research and ignore important variables outside their control. (11)

Change

Knowledge, Experience, Culture > Research > Understanding > Action> Monitoring & Feedback

Habitat loss and predation by feral animals regularly appear as the most reported and important causes of wildlife decline and extinctions in Australia. (3) (12) Yet controls on land clearing are recent, for example, in Queensland the Nature Conservation Act in 1992, the Vegetation Management Act in 1999 and nationally the Environment Protection and Biodiversity Conservation Act in 1999. Prior to that governments were actively encouraging and opening up land by subsidizing ringbarking trees or broader scale land development. For example the Brigalow and other Lands Development Act 1962. (3) We are now on another cusp of change where international drivers are, or should be, having implications for local decisions. If humanity is to address the planetary challenges to achieve anything like a 'soft landing' this can't be avoided. (7)

Political Impediment

Improving knowledge, new science and technology, are essential to address these issues but they alone have no chance of addressing the current decline in biodiversity and faunal extinctions on their own. It will not be until a clearer political focus and multi-national action are energised that this can be achieved. There is no certainty that his will be achieved or is recognised by higher levels of government. The plea by David Attenborough at the Katowice COP24 (13) suggests that the world is far from engaged in this problem.

Our Flow Chart of change requires that balanced, considered and morally tested knowledge and experience can flow through our political framework to sound governance and implementation. Unfortunately all too frequently this fails. Examples abound but a topical case in NSW is the Kosciuszko Wild Horse Heritage Bill 2018. The conservation management plans of KNP have been in place for decades and has generally involved a prolonged consultative process. They are underpinned by the NSW National Parks and Wildlife Act 1974 and the 2006 Kosciuszko National Park Plan of Management. The management plans attempt to balance the competing interests operating in the park and to consider multiple species a number of which are threatened. In 2018 the NSW government passed the Kosciuszko Wild Horse Heritage Bill 2018. This bill was seen by the conservation community as a disaster (14) and a conference of key and eminent scientists was arranged at the Australian Academy of Science, Canberra in November 2018. The group issued an open letter to the minister responsible, The Hon. John Barilaro which said in part,

"The Heritage Bill places a priority on a single invasive species over many native species and ecosystems, some of which are found nowhere else in the world. It is incompatible with the principles that underpin Australia's world-leading protected area system, and with our commitments as a signatory to the Convention on Biological Diversity."

The key points of this bill are that: it has precedence over other existing legislation, the appointment of an advisory group (Wild Horse Community Advisory Panel) which is not science-based and, its intent is to give one species priority status over other species and ecosystems some of which are endangered. The legislation is not well informed or balanced and was dependent on support of minor parties and those with narrow vested interests. This issue has sufficiently engaged the centre of the conservation conscience to mobilise politically and apply pressure to have this misguided Bill rescinded. Groups such as SaveKosci and Reclaim Kosci have been very active.

Role of learned bodies

Great challenges have been addressed in the past like the Apollo Missions and the abolition of slavery but it takes political courage and leadership and decades of focussed effort to attain. Reference to current national and international political resolve provides little evidence that we confronted environmental and conservation issues.

Learned bodies have a particular opportunity, indeed obligation, to contribute to the formulation of public policy through education, engagement and advocacy. The opportunities here are wide including, educating professional colleagues, educating clients and the public and representation on boards and advisory groups. Political advocacy either by ministerial meetings or via support of key advocacy groups are options.

Some professional groups have surveyed their members to determine their views and most support some form of engagement. (15) As the issues of the Kosciuszko Wild Horse Heritage Bill have highlighted the linkage between science and policy is essential for meaningful outcomes yet it can easily be cast aside.

Epithet

If Not Now, When? (Book Title, *Se non ora, quando?* Primo Levi 1982)

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Behavioural Assessment of Welfare

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What is animal welfare?

Animal welfare is considered to encompass both physical and mental aspects; the OIE defines animal welfare as meaning the physical and mental state of an animal in relation to the conditions in which it lives and dies[1]. There are three conceptual frameworks that are used to consider animal welfare: biological functioning, affective state, and natural living[3]. The biological function of animals is generally well understood and accepted by those working with animal production, whereby good welfare is connected with the absence of a physiological stress response, with the animal in good health, with normal biological function and fitness, able to "cope" with its environment, and able to satisfy its biological needs[3]. The mental aspects of welfare are related to the animal's affective state, that is, its emotional state, how the animal feels now, and natural living is related to the concept that animal welfare is improved if the animals can express their normal behaviour[4]. The welfare of an "animal is defined by how well it feels; how well it is able to cope with the physical and emotional challenges to which it is exposed"[5].

How can we assess welfare?

Assessment of biological functioning is the most straightforward, in that we can measure physiological responses of the animal. These may be single point assessments in heart rate, respiratory rate, temperature, and hormones, as well as changes over time and in response to alterations in the environment of the animal. Longer term assessments include measurement of body weight and condition and any changes, reproductive function, and measures of health. It is considered that these factors may provide information about biological costs to the animals in their attempt to cope[3].

Assessment of affective state is harder because we cannot directly ask the animals how they feel, and must instead make assumptions from other measurements and observations. There is support for interpretation of the animals' behaviours in terms of their affective experiences. Behaviour also provides some information about their naturalness.

Many assessments provide information about negative influences on welfare, because it is the response of an animal to a stressor or challenge that provides the opportunity for us to judge whether the animal has the freedom and capacity to react appropriately[6], and to cope, both in a physiological sense and emotionally[5]. It is more difficult to evaluate positive influences; there is little physiological response to indicate optimal or better conditions, and few methods available to evaluate positive affective experiences.

Behavioural assessments

Quantitative measurement: What are the animals doing?

The frequency of behaviours can be measured, for instance by observing the animals, and assessing their activity over time. The behaviours can be expressed as numbers or percentages of animals displaying a particular behaviour at specified time, or in an ethogram or time budget, which calculates what an animal is doing over time (e.g. % of time an animal spends lying, walking or feeding). Health parameters that include a type of behaviour can be included, such as lameness or coughing[7]. It can be assumed that what an animal does provides some information about their welfare; quantitative measurements for different situations or management can allow comparisons, with an interpretation of whether changes are providing for improvement[8].

Preference testing: What do the animals choose to do?

Tests which provide animals with a choice of environment or food or opportunity may allow them to demonstrate the strength of their preference and motivation to perform a behaviour or access a resource. Such testing has been used to make inferences about animal welfare, on the assumption that animals make choices that result in optimal welfare[2]. It is unclear whether that assumption is correct.

Qualitative behavioural assessment: how is an animal behaving?

Quantitative measurements and preference testing may provide information about what an animal is doing but do not indicate the motivations for the activities, or the emotional state of the animal. Qualitative behavioural assessment (QBA) was developed as an integrated measure of how an animal behaves, how it responds to and engages with its environment (reviewed in [9]). This behavioural expression or body language is considered to reflect both the physical/physiological state, and the psychological state, and it has been suggested that QBA was the only measure that captured positive aspects of animal welfare. The use of QBA on cattle in the Australian context was recently reviewed[7].

Integrating assessments

The complexity of assessing the many aspects that relate to animal welfare has led to the production and use of a number of integrated assessments, which consider inputs into the animal management process, such as environment-based and management-based measures, along with outcomes in the form of animal-based measures [10, 11, 12].

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Antimicrobial stewardship: Science and regulation

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Introduction

The adaptability of bacteria in the presence of poisonous agents was first described during the early days of bacteriology in the late 19th century. At the beginning of the 20th century, Ehrlich anticipated the significance of this 'resistance' during his pioneer work on chemotherapy of infectious disease. More than a century later, we are still struggling with how to best manage antimicrobial resistance (AMR) as a threat to the capacity to treat infectious disease with the critically important antimicrobial drugs including antibiotics.

The primary goal of antimicrobial stewardship (AMS) is to optimise clinical outcomes while minimising unintended consequences of antimicrobial use, including the emergence of AMR.[1] This can be understood as a process for delivering best practice in the prescription and use of antimicrobial chemicals, which includes concepts of guardianship associated with ethical prescribing standards within a regulatory framework and of continuous improvement driven by science.

The regulatory perspective

In Australia and most equivalent countries, since the advent of the antimicrobial era in the 1930's, access to and use of antimicrobial chemicals have generally been restricted to prescription and/or supply by authorised professionals. Early reports of the emergence of penicillin-resistant staphylococci as well as adverse effects following some forms of administration confirmed the wisdom of these regulatory restrictions in respect to human treatment.

Human antibiotics had initially been freely accessible over the counter in some countries but a 1959 report from Japan caused major alarm in many countries in respect to both medical and veterinary use of antimicrobial chemicals when published in English in the international literature 56 years ago, in 1963.[2] This watershed paper described the consequential widespread emergence of a form of AMR to multiple drugs which was readily transmissible between Gram negative bacterial species including *Escherichia coli* and shigellae- the latter being important human pathogens in Japan at the time.

Some uses of antimicrobial chemicals in production animals had initially not required veterinary prescription in some countries or jurisdictions and this remained the situation into the late 20th or even early 21st centuries. However, the emergence of significant AMR and the realisation of the implications for human health and well-being have progressively, perhaps belatedly, resulted in the more restrictive requirement that most uses of antimicrobial drugs in production animals now requires veterinary authorisation.

During 2018, the Harmonised Agvet Chemical Control of Use Task Group (HACCUT) developed proposals for harmonising veterinary prescribing rights across the Australian state and territory jurisdictions.[3] The scope included new definitions and regulatory proposals some of which extended beyond control of use regulation. Most were applicable to veterinary chemicals generally; those likely to have significant impact on antimicrobial prescribing are in Table 1. Two proposals were directed specifically to regulating how veterinarians prescribe and use antimicrobials (see Table 2).

Table 1 Proposed HACCUT regulatory requirements likely to have an indirect impact on prescription of antimicrobial veterinary chemicals[3]

Proposal 36: Under the care of a veterinarian A veterinary practitioner must ensure an animal or group of animals is under their care before supplying, prescribing or administering a veterinary chemical.
Proposal 20 presents the relevant definition: **Under the care of a veterinary practitioner** means that: • The practitioner must have been given responsibility for the health of the animal or group in question by the owner or the owner's agent; **and** • The care of the animal or group of animals by the practitioner should be real (i.e. there must be evidence of personally having contact with the animal or group of animals for diagnosis and treatment and of assuming responsibility for the diagnosis, treatment and outcome); **and** • The practitioner must have a thorough knowledge of the current health and treatment status of the animal or group of animals by having: – seen the animal or group of animals for the purpose of diagnosis and establishing a therapeutic need immediately prior to supplying a drug; **or** – visited the premises where the animal or group of animals is kept within the previous three months for the purpose of diagnosing, assessing therapeutic need and treating the animal or group of animals or assessing response to treatment of a disease in the animal or group of animals and is supplying ongoing treatment for that disease in that animal or that herd; **or** – consulted remotely with the assistance of photos or video for the purpose of diagnosis and establishing a therapeutic need, only if the time required for a veterinary practitioner to see the animal or group of animals would compromise the health and/or welfare of the animal or group of animals. Veterinary practitioners must be able to demonstrate through their records that they have taken all reasonable steps to ensure there was a therapeutic need and that the treated animal or group of animals was 'under their care'.
Proposal 39: The mandatory cascade of prescribing by veterinarians A veterinary practitioner must select a veterinary chemical product for use in food or trade species in the following order ("The Cascade"): (a) A veterinary chemical product registered for the food or trade species requiring treatment; **or** if there is no such product: (b) A veterinary chemical product available for use under a current permit for the particular food or trade species; or if there is no such product: (c) A veterinary chemical product registered for any major food or major trade species used off label; or if there is no such product: (d) Under exceptional circumstances, a veterinary chemical product that is: (i) registered by the APVMA for animals other than food or trade species; **or** (ii) registered by the APVMA and used contrary to a restraint or DO NOT statement; **or** (iii) an unregistered veterinary chemical product.

Table 2 Proposed HACCUT regulatory requirements specific to prescription of antimicrobial veterinary chemicals[3]

Proposal 31: Treatment at a lower than registered dose rate Food or trade species must not be treated with an antimicrobial veterinary chemical product at a lower rate than that indicated on the registered product label unless as directed by an APVMA permit.
Proposal 41: Where no registered veterinary chemical product is available A veterinary practitioner, under exceptional circumstances (where no registered veterinary chemical product, in any form, is available to treat a disease in animals of a food or trade species), may authorise use of: (e) a registered veterinary chemical product other than for food or trade species; or (f) a registered veterinary chemical product to be used contrary to a restraint or DO NOT statement; or (g) an unregistered veterinary chemical product; In food or trade species provided that: .... (iv) the treatment is not an antimicrobial veterinary chemical; or .... (xi) if ...... the veterinary chemical product is an antimicrobial, the WHP must be permanent and the advice note must state that the animals may not be slaughtered for human consumption.

Specific responses on behalf of the Australian Veterinary Association (AVA) to individual HACCUT proposals were based on considerations of a) AVA Policy, b) a qualitative risk assessment relevant to different livestock sectors, c) case studies and d) general or composite examples of the impact relevant to veterinary prescribing for production animals.

AVA policies support the harmonisation of veterinary prescribing rights. However, the proposals shown in Tables 1 and 2 were not supported by representative veterinarians from the beef, dairy, feedlot, pig, poultry and sheep/goat sectors nominated by their respective special interest group of the AVA. It was concluded that prescribing guidelines provide a preferable non-regulatory mechanism to deal with the complexity of prescribing antimicrobial chemicals. Responses to specific proposals included suggestions of what might be included in guidelines, in some cases adapting practices from other livestock sectors or in others developing specific protocols to manage risk, and thus support a high level of antimicrobial stewardship across the profession.

A model for prescribing for individual client enterprises

The author has previously advocated a 3-tier approach for veterinarians making decisions on treatment of infectious disease in grazing ruminants (beef cattle, goats or sheep) on individual farms- summarised as Table 3.[4]

This approach is intended to incorporate good microbiological and risk management principles and presumes an ongoing rather than occasional client relationship. The actions and activities are necessarily initiated and directed by the veterinarian, in which it could be regarded as omitting some aspects of the AMS planning approach below.

Table 3: A 3-tier model of decision-making by veterinarians to support the ongoing prescribing of antimicrobial drugs for individual client enterprises[4]

Primary (or **prospective**) **assessment** to determine:	- correct diagnosis, - whether the use of an antimicrobial drug is appropriate, - whether to medicate individuals or groups of animals and, - based on microbiological principles, the drug of first choice.
Secondary (or **retrospective**) **assessment** during the course of treatment to determine:	- whether to cease treatment, - whether to continue with or modify the initial treatment, or - whether to substitute a second-tier antibiotic based on sensitivity testing, taking account of PK/PD parameters and any compatibility issues*. This would take account of the clinical response and the results of any laboratory examinations, and importantly, review whether ongoing antimicrobial treatment is required for the individual, group or cohorts.
Tertiary (or **reflective**) **assessment,** in collaboration with the client, at least once annually to review:	- treatment strategies and protocols on the individual farm, - the results of accumulated data from laboratory tests including emergence of resistance, particularly to drugs of first choice, in that animal population, - the outcomes through adequate treatment records. This might also include utilising industry-wide data from laboratories and public resources, both as a benchmark and to inform subsequent primary decision making.

* The frequent use of injectable depot antibiotics in grazing ruminants presents a challenge in determining the timing and flexibility of retrospective decisions.

Antimicrobial stewardship planning

The development of an AMS plan for enterprises in which there is regular or ongoing use of antimicrobials forms the basis of the guidelines developed for the Australian feedlot industry.[5] This approach incorporates the generally agreed 5 core elements of AMS, or the 5 R's, as the basis of best practice antimicrobial prescribing and use.

Responsibility

The appropriate use of antibiotics and other antimicrobial drugs is viewed as a shared responsibility of the prescribing veterinarian and those engaged in the management, husbandry or treatment of the animals of a livestock enterprise.

This aspect of AMS implies a key role for the veterinarian in an ongoing client relationship with the enterprise. However, some grazing ruminant enterprises utilise more than one veterinarian for different husbandry or treatment protocols, which may necessitate attention to professional ethical obligations of each veterinarian in respect to AMS and other prescribing matters. It is proposed that an agreed, written AMS plan for the enterprise would provide substantial guidance in such circumstances.

Review

A practical understanding of AMS is that it means using "as little as possible, as much as necessary" to ensure that high levels of health and welfare are present throughout the entire life of all humans and animals who might require antimicrobials, including antibiotics, to treat infection.

Effective AMS depends on a process of continuous improvement which should be informed by data or current scientific knowledge. This needs to be supported by regular review of antimicrobial treatments similar to the tertiary assessment advocated above (see Table 3) and of the written AMS plan developed by the enterprise.

Reduce
Considering the risks associated with AMR and residues in meat, the use of antimicrobials should be justifiable on every occasion such a drug is administered to an animal. This includes the drug of choice, determination of the correct dose, frequency and duration, as well as whether it is even appropriate to use an antimicrobial drug.

Anecdotal evidence from the experience of individual veterinarians suggests that some livestock owners have an expectation of veterinarians prescribing or supplying antimicrobial drugs on demand for common conditions, often fostered by non-veterinarians suggesting that farmers 'obtain antibiotics from your vet'. Prescribing for common, easily-recognisable, recurrent disease such as foot abscess in sheep is a challenge for prescribing veterinarians who might defer to having sufficient knowledge of the enterprise to authorise the use of antimicrobials for this purpose.

Refine
In clinical practice, the use of antimicrobial drugs in individuals affected by infectious disease is refined through a process which includes an examination and diagnosis by a trained professional who determines the appropriate drug, and the dose rate, frequency and duration of treatment, supported by clinical records and the opportunity for re-examination.

The treatment of grazing ruminants with antimicrobials is frequently undertaken in circumstances when it is not feasible or practical for an examination of the individual before or during treatment by the veterinarian whose role may be limited to determining the need for treatment of individuals on the basis of a herd or flock diagnosis and directing the dose rate, frequency and duration of the prescribed drug.

There is reliance on the competence of those in charge of the animals to fulfil several of the key roles of the clinical situation, including which animals to treat and the records of treatment. This may require the veterinarian to assess the competence of the person managing treatment and/or to ensure that comprehensive directions beyond the minimum required by regulation are provided.

For many circumstances under which an antimicrobial is administered to grazing ruminants, there are limited choices of drug and on many occasions a determination is made to administer a long-acting antibiotic because it may not be feasible or easy to re-treat the individual for reasons of access or temperament. Not all livestock enterprises have the capacity or capability to manage an individual animal during a course of treatment, which may necessitate treated animals being identified and returned to the mob or flock.

Replace
The objectives of replacement for treatment by antimicrobials include to:
- eliminate or at least minimise the use of medically important antimicrobials, and/or
- utilise treatment alternatives which do not involve the use of this class of veterinary chemical.

In some grazing ruminant enterprises, there is opportunity for replacement (or elimination of the need to treat) by changing management or husbandry practices which contribute to the occurrence including seasonality of some infectious disease such as foot disease in sheep.

However, this may not always be feasible because such practices are often determined by other farm management priorities or events including pasture management and cropping cycles.

Discussion and conclusion

AMS in respect to the treatment of livestock involves significant elements of biosecurity, epidemiology and veterinary public health as well as being directed towards the principle objective of treating infectious disease. Thus, veterinarians are required to exercise a high level of expertise and judgment in prescribing antimicrobial and other veterinary chemicals, taking account of a range of factors including the physiological state of the animals and the disease process within the management systems of the individual enterprise.

This requires a competent veterinarian to consider at least the relative importance of the determinants of prescribing such as (but not limited to) clinical history, examinations, visits to individual locations, diagnostic testing, knowledge of animal health/management systems or the competence of those-in-charge of production animals. Such an approach is complementary to the process of registration of veterinary chemicals by the APVMA, thus enhancing the national framework for managing risk associated with veterinary chemical use.

The principle that veterinarians contribute benefit in managing risk associated with the use of veterinary chemicals seems to be implicit in current control of use regulation in some jurisdictions. This should be a core principle in harmonisation of regulation across Australia and in the national framework for managing risk associated with all AgVet chemicals including antimicrobials.

For antimicrobial prescribing and use, the adoption of guidelines and exercise of professional judgment consistent with and informed by an agreed national AMR plan for the animal sector[6] is preferable to a prescriptive regulatory framework which would substantially limit the ability of veterinarians to contribute to key objectives of AMS.

The limitations of this approach to AMS include that inevitably, individual prescribing veterinarians may not follow or implement guidelines in all circumstances or antimicrobials may be used contrary to best practice. However, this is a manifestation of the challenge for all professions of significant inconsistencies around the exercise of professional judgment across a diverse range of matters impacting both individual benefit and public policy. A range of non-regulatory solutions have been proposed by researchers including risk management or a shift from 'craft' to systemisation for key professional practices.[7] This is an ongoing field of research with the potential to deliver substantial benefit to both community and practitioners, although a consistent conclusion would seem to be that substantial changes in the way professions work and interact with their clients are likely to be required in the future.

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Wellbeing in veterinary students: a student led study

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Introduction

Over the past two decades, considerable attention has been placed on veterinary medical student mental health and stress levels, after early reports identified elevated levels of anxiety and depression.[1-3] More recently, there has been a shift from a reactive focus on depression and perceived stress to a proactive approach, promoting wellbeing and resilience.[4] This is reflected in the Journal of Veterinary Medical Education's dedication of an entire issue to exploring veterinary student mental health, anxiety and depression from a wellbeing standpoint. This special issue explored topics including building resilience,[5, 6] embedded mindfulness,[7] veterinary student wellbeing[8] and stressors and protective factors in veterinary students.[9] Despite this shift, there remains a tendency to synonymise wellbeing with stress management, leaving a gap in the literature where wellbeing in veterinary students is currently not perceived through a holistic lens, accounting for all the major aspects that make up the multidimensional concept. Utilising the ESSENCE wellbeing framework created by Dr Craig Hassed,[10] the aim of this study was to explore how veterinary students understood and enacted behaviours conducive to wellbeing, along with the enablers and barriers affecting engagement with such behaviours.

Research approach

The study posed the following questions:

- How well do students understand and enact behaviours conducive to wellbeing?
- What are the enablers and barriers affecting engagement with such behaviours in the context of being a veterinary student?

A mixed-methods sequential explanatory design model was employed in order to capture both breadth and depth in data to answer the questions. The ESSENCE framework was used to develop a paper-based questionnaire handed out to all 309 students enrolled in the veterinary science program at Charles Sturt University. This particular framework conceptualises wellbeing as comprising of seven dimensions: education, stress management, spirituality, exercise, nutrition, connectedness and, environment. The dimensions of the ESSENCE framework are both unique and inter-related and their application in wellness programs for medical students is supported by empirical evidence.[11] The questionnaire was comprised of Likert scale questions asking students to respond on a scale of strongly agree to strongly disagree. The data obtained was transcribed into Microsoft Excel and descriptive and inferential statistics were obtained. Upon completion of the voluntary questionnaire, students were invited to indicate their interest in the second phase of the study, which was comprised of individual interviews of six students. Of the participants who showed their interest, interviewees were selected to represent a broad range of opinions and contexts, based on their questionnaire responses. The semi-structured interviews tailored to each participant were audiotaped and transcribed. Each transcript was de-identified and allocated a pseudonym for reference. The transcripts were analysed using a directed framework. Ethics approval was sought and obtained from the Human Research Ethics Committee (approval number H18151).

Findings and discussion

A total of 154 (48.9%) returned submissible questionnaire responses were received. Participant demographics were representative of the student population at the university. For ease, "agreed" refers to responses of "agreed" or strongly agreed" on the 5-point Likert scale; "disagreed" refers to responses of "disagreed or strongly disagreed." Amalgamation of the data retrieved from the questionnaires with themes coded within the interview phase of the study revealed four key points worth consideration.

1. **Understanding of wellbeing**

In the questionnaires, participants initially showed a high level of confidence in their understanding if wellbeing (94.2% agreed). However, inconsistencies became apparent as students were challenged to make a personal connection with wellbeing. Whilst an overwhelming number of students understood the importance of stress management (98.7%), only 22.5% of these students agreed they actively made time to manage their stress levels. The ability, or lack thereof, to foster a personal connection and show a well-developed understanding of wellbeing was exemplified further within the interview stage. Whilst some students were limited to a basic textbook definition of wellbeing, other students were able to give a definition that fostered a sense of personal attachment to the concept. A prominent example of this disparity came about when discussion turned to spirituality. The prominent divide noticed in the questionnaire phase was echoed within the interviews. Some of the interviewees were quick to dismiss the notion, struggling to see it as anything more than its affiliation with religion. However, students who had a stronger connection to the idea of wellbeing were able to view spirituality in a broader perspective and discuss how it helped them to stay grounded. Whilst spirituality encompasses more than just its association with religion,[10] the inability of some students to view it as anything more and thus create a personal understanding of the concept meant that they did not fully disclose their appreciation of a holistic outlook on wellbeing.

2. **Student's relationships play a large role in wellbeing**

Within the questionnaire peer support was recognised as helpful in reducing stress (87.7% agreed) and connecting (88.3%). Within the interview phase, students discussed how the shared experience of being in a cohort made them feel more at ease with the challenges being faced; interviewees often referred to the phrase 'being in the same boat'. The positive aspects of those relationships found in this study complement what has already been described briefly within the literature. In Western, Gardner and Yeung's 2017 study, support from both peers and lecturers is acknowledged as being a protective factor against perceived stress.[9] Furthermore, Cardwell and Lewis described how students also used the metaphor of 'being in the same boat' for how they felt supported by their peers. [8]

Peer interactions were not always benign, within the questionnaire only 54.3% of students agreed they learnt wellbeing strategies from their peers and, 20.8% of students agreed they felt judged by their peers for being stressed. This was significantly more likely in students over the age of 24 ($P<0.05$). Whether this is due to internal or external perceptions of being a mature age veterinary student cannot be answered within the realms of this study and presents an opportunity for further research. Universally, interview participants spoke of the expectation to be stressed. One student discussed being in a situation where he felt he was being judged by his peers for not being stressed, whilst other students discussed how conversations with peers would often turn to talking about 'how stressful' the course was. This perspective of peer interaction is less commonly documented within the literature and warrant further exploration. The underlying stress culture identified by these students was seen to have inhibiting effects on the uptake of behaviours conducive to wellbeing and may be an area where future intervention strategies are targeted.

All student participants identified that lecturer support in terms of clear guidance in terms of study requirements and assignments was helpful in reducing stress and allowed them to achieve a greater study-life balance; a finding supported by other studies.[8, 9, 12] However, interviewees expressed how they often felt confusion when it came to their lecturers discussing wellbeing and what was expected of them as a veterinary student. Students identified this as a barrier in the uptake of wellbeing strategies as they struggled to balance wellbeing strategies with study expectations. When discussion in the classroom turned to wellbeing, interview candidates felt that these were often 'throw away' comments made by a lecturer followed by an expectation to do a large amount of study. The complexities of their relationships towards lecturers as described by students in this study has not previously, to the authors' knowledge, been described within the literature. These inconsistencies can create confusion for students who are unable to reconcile the mixed messages. Whilst course workload cannot easily be decreased, and the nature of the degree requires a large amount of time dedication, these findings suggest that the way in which students are taught to find a balance can have a major impact on their ability to implement wellbeing strategies

3. The need for critical analysis of the hidden curriculum

A key and concerning finding of this study involves student beliefs that personal wellbeing may not be compatible with membership of the veterinary profession. The propensity for students to agree that their study comes before many aspects of wellbeing within the questionnaire was complemented by the interview phase where students discussed the idea of the veterinary profession as being an all-consuming career. What emerged is a common belief that veterinary science and wellbeing aren't compatible concepts, and that for one to work the other must be sacrificed. A major driving force in the development of this construct was how veterinary students perceived the clinicians working at the university. At the forefront of this ideation was how students perceived the clinicians working within the university. Zoe exemplified this by saying: "definitely some lecturers will tell us that we need balance, but they're working 25 hours a day". For many students, clinicians at the university are the first long term contact they have with a veterinary professional. The modelling of the profession by these clinicians is an example of what other studies have described as being the 'hidden cirriculum'.[13] The hidden curriculum has a profound effect on how veterinary students learn to be professionals and refers to the unintentionally disclosed information surrounding the culture of an institution.[13] The effect the hidden curriculum appears to have had on the students participating in this study is one that rejects the notion of wellbeing and the veterinary profession being able to mesh together. The implications of this finding are complex and unclear, and it certainly warrants further exploration. It does, however, raise the possibility that there is an underlying culture inhibiting the uptake of wellbeing strategies amongst veterinary students.

4. Striking Balance

The complex and changeable relationships between the themes can be represented by the unifying concept of striking a balance, represented in Figure 1.

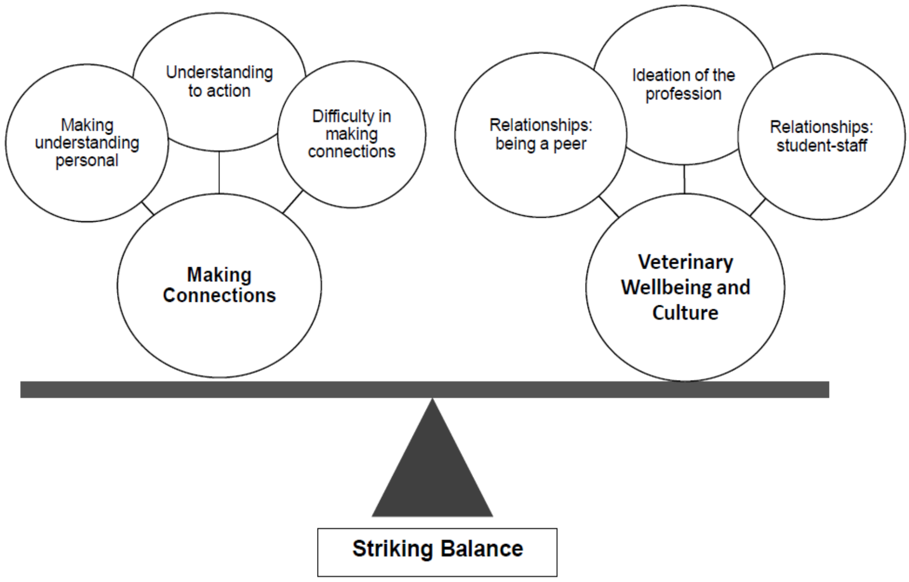

Figure 1. Illustration of interview themes

Finding a balance between looking after personal wellbeing and studying veterinary science was discussed as being a challenge for all interview participants. However, a few participants discussed how they found an equilibrium between the two apparently opposing forces by creating boundaries between their degree and their personal life. Mitch discussed how he intentionally compartmentalised his degree from other aspects of his life in order to gain balance: "vet is one part of my life, when I'm at university I'm focussing on that and I've got my vet friends…you can move over to swimming…where I don't focus on vet". Mitch's ability to maintain his veterinary degree as only a portion of his identity allowed him to maintain a healthy wellbeing and balance within his everyday life. For Luke, instead of relying on peers for support he looked outward to students in other degrees and connected with people outside of the university environment. Whilst this distanced himself from his peers, he felt he was able to better cope with the degree's challenges.

Conclusion

Whilst the study is small, its careful design and holistic wellbeing framework make it a meaningful and valuable contribution to the wellbeing literature in veterinary education. Given the grounding of the evidence within the data, veterinary educators and students can consider the application of this model within their own settings. A major finding of this study and something that warrants further research is the need for both educators and students to critically analyse the hidden curriculum. Although this study was in an educational setting the discussion of whether personal wellbeing is compatible with membership of the veterinary profession is a question for all veterinarians. There remains much to be explored and achieved in improving wellbeing for veterinary students, and there are no simple solutions. This study is unique in veterinary education: it is student-led, is based on a holistic wellbeing framework, and it allows the voices of students to be heard. Its findings can guide practical implementation of wellbeing support programs along with being of interest to students, educators and the wider veterinary profession

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Surviving Wildlife Admissions in General Practice

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Abstract

Vets in general practice will always be faced with wildlife cases. Dealing with these is a challenge in terms of welfare, workload and many other factors. This paper seeks to provide some general and specific advice to veterinary practitioners in Australia and illustrated by some brief case studies.

Introduction

Vets in general practice will always be faced with wildlife cases, whether presented by members of the public, local wildlife rehabilitation carers & organisations, and even owners of private collections. Our decisions whether to attempt to treat, euthanase or refer are affected by a range of factors. These include your;

1. employer's preferred approach
2. existing workload and stress levels
3. perceived value of the animal
4. understanding of the problem and its severity
5. experience and confidence in dealing with this problem
6. prospect of release back to the animal's own local area

I will examine these issues in this talk with a focus on welfare, relevant conservation and the over-arching issue of taking care of yourself.

Triage

From the French to sift or sort, triage is an important concept you are all familiar with. In any situation where the total number of sick patients exceeds the available resources triage is the process of prioritising patients so that you can treat as many as possible[1]. Even in the GP situation where you are likely to only have a single wildlife patient to treat at a time, the concepts of rational triage should be applied.
Triage levels:

1. No significant injuries or clinical signs
2. Treatable wounds or pathology
3. Severe or multiple problems

Your focus should only ever be on level 2 triage patients. However, differentiating them from level 3 patients can be a challenge and that will be our focus further on.

Level 1 patients are often avian window strike victims that have been concussed to varying degrees and have recovered by the time they are examined. These animals should be released back at the site of capture with no delay. The other common level 1 admission is the "orphaned" baby bird. These patients usually fit into 2 subgroups:

1. Healthy birds that have fallen out of the nest near fledging. These animals should be refused admission. Provide information to allow the "rescuer" to make a bucket nest and do not let them talk you or your team into admitting the bird. Altricial birds raised by even the most experienced wildlife carers are very poorly equipped to survive when eventually released into the wild.

2. Unhealthy birds that have been rejected by their parents. Birds with congenital problems or exacerbating disease are frequently ejected from the nest by disarmingly pragmatic forebears. There is nothing to be gained from trying to save these patients.

1. Your employer's requirements.

Attendees to a session like this are unlikely to work in practices where they are completely discouraged / banned from trying to treat wildlife. However, many practice owners will provide begrudging support to clinicians wanting to perform time and resource consuming procedures on wildlife with no budget. You can turn that into more enthusiastic support if you make it work for your clinic. If this is an interest of yours, offer to become the clinic's wildlife champion[5, 11]. This improves practice efficiency and helps ensure patients aren't forgotten as "someone else's problem".

The most practical, and enjoyable, way to make wildlife valuable to your practice is to take pictures of all wildlife you treat and post the best pictures on your clinic's social media pages. If your boss is so firmly rooted in the 20th century that there is no clinic social media at all offer to start and run a page. A Facebook business page is possibly the most effective initial toe in the water. There isn't a lot of time overhead in posting the occasional wildlife case that comes before you. These are universally well received by the Facebook general public and can generate enormous amounts of goodwill and community appreciation. Who knows, you may even bring your boss into the 21st century.

When dealing with wildlife that is brought to you by carers either working singly or within rehabilitation organisations it is appropriate to charge fees, though most expect a heavily discounted or cost recovery only basis. For most of you your practice probably already has a wildlife fee structure you are following.

2. Your existing workload and stress levels

The single most important thing I hope everyone takes away from this session is this; *The only one who really must survive in each wildlife case is you*. Treating another dog attack injury in a bandicoot really isn't as important as you getting home at a reasonable time and in a reasonable frame of mind. Compassion fatigue and burnout begin with an unreasonable determination to do all the things - regardless of how you are feeling.

Whenever you are deciding whether to put significant time into a wildlife case take an early and realistic look at your day's schedule. If you have a full load of client pet cases to work through don't leave the animal till last, hoping you can finish everything with enough time to assess and treat. Instead take a couple of minutes to perform a quick initial assessment[12] and, unless it has a single mild problem you are confident you can treat quickly, allowing you to consider it a level 2 triage patient, do not take it on. If level 1 – send it on to a local wildlife rehab organisation or simply organise immediate release. If it's level 3, euthanase or refer to a wildlife competent clinic that can take it[10]. If you do decide it is a straightforward level 2 patient make the time to immediately institute some simple supportive therapy – fluids and narcosis primarily – and schedule time for specific diagnostics and treatment as soon as the patient is stabilised, and the paying clients have been taken care of.

Consider that all animals in the wild, no matter where they fit in the food chain, end their lives as they are eaten by something else. The wildlife you put to sleep may not be able to contribute their bodies to the trophic pool or their DNA to the genetic pool, but they will be the few individuals that enjoy a humane death. It is always a compassionate act.

3. The perceived value of the animal

Working with individual animals has extremely little general conservation value[4]. However, the bigger picture includes a collection of people involved with any animal from initial capture and transport, to assessment and treatment, to rehabilitation and finally release. All of these people have their own views on the value of the animal and draw significant benefits from their involvement with the process. In addition, there is an educational benefit to yourself and everyone else involved with the case[8] as well as those who simply hear about it – perhaps on your Facebook page.

The educational value of wildlife casualties varies depending on how interested you are developing the skills involved in treating them. If you want to develop exotic species medicine and surgery skills, seeing injured wild birds and reptiles is extremely useful.

In rare situations you may be presented with endangered species casualties. In some of these cases you may be required to refer the animals on to a particular centre, such as with carnaby's black cockatoos in WA which must be treated at the Perth Zoo hospital. With other endangered animals, that you can treat, there is obviously a greater value placed on successful treatment[8]. If other factors described here limit your ability to treat, again, you should consider referral if possible[10]. Depending on where you are, referral to a reputable wildlife rehabilitation centre may be appropriate.

Introduced and pest species are a special and often contentious problem[4]. There is absolutely no good reason to attempt to rehabilitate and release any feral or invasive species. Valuable resources are used, and the environment doesn't benefit. Members of the public who bring in an injured feral pigeon often have an enormous amount of trouble in appreciating that the animal is actually an invasive species and, regardless of its injuries, should be euthanased immediately. Even if you want to practice a wing pinning, the pigeon should be euthanased post surgery.

Every practitioner should do their best to educate the public on these issues, but you will inevitably encounter individuals whose minds are entirely closed to conservation science and remain convinced that the feral animal in the cardboard box they are clutching has a right to life and must be saved. When I encounter these types of people presenting feral and invasive species, I usually take the animal promising to assess it and do what I can to save it. I will then document that the nature of its condition meant it was non-releasable and it had to be euthanased on welfare grounds.

4. Problem recognition and severity

Successful outcomes don't always require successful diagnosis of the problem, but it sure can help. Any animal presented thin and weak, lacking any identifiable injury, is likely to be at the end of its natural life, or suffering from a natural disease process, or both.

A robust rule of thumb to follow is that if humans haven't caused the problem, humans shouldn't be trying to fix it[3].

Importantly, if an animal is neither endangered nor releasable, it should be euthanased. Some notable examples of injuries that are rarely viable for release include:

1. Substantial comorbidities of any type. "Two strikes and you're out"
2. Significant fractures of the distal wings (carpometacarpus) of most flighted birds[12]
3. Open (compound) wing fractures with significant bone necrosis or contamination of most flighted birds
4. Significant necrosis or loss of the patagium of bats and most flighted birds.
5. Maxillary/mandibular fractures in the echidna[12]
6. Loss of an eye in hunting birds
7. Wing fractures in microbats

8. Juvenile magpies with bald heads and a history of being attacked by other magpies
9. Severe cachexia, in any individual

The patient that presents with non-specific malaise should initially be treated with non-specific support therapy:

1. Subcutaneous fluids
2. Narcosis and/or NSAID administration
3. Dark, quiet environment

Additionally, collecting samples of blood, urine and faeces for assessment is valuable where possible. Assessment of haematocrit, serum total protein and examination of a peripheral smear is moderately time consuming but requires just a few drops of blood and remains a cost-effective way to get very useful information on your patient.

Note that administration of antibiotics should *not* be considered first-line supportive therapy without clear evidence of bacterial infection. In particular, the ever popular enrofloxacin should not be used routinely with injured wildlife. The AVA's guidelines on use of antimicrobials lists enrofloxacin as a third line drug in dogs and cats and its use is banned in food producing species. Fluoroquinolones are particularly at risk of generating resistance[7] and while its widespread use in wildlife care has yet to produce confirmed cases of resistant organisms the precautionary principle dictates that we choose alternatives wherever possible.

5. Your experience and confidence in treating the problem

While the general practitioner is ill-advised to just take on a cruciate repair with no previous experience in a client's pet, the same is not the case in treating wildlife injuries. If you have treated traumatic wounds in domestic species, you are quite well prepared to treat them in pretty much any species[2]. Suture techniques, orthopaedic repair techniques and wound dressing techniques differ in both avian and reptilian patients but rereading the literature on these details and then simply giving it your best shot does two very good things. First, it is likely to give the animal its best chance of being able to continue its life and, second, it provides you with an excellent learning experience.

Avian wounds are prepared for surgery by plucking minimal feathers, not clipping, and using masking tape to hold back others that overhang the site. Post-operative infection is generally less of an issue in birds but extended anaesthetic time is more of an issue – increasingly so the smaller the patient. So, keep aseptic prep to a minimum and don't use alcohol. Debride thoroughly and close wounds with the finest absorbable monofilament at your disposal[9]. Packet suture is a cost issue but essential to minimise skin trauma. Keep left over packet sutures from paying clients in a chemical steriliser (chlorhexidine etc) for reuse on wildlife. Muscle tears can be gently apposed with horizontal mattress sutures[9]. Avian skin is extremely thin but extremely stretchy. Wound tension is rarely a problem unless there has been substantial tissue loss. Deficits tend to look enormous until you start to tease out the edges and start suturing. Suture in a continuous pattern with fine spacing and use ophthalmic or similar fine instruments to help ensure your sutures are close to the edge of the skin[9].

Reptilian wounds are prepared with thorough debriding and are often best left to heal by secondary intention. If there is a closable deficit and you are sure debriding is complete, appose the edges in a completely everting pattern with horizontal mattress sutures. Again, swaged on needles from packet sutures are better. The scales of bobtail skinks are impressively difficult to drive needles through. Often it is necessary to guide your needle between scales rather than through them, but even then, bent needles are a constant problem.

6. Prospect of successful release

Crucial to successful rehabilitation in every case is release back into an environment that is not hostile to the animal. Most species have no hope of survival if released into territory that is foreign to them. Therefore, it is important that the exact location of the animal's initial capture is recorded when it is presented to the veterinary practice. Always collect the person's name, phone number and the specific location they found the animal. If nursing staff have admitted an animal without this information, it is far kinder to immediately euthanase. Establishing a standard wildlife admission form that is stocked at the reception desk fits well with standard reception procedures and will minimise the likelihood of this occurrence[5].

A depressingly common situation in many Australian urban areas are wildlife refugees. Bushland is cleared for urban development and wildlife are pushed out, forcing them into conflict with vehicles, pets and other hazards. There is essentially nothing that can be done in these situations as there is nowhere for these animals to be released into. Choosing some other equivalent patch of bush for release is no solution as it will already be at carrying capacity for that species and the incumbent residents will hardly be thrilled to see their cousins competing for scarce resources. In this situation again, euthanasia is the most humane call.

In the ideal situation, having completed primary triage and treatment you will be able to refer the animal on to a wildlife rehabilitation organisation for the final stages of care and release. These centres are becoming increasingly sophisticated and sometimes even have volunteer veterinary staff. So long as you have provided a care plan, suitable medication and an appropriate release location, you can wave goodbye with the satisfaction of a job well done.

Case studies

Case 1 – Failed orthopaedic repair in a private collection kangaroo

Signalment

Harry Hopper – Adult male castrated western grey kangaroo *(Macropus fuliginosus)*, 35 kg. Age uncertain.

History

November 2015 was presented to another vet hospital with an open fracture of the right forelimb. It was noted there was dirt in the exposed ends of the bones. Nevertheless, the clinician went on to pin the radius and ulna and close the wound.

The owners noted it never settled down, remaining swollen despite two courses of clindamycin.

Procedure

Seen November 2016, by this time the right antebrachium was firm and variably swollen through its entire length and discharging pus from a ventral fistula.

Sedated with 1.6ml zoletil, transferred mostly into a very large pouch and transported back to base.

There it was transferred to Iso, prepared for surgery including IVF via the lateral tail vein, and the limb amputated through the proximal third of the humerus, leaving plenty of muscle belly to cover the stump.

Harry was then directly transported back to the owner's property. He enjoyed an excellent recovery.

Video

Case 2 – Fence entanglement in a raptor

Signalment

Wedge-tailed eagle – Adult female wild Wedge-tailed eagle (*Aquila audax*), 4.8kg. Age uncertain.

History

Rescued from entanglement in a barb-wire fence.

Multiple lacerations to legs, right wing and caudal abdomen. Some superficial damage to leg muscles. Severe contamination of all wounds with blood, sand and other crud.

Procedure

Iso GA after transfer from pet-tainer with restraint focused on the legs.

> Safety note – If you or your team are gripped by a powerful raptor do NOT try to force the claws open – you won't win. Simply release any hold anyone has on the bird entirely and provide it space to get away. It will likely release immediately and attempt to fly away. You can usually net it again.

Extensive process of flushing, cleaning and debriding the multiple wounds. All feathers associated with wounds are plucked not cut as this stimulates new regrowth. Finally, all skin deficits were closed with 4-0 PDS in continuous patterns.

Post op medication included 7 days enrofloxacin and 4 days meloxicam.

Case 3 – Chronic wound on an Emu's wing

Signalment

Adult female entire Emu *(Dromaius novaehollandiae)*, 28kg, Age uncertain.

History

A wound had been developing for weeks to months on the left wing of an emu resident at a local wildlife conservation centre. Various attempts had been made to treat the wound topically – including flamazine and fly repellent products but it continued to get worse.

I visited the property and attempted to sedate the animal with 1.5ml zoletil IM. 15 minutes later the bird was ataxic but far from handleable. A distinct difficulty in sedating emus is the risk of partial sedation exposing the animal to self-trauma as they stagger, fall and then flail – often wildly. So I doubled the dose giving another 1.5ml and had just enough control to examine the wing, discovering the entire tip, including some of the distal carpometacarpus, was necrotic.

Procedure

Conservative care is never easy in a semi-wild emu, however, amputation of a wing in a flightless bird is obviously not a problem. More zoletil was poured in as the animal was proving remarkably resistant, and the bird was transferred via closed ute tray to the Wattle Grove Vet Hospital. I followed behind as there was substantial risk of the patient getting leggy in the tray. Turned out the patient wasn't the real risk (video). The surgeons at Wattle Grove were too busy to assist but they graciously allowed me access to their facilities to amputate the necrotic tip.

Post op medication was just meloxicam for 3 days. The bird did very well.

Case 4 – Lawnmower wound on a bobtail

Signalment

Adult male entire bobtail *(Tiliqua rugosa)*, 365 grams, age uncertain

History

Lawn mower injury. This is a very common cause for wildlife admission in areas where these slow-moving reptiles persist. In this case the animal had sustained a deep wound to the right side of the neck and head. The wound was 5-7 days old by the time it was presented and had substantial tissue loss plus necrosis.

Procedure

The severe nature of this wound required multiple surgeries over about 5 weeks. Every week the reptile was represented from Kanyana Wildlife Rehab Centre, and anaesthesia was achieved with IM alfaxan. The wound was cleaned, debrided, sutured and dressed. Key to healing in these large reptile wounds is duoderm, sutured in place. The biggest challenge is ensuring continuous contact over concave surfaces. Anywhere that you don't have contact between the wound and the duoderm healing is delayed and infection tends to take hold. Duoderm is wonderful stuff for this job but you need to be persistent about making it follow the shape of the wound surface. Even small areas that are left with an air gap between the granulation tissue and the dressing will be visibly worse at your next change.

If you are dealing with a wound that is too topologically complex for duoderm coverage you need to use flamazine cream instead. The downside is then you need to ensure the wound is cleaned and redressed every day or two.

Case 5 – Dog attack wounds on a bluetongue

Signalment

Western blue-tongued skink *(Tiliqua occipitalis)*, 620 grams, age uncertain

History

This reptile was reportedly rescued from the mouth of a dog. The skink was brought in directly by a member of the public – a rather rare find in Perth's hills.

Examination showed an obtunded reptile showing signs of severe pain. Surprisingly the mandibles were intact as was the mandibular symphysis. That was the end of the good news. Multiple tooth wounds had penetrated puncturing through the nasal, pre-frontal and frontal bones into the cranium and nasal concha with significant bony displacement.

Immediate euthanasia was elected.

Case 6 - Wing fracture in a little eagle

Signalment

Little eagle *(Hieraaetus morphnoides)*, 860 grams, age uncertain

History

Bird found on the side of the road, unable to fly, in the western Wheatbelt. The animal had been in care with a specialist raptor carer for just under 24 hours prior to presentation. The left wing had been bandaged to the body and appropriate fluids given. They hadn't succeeded in getting it to eat.

Exam suggested an open humeral fracture and quite marked tissue damage from the fractured bone ends. Radiographs confirmed a comminuted spiral fracture of the proximal third of the left humerus with a single loose fragment about 10mm long. In addition, there was some bruising on the sternum and a mild degree of epistaxis. There were no other significant injuries and the body condition was good.

Procedure

A tie-in fixature, or hybrid fixation, was elected. In this process an intramedullary pin only slightly thicker than 50% of the medullary cavity, but well over double the length of the intact humerus, is retrograded from the fracture site and gently hand drilled out through the proximal humerus just distal to the shoulder joint. The pin is advanced all the way through to allow the fracture to be reduced and then normograded back into the distal humerus to seat gently into the elbow knuckle. Going too far will damage tendon insertions at the elbow. Now the pin is carefully bent around 90 degrees where it exits the shoulder, and again another 90 degrees about 20mm from the first bend so the pin now runs back parallel and dorsal to the humerus. The pin is then cut just before the elbow. With the wing flexed up in a closed position, so it is rotationally neutral, 21g 1.5" hypodermic needles are advanced in line with the 2 halves of the pin into the humerus. One is placed about 2 bone diameters from the shoulder joint and a second one a similar distance from the elbow joint. Using fingers to hold the plastic needle base you can hand drill through the cortex with a combination of clockwise and anticlockwise motions. The biggest challenge is getting started on the highest point and not slipping off the cortex surface and stabbing through the soft tissues (including your own!). The IM pin can get in the way but wasn't a problem in this case. The needle is again finger-drilled into to the ventral cortex so that the tip is just protruding on the other side, but the bevel ideally won't have gone all the way through the cortex. This process was repeated at the distal end of the humerus. The needles were long enough to allow the needle hubs to be cut off.

Selleys "Knead it" epoxy putty was then used to rigidly fix the needles to the IM pin. This sets in minutes and the entire apparatus provides excellent rotational and angular stability. The open wound at the mid-humerus had been cleaned and debrided at the beginning of the procedure and was left to heal by secondary intention. Rather than bandaging the wing, the tips of the flight primaries are simply taped together using masking tape. Masking tape is the preferred choice for most adhesive restraint on birds as it is stable yet easy to remove, leaving no sticky residue. Never use Elastoplast type bandage. Wing-tip taping works very well for these types of injuries as it provides adequate stabilisation, is well tolerated, yet still allows some movement, slowing the progression of joint disease from immobility – a process that happens very rapidly in avian joints.

Butorphanol was given as a single injection on recovery and meloxicam was given for 3 days after surgery. Doxycycline was given in drinking water for 2 weeks post-surgery.

The prosthetic apparatus was removed 6 weeks post-surgery. Care must be taken deconstructing the prosthesis, cutting the pin and the needles where they protrude from the Knead-it. The individual components can then be extracted from the wing.

The patient was free-flying within a week but was kept in a large enclosure for several more weeks prior to release.

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Faecal worm egg count reduction testing: best practice protocols

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Following the recognition that anthelmintic resistance was a common and costly impost on the livestock industries, routine testing for drench effectiveness has been a standard recommendation[1,2]. Drench effectiveness must be assessed on an individual property basis, as resistance patterns vary considerably even within a district, due to differences in the selection pressure for resistance in relation to local environments, drenching programs and animal management factors. Failing to ensure adequate drench efficacy has significant consequences for sheep health and production, and for further increases in drench resistance.

Generally-agreed guidelines for faecal worm egg count reduction tests (FWECRTs) have been available for many years [3,4], with testing recommended at intervals of two to three years, and ideally with the input of a livestock adviser. While the test protocols are conceptually simple and relatively practicable to implement, the adoption by livestock owners at present is very low. It is therefore important that best-practice test procedures are followed, to ensure that livestock owners receive the most useful and applicable information possible.

Resistance or efficacy ?

The concept of anthelmintic resistance applies specifically to an identified worm species (or for FWECRT, genus), rather than an undifferentiated worm population. In contrast, the post-treatment reduction in total numbers of a mixed-species population indicates efficacy only against the particular worms present at the time of testing.

Worm egg count reduction tests without differentiation to genus must therefore be interpreted with caution, as failing to distinguish between resistance and efficacy has two potentially detrimental consequences:

- Resistance may not be detected for a species (or genera) present as a small proportion of the mixed population. A common example is where an ML-resistant *Teladorsagia* is present as a small minority but *Trichostrongylus* (rarely ML resistant) dominates: the undifferentiated reduction will typically be greater than 95%, but often considerably less for *Teladorsagia*.
- The severity of resistance varies between species and genera, but large variations in proportions often occur between mobs and seasons. Unless a particular drench provides a complete reduction in egg count, the wider extrapolation of test results for a mixed-species population at one time-point is likely to mis-represent the drench efficacy in other situations.

Definition of resistance

Anthelmintic resistance has traditionally been defined as a reduction in egg count of less than 95% for an identified worm species[2,3,4]. This is a conservative figure that allows for the uncertainty of mean worm egg counts due to test limitations and potential mis-dosing, and that there are often wide confidence intervals around the percentage reduction figure.

For control purposes, an efficacy of only 95% is of value only as a remedial treatment for clinically-affected sheep or where immediate reinfection was expected (high refugia situations[5]). For strategic treatments aimed at minimising pasture contamination with worm larvae, any significant worm survival can derail the long-term preventative effect and increase the level of resistance[5]. Drench selection is best planned by an informed adviser, as it cannot be assumed that any drench providing an egg count reduction above 95% is suitable for routine use.

By convention, WECRT results are calculated using arithmetic means, despite the typically skewed nature of the egg count data, as using geometric means moderates the influence on the mean of occasional very high counts, and can underestimate the importance of resistant worms[6].

Test design

For the usual WECRT format in Australia, animals are sampled only once, at 10 – 14 days after dosing, and the mean worm egg count in each treated group compared to that of an un-dosed control group[2,3]. For especially rigorous assessments, such as for new product registrations, both pre- and post-treatment counts are taken, to allow for changes in control group means over the test interval[1,2].

The need for a control group is a subject of debate, and some recent analyses suggest that for the greatest likelihood of detecting reductions of less than 95%, identified animals should be sampled at both dosing and post-treatment, with no control group[7,8]. The rationale is that changes in treatment group counts over time will not always parallel those of control groups, due to worm density-dependant effects on egg production rates[8]. The post-treatment comparison with control group methods was least sensitive where there was a high dispersion level (low negative binomial statistic, k), and especially so in the critical efficacy range of 80- 95%.

However, a recommendation for the pre- and post-treatment sampling protocol obviously entails considerably more field effort and laboratory time and cost, and is likely to be a barrier to adoption of WECRTS by farmers. A practical approach for general farm testing may be to retain the post-treatment-with-control protocol, but to emphasize that for the most reliable results, all animals dosed should also be sampled; this is especially important where the control counts are significantly over-dispersed (seen as a large proportion of zero counts). An alternative, most applicable where group sizes are small, would be to sample and count a number of identified animals at dosing, and then re-sample only a smaller numbers of high-count individuals.

Number of sheep sampled

Test protocols in Australia generally recommend a minimum of 10 animals per test group[1,2] as worm counts typically follow a negative binomial distribution ("skewed"), and small sample sizes will often mis-represent the mob mean. A larger number than the minimum required is usually dosed, to allow for absentees when re-mustered for sampling, and for insufficient faecal samples.

More recently, it has been suggested that, the number of eggs counted at the pre-treatment sampling is more critical than the number of animals sampled[8], because low or zero counts contribute nothing to the reduction calculation. According to a modelling-derived calculation guide[8], for a drench of 90% efficacy, counting 100 eggs (where one egg detected indicates 50 eggs in the sample) provides confidence intervals of 83 - 94%, whereas counting half that number gives confidence intervals of 78 – 95%. In field situations, the latter estimate would be a reasonable basis for drench decisions, especially as confidence intervals are tighter as the percentage increases above the minimum 95% limit.

The advantage of this approach is that fewer animals are required, provided that the initial egg count is sufficiently high, but it requires that the same animals are sampled pre- and post-treatment. Further, sampling a very small number may lead to bias due to random variations in egg output of individual animals, and where resistance levels vary between the worm population in host animals. If following this approach, it may be reasonable to follow the guidelines for anthelmintic efficacy evaluations[4], which require a minimum of around 7 individuals.

Pre-treatment worm egg counts
The chief requirement is that worm burdens are high enough to provide egg counts sufficient for reasonable test sensitivity, but not to cause clinical parasitism. The generally recommended minimum mean count is around 200 eggs epg for each of the major "scour worm" genera regions (*Teladorsagia* and *Trichostrongylus*), and 500 epg or more for *Haemonchus*.

If possible, egg counts should be checked before testing begins, to confirm adequate counts. Where counts in mobs to be tested are low and unlikely to increase within a short time, group mean counts can easily be increased by counting more chambers of the slide. A modest count of 200 epg becomes 400 epg when 2 chambers per sample are counted, although this approach increases the laboratory cost.

Sheep selection
Lambs aged around 6 months or less are usually ideal as egg counts are most likely to be adequate, and the range of counts within a group relatively uniform, in worm-naïve animals. Tests are therefore often conducted as lambs are weaned, but if counts at that time are too low, a small group of animals in good condition can be left untreated, with minimal effect on the whole-flock control program.

It is important that test sheep have not been recently drenched with a product of any anthelmintic group to be tested, to prevent an over-estimation of resistance due to worms that survived treatment. A relatively safe time period is to allow 2 months after drenching if on green pasture (and exposed to continual larval intake), and to avoid using sheep drenched while on dry pasture, or those treated with long-acting products. Samples should be of similar faecal consistency (and never fluid), to avoid random differences in egg count due to variation in dry matter content.

To ensure that the results are representative of the whole-farm situation, the test mob will ideally have grazed more than a single paddock, or their mothers will have done so. Ewe flocks should have received drenches typical of the property practice, and if they have received long-acting treatments, the lambs should be allowed to encounter other worm populations on the farm before testing.

The mob to be tested should be as uniform as possible in size, so there is minimal weight variation, and drenched to the heaviest in the group, as calculated for each test group. Randomisation to treatment groups is important but simple, and even sequential treatment with each drench type along a race is adequate, providing that the sheep are of reasonably even weight.

Which drenches to test ?
A compromise is needed regarding the number of drench products tested: a large number of test groups may discourage testing due to the work effort required, but too few may not effectively indicate available drenching options.

In most situations, a basic testing panel testing should include moxidectin and a triple-combination product that contains abamectin, and if used more than occasionally, the newer products "Startect" and monepantel. Abamectin is often included because of its role in multi-active products, if those are not tested themselves, or where use is contemplated as a sole active (increasingly less common). From this panel, a good estimate can be made of the likely result of drench combinations not specifically tested.

There is little justification for considering the older anthelmintics - benzimidazoles, levamisole and ivermectin – for routine use, due to the severity of resistance and their role as components of many combination anthelmintics (although levamisole as a single drench may still be effective against H.contortus). However, an alternative approach to resistance testing is to include the individual actives of combination drenches, as the basis of calculations for efficacy[9]. A practical limitation on this may be the availability of small quantities of the older drenches, and the number of drench actives needed to cover the likely spectrum of options.

In winter rainfall regions, where *H.contortus* is only occasionally seen and rarely a problem, large differences between pre-treatment means between groups can occur, due to sporadically-distributed extreme counts, which distort reduction results when compared with a control group. If the proportion of *H.contortus* is likely to be high, it can be removed with closantel at the time of treatment, and where present in small proportion, larval differentiations are especially essential. In summer rainfall regions, closantel should be tested against *H.contortus* as a routine.

Treatment to sampling interval

The usual recommendation for the final sampling is to allow 10 to 14 days after treatment: sampling too early risks an underestimate of resistance if treatment temporarily reduces egg production in resistant worms, but if left for longer than the minimum pre-patent period of strongyle species (about 16 – 17 days) eggs may derive from newly-developing worms. The post-treatment period of 10 days was established once the long-acting effects of benzimidazoles on Teladorsagia were recognised, but the ML drenches appear to have a longer period of potential suppressive effect on egg excretion (L. le Jambre, pers. com.).

While it unlikely that viable ML-resistant worms would produce no eggs at 10 days after treatment, a quantitative difference in egg production over time appears likely, and it is therefore recommended that where possible the final sampling is at 14 or even 15 days.

Worm egg count procedure: individual or bulk counts ?

Although it has been established that the mean worm egg count results are equivalent whether samples are processed using the McMaster technique for individual counts or a composite-sample “bulk” method[10], debate continues regarding the appropriate approach for drench resistance testing. Although more labour-intensive and therefore costly to perform, individual counts have the advantage that all positive counts are evident, and where a single count occurs in groups where complete efficacy is expected, the possibility that it is explained by mis-dosing of the animal can be seen. Against this, the lower cost of bulk counts allows larger sample numbers per group to be included.

In general, to expedite laboratory processing and reduce costs, bulk counts are appropriate, especially for groups dosed with drenches to which resistance is likely. As mis-dosing is uncommon in resistance tests (as operators are aware of the need for careful treatment), it may be sufficient to ensure that samples are not discarded before results are available, so they can be re-tested on an individual basis if queries arise over a particular group. (Best-practice laboratory procedure is to retain samples as a routine, until the case is finalised.)

DrenchTest or DrenchCheck ?

As an alternative to the WECRT (DrenchTest), the DrenchCheck is a simple method of testing the efficacy of a single drench, requiring little effort and minimal cost[9]. Faecal samples are taken from sheep when yarded for drenching, and then (ideally) 14 days later as paddock samples from the mob. Over the course of a year, a number of drenches can be easily assessed, preferably with a worm identification test to indicate the species tested.

The DrenchCheck format is most effective where the results indicate high efficacy, as it is not quantitative if less than a complete reduction in worm egg count is obtained (because almost always, different sheep are sampled at each occasion). In contrast, the more objective information from a Drench Test (WECRT) provides a cross-section of drench options at a particular time, and when compared with previous results, enables an assessment of the sustainability of current worm control program.

Optimal test format

The WECRT format outlined in the WormBoss program provides a relatively sensitive indication of drench effectiveness for the minimum effort and cost:

- Dosing on Day 1, with a non-drenched control group, and sampling only at Day 14 (earlier only if essential). Where counts are high and group sizes can therefore be small, the pre- and post-treatment format can be considered.
- Animal selection: animals less than one year of age are preferred, but older animals can be used if counts are sufficiently high and reasonably uniformly distributed (this requires individual worm egg counts). There should be the minimum weight variation within the group to avoid excessive over-dosing.
- Drenches tested: the drenches should be selected as likely to be effective from recent observations and appropriate for use in a sustainable program. Alternatively, a range of single actives can be included, and the combination results calculated.
- Group size: up to 20 sheep dosed and sampled provides a sensitive test, which is practical and cost-effective when egg bulk counts are used. A minimum group size would be 10, unless the animals are identified and pre- and post-treatment samples are taken – in this case, calculations are best based only on those with positive counts (minimum 50 eggs counted), with a minimum number of around seven per treatment group.
- Mean counts: the minimum of 200 epg for each important genus of the scour worm species is at the low edge of sensitivity, and up to 500 epg is desirable providing that faecal samples are not very soft or fluid. For *H.contortus*, a minimum of 500 epg is easily achievable and much higher counts are feasible before there is a risk of adverse effects.
- WEC methods: whether done by bulk or individual counts depends on laboratory policy, but bulk egg counting allows a larger number of samples per group to be processed at a cost lower than 10 individual counts. Where positive counts occur where not expected, the result can be verified by re-testing individual samples from retained material.
- Worm species/genus identification: this should be mandatory for the control group, to indicate the genera tested, and for any groups where the reduction is less than 100%. In major *H.contortus* zones and if only this species is of interest, the lectin binding assay provides an especially accurate figure. Molecular tests, where available, indicate the complete species range for each group. Costs can be reduced by not proceeding with identification tests in groups with a complete reduction in egg count.

Resistance testing is best conducted with the oversight of a veterinarian or livestock adviser to ensure optimal test procedures, and an informed interpretation of the results and their implications for a worm control program. The effort and cost involved are very small in relation to the benefits of using effective drenches, and the potential penalties if resistant worms survive.

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Aspects of assessing pain during disease and operations in farm animals

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The terms welfare, health, stress and pain are the same concepts whether we are considering a person, a calf or a trout. This point has been emphasised by many animal welfare scientists and is one of the messages of "one welfare"[1]. The welfare of an individual is its state as regards its attempts to cope with its environment[2] and pain is an aversive sensation and feeling associated with actual or potential tissue damage[3].

Whilst welfare ranges from very good to very poor, most people focus on the various forms of suffering when considering our obligations towards the animals that we keep. The problem often expressed in relation to pain in species other than man is that the animals cannot tell you when they are in pain or how bad it is. The major method used in human pain studies is self-reporting, for example on a scale from no pain to very severe pain. This method can be unreliable because people can lie or deceive themselves in relation to pain. Perhaps measures of observed behaviour or physiological change in people, like those used in non-human studies, will in future be considered more accurate than human reporting.

Some people think that pain is a feeling limited to humans, or to mammals, but many studies of anatomy, physiology and behaviour show clearly that pain systems are very similar in all vertebrates, cephalopod molluscs and decapod crustaceans[4,5]. There are variations in the area of the brain that does the pain analysis but little variation in the function. A further misconception is that animals such as cattle do not feel pain because they have thick skin. The simple observation that cattle react to individual mosquito bites demonstrates that the skin thickness does not prevent responses to painful stimuli. Thicker skin can reduce the likelihood or extent of abrasions following some contacts but the nociceptive cells under the skin function fully in animals like cattle.

Sophisticated behavioural measures are being used more and more in studies of pain. However, there are problems in pain recognition which make comparisons between species difficult. Severe pain can exist without any detectable sign. For example, a major response of rabbits that are in pain is inactivity[6]. Individuals within a species vary in the thresholds for the elicitation of pain responses and species vary greatly in the kinds of behavioural responses that are elicited by pain. Hence it is important to consider which behavioural pain responses are likely to be adaptive for any species that is being considered. Humans, like other large primates, dogs and pigs, live socially and can help one another when attacked by a predator. Parents may help offspring and other group members may help individuals who are attacked or otherwise in pain. Hence, distress signals such as loud vocalisations are adaptive when pain resulting from an injury is felt. Those species which can very seldom collaborate in defence, like the smaller ruminants, do not have obvious responses to pain as these are maladaptive. However, subtle changes in facial expression can be useful indicators of pain, for example in rabbits, rodents, sheep, goats and horses[7,8]. The sheep pain facial expression scale involves scoring five facial areas; orbital tightness, cheek tightness, ear position, lip and jaw profile, and nostril and philtrum position. Sheep with footrot, mastitis, or pregnancy toxaemia showed grimace responses and other indicators of pain. When farm ruminants are in pain because of farm operations, the increased cortisol production and an increased occurrence of a range of pain-related behaviours[9,10,11,12,13] can be quantified. For example, pain-related behaviours in calves include: head-shaking, ear-flicking, head-rubbing, inert lying, alterations in gait, amount of walking, licking scrotum, lifting hind leg, abnormal lying, rapid transitions between behaviours and reluctance to go to the food trough. Measures of brain activity can also be used[14] and pain can be prevented using anaesthetics and analgesics[15]. For the general public, pain prevention during farm animal operations such as castration and disbudding is demanded more and more, whilst most people think that more extreme operations like mulesing should be illegal. Consumers refuse to buy specific animal products unless pain is prevented and good welfare guaranteed.

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Sentience, welfare, ethics and human entertainment

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It has sometimes been thought acceptable to watch gladiators fighting, as long as they are slaves, to laugh at shows involving physically deformed and mentally subnormal people, and to remove livelihoods and dwellings to make way for modern developments as long as the people concerned are poor or black. There are various parallels when non-human animals are used for, or affected by, human entertainment activities. These issues raise questions about which individuals and communities have value and deserve respect. It has been written that God gave us dominion over the world but does "us" just mean humans and what should dominion mean? Is it right to focus greatly on humans when terms like health and welfare mean exactly the same for humans and other animals? Which humans and which other animals are sentient and to what extent does the answer matter?

Sentience means that the individual has the capacity to have feelings. A sentient being is one that, in order to have feelings, has some ability: to evaluate the actions of others in relation to itself and third parties, to remember some of its own actions and their consequences, to assess risks and benefits and to have some degree of awareness[1,2]. Recent advances in our knowledge of the complex nature of the behaviour, physiology and neurobiology of all vertebrate and some invertebrate animals tell us that their behaviour is far from just reflexive so they are sentient and have the ability to experience pain and other positive and negative feelings. For example, all fish have sophisticated learning abilities and almost all abilities reported for primates can also be found in fish[3]. The area of the brain that processes emotional function in fish may be the medial and lateral telencephalic pallium or the habenula instead of the equivalent mammalian region but it is the complexity of brain function that is the scientific issue, not the anatomical area[4,5]. When deciding which animals should have legislative protection the question of sentience and indeed whether an animal can suffer is a key factor.

If we know that sheep can recognise individual sheep and people and remember them for over a year, several mammals and birds can learn what is in a mirror, some birds and fish can alter their behaviour as they predict what is likely to happen in the future, some spiders can remember an evaluation and use the information later, does this alter our treatment of such animals?

Animals are among the most sought after tourist attractions and the impact on them is a matter of concern to an increasing number of people. Tourists and other people may chase wild animals with packs of dogs, shoot them accurately or inaccurately with guns, catch them on hooks or with harpoons, watch them in circuses or zoos, disturb them to varying extents in order to see them in their home environment or cause them to be caught in order that they can be kept as pets. The impacts on animal welfare of all of these human impacts can be scientifically measured. Another impact of tourism is the killing of many animals in case the tourists do not want to encounter them in their hotel room, swimming pool, etc. People seeking entertainment that involves or affects animals should know the impact on the welfare of those animals and consider whether or not it is morally acceptable[6].

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Some animal production methods are unsustainable: factors include poor welfare

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There is an urgent need for sustainable animal production systems. A system or procedure is sustainable if it is acceptable now and if its expected future effects are acceptable, in particular in relation to resource availability, consequences of functioning, and morality of action[1]. What might make any animal usage system unsustainable? The system might involve depletion of resources such that a resource becomes unavailable or a product of the system might accumulate to a degree that prevents the functioning of the system. However, any effect which the general public find unacceptable makes a system unsustainable. A production system might be unsustainable because of inefficient usage of world food resources; adverse effects on human welfare, including health; poor animal welfare; harmful environmental effects, such as low biodiversity or insufficient conservation; unacceptable genetic modification; not being "fair trade", in that producers in poor countries are not properly rewarded; or damage to rural communities[2]. Consumers might judge, because of any of these inadequacies, that the quality of the product is poor.

Inefficient use of world resources is likely to become much more important as a factor affecting plant and animal production[3]. Techniques such as life cycle analysis will have to be used in order to justify continuation of a system[4]. One consequence of this is that animals that eat leaves and other food that humans cannot eat, such as ruminants and herbivorous fish, will become more important than animals that are carnivores or that eat grain. Efforts to reduce greenhouse gas production by ruminants will be important and the trade-off between resource efficiency and greenhouse gases will have to be considered. Animal and plant production systems that use the resource inputs more efficiently have a less damaging effect on externalities such as land use, water use, nitrogen and phosphorus pollution and greenhouse gas production so they can make more land available for conservation[5].

Animal welfare is a component of sustainability and good quality of product. For many in the general public, animal welfare is the most important aspect of sustainability after human welfare. Systems with negative impacts on animal welfare are unsustainable and associated with poor product quality[6,7]. Three-level plant production, including pasture, shrubs with edible leaves, and trees that may also have edible leaves, are an example of a silvopastoral system. The production of leaves and other material that can be eaten by the animals is much greater than can be achieved by pasture-only systems. Results presented from tropical and sub-tropical studies show that production of cattle and other animals can be better, water and land use reduced, biodiversity much increased, animal disease reduced, and animal welfare improved in three-level silvopastoral systems[8,9].

In the near future, unsustainable practices in animal production will include: pollution of waterways by farm products, soil damage that releases greenhouse gases, avoidable food waste, production in which poor farmers are exploited by large companies, feeding fish to fish, feeding grain to ruminants, feedlots, individual confinement of sows and calves, battery cages for hens, force-feeding, high stocking densities, genetic selection for the highest milk yields in cows and for muscle growth in broilers, avoidable long distance transport, slaughter without stunning.

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The welfare of animals during transport

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An animal transport journey should be regarded as commencing when the first animal is loaded onto a vehicle and as ending when the last animal is unloaded. The journey includes any resting or transfer periods and a new journey starts only after 48 hours of adequate recuperation. There are many scientific studies of the welfare of animals during and after transport. The principal measures are heart-rate; glucocorticoids and other hormones and proteins in blood, saliva, urine or faeces; a range of behaviour measures such as avoidance movements, pain grimaces or inability to show normal movements; injuries that can be scored such as bruises, cuts or broken bones; immunosuppression; extent of pathology and survival rate[1].

Factors affecting welfare during transport include: knowledge and attitudes of staff involved, laws, codes of practice, training, species, breed, previous experience of the animals, vehicle design, space allowance for animals, duration of journey, tying, mixing, staff payment method, planning of journey, handling during loading and unloading, driving quality, bad physical conditions during journeys and quality of animal monitoring[1,2].

If those with responsibility for animals during transport receive training, this has major beneficial effects for animal welfare. Those responsible include owners, buying or selling agents, transport companies, vehicle owners and loading facility or slaughterhouse owners as well as animal handlers and vehicle drivers.[3] The greatest animal welfare problems during transport are: poor handling of animals leading to pain and fear, insufficient space on vehicles for animals to stand without contact with other animals or to lie down, poor conditions during the journey because of bad driving or bad physical conditions, or fatigue and exhaustion because the journey is too long. Maximum road journey times should be 4 hours for poultry, 8 hours for pigs, 15 hours for sheep, cattle and horses.[4]

Transport by ship can involve good conditions for the animals when the sea is calm, the stocking density low and the food appropriate. However, sea conditions can be very bad and on some ships, the stocking density is high enough for welfare to be poor throughout the voyage. The food provided for transported animals has sometimes been so different from that which the animals normally eat that they did not eat it and some died of inanition. Especially on longer journeys, ammonia concentration in the air breathed by the animals can be high enough to cause respiratory disease. The combination of high stocking density, inadequate ventilation and high external temperature can lead to very poor welfare and high mortality. A particular risk when the animals are imported by some countries is the use of cruel handling methods[5]. The risk of major problems is so great that journeys of more than 24 hours duration should be avoided.

When deciding whether or not a particular animal transport system or practice should be permitted, the risk of poor welfare should be assessed. Publicity about events that have very negative consequences for animal welfare can be very costly for commercial companies, or for whole countries, because large numbers of consumers may refuse to buy the products of the company or country[6]. Live export from Australia by ship is already resulting in some people refusing to buy any Australian goods and the number of people doing this is likely to rise. Carcass transport does not lead to such trade problems.

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Tourism and marine mammal welfare

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Tourist boat trips to observe seals, cetaceans, sea-otters, manatees, dugongs and polar bears have the general effect that the people who profit from such trips and often the governments of the countries involved want to conserve the animals. The public who might wish to visit such animals are in favour of conservation. As a consequence, most countries see more benefit in conservation than in killing marine animals, for example Argentina and Brazil. Since most marine mammal chasing and killing procedures cause very poor welfare, often for long periods, avoidance of killing reduces the amount of poor welfare in the animals[1].

Whilst some land animals are the subject of hunting for "sport" or for trophies, marine mammals are not a major target in this way. As with rhino horn, bear gall-bladders, tiger penises etc., there is some demand for marine mammal body parts. This can lead to poaching, even when most people want to protect the animals. However, for most species the demand is not great.

Marine mammals have been kept in captivity to impress people for three thousand years. Tourists are often paying customers at circuses, zoos and aquaria. The welfare problems are greatest for all animals taken from the wild and also when the keeping conditions are inadequate for meeting the needs of the animals. Training animals may make the life of the animal more interesting or may be associated with fear. Some captive cetaceans and most polar bears show stereotypies, many captive marine mammals have health and reproduction problems and the life expectancy of captive cetaceans is less than that of wild animals. All of these are measures of poor welfare[2,3,4], Some countries now ban the keeping of cetaceans for animal welfare reasons.

Boats taking tourists to see marine mammals using well-controlled observation methods may cause no negative effects on the animals. However, boat approach for observation, chasing by boats and swimming with the animals may cause: avoidance behaviour, reduced time for foraging, social disruption, injury, increased disease, reduced breeding success or reduced life expectancy. Dolphin populations declined where dolphin watching occurred, but there was no decline in a matched population where there was no dolphin watching. Since all of these negative effects can be avoided, it is essential that there should be codes of practice and laws to avoid poor welfare in the animals. Swimming with dolphins always seems to lead to avoidance of swimmers and sometimes to worse effects on the animals. If tourists are given good information about why not all expectations can be realised, they usually do not make demands that could lead to harm for the animals[5,6,7].

Other tourist activities, such as fishing tourism, may result in marine mammals being killed as the animals are perceived to be competitors with the humans who wish to fish. Sometimes, boats hit and injure or kill marine mammals.

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Welfare, sentience and law: an international perspective

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Most of the public think that it is important to use sustainable systems for all activities and that, whenever animals are used, their good welfare is an important aspect of sustainability and product quality. Consumers exert pressure on food retailers and governments to ensure that the worst practices do not continue. Animal welfare science has developed rapidly so scientific evidence can be used in codes and laws.

Laws relating to animal welfare generally refer to sentient animals that have the capacity to have feelings. Human opinion as to which individuals are sentient has changed over time in well-educated societies to encompass: firstly all humans instead of just a subset of humans, and then also: (a) certain mammals that were kept as companions, (b) animals that seemed most similar to humans e.g. monkeys, (c) the larger mammals, (d) all mammals, (e) all warm blooded animals, (f) all vertebrates and (g) some invertebrates. New knowledge about brain function and welfare has tended to show that the abilities and functioning of non-human animals are more complex than had previously been assumed so there should be some re-appraisal of which animals should be protected[1].

EFSA reports are commissioned before changes in E.U. legislation and have pioneered objective review of animal welfare issues using risk and benefit analysis. The sequence of procedures during the analysis of risks or benefits is: first to list factors (hazards if negative), second to calculate exposure, third to estimate uncertainty. The analysis may be quantitative if sufficient numerical information is available, or qualitative if it is not. The inclusion of risk analysis in scientific reports and opinions produced by EFSA and other organisations has helped decision makers to take appropriate action, for example to minimise animal disease and improve animal welfare[2,3].

What do we need from animal welfare law? Most people would say that the law should prevent people from causing poor welfare in animals: pain, fear, other suffering, severe disease, distress caused by environments which do not meet the animals' needs, or distress caused by the genetic selection used in breeding. In reality, the way that a law might do this is principally by acting as a deterrent. People who disobey the law are punished and this becomes known. Laws should provide guidance, not just a mechanism to punish[4]. Key points of the U.K. Animal Welfare Law 2006 are that it refers directly to animal welfare and that it refers to people having a duty of care to the animals covered by the law. The effectiveness of laws and codes depends on the attitudes of people to them and on the efficacy of enforcement.

Whale welfare can be assessed using many of the measures that are used for other animals[5]. Whales are sentient, good at learning and have a pain system. In relation to the whale hunt, studies of welfare should consider the effects of: the disturbance resulting from the approach of humans in boats, chasing by boats, a harpoon entering tissue, pulling on the line attached to the harpoon, tissue damage by an explosive harpoon and procedures during capture of individuals after they have been pulled to the whaling ship. After these matters had been discussed in the informal meetings, animal welfare was placed on the agenda of the I.W.C.

The rules of the World Trade Organization (W.T.O.) do not specify that animal welfare is an accepted ground for restricting trade but they do specify public morality as such a ground. The European Union banned trade in seal products on animal welfare grounds because of public pressure about how young seals were killed for their fur. Seals can be killed humanely, for example by a veterinarian in a laboratory or zoo. Seals on ice floes are often not humanely killed when they are clubbed or shot and, in these conditions, it is not possible to be certain of doing so. Some will be conscious, some will be unconscious and some will be dead but identification of unconsciousness and death is difficult. Without bleeding by cutting a major blood vessel, death cannot be certain. Skinning a conscious animal will cause extreme pain. The W.T.O. found that the E.U. seal law does not violate the Technical Barriers to Trade (TBT) agreement because it fulfils the objective of addressing E.U. public moral concerns on seal welfare. The E.U. ban on seal products was found not to violate the general Agreement on Tariffs and Trade (GATT). This ruling has consequences for other animal welfare issues[6].

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Global Burden of Animal Diseases: problem, solution and legacy

Global Burden of Animal Diseases: problem, solution and legacy

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Problem

A consistent and comparative description of diseases, the risk factors that cause them and the effectiveness of intervention strategies that are designed to mitigate these diseases, are important inputs into decision-making and planning processes. For human health, the global burden of diseases, injuries and risk factors study (GBD) has created a large comprehensive dataset used to measure epidemiological levels and trends worldwide. To date however, there is no systematic way to capture and measure losses associated with animal diseases, nor are the data on expenditure on disease mitigation analysed in a way that enables comparisons to be made. A system is required that regularly collects, validates, analyses, and disseminates information on livestock production and animal health economic effects to achieve evidence-based policy making and impact on the Sustainable Development Goals on health, nutrition, environment, and poverty.

Solution

The Global Burden of Animal Diseases (GBADs) program to address this problem was initiated in 2018 [2]. The program will estimate the losses attributable to diseases in livestock, as well as expenditure on disease mitigation; providing a total cost estimate of animal disease. This will be achieved by establishing standards and methods for animal disease statistics, data collection and data analysis, as well as the development of an integrated animal health database that is regularly updated and freely available.

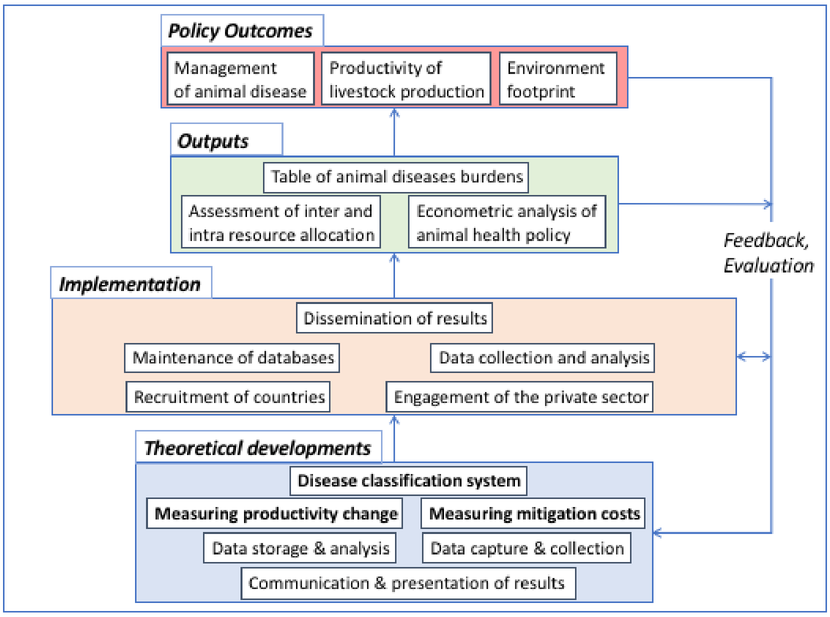

Figure 1. Schematic representation of the GBADs development, implementation and expected outputs

Legacy

The GBADs program will measure burdens to improve animal health through the establishment of: (i) a global standard for animal disease classification; (ii) information on the burden of animal health problems that can be differentiated between production loss, expenditure, trade and human health; and (iii) information on the current allocation of resources between diseases and within specific disease control programs. This will enable the assessment of animal health investments, the ability to perform economic analysis of section, national and global animal health policies, and the incorporation of social and economic analysis in animal health policy. GBADs will measure to improve animal health at local, national and global levels.

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Foal resuscitation – What is possible in the field?

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Acute resuscitation of neonatal foals is required most often in one of two scenarios: cardiopulmonary arrest in the immediate post-foaling period, and as a result of critical systemic illness in the neonatal period (most commonly the first 48-96 hours of life). These two scenarios tend to have different causes, require different treatment strategies, and often have quite different outcomes.

Preparation both in terms of training, mental preparation, and equipment, go a long way to improving outcomes for these foals.

Birth resuscitation

Neonatal foals can suffer cardiopulmonary arrest for a variety of reasons, including hypothermia, hypoxia, premature placental separation ('redbag') or congenital disease. Many causes are unpredictable prior to foaling. However, in up to 50% of cases, neonatal compromise or impending arrest in the immediate post-partum period may be suspected. These include prior maternal disease or signs of foetal distress (placentitis, meconium in the amniotic fluid) or dystocia. A maternal history of prior pregnancy loss or dystocia should also warrant preparation for foal birth resuscitation. Dystocia occurs in up to 12% of foalings,[1] with higher incidences reported in larger breed mares. When second stage labour is prolonged (greater than 40 minutes) foal survival is significantly lower.[2,3]

When presented with a dystocia, a high-risk pregnancy, or even a mare foaling without apparent complication, preparation is key to a good outcome. While this may involve a highly-resourced, coordinated team in a referral setting, field-based resuscitation can still be very effective for foals where the dystocia is resolved quickly. A specific box can be used and updated prior to each foaling season with the following items, most of which can be easily and cheaply obtained from veterinary suppliers or large online auction houses beginning with E:

- Towels to dry the foal
- Suction bulb/60 mL syringe and tubing
- Endotracheal tubes (55cm, a variety of sizes 7-10mm internal diameter)
- Self-inflating bag-valve mask (e.g. Ambu-bag)
- 1L fluid bags
- O_2 cylinder with regulator and tubing?
- Needles, syringes, stethoscope, pen torch
- Lactatometer/glucometer

APGAR scores are formalised methods to evaluate viability in newborn infants. Modified APGAR scores for foals have been described which evaluate the heart and respiratory rates, muscle tone and response to noxious stimuli at several timepoints. These scores can allow early detection of compromise requiring resuscitation. As a reminder, the mean time for foals to stand is 57 minutes and to nurse is 111 minutes.

Score	0	1	2
Heart rate	Absent	<60 beat per minute, irregular	>60 beats per minute, regular
Respiratory rate	Absent	Irregular	Regular
Muscle tone	Limp, lateral	Some flexion	Active, sternal
Reflex: nasal stimulation, ear tickle	No response	Grimace, weak ear flick	Sneeze/cough, flick/head shake

Score 7-8 = Normal; score 4-6 = mild to moderate compromise - stimulate, intranasal O2, ventilate; score 0-3 = severe compromise - begin CPR

Figure 1. Modified APGAR scoring system for foals.

Cardiopulmonary arrest

Commonly cited indications for cardiopulmonary resuscitation include gasping for more than 30 seconds or no breaths at all, a heart rate less than 60 beats per minute or no palpable pulse. However, not all foals should be resuscitated. Comorbidities such as severe congenital neurologic, cardiac or musculoskeletal disease, the inability to provide aftercare, severe prematurity, or severe financial constraints might preclude resuscitation.

Cardiopulmonary resuscitation

Due to the high incidence of respiratory arrest, the initial management in foals in the immediate post-partum period should focus on airway management. Biosecurity concerns mean that mouth to nose resuscitation is no longer recommended. The membranes should be cleared from the nose and mucus suctioned using a bulb syringe or 60mL syringe and rubber tubing. Suctioning should continue for less than 10 seconds due to vagal reflexes that can exacerbate bradycardia. Rubbing with towels is usually sufficient to stimulate spontaneous breathing. If this does not occur, endotracheal intubation is recommended to deliver positive pressure ventilation. This is best performed with a 55cm long 8-9mm internal diameter tube. Two brief attempts via the nose should be attempted with the foal's head and neck in extension. If these are unsuccessful, then orotracheal intubation should be attempted. Low-volume, infrequent breaths (no more than 500mL, 6-10 times per minute) can be given by a self-inflating bag-valve mask device. Oxygen is not required for appropriate ventilation, and in some instances is associated with harm in neonatal infants. While it may be useful if available, it should not be continued for long periods of time at 100%.

The ideal rate and technique for cardiac compressions in neonatal foals is unknown, although extrapolation from human medicine is fair. The ideal rate in humans is approximately 120 beats per minute, with a depth of approximately 4.5cm, centred over the left ventricle. Cardiac compressions in foals should occur just behind the elbow, with the foal in lateral recumbency against a hard surface. If rib fractures are present, the side with the fractures (or with the more cranial fractures if both sides are affected) should be placed downwards. Pauses in compressions should be kept to a minimum, as cardiac perfusion rapidly diminishes once thoracic compressions stop.

Controversy exists over whether cardiac compressions should occur during bradycardia; it makes more physiologic sense to use positive chronotropes like vagolytics in this instance. One example is glycopyrrolate at 2.2 μg/kg (note units) IV. Given the high incidence of respiratory arrest in neonates, doxapram can also be useful to improve respiration, and has been shown to be more effective than caffeine in neonatal encephalopathy[4] and respiratory acidosis.[5] Doxapram can be given at 0.5mg/kg IV. Adrenaline, if required, can be given at 0.01mg/kg (0.5mL of 1:1000 for a 50kg foal) every 2-3 minutes as required. However, recent studies in human medicine have cast doubt on the routine use of this dose in cardiac arrest management.[6] While defibrillation is the treatment of choice for ventricular fibrillation, it is rarely available in the field. The presence of ventricular fibrillation in foals denotes a grave prognosis, as it does not have as reversable a cause as coronary artery disease in humans.

During resuscitation, having a scribe is very useful to document vital parameters, medications administered and timings. Chest compressions are tiring, and operators should be frequently changed to avoid fatigue and poor-quality compressions. Signs of adequate resuscitation include return of a central (and subsequently peripheral) pulse and non-dilated pupils. In a hospital situation, end-tidal CO_2 and diastolic blood pressure are good tools to monitor the effectiveness of resuscitation. Resuscitation should cease when the heart rate is greater than 60 beats per minute and spontaneous breathing is regular, with a normal rate and depth. Alternatively, if the above has not occurred by 15-20 minutes post-arrest, return of spontaneous circulation is unlikely to occur.

Fluid therapy is typically not required during birth resuscitation as hypovolaemia is unlikely. Foals are born with increased body water while haemorrhage is the most common cause of hypovolaemia in newborns and is usually obvious.

Data for overall success rates in birth resuscitation are only available from referral centres. These suggest a survival to hospital discharge of 26% overall, which rises to 53% in cases that present with bradycardia only.[7] However, it may be that simple cases that respond well to basic life support are never presented to hospitals and so the survival rate may be higher than this.

Critically ill foals

The most important differentials in the young collapsed foal are sepsis, neonatal encephalopathy, prematurity, gastrointestinal disease (enterocolitis), neonatal isoerythrolysis, uroabdomen (usually presents a little later) and trauma.

These foals most often present in the first 48 hours, with mortality being highest during this time. Due to the, often rapid, progression of disease in these foals, resuscitative treatment is often required during the initial triage. A primary survey is targeted towards the most life-threatening problems, typically cardiovascular and respiratory. This may include the demeanour and recumbency status of the foal, evidence of perfusion status including mucous membrane characteristics, peripheral pulse amplitude, extremity temperature and venous fill, evidence of respiratory compromise such as an altered rate and pattern, and basic clinicopathologic findings such as glucose and lactate concentrations.

It should be noted that the purpose of the primary survey is not to obtain a definitive diagnosis, but to triage and address the immediate threats to life. Once the foal is stable, or fluid resuscitation has begun, a more detailed examination of each body system, along with history taking and diagnostics as appropriate, can take place. In cases where treatment is delayed, or illness sufficiently severe, cardiovascular collapse and subsequent cardiac arrest can occur. A paradigm shift has occurred over the last two decades in human medicine with regards to the resuscitation of non-traumatic cardiac arrests. Traumatic cardiac arrest management has also undergone many changes, although has a completely different focus and algorithm that is not as relevant here.

The main initial focus in cases of cardiac arrest in critically ill foals differs to that found in foals that arrest during the birthing process. Specifically, the initial emphasis if a pulse is lost as is immediate cardiac compressions. These should take place at the expense of securing an airway in the first instance, in contrast to the order in birth resuscitation. Causes of cardiac arrest in young, sick foals include hypovolaemia, hypothermia, hypoglycaemia, hypoxia, acidosis and bradycardia (and thus hypotension and poor myocardial perfusion). Therapy of the initial crashing foal is thus aimed at these factors. Unfortunately, the prognosis for survival in critically ill foals that arrest is typically less than 3-4%.

Due to the high prevalence of sepsis as a cause of critically ill foals, early initiation of broad-spectrum antibiotics is imperative. Research from around the world has demonstrated both an increase in the frequency of Gram-positive infections and a decrease in sensitivities to commonly used antibiotics. As such, antibiosis should be provided prior to confirmation of a septic process. While blood and other tissue cultures can be helpful in guiding therapy, antibiotics should not be delayed such that samples can be obtained.

Hypovolaemia occurs through decreased drinking, losses associated with the primary disease process (e.g. diarrhoea), and insensible losses. Sepsis also leads to a distributive shock state due to uncontrolled vasoplegia, leading to a 'functional' hypovolaemia. The aim of the initial fluid resuscitation is not to completely restore the entire fluid deficit, but to provide enough circulating volume to maintain perfusion to vital organs. Boluses of 20mL/kg (1L for 50kg foals, conveniently) can be administered with re-evaluation of vital parameters and perfusion after each bolus. Adequate perfusion will also lead to normalization of blood pH. If improvement is not observed, vasopressors are likely required, necessitating hospitalisation. Inotropes may also be required if bradycardia persists.

Sepsis can lead to insulin resistance and hyperglycaemia, but hypoglycaemia can often occur due to the relatively low glycogen stores that newborn foals possess. Adding 20mL of 50% glucose to the first bag of fluid provides a safe quantity of glucose for this purpose.

Sick foals, particularly septic and premature foals, have poor thermoregulatory function, and lose heat easily due to their high surface area to body mass ratio. Active warming should be avoided due to inadvertent peripheral vasodilation, but warmed fluids can be helpful in preventing further heat loss. One litre of fluids heated for 1 minute in a 1000W microwave and mixed very well is usually safe to administer. Passive warming through blankets and insulating bedding are also important. This should be considered if the foal is being transported to a referral institution post-resuscitation.

Hypoxaemia is most commonly treated with intranasal oxygen using nasal prongs or narrow (e.g. feeding) tubing secured to the foal's nose at 8-10L/min. A humidifier is not required for short periods of time (<1hr).

Conclusions

As is hopefully clear from above, many of the above problems can be anticipated based on prior maternal disease or foaling history. As such, educating on-farm personnel and ensuring basic resuscitation skills and equipment can markedly improve the outcome for many foals.

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Surgical extractions and avoiding complications

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- *Introduction*
- *Indications for tooth extraction*
- *Surgical extraction techniques*
- *Minimally invasive techniques*
- *Avoiding complications*

Introduction

Tooth extraction (exodontia) is performed commonly in small animal practice.
In the past, often due to poor extraction technique and/or inadequate instrumentation, exodontia had been considered a difficult and time-consuming procedure to perform. This lead some veterinarians to only extract very mobile periodontally compromised teeth and ignoring the fractured teeth with pulp exposures or feline tooth resorptions or other dental pathology requiring tooth extraction. Because extracting these teeth required a lot more effort, time and complexity, these teeth were left in situ to the ultimate detriment of the animal.

Today, due to the better training of our veterinary graduates, better technique and veterinary sourced instrumentation, extractions can be performed with a higher degree of predictability and less complications.

Minimally traumatic extraction protocols refer mainly to those extractions that do not involve raising a mucoperiosteal flap nor alveolar bone removal. Multi-rooted teeth can still be sectioned with a bur and extracted as single tooth units under this protocol.

With any extraction procedure, informed consent from the owner is required prior to performing one extraction or multiple extractions. The owner should be offered other options such as root canal therapy for endodontically involved teeth, or advanced periodontal therapy for periodontally involved teeth.

The commonest indications for extraction are:

- Advanced periodontal disease especially with tooth mobility and furcation involvement
- Complicated crown/root fractures and subsequent periapical pathology
- Traumatic occlusion
- Tooth resorptions in cats and dogs
- Persistent deciduous teeth
- Dental caries

- Supernumerary teeth
- Impacted teeth

Why can tooth extractions be so difficult?

Tooth extractions in dogs and cats can be difficult due in part to the large root/crown ratio, root curvature and in the case of multi-rooted teeth, the divergence of the roots. As mentioned before, poor technique and inappropriate instrumentation may also lead to extraction difficulties.

The use of *controlled* force is imperative to the successful delivery of the tooth. Impatience, frustration, combined with inadequate knowledge of oral anatomy including tooth/root anatomy is a recipe for disaster and iatrogenic damage.

Some of the complications seen in exodontia include fractured roots, soft tissue trauma, haemorrhage, oronasal fistula and jaw fracture. These complications are often due to poor treatment planning and extraction technique and can be minimized by following appropriate extraction principles. Careful planning, patience and atraumatic technique are essential components in successful exodontia.

Extraction techniques (simple and surgical)

Simple or closed tooth extractions are performed without raising a periosteal flap and alveolar bone removal. Surgical or open tooth extractions involve some form of flap and bone removal.

To perform a successful extraction, one needs to consider:

- Patient placed in an ideal position for extraction. Often lateral recumbency is the preferred position for tooth extraction
- Accessibility to the site- adequate access to the operative site is essential for success
- Good visibility- good lighting is essential. Handpieces with a fibre-optic light source also aid in visibility during surgical extractions. Magnification can also help.
- Knowledge of tooth attachment, and crown/root anatomy
- Knowledge of extraction techniques and instrumentation
- A preoperative intraoral radiograph is helpful to assess root structure and root angulation, root pathology, proximity of vital structures, canals and spaces, position of the furcation in multi-rooted teeth. A postoperative radiograph may be necessary to confirm complete removal of the tooth

Due to the large root/crown ratio in domestic pets, the removal of the lateral (buccal) wall of alveolar bone overlying the root may be a necessary component of extraction
In multi-rooted teeth, sectioning of the tooth into individual root segments aids in the tooth's removal.

Instrumentation

The use of both power-driven equipment and hand instruments is considered essential for a successful tooth extraction. Good surgical lighting, fibre-optic or LED light source on the high-speed handpiece and magnification for the operator are considered very useful in the extraction procedure.
The use of air driven or electric driven equipment is ideal for sectioning teeth as well as removing the buccal (lateral) bony wall.

In the case of air driven equipment, the water-cooled high-speed handpiece (350,000 RPM) with a tungsten-carbide round bur is used to remove the buccal bony plate, and with a straight or tapered fissure bur (FG #701 or #701L bur in dogs or FG #699 in cats), to section multi-rooted teeth. Round burs are used to remove alveolar bone- sizes FG ½ to 8 can be used depending on the size of the animal.

When using electric driven equipment, the slow speed handpiece allows the bur to spin at up to 30,000 RPM but with better torque than the air driven handpiece. Electric driven equipment can be used for sectioning teeth, removal of the lateral bony plate and with a *prophy* attachment, for polishing teeth. However, because there is no water spray, the burs must be cooled by dripping saline or water onto the bur head. This avoids thermal trauma to the underlying bone. The electric driven equipment is a cheaper alternative to air driven units for those veterinarians who only want to perform extractions and polish teeth post scale and clean.

A size 11 or 15 scalpel blade is required to sever the gingival attachment to the tooth as well as incising mucosa for various mucoperiosteal flaps.

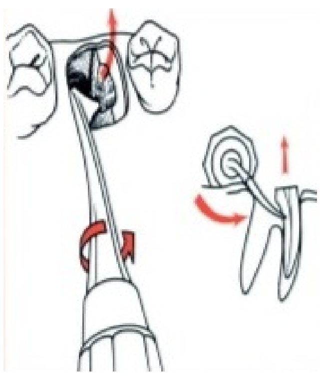
There are various periosteal elevators, with the number 9 Molt being a common choice for flap procedures. The Molt elevator has two different ends, a pointed sharp end for raising the interdental gingival papilla and a flatter, broad end for raising the mucoperiosteum off the bone. There are other smaller elevators that are more suited for small dogs and cats. Periosteal elevators can also be used for supporting the mucoperiosteal flap while alveolar bone is removed. Minnesota retractors can also be used to protect and hold the cheek, lips, mucoperiosteal flap or tongue out of the way when bone removal is occurring.

Adson tissue forceps are fine forceps used for holding the mucoperiosteal flap as it is being raised or when suturing the flap back.

There are several types of dental elevators of varying sizes. The commonest dental elevators are the straight Bein elevator or the winged elevator (developed by Dr Robert Wiggs). Dental elevators are composed of three parts, the handle, the shank and the blade. Most veterinary elevators are of a straight or slightly curved design. The blade of the instrument has both a concave and convex side with the concave side placed against the tooth surface either perpendicular to the tooth or along the long axis of the tooth. Select an appropriate sized elevator for the circum-ference of the tooth root to be extracted. The instrument is held by the handle with a tennis racquet grip with the index finger resting along the shank and blade of the elevator acting as a finger stop to avoid slippage. Winged elevators have a fine blade which needs to be kept

Man lifting a stone with a lever

sharpened to assist in cutting the periodontal ligament fibres (luxation). Winged elevators are also spoon shaped and hug the root surface well, allowing for a non- slip wriggling/twisting action to get as close to the apex of the tooth as possible. Human Cryer elevators have a sharp terminal point that can be used as a class 2 lever (see diagram below).
Elevators can be used as levers. They are classified as either class 1 or 2 levers.
Class 1 levers act like using a crowbar with a fulcrum point to move a large object such as a rock. The longer the lever arm (shank of elevator), the more force that can be applied to the object (tooth root). You should only use the elevator as a lever when the tooth is mobile, and you should only lever from close to the apex of the tooth.
Class 2 levers (wheel and axle) can also assist in the delivery of the tooth or tooth root from the alveolus. The effort is applied to the circumference of a wheel, which turns the axle, to raise a weight (tooth root). This levering technique is often used after wedging a dental elevator at several application points, between the tooth and the alveolar bone in the periodontal ligament space, to expand the alveolus and loosen the tooth. The dental elevator (often a Cryer elevator, which has a sharp point on the terminal blade) then inserts into either a natural defect in the tooth root or a man-made defect (made with a high-speed bur). Then in a rotational movement of the Cryer elevator, with the crestal bone as the fulcrum point, the root is rotated out of the alveolus.



Sharp fine dental elevators or “root tip picks” are used to extract fractured root tips and root remnants from the alveolus. Sometimes an Endodontic Hedstroem file can be used to retrieve a fractured root. The file is placed into the pulp canal of the loose fractured root and once engaged the file is withdrawn from the alveolus, hopefully with the root tip attached to the file.

Extraction forceps consist of a handle, hinge and beaks for grasping the tooth. There are now many different types of extraction forceps made specifically for veterinary dentistry.

Bone curettes can be used post tooth extraction to debride the alveolus of any pathology including granulation tissue.

Management of pain can be accomplished peri-operatively with opioid premedication and local anaesthetic nerve blocks, followed by post-operative pain management with either non-steroidal anti-inflammatory or opioid drugs.

Basic steps/*controlled* force

When performing dental extractions, certain steps should be followed.

Firstly, the gingival attachment to the tooth (usually at the cementoenamel junction of the tooth) should be severed with a number 11 or 15 scalpel blade. This frees up the gingiva from the tooth surface and allows for the introduction of a dental elevator (winged elevator of various sizes, Bein elevator or Coupland chisel 2mm. or 4 mm.). It is imperative when using elevators that the jaw and soft tissues be supported by the fingers and palm of the free hand to avoid iatrogenic jaw fracture. The dental elevator should be grasped in a tennis racquet grip with the index finger extending along the blade of the instrument to act as a stop, should the instrument slip while elevating. This grip also allows the operator to control the instrument better, and to avoid the application of excessive force. Elevators are angled at 10-20 degrees to the long axis of the tooth to avoid slipping. Once introduced, the elevator can be directed parallel to the long axis of the tooth. Elevators should have sharp cutting surfaces to facilitate severing the periodontal ligament (PDL). The elevator is placed between the gingiva and the tooth and controlled pressure is applied as the elevator is swung in a small arc.

It is sometimes necessary to remove part of the mesial or distal wall of the tooth to be extracted to allow for the introduction of the dental elevator. This may need to be done when teeth are in very close contact with each other such as the incisor teeth or maxillary or mandibular caudal premolar/molar teeth. Another option is to introduce the dental elevator perpendicular to the long axis of the tooth above the contact point between the tooth to be extracted and the neighbouring tooth and once a purchase point is established, start to run the dental elevator along the long axis of the tooth root.
The dental elevator is pushed along the root surface, severing the PDL. Once the elevator has moved sufficiently along the root surface, leverage force can be applied to the root.

At the end of each leverage arc, the elevator is held for about 30 seconds, so that the PDL fibres are cut. Haemorrhage caused by the elevator placed in the PDL space will aid in tooth delivery by expanding this space. The dental elevator can therefore be used not only to cut through the periodontal ligament but also as a lever or luxator, especially in the extraction of multi-rooted teeth. The leverage force is applied as close to the apex of the tooth as possible, and this force tears the PDL fibres. However, avoid levering the tooth until it has been loosened with the elevator. Do not use the elevator against a tooth that is not going to be extracted. Only lever against a tooth to be extracted or use crestal alveolar bone as a fulcrum point. Elevators can also be used as levers between sectioned tooth roots with the elevator applied perpendicular to the tooth and at the level of the crestal bone. This form of leverage must be finely controlled to not fracture a tooth root. Sometimes the tooth can be extracted by levering the tooth out of the alveolus without the application of extraction forceps.
For most teeth however, teeth are finally extracted with extraction forceps. When the tooth has been sufficiently loosened, and only then after loosening, can the extraction forceps be used.
The extraction forceps are placed as apical along the tooth as possible, to avoid root fracture, and are used to gently move the tooth one way, hold for a few seconds and then to move in the opposite direction and hold. Apical force with the forceps will also aid in PDL space expansion and aid in extraction. Once the tooth has been sufficiently loosened, it can be gently extracted with extraction forceps. Force should be avoided when using extraction forceps. If the tooth cannot be extracted easily with the forceps then more elevation is required, before retrying with the forceps.

Knowledge of the shape and angulation of the tooth root will assist in the extraction process. Conical roots will accept rotational forces from the extraction forceps as well as rostro-caudal and bucco-lingual forces. Teeth such as canines due to their oval cross-sectional shape accept rostro-caudal movements. It is only when the canine tooth is quite loose, that careful rotational force can be applied. Care needs to be taken when extracting the maxillary canine tooth especially in dolichocephalic dog breeds, to prevent iatrogenic perforation of the medial (palatal) cortical plate of bone causing an oronasal communication. Maxillary and mandibular incisors can be elevated and extracted with a bucco-lingual movement before any rotational force is applied.

With simple extractions, especially of single rooted teeth, it is not always necessary to suture the alveolus closed.

If mucoperiosteal flaps have been raised, they can be sutured back with a fine needle holder and 4/0 to 5/0 absorbable suture material such as Monosyn® (glyconate monofilament) or coated Vicryl® (braided polyglactin 910 or coated Vicryl plus® with antimicrobial) with a reverse cutting needle attached. Simple interrupted and tension free suturing techniques are recommended. The release of the periosteum at the base of the flap may be required to allow for tension free suturing. Alveoloplasty (smoothing over rough bony edges) is often performed prior to suturing.

Sometimes haemostatic agents such as Gelfoam® (purified porcine skin gelatine) are placed into extraction sites to promote haemostasis.

The use of osteoinductive agents (Synergy™, Consil® and other materials) for bone fill in an extraction site is not often indicated, unless performing extractions of large canine teeth where the alveolus dead space is large, or a cystic lesion is involved with the tooth extraction.

Surgical tooth extraction (canine tooth)

The canine tooth has a very long root making extraction a difficult and time-consuming process. Even with advanced periodontal disease and alveolar bone loss, there is still a large amount of periodontal attachment present.

The best method of extracting canines is to raise a mucoperiosteal flap (various techniques available including trapezoidal, triangular or envelope flaps) and bur away the lateral bony plate overlying the root, to aid in tooth removal. The vertical mucosal incision is best made well away from the tooth to prevent suturing over a dead space when closing. For trapezoidal flaps two vertical releasing incisions are made. For triangular flaps, one vertical releasing incision is made connected to a horizontal incision around the cervical areas of the tooth (teeth). Envelope flaps have only a horizontal incision made around the necks of the teeth with no vertical releasing incisions required. The full thickness flap is raised with a periosteal elevator and then a round bur in a slow or high-speed handpiece is used to remove the lateral alveolar plate (remember to ascertain where the apex of the canine is, by palpating the alveolar jugum). Care should be taken to avoid damaging the root of the maxillary 1st premolar when extracting the canine tooth. Once the plate of bone is removed, an elevator of appropriate size can be introduced, and the tooth loosened mesially, distally, labially and lingually. Be careful when using an elevator palatally for the maxillary canine tooth, to avoid an iatrogenic oronasal communication being created. Purchase points for the dental elevator can be made on the mesial (rostral) and distal (caudal) sides of the canine tooth, using a high-speed handpiece and round bur to create concavities in the bone next to the canine tooth root. Alternatively, a large triangular flap can be raised, and the labial plate of bone removed all the way apically to the apex of the canine tooth (the tooth is then elevated labially or laterally). Once the tooth has been loosened, the extraction forceps are used to grasp the tooth as far apically as possible and a mesial-distal rocking action followed by slight amounts of labial-lingual rotation is performed which will further loosen the tooth. When the tooth has become very mobile, extraction forceps can be reapplied as far apically as possible and the tooth extracted.

The alveolus is then curetted and flushed out with saline or chlorhexidine gluconate to remove any debris and then the wound is closed with absorbable simple interrupted sutures. Before suturing, a releasing incision is made in the periosteum at the apical extent of the flap. This allows the flap to be freed up and avoids excessive suture tension. Healing should be uneventful and rapid.

The mandibular canine tooth can be one of the more difficult teeth to extract in both dogs and cats. One approach is to raise an envelope or triangular flap on both the buccal and the lingual sides of the tooth taking care to avoid the mental nerve and blood vessels exiting the mental foramen (about level and ventral to the mesial root of the 2nd premolar tooth in dogs). Also, care must be taken to avoid damaging the 1st premolar tooth when removing alveolar bone close to its root. Some veterinary dentists sacrifice the mandibular 1st premolar to allow better access to and bone removal for the mandibular canine tooth. Caution needs to be exercised to avoid separating the mandibular symphysis or fracturing the rostral mandible. I usually cut a bony gutter around this tooth especially on the lingual side, to allow the introduction of dental elevators.

Multi-rooted tooth extraction

The example of a multi-rooted tooth used here will be the maxillary fourth premolar (carnassial tooth); however, the principles apply to any multi-rooted tooth.

Again, a mucoperiosteal flap is raised after the gingival attachment of the tooth is severed. Be careful when making the mesial releasing incision for the flap, to avoid the infraorbital foramen, with its rich supply of vessels and nerves. The foramen can be palpated very easily being just mesial to the maxillary fourth premolar about level with the distal root of the third premolar; however, there can be breed/species variation.

Once the flap is raised and the furcation identified, the tooth is sectioned with a fissure bur (i.e. # 701 or #701L) starting at the furcation and running the bur at right angles to the tooth towards the coronal tip.

When the mesiobuccal and distobuccal roots have been separated, a dental elevator is directed at right angles to the tooth. It is used as a wedge to tear the PDL fibres. Sometimes, the removal of the lateral (buccal) bony wall overlying the distobuccal root will aid in its removal.

Then, the mesiobuccal and mesiopalatal roots are separated with a fissure bur starting at their furcation and running the bur towards the crown. Alternatively, the bur can be directed in an angled mesial-distal direction just missing the distal coronal edge of the maxillary third premolar and starting coronally, the bur is run apically until the two mesial roots are separated. Another method, employed by some veterinary dentists, is to remove the mesial crown of the tooth (coronectomy) to visualise the furcation area between the mesiobuccal and mesiopalatal roots. Once separated, the mesiobuccal root is removed first, followed by the mesiopalatal root. The mesiopalatal root is very delicate, often curved and easily fractured. Be careful! Sometimes it is necessary to remove the inter-radicular bone between the mesiobuccal and mesiopalatal roots. Removing the crown overlying these two roots may also be helpful in identifying the furcation between the roots. However, this step may make it more difficult to elevate and remove the mesial roots.

Quite often, when closing the gingival flap, one must perform alveoloplasty first, to smooth over the rough bone edges and allow closure of the wound without tension.

Minimally invasive extraction techniques

More recently a new mechanical extraction device (iM3 Vet-Tome®) has been introduced. This instrument has a mechanically driven blade of various sizes that passes along the periodontal ligament space, thus loosening the tooth without the need for buccal or lingual alveolar bone removal. The outcome is a less traumatic tooth extraction that will allow for less post-operative pain and quicker healing of the extraction socket.

iM3 Vet-Tome® is a mechanically driven periotome. It is initially introduced at about 45 degrees to the root surface onto the mesial or distal surfaces of the tooth. In a side sweeping action, the iM3 Vet-Tome® is used in short bursts to cut along the periodontal ligament to close to the apex of the tooth and will eventually cut 360 degrees around the tooth, normally without the need to raise a mucoperiosteal flap, nor usually without the need to remove bone. There are various width blades that come with the iM3 Vet-Tome® and these can be matched to the size of the tooth. Once the tooth has become loose, dental elevators can be used to further loosen the tooth, following the path that the iM3 Vet-Tome® created. Finally, with the use of extraction forceps, the tooth can be extracted. The socket can be sutured, whilst maintaining the four bony walls of the socket.

What to do if a root fractures during extraction?

This can be a common occurrence during tooth extraction. Removal of the whole tooth is the gold standard to avoid postoperative complications. However, if a root fractures, an intraoperative intraoral radiograph may assist in your decision making. The decision on whether to search for the root or not will be based on the answers to such questions as

- Risk/benefit analysis-Will I do more harm than good in trying to find the root fragment? - consider referral
- Was the tooth's root canal system infected- if so then any remaining tooth structure must be removed
- How much of the root is remaining? The more root remaining, the greater the need to remove it
- Do I have the expertise/equipment to remove the root?

Ideally, the remaining tooth root should be removed with the following provisos:
If the tooth was periodontally compromised and the root fragment is below the alveolar bone level, consider leaving the root behind if removal is difficult or the risk of iatrogenic damage is great. Suture the gingiva closed, tell the owner, offer referral or monitor the site for uneventful healing. A postoperative radiograph should be taken to confirm the root position.

If the tooth was endodontically compromised and therefore infected, then the root fragment must be removed. Again, consider referral.
If the root is ankylosed due to internal/external resorption, then leaving the root behind is acceptable, but again one must inform the owner and monitor the patient for uneventful healing.

Avoiding complications associated with extractions

Complications can occur before/during/ or after extraction

Any adverse, unplanned event that tends to increase morbidity above what would be expected from an operative procedure under normal circumstances is a complication.

Factors involved in complications (see also Common complications seen in tooth extractions)

- *Patient factors* including sick, debilitated or immune-compromised patients
- *Surgeon factors* including inexperience, poor planning, use of excessive force, poor technique, lack of patience
- *Procedural factors* including inappropriate tools, difficult access.

To minimize extraction complications:

- <u>DO</u> have a plan i.e. cut multi-rooted teeth into single units. A good knowledge of the local anatomy including tooth anatomy is important
- <u>DO</u> have patience. Extractions take time
- <u>DO</u> use atraumatic techniques
- <u>DO NOT</u> use excessive force. The more controlled the force, the less chance of a complication occurring such as slippage of the instrument or fracturing a root

In summary,

- Be patient, do not use excessive force to extract teeth as this will result in root fracture
- Cut the mucosal attachment first, and then raise a mucoperiosteal flap to expose the lateral bony plate
- Section all multi-rooted teeth into single rooted segments for ease of extraction
- Know your anatomy, the proximity of the orbit, infraorbital foramen, nasal cavity, mandibular canal etc.
- Use finger/palm rests to prevent soft tissue damage or jaw fractures
- Do not use extraction forceps until the tooth has been sufficiently loosened with dental elevators
- Smooth over any sharp edges of bone and then place tension free absorbable sutures

The use of local anaesthesia in dentistry

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- Introduction
- Benefits of local anaesthesia
- Local anaesthetic agents
- Anatomical landmarks for local anaesthetic blocks
- Regional nerve block techniques
- Complications/adverse events

Introduction

Most dental procedures are elective and therefore conducive to pre-emptive analgesia techniques.
In human dentistry, the combination of local anaesthesia and post-operative pain relief with either non-steroidal anti-inflammatory drugs (NSAIDs) or opiates such as codeine are routinely used.
In veterinary dentistry, the use of local anaesthesia and post-operative NSAIDs works very well in managing peri-operative and post-operative pain. Local anaesthetics reversibly block impulse conduction in nerve fibres.
The types of dental procedures that require pain management include orofacial trauma cases, acute periodontal infections, gingivostomatitis, oral tumours and tooth extractions.

Analgesia protocols for dental patients

A good protocol for the management of dental pain includes:

- Opioid premedication (Methadone, Buprenorphine, Butorphanol);
- The use of local anaesthetic agents before any noxious stimulus and possibly at the end of the procedure;
- The use of NSAIDs* (Carprofen, Deracoxib, Ketoprofen, Meloxicam) and/or an opioid at the end of the procedure;
- 24−48 hours NSAIDs post-operatively.

* Care should be exercised in patients with renal disease, liver disease, gastric ulceration, hypotension, bleeding disorders
Opioids act centrally, at the level of the spinal cord and peripherally to alter pain response.
NSAIDs act both centrally and peripherally.
Opioids, local anaesthetic agents and NSAIDs can all act synergistically to produce good analgesia.

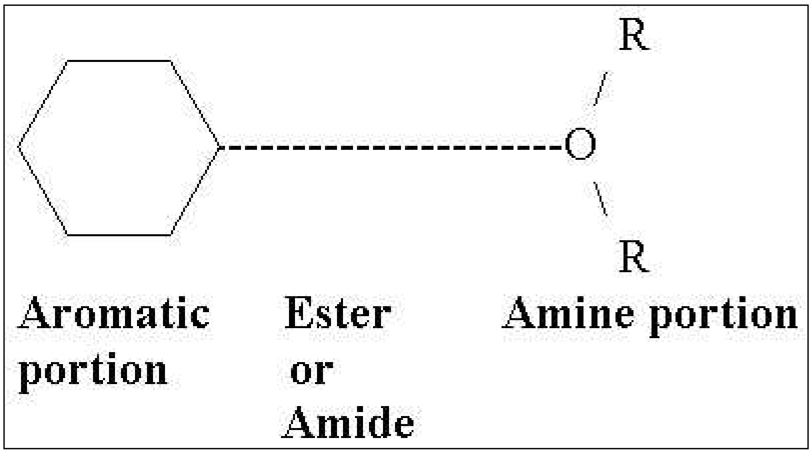
Benefits of Local Anaesthesia

Regional nerve blocks can be used for adjunctive/pre-emptive analgesia under general anaesthesia. They offer a low-cost method of achieving excellent analgesia in the oral cavity.
Pain during oral procedures arises both at the time of the noxious stimulus, i.e. surgery (1st phase), and is followed by a more prolonged pain response due to inflammation and release of inflammatory mediators that occurs during the surgery, and this pain can last for several days, if not longer (2nd phase).

Noxious stimuli cause a stimulation of the peripheral sensory nerve endings which leads to excitation of these nerve endings and transmission of excitatory impulses to the dorsal horn of the spinal cord. Here N-methyl- D-aspartate (NMDA) receptors are triggered and the stimulus is directed up the spinal cord to the cerebral cortex. With continued stimulation of the peripheral sensory nerve endings, the threshold for firing of these peripheral nerve endings is lowered and this induces a state of hyperalgesia with increased stimulation of the NMDA receptors, leading to a further amplification of the pain response or what is referred to as “wind-up” pain. The NMDA receptors remain hypersensitive, even after the peripheral noxious stimulus has ended. This central sensitisation can last for days if not weeks.
Modification of the pain response can be done either centrally, NMDA receptors (Opioid, NSAID), or peripherally (local anaesthesia, NSAIDs), or both.
Local anaesthetic agents prevent the transmission of painful stimuli to the dorsal horn NMDA receptors or higher receptors in the brain itself. Therefore, these agents are excellent for preventing “wind-up” pain (pre-emptive analgesia). This will allow better pain management as well as lowering the dose or reducing the treatment course for further post-operative analgesia.
Vasoconstrictor agents (often adrenaline) added to the mixture lengthen the duration of action of the local anaesthetic drug by slowing vascular uptake of the agent. Vasoconstrictors also reduce the chance of systemic side effects due to this reduced uptake of drug by the vascular system. Ischaemia is not normally an issue encountered in the oral cavity when a vasoconstrictor is used with a local anaesthetic agent.
The injection techniques are easy to master and can give up to 4–6 hours of anaesthesia/analgesia after one injection. Local anaesthesia complements general anaesthesia and allows for a lower concentration of the inhalant anaesthetic agent, which may be beneficial when dealing with older or more debilitated pets.
The nerves most commonly blocked for oral surgical procedures include maxillary nerve, mandibular nerve (inferior alveolar nerve), infraorbital nerve, greater palatine nerve and the mental nerve.

Local anaesthetic agents

Local anaesthetic agents are both water (amine portion) and lipid soluble (aromatic portion). Water solubility is required to get the anaesthetic agent to the site of its action and lipid solubility allows the active agent (unionized form or electrically neutral form) to migrate through the hydrophobic lipid-rich axonal membrane into the axon. Once in the axon, the agent transforms into the ionised form and binds to the sodium channels. Local anaesthetic agents are used in veterinary dentistry primarily as nerve blocks or for mucosal infiltration. Local anaesthetic agents are either ester-linked (cocaine) or amide-linked drugs based on their chemical structure. Ester-linked anaesthetic agents are rarely used today in veterinary and human medicine due to factors including possible allergic reaction, short duration of action due to being broken down by pseudocholinesterases and the potential cause of acquired methaemoglobinaemia.



Local anaesthetic agents are weak bases and at body pH can diffuse through tissues. They stabilise neuronal membranes and prevent depolarisation and the production of an action potential by blocking sodium voltage-gated channels within the membrane. This prevents the initiation and conduction of the electrical impulse along the nerve fibre (prevents depolarisation).
Local anaesthetics preferentially block pain and autonomic neurons especially A∂ (small

myelinated fibres) and C (unmyelinated fibres) nociceptors. Local anaesthetics need greater concentrations to penetrate (causing nerve blockade) into larger diameter nerves such as maxillary and mandibular nerves or motor nerves.

The two most commonly used local anaesthetic agents in oral surgery are Lignocaine hydrochloride (Lignocaine, founded in 1943) and Bupivacaine HCl (off-label use in dogs/cats). Both drugs are amides and work best in either a neutral or basic pH environment. Acidic environments, as seen when infection is present, affect the ability of the local anaesthetic agent to transform from an ionised state (due to low tissue pH) to a unionised state to penetrate the axonal membrane.

Onset of action of LA

The pKa is the pH at which the drug is 50% ionised and 50% unionised. Ionised drugs are poorly lipid soluble.

The closer the pKa is to local tissue pH (usually 7.4), the more unionised the drug is, or, the higher the pKa, the more ionised. Because all local anaesthetics are weak bases, those with a pKa near physiological pH (7.4) will have more molecules in the unionised lipid soluble form (e.g. lignocaine) and therefore have a more rapid onset of action.

Importance:

- lower pKa equals better absorption into the axon (lignocaine)
- higher pKa equals more effective blockade within the axon (Bupivacaine)

Lignocaine without adrenaline can last up to one hour and Bupivacaine without adrenaline can last around four hours. Both Lignocaine and Bupivacaine are metabolised by the liver and excreted through the kidneys. Both agents are protein bound and their duration of action depends on how highly protein bound they are (the more potent the LA, the more protein bound it is). Bupivacaine is very highly bound to neuronal membrane proteins and thus has a longer duration of effect when compared to Lignocaine. Lignocaine has a faster onset of action after administration (about 5 minutes) when compared to Bupivacaine (up to 10 minutes).

This is because more of the Lignocaine is in the lipid soluble unionised form and acts more rapidly in preventing nerve depolarisation (see above).

The addition of sodium bicarbonate to the anaesthetic agent (1 ml of 8.4% sodium bicarbonate per 10 ml of Lignocaine) can accelerate the onset of action of the drug by raising the pH of the drug solution and allowing for more of the drug to be in the unionised form to penetrate the axonal membrane. However, the addition of 1-part sodium bicarbonate to 9-parts Lignocaine will shorten the shelf life of the product. Also, do not exceed a 10% sodium bicarbonate concentration, otherwise precipitates may occur within the syringe.

On the other hand, dilution of the agent with 0.9% saline will affect the concentration of the drug and therefore affect onset of action and efficacy.

Any dilution of anaesthetic agent with sodium bicarbonate or saline should be done immediately prior to the injection being given.

Some veterinary dentists like to mix anaesthetic agents together. The main reason is to get a more rapid onset of anaesthesia (Lignocaine) combined with the longer duration of anaesthesia (Bupivacaine). However, there are few clinical studies showing any benefits of mixing Lignocaine 2% with Bupivacaine 0.5%. The author does not recommend the mixing of different anaesthetic agents (so called anaesthetic “cocktails”). Lignocaine can always be given again if required, about an hour into the surgical procedure without any adverse effects.

It should be remembered that the larger the diameter of the nerve fibre, the greater the concentration of anaesthetic agent is required to produce anaesthesia. A greater volume of anaesthetic agent may be required for the maxillary and caudal mandibular blocks.

The maximum accumulative dose for lignocaine hydrochloride is 4mg/Kg bodyweight in dogs (2mg/Kg cats) and for Bupivacaine is 2 mg/Kg body weight (may need to consider lower doses for immature animals as is recommended in children due to differences in pharmacokinetics and slower clearance by the liver).

A 5 Kg canine patient could receive 1.0 ml of lignocaine hydrochloride or 2ml of bupivacaine, total dose.

A tuberculin/insulin syringe and 25−27gauge ¾ to 1¼ inch needle plus local anaesthetic agent of choice (lignocaine 2% or bupivacaine 0.5% without adrenaline) are routinely used in the delivery of regional nerve blocks.

Anatomical landmarks for nerve blocks

The afferent sensory nerves that detect pain in and around the oral cavity are branches of the trigeminal or fifth cranial nerve (V) nerve. The trigeminal nerve arises from the trigeminal ganglion in the brain. The nerve has three divisions. The most dorsal is the V1 division which exits through the supraorbital fissure and branches into the lacrimal, frontal, nasociliary, supratrochlear and supraorbital branches. The maxillary division (V2) originates at the trigeminal ganglion and exits the foramen rotundum and branches into the maxillary, zygomatic, greater and lesser palatine and infraorbital branches. The third division (V3) arises from the trigeminal ganglion and exits the foramen ovale and has sensory branches including the buccal, lingual inferior alveolar nerve as well as a motor branch to mylohyoid which is motor to the anterior belly of the digastric muscle and the mylohyoid muscle.
The nerves of interest for regional blocks in the oral cavity include the maxillary, the infraorbital, the greater palatine, the inferior alveolar and the mental nerves. The maxillary nerve is blocked as it courses through the pterygopalatine fossa, the infraorbital nerve blocked within the rostral portion of the infraorbital canal. The greater palatine nerve can be blocked as it exits through the greater palatine foramen onto the hard palate and this will block sensory innervation to the hard palate and palatal mucosa.
The inferior alveolar nerve courses ventrally and enters the medial aspect of the mandible at the mandibular foramen. As this nerve continues through the mandibular canal, sensory branches exit providing sensory innervation to the teeth of the mandible. Rostrally (mesially) the inferior alveolar nerve branches into the mental nerves, which exit the mandible through the three mental foramina (the largest of which is the 2nd). The inferior alveolar nerve continues in the mandible to supply sensory innervation to the mandibular incisor and canine teeth. The mental nerves supply sensory innervation to the lower lip, chin and labial gingiva and mucosa.

Regional nerve blocks techniques

There are 5 major regional nerve blocks:

- Caudal Maxillary (Maxillary) block
- Rostral Maxillary (Infraorbital) block
- Major Palatine infiltration/block
- Caudal Mandibular (Inferior alveolar) block
- Rostral Mandibular (Mental) block

Of these the two major blocks are the caudal maxillary and caudal mandibular blocks.

Caudal Maxillary (Maxillary) nerve block

Intraoral approach:
The anaesthetic agent is placed into the pterygopalatine fossa region. This fossa is bounded by the sphenoid, palatine and maxillary bones. The maxillary nerve exits the foramen rotundum in the skull base and divides into the infraorbital, zygomatic and pterygopalatine sensory nerves. The maxillary artery and vein run with the nerve.
This block affects sensory perception to all the teeth of the maxilla (through the superior alveolar nerve branches off the infraorbital nerve), maxillary bone and the buccal and palatal mucosa and soft palate (the major and minor palatine nerves are also blocked) on the injected side. It also anaesthetises the labial mucosa in the canine/incisor area as well the upper lip and nares on that side. It may also anaesthetise the mucosal lining of the nasal cavity on that side.

Firstly, a short (5/8 inch for 25G) 25G or 27G needle is bent at a 45-degree angle. Insert the needle with the tip perpendicular to the hard palate behind the 2nd molar tooth or in cats at the demarcation between the hard and soft palates just medial to the maxillary 1st molar tooth. In cats and brachycephalic dogs, the needle should only go into this area about 5mm. In large dogs the needle can go in up to 15mm. Run the needle adjacent to the edge of the hard palate bone and in a dorso-rostral direction perpendicular to the hard palate. The injection site should be just above the apices of the 1st/2nd molar teeth. Aspirate to make certain that the needle is not in a blood vessel and slowly inject. There are a number of blood vessels in this area and care must be taken to avoid haematoma formation and possible ipsilateral exophthalmos. It is also a good idea to lubricate the eye on that side in case a branch of the facial nerve or the ophthalmic branch of the trigeminal nerve is affected, and the blinking reflex is lost for the duration of the local anaesthetic action. Be especially careful of avoiding globe penetration in brachycephalic breeds of dog and cats because the globe in these animals is much larger.

Extraoral approach (not recommended)
Insert the needle through the skin at a 90-degree angle, in a medial direction, ventral to the border of the zygomatic arch and about 0.5 cm caudal to the lateral canthus of the eye, and then advance it toward the pterygopalatine fossa. Frequently, the needle will contact the ramus of the mandible; if you do so, walk it off the ramus cranially. Slowly inject the local anaesthetic after aspiration. This technique is too difficult in cats.

Rostral Maxillary nerve block (Infraorbital nerve block)

Intraoral approach: The rostral infraorbital nerve block stops sensory innervation to the maxillary teeth from PM3 to the 1st incisor (middle and rostral superior alveolar nerves). If the caudal component of this nerve block is used (a needle is passes to the level of the medial canthus of the eye through the infraorbital canal), the maxillary 1st and 2nd molar teeth may also be blocked (caudal superior alveolar nerve branch, although this may not always be effective- the caudal maxillary block should then be considered). The block also anaesthetises the buccal/labial mucosa, upper lip and skin to the level of the infraorbital canal, as well as the nares and maxillary bone on that side, but not the hard or soft palate mucosa.
The infraorbital nerve branches from the maxillary nerve (the other branches are the major and minor palatine nerves) at the level of the pterygopalatine fossa. The infraorbital nerve enters the maxillary foramen and exits at the infraorbital foramen. Prior to entering the maxillary foramen, the infraorbital nerve gives of the caudal superior alveolar nerve that supplies innervation to M1 and M2 teeth. The nerve branches within the infraorbital canal into the middle superior alveolar branch (PM teeth 1, 2, 3, 4), rostral superior alveolar branch (canine, incisors) just before the infraorbital nerve exits the infraorbital canal through the infraorbital foramen. The nerve then branches into rostral and lateral nasal branches supplying the buccal and labial mucosa, as well as the upper lip and nares. There can be cross over from the other side so sometimes the central incisors may not be completely anaesthetised. A splash or infiltration of local anaesthetic in the central incisor area will alleviate this issue.
The infraorbital foramen is usually easily palpated about level with and dorsal to the distal root apex of the maxillary 3rd premolar (usually more rostral in cats). The needle can be advanced a few mm. caudally into the canal via the foramen. Aspirate and then inject slowly into the canal with finger pressure over the foramen. This will allow for diffusion of the anaesthetic agent caudally along the canal to anaesthetise the caudal premolar teeth. As mentioned before, in cats and brachycephalic dogs, the infraorbital canal is very short (ends about level with medial canthus of the eye) and the needle **should not** be advanced too far caudally.

Major Palatine nerve block

This nerve block blocks sensory perception to the mucosa and bone of the hard palate rostral to the maxillary 4th premolar. The injection site lies halfway in a buccal-palatal (lateral-medial) direction from the mid palatal suture to the midpoint of the maxillary 4th premolar in dogs and slightly more rostral (mesial) at the level of the mesiopalatal cusp tip of the 4th premolar in cats. The foramen is usually not palpable, so anaesthetic agent is infiltrated near the foramen and reaches the foramen via diffusion. Be careful re the hub of the needle dislodging, because the palatal mucosa is firmly attached to the underlying bone and you will be injecting with a reasonable amount of force.

Caudal Mandibular (inferior alveolar) nerve block

The caudal mandibular nerve block blocks all sensory innervation to all the mandibular teeth, buccal soft tissue (sometimes infiltration around buccal nerve* is required also) and bone on that side. Due to the proximity of the lingual nerve to the inferior alveolar nerve, the lingual nerve may be inadvertently blocked also (may be dependent on the volume of local anaesthetic agent given). This blocks sensory perception to the soft tissue on the lingual surface of the mandible and to one half of the rostral 2/3 of the tongue to the midline (see complications section).
*The buccal nerve courses over the caudal extent of the body of the mandible near the angle of the mandible.

Extraoral technique

The facial vascular notch (poorly defined in cats) is on the caudal ventral part of the mandible rostro-dorsal to the angular process.
The notch may not always be palpable. If not, then a vertical line from the midpoint of the zygomatic arch (or lateral canthus of the eye) to the ventral mandible identifies the notch and the mandibular nerve usually lies along this imaginary vertical line.
The needle is inserted extra-orally ventral to this vascular notch and advanced along this imaginary vertical line along the medial (lingual) side of the mandible to half the dorsoventral height of the body of the mandible at the level of 1st molar. The needle should then be near the mandibular foramen where the mandibular nerve (branch of the trigeminal nerve) enters the mandible on the medial (lingual) side.

Intraoral technique

The mandibular foramen can be palpated on the medial (lingual) surface of the mandible in medium to large dogs. It lies about half to two-thirds of the way along an imaginary line drawn from the mandibular 3rd molar to the angular process of the mandible, which is the most caudal ventral point of the mandible.
The needle is advanced along this imaginary line from the ipsilateral mandible along the lingual surface of the mandible. One finger can lie on the foramen and the needle advanced to this point. The syringe is then aspirated, and the injection given slowly around the foramen.
An alternative technique is to place a needle ipsilateral behind the 3rd molar tooth in dogs at the angle of the mandible. In cats, the needle is inserted at the angle of the mandible behind the mandibular 1st molar tooth. The needle should be angled at approximately 30 degrees to the horizontal ramus of the mandible and inserted to a distance which is equal to the height of the mandible from the dorsal to ventral cortex, in the mandibular 1st molar region. The needle should hug the medial surface of the ramus of the mandible.

Rostral Mandibular (Mental) nerve block

This block will block all teeth, bone and labial gingivae rostral to mandibular PM2 in dogs.
The middle mental foramen is the largest of the three mental foramina in dogs (rostral, middle and caudal).
The foramen is usually located on the labial side at the level of the mesial (rostral) root apex of the mandibular 2nd premolar or caudal (distal) to the labial frenulum in small dogs. In dogs, it is located about 1/3 the height dorsal to the ventral border of the mandible. In the cat it is level

with the labial frenulum, approximately equidistant between the mandibular 3rd premolar and the canine tooth and about half way in a dorsoventral direction.
The needle is inserted from a rostro-caudal direction into the foramen and the syringe is aspirated to avoid injecting into a blood vessel. The injection is given into the foramen and finger pressure applied over the injection site for about one minute.

Complications and adverse events

Local anaesthetic complications

Local effects

Haemorrhage: haematoma formation from a damaged blood vessel can lead to globe protrusion when performing the caudal maxillary block. Often caused by too large a gauge needle, going too deep into the pterygopalatine fossa or not aspirating prior to injecting local anaesthetic agent (LA)
Globe penetration: Usually associated with caudal maxillary nerve block. This may lead to inflammation/haemorrhage within the globe and eventually blindness. Can also occur using the rostral maxillary block in cats or brachycephalic breeds of dog, when the needle is passed too far caudally along the infraorbital canal.
Tongue chewing post caudal mandibular block
Paraesthesia: Causes of temporary or persistent paraesthesia include direct nerve damage following needle penetration of the nerve whilst injecting local anaesthetic agent or the development of haemorrhage or haematoma around the nerve sheath leading to necrosis of the neural tissue. It may be very difficult to ascertain that an animal has any loss of sensory perception to lips etc.
Facial nerve paralysis: Also misdirected delivery of LA can cause anaesthesia to branches of the facial nerve leading to temporary loss of blink reflex in the eye. Temporary blindness may occur when LA agent inadvertently infiltrates around optic nerve.
Scarring within a canal: Has been reported after multiple infraorbital blocks May lead to persistent infraorbital nerve damage.

Systemic effects
These are usually associated with delivery of LA into a blood vessel and effects on cardiac output. If overdose occurs: seizures or hypotension leading do cardiorespiratory arrest
Bupivacaine HCl is more cardiotoxic than lignocaine and inadvertent intravenous injection of Bupivacaine HCl should be avoided.

In summary:
Local anaesthetic techniques:
- Are easily mastered with a little practice and knowing the anatomic landmarks;
- Allow you to use less anaesthetic agent; lighter anaesthesia results in quicker recovery;
- Be careful and avoid overdosing;
- Always aspirate before injecting and during injecting;
- Follow up with post-operative pain management.

Common Avian Presentations in Veterinary Practice

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This paper will discuss the avian diseases you could expect to normally encounter in veterinary practice in Australia.

Any avian examination begins with first contact with your practice. Appendix 1 contains an approach to clinical examination that demonstrates how to perform a clinical examination You need a structured process to begin your diagnosis and this is a well established method.

INFECTIOUS DISEASES

Viruses
- Psittacine Circovirus - Beak and Feather Disease
- Polyomavirus

Bacteria
- Salmonella & E.coli
- Pseudomonas
- Psittacosis/Chlamydia

Fungi
- Aspergillosis
- Macrorhabdosis (Megabacteria)
- Candidiasis

Protozoans
- Trichomonas
- Giardia
- Coccidiosis

Parasites
- Ascarids
- Capillaria
- Cestodes
- Cnemidocoptes
- Air Sac mite

PSITTACINE BEAK AND FEATHER DISEASE

Aetiology

Viruses of the family Circoviridae

Signs & Symptoms

Signs may vary greatly. Generally a disease of young birds but may appear in older birds.

Feather changes, lesions of the beak and occasionally of the nails, and are usually symmetrical.

Different forms of the disease seem to be influenced by the age of the bird when symptoms first appear.

Peracute – seen in neonates

- Septicemia
- Pneumonia
- Enteritis
- Rapid weight loss and death

Acute - usually reported during first feather formation

- Depression
- Sudden changes in developing feathers including necrosis, fractures, bending, bleeding or premature shedding of diseased feathers.
- May not show feather symptoms but develop crop stasis and diarrhoea followed by death.

Chronic

- Progressive appearance and abnormally developed feathers during each successive moult.
- Dystrophy of powder down feathers, found on dorsal aspect of each hip. Unlike most feathers they do not display dystrophy following a moult, Powder down feathers are constantly growing and display dystrophy earlier. Lack of powder down also leads to dull, stained and soiled feathers.
- Retention of feather sheaths
- Haemorrhage within the pulp cavity
- Fractures of the proximal rachis and failure of developing feathers to ex-sheathe.
- Short clubbed feathers
- Deformed curled feathers
- Stress lines within vanes and circumferential constrictions may also be present.
- Replacement feathers are increasingly abnormal and birds may become bald as feather follicles cease to function.
- Changes in beak: progressive elongation, fractures necrosis and ulceration.

Laboratory Findings

- Basophilic intranuclear and intracytoplasmic inclusion bodies are seen on haematoxylin and eosin staining of sections of beak, feathers, thymus and bursa in birds
- Immuno-histochemical staining with viral specific antibodies to confirm intracytoplasmic basophilic inclusion bodies
- DNA probes are used to detect Beak and Feather Disease Virus (BFDV) nucleic acid. PCR is regarded by many as the most sensitive test.
- Haemagglutination tests can be run to determine immunologic response to PBFD virus.
  - Circovirus antibody Titre detected by Haemagglutination Inhibition (HI) test

– Circovirus antigen Titre detected by Haemagglutination (HA) test

Table 1: **4 possible outcomes for HA & HI testing :**

	Virus Antigen (HA) positive	**Virus Antigen (HA) negative**
Antibody (HI) positive	Active virus carrierMay occasionally be a bird that has completely or partially recovered – more common in: Budgerigars, Rainbow Lorikeets, African Lovebirds, King Parrots and Eclectus parrots.	Has been exposed to BFDV and has recovered Consistent with previous disease within an aviary, particularly if it is a closed aviary
Antibody (HI) negative	Peracute or chronic psittacine circovirus disease case Active carrier Unlikely to recover	Has not been exposed Agar gel immunodiffusion tests can detect precipitating antibodies to PBFD virus.

Prevention

- The major routes of infection are from dust from faeces, feathers and from parental feeding of young. The virus may be present in all body excretions and secretions as well as shed from GIT mucosa, skin and feathers
- Any bird which has been exposed to possible sources of infection should be isolated.
- The tests mentioned above such as HA, HI and PCR for BFDV should be used to test all birds of susceptible species to rule out latent infection.
- The PCR can also be used to test the aviary environment and equipment for possible contamination.

Treatment

- Stress free environment and supportive medical care.
- Isolation from other susceptible birds.
- No known specific treatment at this time.
- Euthanasia is often recommended

POLYOMAVIRUS

Aetiology

- Polyomavirus and commonly known as Psittacine Papovavirus and Budgerigar Fledgling Disease

Signs & Symptoms

- Depression
- Anorexia

- weight loss
- delayed crop emptying
- regurgitation
- diarrhoea
- dehydration
- subcutaneous haemorrhages
- dyspnoea
- polyuria
- ataxia
- tremors
- paralysis
- Common at the time of weaning and fledglings usually die 12 to 48 hours after developing symptoms.
- Chronic form has intermittent symptoms.
- Carrier state is possible.
- Can affect any parrot, but most commonly seen in macaws, conures, eclectus parrots and caiques

Laboratory Findings

- Staining of lesions using viral specific antibodies or polyomavirus specific DNA probes.
- Immunodiffusion and virus neutralisation to detect polyomavirus antibodies in exposed birds.
- This antibody response is transient and birds revert to sero-negative over a short period of time.
- Best test for birds shedding polyomavirus is a cloacal swab.

Prevention

- Polyomavirus survives well in most environments and unaffected by many disinfectants.
- Transmission can take place through feather dust and excrement and through parental feeding of chicks.
- Isolate any birds tested to be shedding the virus.
- Interrupt the breeding cycle with a rest of several months since the disease seems to be self-limiting.

Treatment

- No confirmed therapies are known.

<u>CHLAMYDIA</u>

Commonly referred to as Psittacosis or Parrot Fever

Aetiology:

Chlamydia psittaci (formerly *Chlamydia psittaci, and Chlamydophila psittaci*)

Signs & Symptoms:

➢ *Adult birds*: Progressive emaciation, greenish diarrhoea, occasional conjunctivitis and high levels of urates in droppings.

- *Young Birds*: Rough plumage, low body temperature, tremor, lethargy, conjunctivitis, dyspnoea, rales, coryza, sinusitis, emaciation, dehydration, yellowish to greenish dropping or greyish watery droppings.
- Death usually follows within 8 to 14 days.
- Spontaneous recovery is rare.
- Zoonotic potential.

Laboratory Findings:

- ELISA test for antibodies (Immunocomb) is very sensitive and may have false positive results; cloacal/choanal and conjunctival swab be should used for testing
- PCR
- Immunofluorescent testing with commercially available conjugates are very useful.
- Culture of the organisms and cell culture medium.
- Antibody detection via serology. A positive serology test indicates exposure to chlamydia. To confirm active infection you require a rising or persistently high titre and this must be related to the bird's clinical signs, antigen detection and haematology changes (moderate anaemia, leucocytosis, absolute or relative heterophilia, and monocytosis) as well as changes to blood chemistry (elevated CPK, AST, LDH, TPP and bile acids if liver is involved or elevated uric acid if kidney is involved).
- Direct complement fixation to detect IgG – best used with paired serum samples.

Prevention

- Regular cleaning and disinfecting program.
- Suggested disinfectants: Benzalkonium chloride, hydrogen peroxide, 1% formalin.
- Prophylactic treatment using member of tetracycline family of drugs

Treatment

- Tetracyclines: Doxycycline is preferred. Some still use Chlortetracycline or Oxytetracycline
- Some reported success with Azithromycin or Enrofloxacin.

BACTERIAL ENTERITIS (COMMONLY SALMONELLA AND E. COLI)

Aetiology

- Gram negative bacteria most often coliforms, *Salmonella* and *E.coli*.

Signs & Symptoms

- Ruffled feathers - diarrhoea - listlessness - weakness - shivering - vent picking
- The severity of the illness depends on the age of the bird, the virulence of the bacteria, stress and the degree of contamination.
- Affected birds may be carriers showing no disease symptoms. These carriers may spread the disease to their offspring and may become acutely ill as a result of stress.
- Newly hatched and juvenile birds, with their less developed immune systems, are usually acutely ill and frequently die.
- Chronic infections in adult birds may form abscesses, fail to hatch eggs, have changes in eating habits and may intermittently pass contaminating bacteria.
- In *Salmonella*, droppings are coloured a Sulphur yellowish green which is very much a diagnostic sign for this microorganism.

Laboratory Findings

- Live bird: Several specimens of faecal material or blood collected over several days are cultured in order to identify the bacteria involved.
- Dead bird: intestinal contents, liver, blood and spleen are also cultured. Sensitivity testing should be performed since enteric bacteria are often resistant to several antibiotics.

Prevention
- Keep water and feed dishes free of faecal material.
- Identify and cull carrier birds.
- Control contact of the aviary or bird with outside birds.
- Careful disposal of contaminated materials.
- Minimise Stress.
- People working with contaminated material should practice good hygiene.

Treatment
- Start treatment at once based on clinical signs.
- Injections and oral fluid replacement is often necessary. Lactated Ringer's solution is best.
- Broad-spectrum antibiotics should be started when the culture is taken. Oral and injectable antibiotics should be given simultaneously in severe cases.
- The sulfa drugs are good to use orally. Kanamycin and Gentamicin are usually effective by injection. When Gentamicin is used do not allow dehydration or renal toxicity may occur.
- If necessary, the antibiotics can be changed after sensitivity test results are known. In many cases the antibiotic needs to be administered for 3-8 weeks.
- If diarrhoea is severe Kaopectate or Pepto-Bismol may be given orally with 2 to 3 drops in mouth three times per day.
- Water consumption should be monitored to prevent dehydration.
- Maintain a stress free environment.
- An incubator or a heating pad under the cage should be used to maintain the temperature between 30 $^{\circ}$-32° C. If the heating pad alone won't maintain the temperature, place the cage in a box and the box on a heating pad with a thermometer in the back of the box in order to monitor temperature.

COCCIDIOSIS

Aetiology
- An enteric infection by one of the host-specific coccidia.
- Genera: *Eimeria, Isospora, Caryospora*, *Tyzzeria*,*Wenyonella.*
- Not all species are pathogenic, so birds may be asymptomatic carriers.

Signs & Symptoms
- Diarrhoea with or without blood and mucous.
- Weight loss
- Anaemia
- Dehydration
- Depression
- Ataxia
- Incoordination
- Sudden death

- Soiled vent and swollen abdomen
- Faeces ranges from bloody and greenish to watery and brown.

Laboratory Findings
- Impression smears of intestinal contents at Post-mortem examination or faecal flotation will reveal oocysts.
- Diagnosis may be more difficult in Budgerigars as they often are ill before they reach the oocyst producing stage.

Prevention
- The coccidial life cycle is direct as the birds ingests the sporulated oocysts from the environment. Cleanliness of the environment, food and water is most important.
- Stress caused by factors such as overcrowding also contributes to response to infection.
- Do not mix young birds with adults
- Use strategies to reduce exposure such as using concrete floors, or suspended wire floors, regularly cleaning the environment, All water and food containers should be kept off the ground.

Treatment & Medication
- Advanced cases are difficult to treat and often result in death.
- Coccidiocides:
  - Baycox (Toltrazuril) 25mg/ml. Use 3ml per litre drinking water for 3 days.
  - Amprolium + Ethopabate

<u>MACRORHABDOSIS (MEGABACTERIA ASSOCIATED DISEASE)</u>

Aetiology
- A yeast infection of the outflow of the Proventriculus by *Macrorhabdus ornithogaster*, formerly known as Megabacteria.
- Can affect most bird species but is common in parrots, canaries, ostriches and poultry. Not all species are pathogenic, so birds may be asymptomatic carriers.

Signs & Symptoms
- SBL (Sick Bird Look)
- Chronic wasting disease
- Neck-stretching and vomiting (usually mucous covered seed, but may also contain blood)
- Droppings range from slightly loose to diarrhoea (soft, watery, bulky), occasionally dark and tarry.
- Polyphagia - Birds appear to have a ravenous appetite but close observation reveals that they are not swallowing the seed.
- Acute form: die within a few days
- Chronic form: become progressively emaciated and debilitated over weeks to months and may die or appear to recover then died when they relapse weeks to months later

Laboratory Findings

- A rod-shaped to filamentous organism, Gram positive and Periodic Acid-Schiff (PAS) positive.
- Commonly located at the junction of proventriculus and ventriculus at post-mortem examination.

- Gram stain or wet mount
  - On crop aspirate or vomitus on crop aspirate or vomitus as is the easiest screening test to perform
  - Impression smears of proventricular outflow at Post-mortem examination
  - faeces (f not apparent on first sample, repeat on pooled faecal samples collected over 5 days)

Prevention

- Quarantine and examine droppings of all new arrivals
- Avoid mixing Budgerigars, European Goldfinches (*Carduelis carduelis*) and European Greenfinches (*Carduelis chloris)* with other birds, as these species have been implicated in causing outbreaks in aviaries with mixed species
- Reduce stress
- Remove eggs from infected parents and foster under non-infected birds is successful in preventing transmission

Treatment & Medication

- Resistance and relapses are common with all treatments
- It does not appear to be transmitted in eggs. Using artificial incubation and rearing chicks in isolation can break the transmission cycle.
- Amphotericin B orally for 30 days 100mg/kg PO q12h.
- Sodium benzoate.
  - Has been used with varied results.
  - It has been toxic in finches and canaries
  - This is a food disinfectant, not an antibiotic. Dissolve 0.5 teaspoon of powder in 1 litre water. Make fresh daily. Use as only source of water. If birds will not drink reduce to 0.25 teaspoon in 1 litre water and once drinking increase to 0.5 teaspoon per litre. Recheck droppings for Macrorhabdus after 14 days. If still organisms present gradually increase to 1 teaspoon per litre. Check droppings after 14 & 30 days

HELMINTH PARASITES OF ALIMENTARY SYSTEM

1. ASCARIDS

Aetiology

- *Ascaridia* species. Commonly called Roundworms
- Common in psittacines (especially Australian parrots), poultry and wild birds
- Usually found in small Intestine, particularly in the duodenum. Can cause intestinal blockage or rupture in parrots, particularly budgerigars, Princess parrots (and other members of the *Polytelis* genus), grass parrots (*Neophema* and *Psephotus*), Rosellas and Major Mitchell's cockatoos.
- relatively large (3-5cm long) and easily located at post-mortem examination.

Signs & Symptoms

- SBL (Sick Bird Look)
- Droppings range from slightly loose to diarrhoea (soft, watery, bulky), occasionally dark and tarry.
- Polyphagia – some birds have a ravenous appetite Acute form: die within a few days

Laboratory Findings

- Routine faecal flotation is usually diagnostic as they shed large numbers of typical ascarid eggs. Eggs may not be apparent if the ascarids are all juvenile and not yet egg producing.

Prevention

- Quarantine and examine droppings of all new arrivals
- Avoid mixing budgerigars, Princess parrots (and other members of the *Polytelis* genus), grass parrots (*Neophema* and *Psephotus*), Rosellas and Major Mitchell's cockatoos with other birds.

Treatment & Medication

- Fenbendazole (Panacur 25, 25mg/ml) 25-50mg/kg PO sid x 3-7 days, or 100mg/kg PO once.
- Moxidectin (Cydectin Oral Drench 1g/L) 400-1000 mcg/kg PO
- Levamisole (many brands) 15mg/kg PO once
- Oxfendazole (Oxfen LV, 45.3 g/L) 20mg/kg PO once or 2.5ml/L Drinking water
- Ivermectin (Ivomec 0.8g/L) 0.1cc/100g PO by gavage needle

2. CAPILLARIA

Aetiology

- Commonly called hairworm or wireworm.
- *Capillaria* species: *C. annulata, C. contorta* and *C. obsignata* are parasites of crop and oesophagus of pheasants, quail, ducks and turkeys. Also a problem in pigeons, African Lovebirds and South American Conures.
- Adult Capillaria cause damage by burrowing under the epithelium creating inflammation and haemorrhage. The eggs cause a local inflammatory response and are released with the sloughed mucosa.
- They are relatively small and easily overlooked at post-mortem examination.

Signs & Symptoms

- SBL (Sick Bird Look)
- Weight Loss and diarrhoea
- Pigeons have poor performance in races

Laboratory Findings

- Routine faecal flotation may not be diagnostic with normal salt solutions
- Eggs are intermittently shed in only small numbers. Typical bipolar eggs.
- Use repeated wet mounts to detect the eggs.

Prevention

- Quarantine and examine droppings of all new arrivals

Treatment & Medication

- Moxidectin (Cydectin Oral Drench 1g/L) 400-1000 mcg/kg PO
- Oxfendazole (Oxfen LV, 45.3 g/L) 20mg/kg PO once or 2.5ml/L Drinking water
- Levamisole (many brands) 15mg/kg PO for 3days

3. CESTODES

Aetiology

- *Choanotaenia* (finches), *Raillietina* (poultry) and other host-specific species
- Tapeworms are an uncommon problem and most infestations involve only small numbers of the parasite that do not harm the host.
- They are more common in finches, cockatoos and racing pigeons.
- The adult tapeworm lives in the lumen of the intestine and segments are shed with the faeces. These segments are usually ingested by a range of intermediate hosts - arthropods, molluscs or annelids.
- They are easily seen at post-mortem examination.

Signs & Symptoms
- SBL (Sick Bird Look)
- Weight Loss and depression
- Occasionally segments can be seen protruding from the bird's vent.

Laboratory Findings
- Routine faecal flotation are not diagnostic with normal salt solutions

Prevention
- Quarantine and treat all new arrivals
- Use insecticides such as pyrethrins to control possible intermediate hosts, by spraying the environment – especially small cracks that provide protection.

Treatment & Medication
- Praziquantel
- Niclosamide
- Fenbendazole

<u>CNEMIDICOPTES (SCALEY FACE OR SCALEY LEG MITE)</u>

Aetiology
- *Cnemidocoptes pilae* (budgerigar, canary and parrots) and *C. mutans* (poultry) – causes bird mange
- Can affect domestic chickens and other galliformes, Canary, budgerigar, pigeons, Kakariki, Neophemas, cockatoos
- Common name is different in some species:
  - Scaley face in Budgerigar
  - Scaley leg in chicken
  - Tassle foot in canary (elongated encrustations on legs and plantar aspect of feet)

Signs & Symptoms
- The mites burrow under the epithelium causing raised white to yellow crusting and honeycomb-like encrustations on cere, eyelids, beak and legs. Occasionally cause encrustations at elbows, vent and skin (commonly apterylae of wings).
- In budgerigar often causes beak deformity and anorexia
- May resemble flour on the skin of Sulphur-crested cockatoos.
- Some birds with the problem on feet may be lame and develop secondary bacterial or fungal infections

Laboratory Findings

- Crush preparation of encrustations in oil will reveal the mite

Prevention

- Quarantine and examine droppings of all new arrivals
- Avoid mixing Budgerigars, European Goldfinches (*Carduelis carduelis*) and European Greenfinches (*Carduelis chloris)* with other birds.
- Reduce stress
- Remove eggs from infected parents and foster under non-infected birds is successful in preventing transmission

Treatment & Medication

- Moxidectin 200 mcg/kg PO once weekly until controlled
- Ivermectin 400-1000 mcg/kg PO once weekly until controlled

<u>AIR SAC MITE</u>

Aetiology

- *Cytodites nudus* and other sarcoptiform mites.
- Can affect passerine birds (finches and canaries) as well as other birds – turkey, pheasant and domestic chicken.
- Most common in Canary, Gouldian Finches, European Goldfinches and Cordon Blue Finch.

Signs & Symptoms

- Respiratory signs are present only in severely affected birds
- Transilluminate trachea: (spray overlying feathers with methylated spirits and then shine a bright light source through the skin overlying the trachea) see tiny red dots moving within the tracheal lumen
- Respiratory "click" apparent with careful auscultation or if you stand still in an aviary on a quiet evening.

Laboratory Findings

- They are difficult to visualise on post-mortem examination (use magnification and examine trachea and air sacs carefully.
- Easily missed histologically.

Prevention

- Quarantine and treat all new arrivals

Treatment & Medication

- Ivermectin 400-1000 mcg/kg PO once weekly until controlled, (Ivomec for sheep, 0.8g/l) 1 drop into the beak once weekly until controlled
- Moxidectin 200 mcg/kg PO once weekly until controlled
- Pyrethrin spray into a localised area for the birds to inhale. Repeat weekly.

<u>ASPERGILLOSIS</u>

Aetiology

- *A. fumigatus*, *A. flavus* and *A. niger*

Signs & Symptoms

- Emaciation

- respiratory distress
- neuromuscular disease
- abnormal droppings
- regurgitation
- voice changes
- poor appetite
- nasal discharge
- gout
- conjunctivitis

Laboratory Findings
- Fungal culture and identification combined with presence of lesions.

Prevention
- Minimise stress and overcrowding.
    - Malnutrition from lack of proper diet.
    - Poor ventilation.
    - Disinfectant fumes, cigarette smoke, ammonia, and other toxic chemicals
    - Transmitted by spores, which are often present in the environment.
    - Healthy unstressed birds are generally resistant to fairly high concentrations of spores.
    - Young, old, unhealthy and those on antibiotics or corticosteroid therapy are frequently infected.
    - Reduce contact with nesting materials, etc which may be contaminated with mould or spores.
    - Feed should be free of fungal growth. May be found in corn and grain products as well as manufactured pellets or extruded food. Improper storage of these food products will greatly increase contamination.

Treatment & Medications
- Removal of stress factors.
- Combinations of Flucytosine, Fluconazole and Itraconazole are recommended.

<u>CANDIDIASIS</u>

Aetiology
- *Candida albicans*, a yeast.

Signs & Symptoms
- General unthriftiness, increased appetite, vomiting and weight loss, especially in babies.
- May affect crop and oral mucosa, intestines, eyes, lungs.
- Lesions are raised white patches that are easily removed with a cotton swab.
- The underlying mucosa is thickened and roughened and therefore cannot function properly.
- Rhinitis and beak necrosis may also occur if the yeast infects the otopharynx and spreads to the nares or the upper beak causing the lower beak to grow abnormally.
- Crop and oesophagus contains lesions.

Laboratory Findings
- Culture of the characteristic raised white colonies.

- Identification of yeast-like cells on microscopic examination of a gram stain.
- Abundant growth must be present to be diagnostically significant.
- It is often secondary to prolonged antibiotic therapy.

Prevention

- Careful removal of all food residue from the environment.
- Provide adequate Vitamin A in diet
- Care should be taken to ensure the cleanliness of everything used in handfeeding.
- The infection can be transmitted by secretions of the mouth therefore infected birds, both babies and adults should be isolated from healthy individuals.
- Many breeders use Nystatin suspension in a gavage as a preventive.
- After handfeeding babies should have mouths swabbed with clear water or water to which Vitamin K has been added. This can be followed by a 0.1cc dosage of Nystatin as preventive treatment. This suspension can also be given at the rate of 1 ml per litre of water for 5 days.

Treatment

- Nystatin is the most commonly used drug to treat candidiasis. It comes in suspension or a premix that is added to feed. The suspension seems to be the most effective.
- Other commonly used drugs include Ketoconazole, topical iodine and Nolvasan (Chlorhexidine).
- Selection of the drug of choice depends on the severity of the infection, physical condition of the birds and susceptibility of the particular infection. Yogurt added to handfeed formula in addition to the Nystatin treatment is effective with babies; serving water mixed with yogurt would be used with adults.

<u>TRICHOMONIASIS</u>

Aetiology

- A Protozoan with flagella known as *Trichomonas gallinae*

Signs & Symptoms

- Inflammation, white plaques on the gastrointestinal mucosa, necrosis with accumulation of cheesy material that may occlude the oesophagus and trachea.
- *Young birds*: Crop stasis, poor growth and high mortality. Often infected through parental feedings.
- *Adult birds*: Emaciation, dyspnoea and vomiting are common signs. Usually infected by contaminated food and water.
- A carrier state is possible.

Laboratory Findings

- Identification of trophozoite in warm saline, wet mount prepared from a swab of the oesophagus.
    - These preparations must be examined as soon as they are prepared or the organism may not be recognised.
        - Wright's stain or Trichrome staining is also possible.
        - Cysts do not occur in this organism.
- Diagnosis may be more difficult due to intermittent shedding of the organism. Repeated crop washes may help.

Prevention

- Cleanliness of the environment, food and water is most important.

- Overcrowding may also contribute to infection.

Treatment & Medication
- Advanced cases are difficult to treat and often result in death.
- Imidazole group of medications which can be used, are:
  - Ipronidazole
  - Metronidazole
  - Ronidazole
  - Dimetridazole
  - Carnidazole.

GIARDIA

Aetiology
- A protozoan with flagella. *Giardia psittaci* - commonly found in faeces of asymptomatic adults suggesting an asymptomatic carrier state.

Signs & Symptoms
- Loose, malodorous stools
- mucoid diarrhoea
- debilitation
- gram negative enteritis
- anorexia
- depression
- recurrent yeast infections
- eosinophilia and hypoproteinaemia
- dry skin and feather picking
- can cause poor growth and high mortality in neonates.

Laboratory Findings
- There are two stages in the life cycle of Giardia: the trophozoite and the cyst. Both can be identified in faecal smears stained with Carbol fuchsin or iodine or by flotation techniques with zinc sulfate. These preparations must be examined within ten minutes of collection or the organism may not be recognised.
- Trichrome staining may also be used.

Prevention
- Keep the aviary as clean and dry as possible. This will reduce the viability and number of cysts.
- Contaminated water supplies may be a source of infection. The cysts survive the standard chlorination of water.

Treatment & Medication
- Ipronidazole
- Dimetridazole
- Ronidazole
- Metronidazole

PACHECO'S DISEASE

Aetiology
- A member of herpes virus family

Signs & Symptoms

- Lethargy is often the only sign of sickness seen before sudden death.
- Some birds show yellowish, watery diarrhoea 10-12 days before death followed by inactivity, anorexia,
- lethargy and ruffled feathers. This phase may only last a few hours.
- Because of the acuteness of Pacheco's many bird owners come home to find their previously healthy bird dead.
- A Carrier state is also possible.

Laboratory Findings

- At necropsy the liver and/or spleen may be enlarged with multifocal or widespread necrosis.
- Eosinophilic intranuclear inclusion bodies are seen in hepatocytes and sometimes in the spleen.
- The inclusion bodies in the liver are distinctive and characteristic; their absence even with characteristic symptoms and lesions do not allow a definite diagnosis to be made.
- The virus can be isolated from frozen liver, kidney, spleen, intestinal or faecal sample by a qualified laboratory.

Prevention

- Isolation of any bird suspected of having or being exposed to Pacheco's.
- Regular cleaning and disinfection of the aviary.

Treatment & Medication

- Acyclovir has been shown to be effective for at least some strains of Pacheco's Disease virus.
- The treatment is by gavage or by powdered acyclovir added to the food and is most effective in reducing morality in flock outbreaks if started before clinical signs develop.
- This treatment may cause considerable nephrotoxicity and should be administered carefully in patients with nephropathies.
- An inactivated Pacheco's Virus vaccine is available in the United States. There have been frequent reports of granulomas and paralysis following the use of this vaccine and is intended for use only in high-risk patients.

NON-INFECTIOUS DISEASES

Behaviour Problems

- Feather Picking
- Screaming

Dietary Problems

- Obesity
- Egg-binding
- Metabolic Bone Disease

Toxicities

- Heavy Metal poisoning

OBESITY

Aetiology

- Overfeeding, particularly high fat foods such as sunflower seeds.

Signs & Symptoms

- Excessive weight
- Lipomas and collection of fat over body

Laboratory Findings

- Fine Needle Aspirate and cytology of mass reveals fat globules and no sign of neoplasia.

Prevention

- A good discussion of captive diets is provided in the supplemental reading: *Feeding Birds: Nutrition and Feeding Strategies; Nutritional Disorders*, by Dr Stacey Gelis in Birds 200, PGF Proceedings 2000..

Treatment & Medication

- Place on a diet
- There is a strong swing towards pelleted diets forming the majority of the diets being fed, particularly in pet birds, although it is now becoming more prevalent in aviculture as well. The current recommendation is 60-80% of the diet being pellets and 20-40% being vegetables and fruit with the small balance made up of other treats.
- For birds that will not eat pellets, provide a good quality **budgie seed mix** or a seed mix containing **Hulled oats and French White Millet or Japanese Millet**, to make up the basic part of the diet. seeds should be clean, dry, be free of rodent droppings and insects, and stored in airtight containers.
- If you wish you may give your pet parrot a maximum of 3-4 of the Sunflower or similar seeds, as a training treat once a day.
- Suggested Major Supplements (these are added as a main part of the diet to replace seeds)
  - Dark green, leafy vegetables; spinach or silver beet, endive, Chinese cabbage (bok choy), celery, parsley, thistle, dandelion or native grasses and their seeding heads. In most cases, lettuce is not recommended
  - Broccoli, capsicum, tomato, and sweet corn or corn-on-the-cob. Feed raw or parboiled.
  - Carrot, peas, beans, pumpkin and sweet potato. Feed raw or parboiled and can be grated.
  - Sprouted seeds: alfalfa, mung beans, cress etc, or normal budgie seed mix can be sprouted.
  - Small amounts of fruit: apple, peach, pear, plums, apricot, honeydew melon, rockmelon, mango, paw-paw, grapes, cherries, mandarin, orange, nectarines, figs, raisins, sultanas, currants, strawberries, raspberries, blueberries, kiwi fruit, loquat.
  - **Avoid cabbage, cauliflower, brussel sprouts, rhubarb leaves and avocado as they are toxic.**
- *Suggested Minor Supplements* (these are added as only a small part of the diet to add interest for the bird or during the breeding season)
  - Animal protein supplement (give each hen ½-1 tsp daily in the breeding season - July-January): Egg and biscuit mix, boiled or scrambled eggs, plain cake (eg. Madeira cake), small pieces of meat or a chop bone , fresh whole-grain or wholemeal bread.
  - Calcium source: shell grit, cuttlebone, products e.g. Calcivet®, Calcium Sandoz Syrup®.
  - Flowers and nuts (cones and seed pods) from native trees: *Grevillea*, *Acacia* (wattle), *Callistemon* (bottlebrush), *Banksia* cones, pine cones, *Casuarina* cones, *Hakea* seed pods.

- Branches from native trees (leave the leaves attached) can be used as perches and birds enjoy chewing the bark. Common ones are: *Hakea*, *Grevillea*, *Acacia* (wattle), *Callistemon* (bottlebrush), *Banksia*, *Eucalyptus* (gum trees), *Casuarina* (She-oaks), *Leptospermum* (Tea-tree) and Illawarra Flame Tree. Most non-native trees should be avoided, two exceptions that may be used are Pine trees and the Jacaranda.

FEATHER PICKING IN PARROTS

Feather plucking is common, and often a difficult problem to diagnose and treat. Typically, the bird has normal **head feathers** with feather loss or damage only in areas the bird can reach with its' beak. Anything from small patches, to the whole body may be affected. It occurs commonly in Sulphur Crested Cockatoos, Galahs and Cockatiels, but any species can be affected. There are many causes of feather plucking, so a thorough investigation may be needed to get an accurate diagnosis. Without an accurate diagnosis, effective treatment is very difficult. Unfortunately, there is no single *"magic cure-all"* for feather plucking birds. Listed below, are some of the causes of feather plucking and possible treatments.

Diseases Causing Feather Picking

1. **Allergies:** recent work has shown that some birds are allergic to certain seeds (oats, sunflower and canary seed), and house dust mites. Some birds respond to 'elimination diets', ie. the only seed permitted was millet seed, and the general diet was improved with the addition of fresh fruit and veg. Central heating has been associated with increased dust and allergies in people and birds, as well.

2. **Mites and Lice:** often blamed but rarely a problem. Generally easily diagnosed and treated.

3. **Intestinal Parasites:** worms and protozoal parasites frequently cause feather plucking, especially in Cockatiels and Budgies. Diagnosis and treatment is usually quick and effective.

4. **Low Humidity:** Tobacco smoke and central heating have been blamed for drying out feathers and causing excessive preening. Regular, light water sprays can be beneficial.

5. **Skin Infections:** infectious dermatitis may cause feather plucking or may be the result of it. Beak and Feather Disease often causes feather loss and feather plucking.

6. **Psittacosis:** although pneumonia and gastroenteritis are the most common symptoms associated with psittacosis, many birds pick their feathers as well. Antibiotics are usually curative.

7. **Malnutrition:** common in parrots on seed-only diets. These lead to vitamin, mineral and protein deficiencies which frequently cause feather plucking. Improving the diet will cure the problem.

8. **Cancer:** is common, especially in Budgies, Galahs and Sulphur Crested Cockatoos. The birds will pick at skin cancers or areas of skin overlying tumours. Severe, self-inflicted wounds can result. Many tumours seen in birds can be successfully operated on.

Psychological Causes of Feather Plucking

When all medical causes of feather plucking have been ruled out, psychological causes are considered.

1. **Boredom:** parrots are very intelligent and get bored easily. This is common cause of severe feather plucking. A change of scenery, people or other birds for company, interesting toys, chewable food (such as fruit and vegetables and non toxic wood) to chew, and TV or radio can all help to stimulate bored birds.

2. **Overcrowding:** leads to stress and aggression. This may cause birds to pluck themselves or other birds. Be very careful when placing a new bird into a cage with an existing bird.

3. **Environmental change:** although birds enjoy stimulation they also need routine. Sudden or constant changes can often lead to stress. Frequent cage movements or new toys may cause this.

4. **Poor wing clipping:** may cause a bird to chew at the feathers and over preen.

5. **Sexual frustration:** many single birds may pick their feathers in the breeding season.

However, *don't* introduce a 'mate' without sound advice as this may actually make the problem worse.

Treatment of Feather Picking

This can be a difficult problem to diagnose its cause and so arrive at a treatment regime. There are many causes of feather picking and you need a full work up to rule out causes other than behavioural. If medical causes are identified, treat them before embarking on behavioural treatments.

You have worked up your case and arrived at your diagnosis that a behavioural problem is the underlying cause of the feather picking. What do you do now? You need to discuss the options with your client and make your assessment of which treatment is likely to meet with some success. In all cases you need to be guarded in your prognosis - clients are often quite disappointed when their bird does not respond and they need to be prepared for all outcome possibilities.

Screaming

Screaming is reported as being responsible for more birds being abandoned than any other problem. There is often a fine line between normal vocalisation (calling and squawking) and the level when it should be regarded as a behavioural problem (screaming). This problem arises because suppression of normal vocalisation may cause the bird to become stressed. A normal bird does not squawk constantly[1]. Most birds will vocalise and "sing" at dawn and dusk with short bouts during the day. They rarely vocalise after dark. In many instances the birds are vocalising because they have learnt that it is good technique for gaining attention from their owners - feeding them; coming over to the cage or even negative attention such as: throwing something at them; yelling at them; squirting them with a water pistol.

- As soon as screaming occurs, cover the bird with a dark cloth, turn out the light or place it in a darkened room and close the door[1].
- When the screaming cease, open the door and uncover the bird. Speak to it very softly. Teach it that a soft voice attracts you, not a loud one.
- When you are in another room in the house, call to the bird in a gentle singing voice. Eventually, it will mimic you and call back to you in the same style of voice. If the voice is appropriate then you can answer it. If it is inappropriate - ignore it and keep talking in your singing voice.

EGG-BINDING

Aetiology

- Failure of the egg to pass through the oviduct at a normal rate. This may be physiological or obstructive. Dystocia is more advanced stage of egg-binding and is due to the egg being stuck in the distal oviduct.
- A common problem in finches, canaries, lovebirds, cockatiels and budgerigars.

Signs & Symptoms

- Sudden death
- Abdominal straining
- Often bird is on the floor of the cage
- Bilateral leg paralysis/paresis

- Penguin-like stance – especially budgerigar
- Swollen abdomen profile

Laboratory Findings

- Detect on physical examination, ultrasound or radiographs.

Prevention

- Feed balanced diet and provide access to sunlight
- Provide appropriate nesting box

Treatment & Medication

- Prophylactic antibiotics – Amoxycillin, Enrofloxacin
- Lubricate cloaca with creams such as Chlorhexidine or antibiotic eye ointments
- Calcium supplements
    - Calcium Borogluconate 50-100mg/kg IM.
- Glucose/electrolyte supplementation – 10% body weight spread over 24 hours
- Place in a humidicrib at 25-30°C and 70% humidity
- Simple cases should respond to heat, humidity and Calcium supplement within 1-2 hours. The smaller the bird the more critical they become in a short time. Observe the patient regularly and provide more treatment as required.
- Advanced cases may require aspiration of egg contents to collapse through to laparotomy.

<u>HEAVY METAL POISONING</u>

Aetiology

- Ingestion of excessive Zinc or Lead – usually from galvanized wire or dishes.

Signs & Symptoms - none are specific for heavy metal.

- Dyspnoea
- Sudden death
- Nervous disease signs
    - Seizures
    - Drooped wings
    - Lethargy & ataxia
    - Star gazing
    - Head pressing
    - Unable to fly
    - Paralysis
    - blindness
- GIT disease signs
    - Bright green droppings (biliverdinuria)
    - Diarrhoea
    - Regurgitation
    - Crop stasis
- Urogenital disease signs
    - Haemoglobinuria
    - Haemorrhage

Laboratory Findings

- Collect heparinised blood.

- Excessive blood levels of Zinc (> 33 ppm suspicious, >75 ppm diagnostic) Remember Zinc is an essential nutrient.
- Lead >20mcg/dl (0.2ppm) suspicious, >50 mcg/dl (0.5ppm) diagnostic.

- Non-regenerative anaemia
- Elevated LDH, AST, CK and UA

Prevention

- Remove sources of Zinc and Lead from the environment.

Treatment & Medication

- Stabilise the patient with heat, humidity and fluid therapy
- Anticonvulsants if indicated (Diazepam)
- Cathartics – peanut butter or Metamucil PO.
- Chelation:
  - CaEDTA 35mg/kg IV, IM – sid to bid (5 days son, 5 days off then repeat)
  - BAL (Dimercaprol) 2.5 mg/kg IM q4h x 2 days then bid for 10 days. May be used with CaEDTA

Clinical Examination

Name: ____________________ Street: ____________________

Phone: ____________________ Suburb: ______________ Postcode:

Date: __________ Species: ________________ Age: ____ Sex: M F

Presenting Complaint: ______________________________________

Pet/Aviary*; New/Old*; Behaviour Changes: ______________________

Medications used: ____

Diet: ________________________________ Weight: __________

History: ____

____

____

	N	A	NE		N	A	NE
(1) Position in cage				(6) Legs			
(2) Respiration				(7) Keel			
(3) Head/Eyes				(8) Abdomen			
(4) Feathers/Beak				(9) Vent			
(5) Wings				(10) Auscultation			

Describe abnormal findings noted above:

____

		Fee	Credit	Balance
Faeces Gross:				
Faecal Float:				
Crop Wash:				
Other Tests:				
Diagnosis:	Definite Tentative *			

Treatment:

____

____

* = circle one N = Normal A = Abnormal NE = Not Examined

Exploring the Gap between Student-reported Confidence and Expert-rated Competence for Veterinary Anaesthesia Skills

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Introduction/Background:
Self-evaluation allows students to reflect on how they believe they performed rather than relying solely on performance feedback from supervisors. A mismatch between self and expert evaluation has been described in the medical literature but reports in veterinary literature are sparse. Overconfidence and underconfidence both have the potential to negatively influence veterinary professional practice and patient care. This project sought to establish the presence of the mismatch between self- and expert-perceptions and explore any underlying factors that contribute to the gap. Understanding the underlying causes of the mismatch is important to help inform curriculum change in order to prevent its development.

Methods:
A mixed-methods approach drawing from both positivism and interpretivism was utilised. Of 125 eligible students, 105 and 109, respectively, completed the quantitative and qualitative aspects. Quantitative data were collected regarding student attributes including age, gender, citizenship, and final rotation mark. Final year veterinary students completed Likert-scale ratings of self-confidence in 18 anaesthesia skills at the start and conclusion of their anaesthesia rotation. Veterinary anaesthesia nurses and a resident (experts) completed Likert-scale ratings of competence for each skill for each student at the rotation completion. Qualitative data were collected via anonymous open-ended responses to the question: "In your own mind, how can you tell whether you are confident or not?". Quantitative data were analysed using descriptive statistics, correlations, and linear regressions. Qualitative data were coded and thematically analysed.

Results/Findings:
Student confidence ratings at the end of the rotation were positively correlated with confidence ratings at the start of the rotation (r values ranging from 0.31-0.733). Confidence ratings at the end of the rotation were not correlated with either the expert ratings of competence or the final rotation marks, with overconfidence demonstrated more frequently than underconfidence. There was no influence from age, gender, or citizenship. Data saturation was achieved with qualitative phase of the project and five themes regarding how students gauged their own confidence emerged:
1. The use of emotion as a proxy for confidence
2. Using the automaticity of an action or the anticipatory reaction one experiences when faced with the need to act
3. Using outcomes or external cues to retrospectively determine confidence
4. Appearing confident when you aren't
5. The belief that independence equates to confidence

Discussion:
This project demonstrated the gap between student self-confidence and expert-evaluated competence in veterinary anaesthesia students. Students' overconfidence does not seem to be impacted by physical and social attributes, especially gender, marking a notable difference from the human medical education literature. Instead the mismatch may be explained by more cognitive attributes such as students choosing inappropriate personal gauges of confidence, most notably the belief that one must be completely independent to practice as a confident professional. Educators must promote the notion that excellent professional practice requires interdependence between internal and external cues to self-regulate.

Common dental problems in paediatric patients

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General practitioners should feel comfortable in recognising normal oral structures and dentition in companion animals, and the developmental changes that occur in the first year of life. Prompt identification of pathology and early treatment minimises the likelihood of significant disease or discomfort, and may prevent life long problems.

Common juvenile dental conditions include malocclusions, failure to erupt teeth, crown fractures, and persistent deciduous teeth. The approach to treatment is similar to adult dentistry but greater care and attention to detail is required. Radiography is critical to assessment and successful treatment. Juvenile dentistry is a part of good general practice.

Normal anatomy and eruption sequence of puppies and kittens

The juvenile individual should have an occlusion similar to the adult. That is the midpoints of the mandibular and maxillary dental arches should be in alignment with the mid-sagittal plane of the head. The maxillary incisors should occlude rostral to their mandibular counterparts and the mandibular canine teeth should occlude buccal to the maxillary gingiva equidistant between the maxillary canine tooth and third incisor. Pre-molar teeth should interdigitate with their opposing teeth and be positioned such that the maxillary teeth are buccal to the mandibular teeth.

Note that there are no precursor teeth for the first pre-molar molar teeth in the dog, or for any molar teeth in the dog or cat. Some breeds, especially brachycephalic breeds, may have an occlusion that varies slightly from the standard normal occlusion.

The dental formulae for the dog are:

deciduous – 2 $\left\{ i\frac{3}{3}\ \ c\frac{1}{1}\ \ p\frac{3}{3} \right\} = \ 28$

permanent – 2 $\left\{ I\frac{3}{3}\ \ C\frac{1}{1}\ \ P\frac{4}{4}\ \ M\frac{2}{3} \right\} = 42$

The dental formulae for the cat are:

deciduous – 2 $\left\{ i\frac{3}{3}\ c\frac{1}{1}\ p\frac{3}{2} \right\} = 26$

permament – 2 $\left\{ I\frac{3}{3}\ C\frac{1}{1}\ \ P\frac{3}{2}\ \ M\frac{1}{1} \right\} = 30$

The deciduous teeth erupt within the first 6-12 weeks of life and are exfoliated in a progressive manner to be replaced by the permanent dentition. A full juvenile dentition should be apparent in puppies from 4-12 weeks of age, and in kittens by 6 weeks of age. This coincides with the timing of the initial vaccine courses for these young animals and provides an ideal opportunity for assessment and client education, particularly with respect to safe chewing habits and behaviours.

The permanent teeth begin to erupt in a similar sequence from the sagittal midline of each arcade extending caudally i.e. the incisors are exfoliated first followed by the canine teeth, premolars and molars. Some variation does occur between individuals in their sequence and timing of eruption. A full adult dentition should be apparent clinically by 6 months of age in cats and most dog breeds. General practitioners commonly desex young animals at the age of 5-7 months and at this time the animal should be examined for any eruption pathologies. The most common eruption pathology noted would be persistent deciduous teeth.

Radiographic appearance of the immature dentition

The radiographic appearance of the deciduous teeth is similar to the permanent dentition with the obvious point of difference being the presence of the un-erupted tooth structures within the maxillae and mandibles. It is possible to assess in young pups 8-12 weeks of age if there will be permanent teeth in the series by taking intra-oral radiographs. These can be difficult to assess due to the number of structures present.

Teeth are dynamic, vital structures that continue to lay new layers of secondary dentine within the pulp cavity. As the tooth matures the pulp cavity therefore narrows. An animal with a mixed dentition will have teeth with various sized crowns and pulp cavities, the larger the crown and pulp chamber the younger the tooth (permanent dentition). Partially erupted and immature erupted teeth will not have a complete root. Apexification is not complete till sometimes many months following occlusion especially in large breed dogs.

The alveolar bone increases in density with age and the radicular cortical plate (lamina dura) becomes less distinct.

Common paedodontic conditions

- “missing” teeth

The first thing to assess when an animal is presented for an apparently missing tooth is to determine whether the tooth is truly absent for the animal’s developmental age. There are no deciduous pre-cursors to the first maxillary and mandibular pre-molars in the dog, and none for any of the molars in the dog or cat.

Radiography is essential to determine if the tooth is completely absent and not just un-erupted. In some animals a persistent, thick gingival covering may be partially or completely covering the crown of the erupting tooth. This is termed an operculum and resection of the tissue (operculectomy) will allow eruption to continue.

A fully un-erupted tooth may be impacted or embedded, and requires surgical removal. Failure to remove the tooth may result in a dentigerous cyst developing. Arising from the neck of the developing tooth, these cysts can become quite large, space occupying lesions within the jaw. Dentigerous cysts can be associated with pathological fractures.

Complete absence of all teeth (anodontia) is rare however the absence of one or several teeth (oligondontia), especially in the pre-molar series, is quite common. Oligondontia may be unilateral or bilateral. If bilateral it is most likely to be considered hereditary in origin. Unilateral oligodontia is usually thought to result from a failure of the tooth bud to develop from localised trauma or inflammation.

- trauma

Deciduous teeth are smaller in size than their permanent counterparts and are more prone to traumatic injury. Dog bite attacks and blunt trauma to the head are common insults that may result in fractured teeth and/or jaws. All head trauma cases should have intra-oral radiographs taken for complete assessment.

A fractured deciduous tooth should not be left in situ as it may not be exfoliated at the appropriate time resulting in malocclusion as the permanent teeth are erupted, or the associated peri-apical inflammation may damage the underlying developing permanent tooth. When extracting deciduous teeth, patience and gentle tissue handling is imperative in order to protect the nearby developing dental structures. Consider using antibiotic therapy as appropriate.

Immature permanent teeth have a relatively large pulp chamber and minimal dentine. These teeth are also more prone to trauma as they are not as solid. Bleeding is common when an immature tooth is fractured. Any tooth with an exposed pulp needs endodontic therapy or extraction. A vital pulpotomy may be appropriate especially if apexification has not occurred, to allow the tooth to continue to develop.

Avoid any intraosseous fixation techniques for fracture repair in immature animals as it is almost impossible to avoid damaging the developing dental structures and not penetrate deciduous teeth roots. Soft tissue apposition of damaged tissues and simple intraoral composite splints can be used.

- persistent deciduous teeth

The simple rule of dental succession is that there should not be two teeth present at the same time in the same space. Most of the permanent teeth erupt lingually to the deciduous precursors except the maxillary canine teeth and fourth pre-molars. The permanent upper fourth pre-molar tooth erupts buccally and slightly distal to the last deciduous tooth in the maxilla. The maxillary canine teeth erupt rostral to the deciduous teeth.

Persistent deciduous canine teeth are the most common pathology. Failure to extract the deciduous maxillary canine teeth will result in narrowing of the diastema between the upper third incisor and canine tooth. This results in the mandibular canine tooth making contact with the maxillary teeth.

If the mandibular canine teeth have failed to exfoliate at the appropriate time then a base narrow malocclusion with concurrent palatal trauma will occur as the permanent teeth erupt.

Careful extraction technique with pre-and post-extraction radiographs is required when any persistent tooth is identified. It can be helpful to raise a small flap and perform alveoloplasty if the roots are fractured. Patience is essential. Do not use extraction forceps other than to grasp the very loose tooth.

- eruption gingivitis

Some individual animals will have a more exaggerated inflammatory response during the eruption phase of the permanent dentition. It is usually transient and resolves with consistent homecare e.g. chlorhexidine oral rinses, tooth brushing.

Occasionally a young animal will present with a more aggressive form of juvenile periodontitis and these animals may require professional scaling and polishing of the teeth on a regular basis as well as significant homecare in order to maintain the permanent dentition.

- <u>malocclusion</u>

Young dogs are commonly presented with dental malocclusion, it is rarely seen in cats. They are classified based on whether the jaws are of proper length, and the relationship and positioning of the teeth in those jaws.

Common malocclusions include anterior and posterior cross bites, crowded or rotated teeth, base narrow canines, lance canine teeth, level bites, mandibular brachygnathism and mandibular prognathism. Some malocclusions not causing pain or loss of function do not require treatment.

Interceptive orthodontics for jaw length disparities resulting in loss of the canine teeth interlock is possible in puppies to relieve pain and allow maximal jaw growth if impeded. Clients need to be counselled that the permanent dentition maybe similarly affected, especially in significant malocclusions.

As the permanent dentition arises, selective extractions of non-essential teeth will reduce the incidence of periodontal disease associated with overcrowding. Selective extraction of any affected permanent teeth may also be the treatment of choice for some clients who do not wish to undergo orthodontic work in those malocclusions where there is the possibility to reposition the tooth.

- <u>discoloured teeth</u>

Hereditary failure to produce adequate enamel has been reported but is uncommon, and results in teeth that are easily chipped and worn away. Acquired changes to the enamel are more common such as inflammation or infection during the critical time in the tooth's development, which leads to enamel hypoplasia (more accurately termed enamel hypocalcification). The enamel appears irregularly pitted and discoloured yellow brown in colour.

The presence of certain compounds during development may also affect the colour of the tooth e.g. excess fluoride intake, tetracycline use. Endodontic disease due to trauma with or without a fracture can lead to a pink to bluish-grey tinge to the crown. Treatment varies due to the cause of the discolouration of the tooth.

- <u>abnormally shaped teeth</u>

Common dental anomalies include peg teeth (cone shaped crown) and fusion where two separate tooth buds have joined. Abnormally shaped crowns are not a clinical problem unless they cause overcrowding or allow food material to collect, leading to periodontal disease. Supernumerary roots can occur, along with abnormally shaped or fused roots. These are also incidental findings and only pose a clinical problem when attempting to extract the affected teeth or perform endodontic therapy.

- <u>supernumerary teeth</u>

It is possible that an immature animal is presented because additional teeth are present in the oral cavity. Frequently these are actually not supernumerary teeth but persistent deciduous teeth. Radiographs are essential to differentiate. If the tooth radiographically has a narrower pulp chamber and subsequently more dentine, relative to the other teeth in the series, then this is the older tooth and is deciduous in nature. Small breed dogs especially the brachycephalic breeds are difficult to visually discern the deciduous dentition from the permanent. Supernumerary teeth are not a clinical problem unless they are causing overcrowding and as such should be extracted.

Conclusion

Oral pathology is relatively common in general practice. Prompt identification of pathology and early treatment in young animals minimises the likelihood of significant disease or discomfort and may prevent life-long problems The approach to treatment is similar to adult dentistry but greater care and attention to detail is required. Radiography is critical to assessment and successful treatment. Juvenile dentistry is a part of good general practice.

Minimising microbial growth during liquid storage of stallion spermatozoa

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Experiment 1

A synthetic medium, UoN media,[1] has been developed which allows stallion spermatozoa to be stored at room temperature (RT) for up to 7 days. However, prevention of microbial growth in this medium has been difficult. In the porcine industry, nanoparticle coated storage bags Bactibags™ have been developed to assist in the elimination of bacteria during cold storage for 5-7 days. Bacteria within the solution are attracted to and neutralised by the nanoparticles. The aim of these experiments was to identify whether Bactibags™ could effectively prevent bacterial growth during the liquid storage of stallion spermatozoa at RT. UoN media (patent pending) was initially supplemented with 0.25 mg/mL gentamicin, 50 U/mL penicillin and 50 µg/mL streptomycin.

Three ejaculates from each of four stallions (n=12) were extended 1:2 with EquiPlus™ and centrifuged at 350 x g for 15 min. Sperm pellets were resuspended to 50 x 10^6/mL and divided into three different treatments:

- Treatment 1: Equiplus™ in semen safe syringes at 4 °C (commercial control),
- Treatment 2: UoN media in semen safe syringes at RT
- Treatment 3: UoN media in Bactibags™ for at RT.

Total motility was different ($P \leq 0.05$) between all groups at days 0 and 3 of storage (Table 1) demonstrating the ability of the UoN media to maintain adequate motility at room temperature compared to the commercial control. Interestingly there appeared to be an interaction between the UoN media and the commercially available semen safe syringes. The reason for this is unclear but was perhaps due to an electrostatic charge or the anaerobic environment created by sedimentation.

Treatment	Day 0	Day 3
1	56.3±6.4%,	29.0±5.6%
2	38.3±6.0%	7.5±3.4%
3	80.3±5.1%,	57.5±6.5%

Table 1: Total motility (CASA) at day 0 and day 3 storage

All Treatment 1 replicates demonstrated bacterial growth at 10 days and motility was reduced to zero at 7 and 10 days storage. Bacterial growth was not completely eliminated by storage in Bactibags™, and there was a significant yeast/fungal growth in both of the UoN room temperature groups. The motility of spermatozoa stored in the Bactibags™ was disappointing when compared to other preliminary studies using the UoN media.

Experiment 2

Equipure™ is a commercial density gradient that has been shown to improve the percentage of morphologically normal spermatozoa in an ejaculate in addition to removing seminal plasma, bacteria and some viruses. In the second experiment, Equipure™ centrifugation was employed in an attempt to reduce the overall bacterial component from the start of the storage period to improve antimicrobial efficiency. Additionally, INRA 96™ (containing Amphotericin B) was compared to Equiplus™ to determine whether the extender used for the initial dilution reduced yeast/fungal overload.

Ejaculates (n=12) were divided into four aliquots and diluted 1:2 with either INRA 96™ or Equiplus™ for the following treatments:

- Treatment 1: Initial dilution in INRA 96™ followed by centrifugation at 350 x g for 15 min
- Treatment 2: Initial dilution in INRA 96™ layered over an EquiPure™ gradient and centrifuged at 400 x g for 20 min
- Treatment 3: Initial dilution in Equiplus™ followed by centrifugation at 350 x g for 15 min
- Treatment 4: Initial dilution in Equiplus™ layered over an EquiPure™ gradient and centrifuged at 400 x g for 20 min

Sperm pellets were re-suspended in UoN media supplemented with 100 U/mL Nystatin to a sperm concentration of 50 x 10^6/mL and stored at RT. Sterile pots (10mL) were used for storage in this experiment. Motility (CASA) was assessed and aliquots cultured on blood agar at 3, 7 and 10 days. EquiPure™ centrifugation eliminated bacterial growth at day 7 (0% vs 60% for pooled T2/T4 and T1/T3 respectively; Chi-square, $P \leq 0.05$). Nystatin effectively prevented fungal growth in all samples at day 7 and all but one of each of T3 and T4 (Equiplus™) at day 10. Total motility was improved by EquiPure™ treatment.

Conclusions

Initial experiments showed promising results for the Bactibag™ nanotechnology. However, spermatozoa that were subjected to Equipure™ centrifugation demonstrated superior motility following 7 days incubation at room temperature. The addition of Nystatin to the UoN media appeared to reduce Bacterial and Yeast/Fungal contamination whilst maintaining adequate motility following 7 and 10 days following Equipure™ centrifugation.

The future use of this medium will greatly simplify sperm transport logistics and artificial insemination regimens for Equine Veterinarians.

References

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Change Management Workshop – prepare, manage & reinforce: A practical approach to implementing effective practice changes with your team

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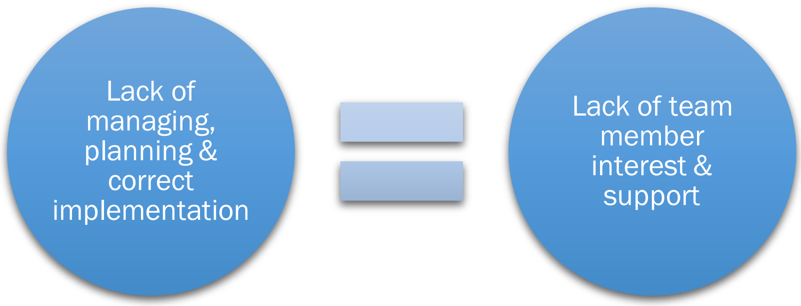
Organisations that want to thrive and survive are innovative. Therefore, effective change management is an absolute must-have skill that is required across all organisations. Engaging the commitment of your team members' to change is the biggest challenge facing every business. The aim of this session is for you to reach an understanding of the following key elements involved within managing change in your workplace:

- Understand the change management process
- Understand people's reactions to change
- Develop plans to integrate change effectively



"Change has a considerable psychological impact on the human mind. To the fearful, it is threatening because it means that things may get worse. To the hopeful, it is encouraging because things may get better. To the confident, it is inspiring because the challenge exists to make things better."
King Whitney Jr.

How good are your change management skills?

Complete the following quiz to assist you in targeting the techniques that will help you the most.

Question		Never	Rarely	Sometimes	Often	Always
1.	When I create a plan for change for those within my clinic, I involve all team members					
2.	All change, no matter what size, has implications for my team members.					
3.	I receive positive support for changes that I want to implement.					
4.	My leadership style directly affects the degree of success in managing change.					
5.	I communicate achievements throughout the clinic so that everyone understands the positive impact of a change project.					
6.	I effectively use mechanisms to identify and track all of the significant change efforts in our clinic					
7.	Misunderstanding and miscommunication are sources of resistance to change.					
8.	Good change management may require me to forecast and plan for change.					
9.	Knowledge of culture is important for managing change.					
10.	During the implementation phase, I am lenient and do not expect my team members to continue to perform at 100%.					
11.	I effectively use a process to ensure that I have the right change efforts to deliver our business strategy.					

12.	To ensure I receive support from my team members, I like to take the time to talk with them about the grounds for any required change.					
13.	I let people get comfortable with changes before I decide if any training is necessary.					
14.	It's harder to manage change effectively when the clinic has previously managed change projects badly.					
15.	When implementing a change project, I set achievable, short-term targets that, once accomplished, will motivate people to persist and keep trying.					
	Subtotal					
	Total					

Points System:

Never: 1 point Rarely: 2 points Sometimes: 3 points Often: 4 points Always: 5 points

Points Outcome:

Score	Comment
46-75	Great! A score within this range indicates you have a strong grasp on the understanding of what makes organisational change successful. It also indicates you have a good understanding of managing, planning, and implementation. We encourage you to review the lesson below and see if there is anything you may wish to "fine-tune" or have better comprehension.
31-45	Doing ok. From this result, we can see you likely have an understanding of the elements of managing, planning, and implementation but actually putting them into practice may not be as consistent. Review the lesson below to see which strategies you can apply.
15-30	Oh dear! This score range indicates you tend to look at the end result but neglect to focus correctly on managing and planning. Good news though….this result shows you have a great opportunity for improvement. Use the informtion below to assist in improving your change management skills.

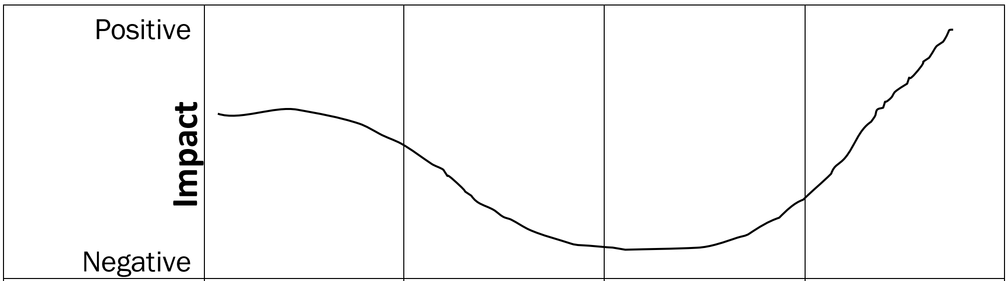
Understanding Change & its Processes

“Change management is the process, tools and techniques to manage the people-side of business change to achieve the required business outcome, and to realise that business change effectively within the social infrastructure of the workplace.”[i]

The main focus is on the bigger impact of change, i.e. the people – your team members as well as those surrounding your department/within your organisation. How they, as individuals and as a team move from the current situation to the new situation. It could be something as minor as a small change in a policy and procedure or something that can have a major impact such as change of leadership or company takeover or acquisition.

The Change Curve

The change curve is a common model that is used to describe the emotions/stages of personal transition from old to new. It helps you to understand how your team may respond to change, which in turn should help you to effectively manage and lead your team and provide the support they need. [ii]



Stage	1	2	3	4
State	Status quo	Disruption	Exploration	Rebuilding
Reaction	Shock, denial	Anger, fear	Acceptance	Commitment

Applying the Change Curve

Naturally, your aim with any organisational change is to minimise the negative impact on your team/organisation. In the application of the change curve model, you want to make the curve shallower and narrower. Use your knowledge of the Change Curve to give individuals the information and support they need. This will increase the likelihood of successful change.

Stage 1 – the change has been introduced, initial reaction may be shock or denial.

Stage 2 – the negative responses – anger, fear, resistance.
Stage 3 – the turning point: exploration.
Stage 4 – the best stage of all – acceptance and embracement. This is where you should start to reap some rewards of all the hard work you’ve put in to ensure a smooth transition. The wheels of productivity and efficiency should start to increase in motion.

Managing Resistance to Change

Managing Team Resistance

1. Expect it and don't be afraid of it
2. Involve interested groups/teams
3. Communicate
4. Provide training and/or coaching

Managing Individual Resistance

Miller and Rollnick, (1991) outline five key techniques for managing individual resistance.

1. Express Empathy
2. Develop the discrepancy between what the person is expressing and the vision for the change project
3. Avoid argumentation
4. Roll with the resistance
5. Support the individual

Implementing Change

"The wise man bridges the gap by laying out the path by means of which he can get from where he is to where he wants to go." John Pierpont Morgan

How do you lead change? How do you see it through to the end? How do you make it successful? World-renowned change expert John Kotter has written an 8 step process for leading change. Each stage acknowledges a key principle identified by Kotter relating to people's response and approach to change, in which people **see**, **feel**, and then **change.**

John Kotter's 8 step model:

- Step 1 • **Establishing a sense of urgency**
- Step 2 • **Creating the guiding coalition**
- Step 3 • **Developing a change vision**
- Step 4 • **Communicating the vision for buy-in**
- Step 5 • **Empowering broad-based action**
- Step 6 • **Generating short term wins**
- Step 7 • **Never letting up**
- Step 8 • **Incorporating changes into the culture**

References

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[1] NB: this model is often attributed to psychiatrist Elisabeth Kubler-Ross from her work on personal transition in grief and bereavement and as such you will likely see the similarities.

Dealing with diarrhoea in the adult horse

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Introduction

Diarrhoea is defined as the passage of faecal material with increased water content, and when acute in onset, it is a potentially life-threatening disorder that requires prompt intervention with appropriate treatment. This presentation will focus on acute diarrhoea in adult horses caused by inflammation of the caecum, colon, or both - generally referred to as 'colitis'.

Causes

There are several potential causes of colitis; these include infectious agents such as *Salmonella* spp., *Clostridium perfringens* and *difficile,* coronavirus, and parasites; the administration of drugs such as antimicrobials or nonsteroidal anti-inflammatories; ingestion of toxic or irritant material; or secondary to other systemic disease such as peritonitis. In a large percentage of cases a definitive diagnosis is not made. The approach to diagnosis hence needs to be systematic and thorough in order to maximise the likelihood of reaching a diagnosis. Ultimately, obtaining a definitive diagnosis is of most importance when dealing with potentially infectious agents, in order to minimise the risk of dissemination of the disease throughout a farm or hospital population.

Pathophysiology

Regardless of the initiating cause, common clinical and pathologic features suggest a common pathophysiological pathway. A combination of mechanisms, including mucosal inflammation and damage, malabsorption, increased secretion across the gastrointestinal wall, reduced transit time of ingesta or osmotic overload of the luminal contents, result in the passage of profuse watery diarrhoea. These mechanisms also lead to protein loss and severe electrolyte abnormalities.

Clinical signs

Clinical signs associated with diseases causing diarrhoea include dull mentation, inappetence, abdominal pain, hypermobile borborygmi, pyrexia, tachycardia, altered mucous membrane colour, prolonged capillary refill time, sweating, and watery, often fetid, diarrhoea. In horses with impending colitis, lethargy, inappetence and abdominal pain are frequently noticed several hours before the appearance of diarrhoea, and thus these cases may be confused with other large intestinal disorders, such as large colon torsion.

Clinical pathology

Clinicopathological findings can assist in assessing the severity of the condition and help target treatment. Typical haematologic findings include hypovolaemia, dehydration, metabolic acidaemia, electrolyte derangements, leukopaenia with a left shift, and toxic neutrophils. Serum biochemical analysis is often non-specific and usually demonstrates azotaemia. Total protein and albumin concentrations may initially be increased as a consequence of dehydration, however, once treatment is initiated to correct dehydration, a hypoproteinaemia often becomes evident. Severe diarrhoea will result in electrolyte losses, particularly sodium, potassium, calcium and bicarbonate. Serum lactate may be increased due to tissue hypoperfusion associated with hypovolaemia and shock. Traditionally, L-lactate has been viewed as a marker of hypoperfusion and is considered an accurate predictor of mortality in different species.

Further diagnostic tests

Faecal culture is a vital step in the identification of a bacterial aetiology. It should be remembered that the causative organism may not be continuously shed in the faeces and so it is advisable to collect multiple samples (ideally 5) over a period of at least three days. PCR tests are now available for the detection of *Salmonella* spp. DNA, *Clostridial* DNA encoding for the clostridial toxins and equine coronavirus. Abdominal ultrasonography can be useful in detecting and characterising the nature of peritoneal fluid, an abdominal mass and intestinal thickening. Normal large colon wall thickness is < 4mm. Rectal palpation can help rule out other large intestinal disorders.

Management

In the absence of a definitive diagnosis, management is frequently empirical, symptomatic and supportive in nature. The management of these cases can be intensive, often requiring hospitalisation.

Firstly, strict biosecurity is of utmost importance in these cases. Isolation is imperative, to reduce the risk of disease transmission in the case of an infectious aetiology. Several other preventative measures are also advised, including rigorous hand hygiene, barrier nursing precautions, and movement restrictions.

Secondly, the initial aims of therapy are to maintain fluid and electrolyte balance in the face of losses imposed by the diarrhoea. The goal of fluid therapy is to improve cardiovascular function, thereby resulting in improved tissue oxygen delivery and organ function. In severe cases, a fluid deficit will often need to be corrected first. An assessment of hydration status can be made using clinical and clinicopathological parameters, including haematocrit, serum total protein concentration, serum lactate concentration, serum creatinine concentration, and urine specific gravity, and should allow an approximation of the degree of dehydration present. This initial fluid deficit should be replaced within 4-12 hours. Replacement with a balanced polyionic crystalloid fluid such as Hartmann's solution is most suitable. In most cases, especially horses unable to eat due to anorexia, the low potassium content of replacement solutions requires the addition of supplemental potassium in the form of potassium chloride (10 – 20 mEq/L).

In acute, severe cases, hypertonic saline may be used to provide rapid restoration of circulating blood volume and haemodynamic stabilisation. It is essential that hypertonic saline is followed by an appropriate volume of isotonic solutions to maintain volume expansion. Once the deficit has been corrected, the fluid therapy plan should provide for basal metabolic requirements and replace ongoing losses from continued diarrhoea. In general, fluid therapy should be continued until the diarrhoea resolves or the patient is able to maintain their fluid balance with voluntary drinking. Frequent monitoring of PCV, serum electrolyte concentration, venous blood gases, BUN and creatinine, and body weight is important to monitor hydration, electrolyte and acid-base balance, and renal function.

The loss of intravascular plasma proteins and reduced oncotic pressure results in fluid accumulation in the interstitial tissues and it becomes difficult to maintain crystalloid fluids in the intravascular space. Administration of colloid solutions is helpful for volume expansion and maintenance of plasma oncotic pressures, which improve tissue perfusion and oxygenation and organ function. Natural colloids, such as plasma, are commonly used. Synthetic colloids such as dextrans, hydroxyethylstarch, or gelatins, are also available.

Thirdly, once fluid therapy has been initiated and the patient is stabilised, ancillary treatments may be beneficial to prevent sequelae, resulting from the systemic inflammatory response. Treatment strategies are aimed at a) reducing systemic and intestinal inflammation, b) controlling pain, and c) promoting mucosal repair.

Reducing inflammation

The non-steroidal anti-inflammatory drugs (NSAIDs) play an important role in modifying the systemic inflammatory response to gastrointestinal disease. The most commonly used NSAID is flunixin meglumine which has a well demonstrated ability to suppress the systemic response to endotoxin, together with potent analgesic properties. Judicious use of flunixin, however, is warranted to avoid unwanted adverse effects on the gastrointestinal mucosa and kidneys.

When administered early in the disease process, polymyxin B has been demonstrated to bind to endotoxin and prevents it from initiating or potentiating the systemic inflammatory response. The use of polymyxin B in humans has been limited by concerns regarding the nephrotoxic potential of this compound. It is thus recommended that horses are adequately hydrated, and serum creatinine monitored when administrating this drug. Pentoxyfylline has well-demonstrated anti-inflammatory properties and may be useful in treatment of diarrhoea cases. The toxin binding properties of di-tri-octahedral smectite (Biosponge) may reduce the degree of systemic toxin absorption and subsequent systemic inflammation, particularly in horses with clostridiosis. Bismuth subsalicylate has been used as an agent to protect the gastrointestinal mucosa and decrease mucosal inflammation and therefore may also be beneficial.

Pain control

Pain control is most commonly accomplished using NSAIDs. NSAIDs that typically target COX-2, such as meloxicam and firocoxib, may be more useful long-term in sparing the gastrointestinal mucosa. Xylazine or detomidine may provide temporary relief of pain. Opioid analgesics, such as butorphanol and morphine, can be used as an alternative to, or adjunctive therapy with, NSAIDs or alpha-adrenergic agents. Butorphanol can be administered either intramuscularly or as a continuous rate infusion (CRI). Lidocaine has been demonstrated to have analgesic and anti-inflammatory effects, in addition to effects as a prokinetic, and is also widely used as a CRI.

Promoting mucosal repair

Misoprostal and other synthetic prostaglandin E1 analogues have been shown in other species to enhance intestinal mucosal healing in models of colitis. The efficacy of misoprostal in hastening mucosal healing in equine colitis however is unproven. Adverse effects such as colic, diarrhoea, abortion in pregnant mares and sweating may however limit its clinical use. Psyllium mucilloid can be added to the diet to increase the production of short chain fatty acids in the colon, which play an important role in maintaining metabolic homeostasis in colonocytes. Omega 3 oils have also been advocated as cytoprotective in the presence of gastrointestinal inflammation.

Use of antimicrobials

The role of antimicrobial therapy in horses with diarrhoea is controversial. Antimicrobial administration has been associated with the development of diarrhoea in many studies and the induction of bacterial resistance is an increasing concern. There is no evidence to suggest that routine systemic use of antimicrobials benefits equine patients with colitis, with the exception of metronidazole in cases of clostridial diarrhoea. Antimicrobial use is however advocated in neutropaenic horses or horses with signs of septicaemia.

Nutritional support

Horses with colitis are typically anorectic. Good nursing care and adequate nutrition are vital. The basic goal of nutritional supplementation is to prevent the development of a negative energy balance in patients unable to meet their nutritional needs through voluntary intake. Most adult horses can be reasonably maintained without nutritional support for a few days. Horses suffering from colitis can often be allowed to ingest feed, provided that substantial ileus is not associated with their condition. Access to high quality grass is ideal. Dietary management usually consists of restricting or eliminating long-stem roughage from the diet and feeding a complete pelleted feed to reduce the mechanical and physiologic load on the colon. Frequent meals are recommended. Corn oil can be added to the pellets to increase the caloric intake without adding bulk.

Prognosis

If left untreated more than 90% of horses with acute colitis die or are euthanised. Horses that are treated appropriately usually respond and gradually recover over a 7- to 14-day period.

Snake envenomation in the horse

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Introduction

Snake envenomation is often suggested, but rarely confirmed, in the unexpected death of horses in Australia. Elapid snakes are the predominant snake family in Australia and comprise many of the world's deadliest snake species (most notably tiger, black, and brown snakes). Although potentially life-threatening, not all cases of elapid snake envenomation in horses result in a fatal outcome.

Clinical signs

Elapid venoms contain potent neurotoxins, cytotoxins, and procoagulants that act to immobilise the snake's prey and start the process of digestion. The predominant activity of the venom – neurotoxicity, myotoxicity or coagulopathy – will depend on factors such as the species of snake and season. Every envenomation is a unique event and outcome will depend on the aforementioned factors, together with other notable factors such as the amount of venom injected, the time course of venom absorption, and geographical location. Neurotoxicity characterised by progressive generalised neuromuscular weakness (staggering gait, muscle fasciculation, recumbency, mydriasis with delayed or absent pupillary light response, ptosis, tachypnoea, dyspnoea, dysphagia and/or tongue paresis) is the principle manifestation of disease in the majority of horses. Other common clinical findings of disease in horses include dull mentation, tachycardia, profuse sweating and mild pyrexia.

Clinical pathology

Myotoxicity is a reliable feature of tiger snake envenomation in humans, dogs and cats, with CK activity often used to aid in diagnostic confirmation. Mild to moderate elevations of CK have been reported in envenomed horses. Notable differences in myolytic activity between elapid venoms are reported, whereby brown snake venom does not have any myolytic activity. Other abnormal clinicopathological findings may include hyperlactaemia and pigmenturia. Although haemolysis is reported in some cases of elapid snake envenomation in horses, venom-induced consumptive coagulopathy (VICC) has not been reported as a major manifestation of disease in horses, which contrasts to the reported finding of VICC in envenomed humans, dogs and cats.

Diagnosis

Elapid snake envenomation can present a diagnostic and therapeutic challenge for veterinarians. Diagnosis requires the use of a combination of history, clinical signs, local knowledge and laboratory tests. Due to the very small size of elapid snake fangs and lack of local reaction to the bite, bite sites are seldom found on animals. If clinical signs are vague or equivocal, laboratory tests involving measurement of serum CK and the use of a commercially available multivalent snake venom detection kit (SVDK) are used to confirm diagnosis of suspected envenomation. The SVDK may also be used as an aid in selecting an appropriate cheaper monovalent antivenom to use. Although the SVDK is reportedly capable of detecting and immunotyping venom from any tissue, body fluid or other biological sample, a bite-site swab is considered to prove the most valuable result, followed by urine, serum, or plasma. The SVDK has been validated for use on equine urine and plasma samples, however, test performance has not been widely evaluated in clinical cases. A negative SVDK result does not rule out envenomation. Results must therefore be interpreted in light of the clinical signs and laboratory data, and knowledge of local snake geographical distribution.

Treatment

Once recognised as a potential diagnosis, prompt medical intervention and intensive care is required to reduce morbidity and mortality. Envenomed animals can clinically deteriorate extremely rapidly. Antivenom use is the cornerstone of treatment of elapid snake envenomation and acts to neutralise unbound circulating venom, thus preventing venom from binding to target proteins and worsening any clinical effect. Antivenom does not, with the exception of some of the pre-synaptic neurotoxins, reverse the clinical effects of venom. There is a perceived misconception that horses require large amounts of antivenom because of their size, thus prohibiting its use because of cost. The dose of antivenom, however, is determined by the dose of venom injected rather than the patient's body size. The titration of multiple vials of antivenom to effect is not uncommon in small animal practice. More recent research in human patients has shown that one vial of antivenom is sufficient to treat envenomation by the most commonly encountered Australian snakes in almost all cases. In horses, the required volume of antivenom is often determined by the clinical response to administration, and in most cases, only a single vial of antivenom is required. It is recommended to administer antivenom diluted in 1 L of an isotonic crystalloid solution over a period of 15-30 minutes. In the absence of definitive snake identification, the appropriate antivenom may be chosen with the aid of the SVDK. In areas where there are two or more venomous snakes, it is recommended to stock polyvalent antivenom.

Supportive care to manage the clinical manifestations of envenomation is also required. Cardiovascular support with intravenous fluids, respiratory support with supplemental oxygen, nutritional support, analgesia and nursing care in recumbent animals may all be necessary. Ensuring renal perfusion through the use of intravenous fluids is the mainstay for preventing acute renal injury secondary to rhabdomyolysis and myoglobinuria. Analgesia is also indicated in horses with myopathy. Infection secondary to elapid snake envenomation is very rare and the use of prophylactic antibiotics is not indicated. Routine use of corticosteroids or antihistamines as a premedicant to the administration of antivenom has also been questioned and is no longer recommended in human medicine.

Prognosis

Observational studies in other species show that administration of antivenom is associated with improved outcomes. A sole observational case series of elapid snake envenomed horses reported a favourable survival rate (86%) in cases that received antivenom. Although the administration of antivenom is associated with improved outcomes, the SVDK and antivenom are expensive, and so their use may be precluded if there are financial constraints. If there are no financial constraints, and access to 24 h care is available, the prognosis for horses with snake envenomation is considered good.

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Total intravenous anaesthesia with ketamine, medetomidine and guaifenesin compared with ketamine, medetomidine and midazolam in horses.

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Introduction

In horses, there is no information directly comparing midazolam with guaifenesin when used in combination with an alpha-2 agonist and ketamine to maintain anaesthesia via intravenous (IV) infusion. The primary aim of this study was to compare total intravenous anaesthesia (TIVA) of ketamine, medetomidine and guaifenesin with ketamine, medetomidine and midazolam in horses. A secondary aim was to evaluate ketamine, medetomidine & midazolam TIVA for horses undergoing castration.

Materials and methods

The initial study was a prospective blinded cross over design. Fourteen weanlings were premedicated IV with medetomidine (7 μg kg^{-1}) and induced with ketamine (2.2 mg kg^{-1}). Horses were randomly allocated to each receive infusions of ketamine (3 mg kg^{-1} hr^{-1}), medetomidine (5 μg kg^{-1} hr^{-1}) and guaifenesin (100 mg kg^{-1} hr^{-1}) (KMG) or ketamine (3 mg kg^{-1} hr^{-1}), medetomidine (5 μg kg^{-1} hr^{-1}) and midazolam (0.1 mg kg^{-1} hr^{-1}) (KMM) for 50 minutes on two separate occasions. Cardiorespiratory variables and anaesthetic depth were assessed at 5-10 minute intervals and recovery times after the infusions ceased recorded. Recovery quality was assessed by three blinded observers with three scoring systems; a composite system, visual analogue scale (VAS) and a simple descriptive scale (SDS). In a second study, five colts scheduled for routine castration were premedicated with IV acepromazine (0.01-0.02 mg kg^{-1}), medetomidine (5-7 μg kg^{-1}) and methadone (0.1 mg kg^{-1}). Anaesthesia was induced with IV ketamine (2.2 mg kg^{-1}) and midazolam (0.06 mg kg^{-1}) and maintained for 40 minutes with an IV infusion of KMM. Surgical conditions were subjectively assessed, and anaesthesia monitoring and assessment of recovery from anaesthesia was as previously described.

Results

Results are reported as marginal mean ± SE. For the initial study, cardiopulmonary variables were generally well maintained with little difference between treatment groups. There was no difference ($p < 0.05$) in recovery times with horses standing 23 ± 2.4 and 20.9 ± 2.6 minutes after the infusions ceased for the KMG and KMM groups, respectively. The quality of the anaesthetic recovery in the KMM group was significantly better than the KMG group when results from the three scoring systems were compared (Table 1). For colts that were castrated, surgical conditions were described as acceptable. Cardiopulmonary data presented as range of mean values at each time point were: HR: 36-44 beats/minute; MAP: 109-117 mmHg; RR: 9-13 breaths min^{-1}; PaO_2: 99-158 mmHg; $PaCO_2$: 53 - 56 mmHg and pH 7.36 - 7.38. Horses stood without incident (mean ± SD) 38.6 ± 13.4 minutes after the infusion was stopped. Recovery scores were 34 ± 4, 83.7 ± 2.9 and 2 (good) for the composite system, VAS and SDS respectively.

Relevance to Australian clinical equine practice

In horses, TIVA using ketamine, medetomidine and midazolam is comparable to anaesthesia maintained with ketamine, medetomidine and guaifenesin and may result in an improved quality of recovery. An infusion of ketamine, medetomidine and midazolam also provides adequate anaesthesia for castration. In Australia, this combination may be useful for short term TIVA for horses given the recent unavailability of commercially formulated guaifenesin.

Table 1: Anaesthetic recovery scores using 3 scoring systems for 14 weanling horses anaesthetised with infusions of ketamine, medetomidine and guaifenesin (KMG) or ketamine, medetomidine and midazolam (KMM).

	Mean	SE	Comparison (KMM – KMG) Difference	SE	p-value
Composite scoring system					
KMG	64.3	3.8	-15.6	3.6	< 0.001
KMM	48.7	3.7			
Visual analogue scale					
KMG	48.6	4.5	14.6	3.6	< 0.001
KMM	63.2	4.5			
Simple descriptive scale					
KMG	3.4	0.2	14.6	3.6	< 0.001
KMM	2.7	0.2			

Results as marginal mean ± SE. $p < 0.05$ statistically significant. Composite scoring system; 0-115 with 0 optimal recovery, Visual analogue scale; 0-100 with 100 optimal recovery, Descriptive scoring system; 1-5 with 1 excellent recovery.

How To Catch An Exotic Disease

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Introduction

The Australian livestock industry, including the beef cattle, dairy, sheep, pig and poultry industries, account for 45% of the gross value of Australian agricultural output and generated in excess of AUD$22 billion in 2015-2016 for the Australian economy.[1] Farming enterprises in Australia provide direct employment for approximately 140,000 persons nationally. The livestock industry also generates significant indirect employment and economic activity through advisory and technical services, road transport, energy, veterinary and pharmaceutical products, finance and insurance, and a wide range of manufactured goods.[1] Livestock disease investigations are an integral part of protecting that industry and all other livestock industries in Australia on several levels. In 2016, WA turned-off 757 000 head of cattle with just under half of these destined for live export (A$400m).[2] Many of Australia's trading partners have very strict import requirements and require evidence that any product entering their country is free of certain diseases and residues. Livestock disease investigations in Australia provide valuable data and evidence of our freedom from these diseases, keeping these export markets open to the Australian economy. Many of the diseases exotic to Western Australia, including diseases like Foot and Mouth disease (FMD) and Classical and African Swine fever (CSF and ASF), would devastate the Australian livestock industry if they were to breach our borders. Everyday livestock disease investigations are a primary method of detection of exotic disease in this country. Consequently, it's vital that veterinarians in the field know not only how to recognise these diseases but also how to maximise the chances of an accurate and rapid diagnosis by sampling appropriately. While it's vital to be able to rapidly and accurately diagnose or exclude the relevant exotic disease, it is also beneficial to the veterinarian, the producer and the Western Australian government to make an accurate endemic diagnosis whenever possible. This paper presents guidelines for sampling during a livestock disease investigation that maximise the chances of a reliable exotic disease exclusion and a useful endemic diagnosis with the same sample set. This paper will also provide a brief update on the status of several exotic diseases across the globe.

Sampling in a Livestock Disease Investigation

The two most important parts of successful livestock disease investigation are consistency and an open mind. At DPIRD we recommend consistently collecting the same set of samples for every disease investigation regardless of the species. Table one (below) illustrates the base sample set recommended by DPIRD pathologists. This sample set allows for diagnosis of a wide variety of exotic and endemic diseases including exotic diseases such as FMD, CSF and ASF. Sampling widely, regardless of the provisional diagnosis, ensures important or alternative diagnoses are not missed due to a lack of diagnostic material. In addition, we routinely classify disease investigations by 'syndrome' and recommend collecting additional samples for certain syndromes in certain species. Table two (below) illustrates additional samples we recommend collecting for investigation of particular syndromes such as abortion and stillbirth, skin lesions and sudden death. Finally, in addition to the physical samples, relevant information such as the location and contact information of the submitter and the owner or premises sampled as well as the case information and associated epidemiological information should always accompany the specimens to the laboratory.

Sample type	Sample to take	Storage/transport
Antemortem samples	Lithium heparin 10mL EDTA 10mL Clotted 10mL Faeces 10g	**Blood samples:** Mix with anticoagulant and chill; **do not freeze.** Draw off serum if prolonged transport.
Fresh tissue (individual containers)	Brain (swab/small section) Liver Lung Kidney Rumen contents (100-200g) Ileal content 10mL Vitreous humour 1mL Faeces/rectal content 10g	**Fresh samples:** Chill/refrigerate; **do not freeze.**
Fixed samples (pooled in formalin - 10:1 formalin:tissue)	Brain (fixed whole) Kidney Liver Lung Rumen Abomasum Small intestine Large intestine Skeletal muscle Heart	**Fixed tissues:** Can be kept at room temperature

Table One: DPIRD Diagnostic Laboratory Services (DDLS) base sample set.

Syndrome	Additional samples
Abortion/ stillbirths	Placenta, foetus - lung, liver, kidney, adrenal, stomach content, skin/eyelid; dam - uterus or uterine swabs
Acute febrile disease	Alimentary sections and local lymph nodes (fixed and fresh), feed (ergot)
Anaemia	EDTA blood, air-dried blood smears, fixed spleen, bone marrow
Congenital defects	Consider whole neonate or grossly affected tissue
Diarrhoea	Multiple alimentary sections and local lymph nodes (fixed and fresh)
Found dead	As for neurological; possible water sample (blue-green algae), plants, vitreous humour
Genital lesions	Fixed/fresh biopsy material, swabs in viral transport media, swabs in culture media
Ill-thrift/ weightloss	Multiple alimentary sections and local lymph nodes (fixed and fresh)
Female infertility	*Campylobacter* swabs, *Tritrichomonas* swabs, uterine swab/biopsy, fixed reproductive tract
Male infertility	Semen, swabs, fixed and fresh testicle/reproductive tract
Jaundice	Fixed spleen and liver, plants (sporedesmin, other hepatotoxins)
Lameness	Arthritis - swabs or fresh joint fluid/synovium, fixed joint capsule/synovium, fixed muscle, fixed bone/joint for deformity/joint lesions Hoof lesions - interdigital and hoof scrapings in Stuart media
Lymphadenopathy	Fixed and fresh lymph node, swabs

Mastitis	20mL milk in sterile jar, chilled
Muscle lesions	Fixed muscle and heart, fixed nerve and spinal cord
Myiasis (flystrike)	Maggots chilled, in alcohol or formalin. Photo in situ
Neurological signs	Fixed and fresh spinal cord segments, EDTA (thiamine), vitreous humour, fat (samples for TSE exclusion) Avian - blood and cloacal swabs in viral transport media (VTM)
Pruritis	Skin, ectoparasites (sheep TSE samples for scrapie exclusion)
Production drop	Alimentary sections and local lymph nodes (fixed and fresh), feed Cows - udder (fixed/fresh), milk Avian - blood and cloacal swabs in VTM (consider submitting the whole bird)
Respiratory signs	Bronchial lymph node, trachea/larynx, feed (ergotism) Avian - blood and cloacal swabs in VTM
Salivation	Oral mucosa/vesicles/erosion (fixed) vesicle fluid (fresh in VTM), feed
Skin lesions	Skin/mucous membrane (fixed) swabs if mass, vesicle fluid in VTM, feed/plants (ergot etc.) Avian - blood and cloacal swabs in VTM
Sudden death	Plants/feed, water, vitreous humour Avian - blood and cloacal swabs in VTM
Weakness / paresis	Fixed and fresh spinal cord segments, fat, vitreous humour Avian - blood and cloacal swabs in VTM

Table Two: DPIRD Diagnostic Laboratory Services (DDLS) additional samples by syndrome.

Foot and Mouth Disease: Additional Sampling Requirements

FMD is a highly contagious disease with the potential to induce economic devastation in the Australian livestock industry should it enter the country. FMD is clinically indistinguishable from other vesicular diseases of livestock and should be considered as a differential diagnosis in any cloven-hoofed animal with vesicular lesions on the feet, buccal mucosa or mammary glands, sudden acute milk drop in diary animals and/or severe pyrexia. In acutely infected animalsthe FMD virus may be detected in virtually any secretion or excretion including expired air, even in animals without detectable clinical signs (i.e. pre-clinical infection). However, following recovery from the acute phase of the disease the virus disappears from most secretions and tissues but can persist in oropharyngeal fluids and lymph nodes, particularly in carrier animals. The preferred tissue for diagnosis of FMD is epithelium from unruptured or freshly ruptured vesicles or vesicular fluid. However, where collection of these particular samples is not possible (i.e. the vesicular phase of the disease has passed), blood in EDTA, clotted blood/serum and/or oesophageal–pharyngeal fluid samples are appropriate. In ruminants oesophageal–pharyngeal fluid samples can be collected using a probang (sputum) cup. In pigs, throat swabs are useful for diagnosis. In deceased animals myocardial tissue or clotted heart blood can be submitted. Swabs and epithelial tags should be preserved in viral transport media (VTM) where possible. If VTM is not available these samples can be submitted fresh and chilled in a sealed container with a few drops of sterile saline. Other samples (i.e. blood, fluids, myocardial tissue) can be submitted fresh and chilled. Samples from suspected cases of FMD should be transported under secure conditions and the receiving laboratory should be alerted to impending arrival as early as possible.

The Swine Fevers: Additional Sampling Requirements

ASF and CSF are highly infectious diseases with very similar clinical, gross and histological presentations. These diseases can affect domestic and wild pigs of all breeds and ages and produce a wide range of syndromes in affected animals. Both acute and chronic manifestations are reported and strain virulence can vary significantly. It is impossible to differentiate between ASF and CSF based on clinical signs or gross findings. These diseases should be considered in any pig with acute febrile disease or haemorrhagic syndrome. Bacterial septicaemias, Porcine Erysipelas, Porcine Dermatitis and Nephropathy syndrome and a range of other common pig diseases can be confused with ASF and CSF; laboratory testing is essential for a definitive diagnosis.

PCR is an excellent, highly sensitive, and rapid technique for the diagnosis of ASF and is often the first port of call in the exclusion of this disease in WA. ASF virus can be detected by PCR very early in the disease process and viraemia can persist for several weeks in pigs acutely or chronically infected with low or moderately virulent strains. Useful diagnostic samples for the diagnosis of ASF include tissues such as tonsil, lymph node, spleen and kidney, EDTA blood and serum. Samples should be submitted chilled.

For the diagnosis of CSF in live pigs, detection of virus through virus isolation or viral nucleic acid by PCR on EDTA whole blood or of antibodies in serum are the methods of choice. In deceased pigs, detection of virus, viral nucleic acid or antigen in organ samples are most suitable. Useful diagnostic samples for the diagnosis of CSF include tissues such as tonsil, lymph node, spleen, ileum and kidney, EDTA blood or serum. Samples should be submitted chilled.

Samples from suspected cases of swine fever should be transported under secure conditions and the receiving laboratory should be made aware of their impending arrival as early as possible.

In Summary

The recommended samples listed above for FMD, ASF and CSF are listed in the OIE disease manuals available on the World Organisation for Animal Health website. In each case several of the diagnostic samples recommended for diagnosis and exclusion of these diseases are included in the base sample list recommended by DPIRD and discussed earlier in this paper. By routinely collecting the samples on this list you will maximise the chances of a reliable exotic disease exclusion or diagnosis and, in most cases, will also get a useful endemic diagnosis with the same set of samples.

Foot and Mouth Disease: An Update

FMD is a transboundary animal disease with a morbidity rate that approaches 100% in cattle. This disease can severely affect the production of livestock and has disrupted regional and international trade of animals and animal products across the globe. The 2001 outbreak in the United Kingdom caused losses of more than 8 billion pounds (approximately AUD$19 billion).[3] Australia estimates that an FMD outbreak controlled in 3 months could cost around AUD$7.1 billion, while a large 12 month outbreak could cost in the vicinity of AUD$16 billion.[5]

In early 2019 China suspended wool trade from South Africa due to a recent FMD outbreak. Approximately 70% of South Africa's exported wool was destined for China prior to the suspension. The Australian wool industry generated AUD$3.9billion in 2018 with approximately 80% of exports to China.[4]

As of early 2019, South East Asia continues to struggle with outbreaks of FMD with numerous detections in the northern regions of Laos. Laos shares it's borders with China, Thailand, Vietnam, Cambodia and Myanmar and has an estimated 1.6 million cattle and 775,000 buffalo.

It is estimated that FMD circulates in up to 77% of the global livestock population, particularly in Africa, the Middle East and Asia and to a lesser degree in South America. Australia, New Zealand, Indonesia, Central and North America, and continental Western Europe are currently free of FMD. However, FMD is a transboundary disease which can occur sporadically in areas generally considered disease free. Countries like Australia are under constant threat of an incursion.[3] In December 2018 the Australian Animal Health Laboratory detected FMD in two separate meat products declared and/or seized at airports.[6] If infected meat products such as this were inadvertently fed to a susceptible species such as pigs these animals could become infected and be the source of an outbreak that could devastate the livestock industry in this country.

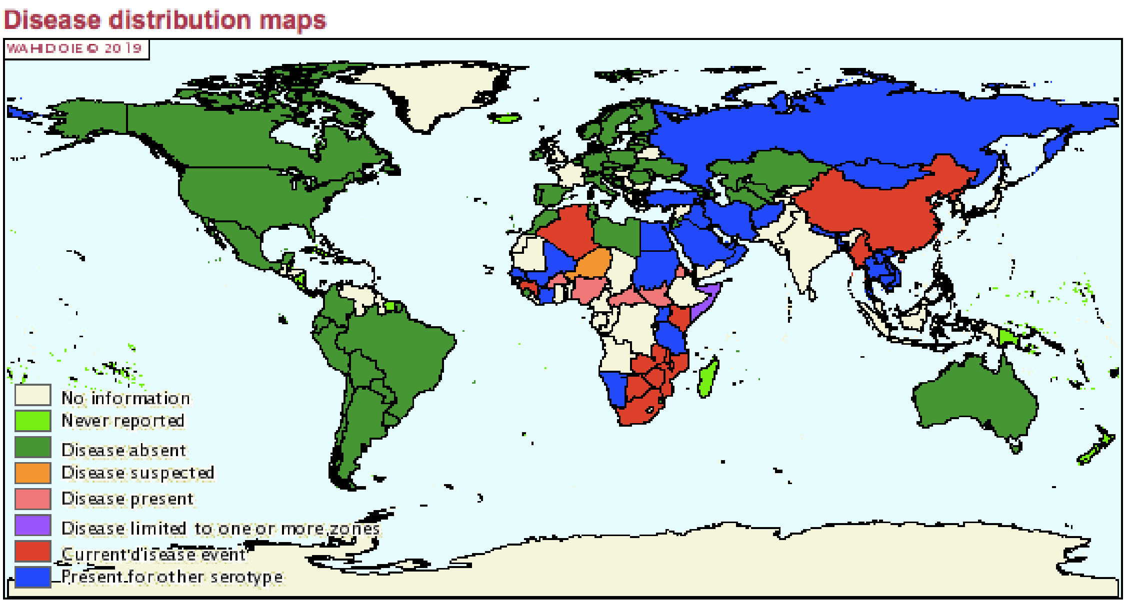

Figure One: Worldwide distribution of FMD in 2018.*

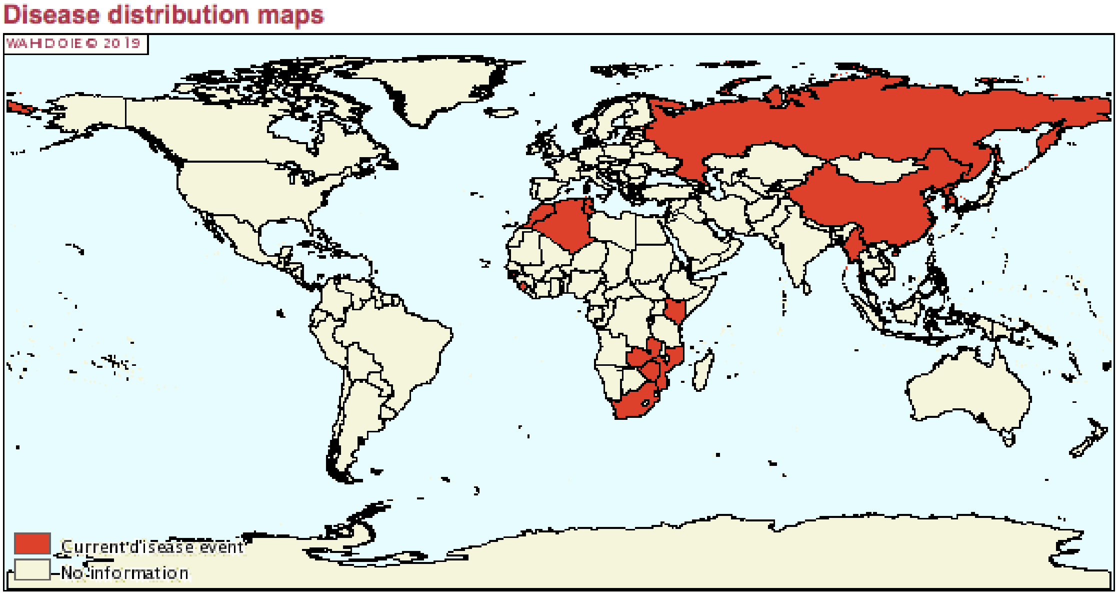

Figure Two: Current FMD outbreaks across the globe; February 2019.*

Classical Swine Fever: An Update

CSF was first detected in the US in the nineteenth century. It has subsequently been eradicated from North America and much of Western Europe. The disease is most commonly transmitted through direct contact with an infected host however contaminated feed and other fomites also represent an important mode of transmission. Persistently infected animals or chronic carrier animals may show no clinical signs of illness but shed virus in their faeces. The CSF virus can persist in pork products for many months when meat is refrigerated and for years when it is frozen. Similar to FMD and ASF, pigs can develop an infection through ingestion of infected pork meat or products. Disease spread has occurred through legal and illegal transport of animals and by feeding swill containing infective tissues.[7]

CSF is currently circulating in Central and South America, Europe, and Asia and parts of Africa. North America, Australia and New Zealand are currently free of the disease. CSF outbreaks occur relatively frequently in Asia and Southeast Asia and a wide diversity of viral strains and pathogenicities are reported.[7] A CSF outbreak was detected in Gifu Prefecture, Japan in September 2018 after 26 years of disease freedom in this country. Eighteen additional outbreaks have been reported subsequently throughout the region with close to 17000 pigs culled since September.[8] CSF also remains a recurring problem throughout Eastern Europe. Similar to ASF, in parts of Europe, the wild boar population may play a role in the epidemiology of the disease. A vaccine is available for CSF which makes control and prevention somewhat more manageable than that of ASF despite this reservoir of virus.[7,9]

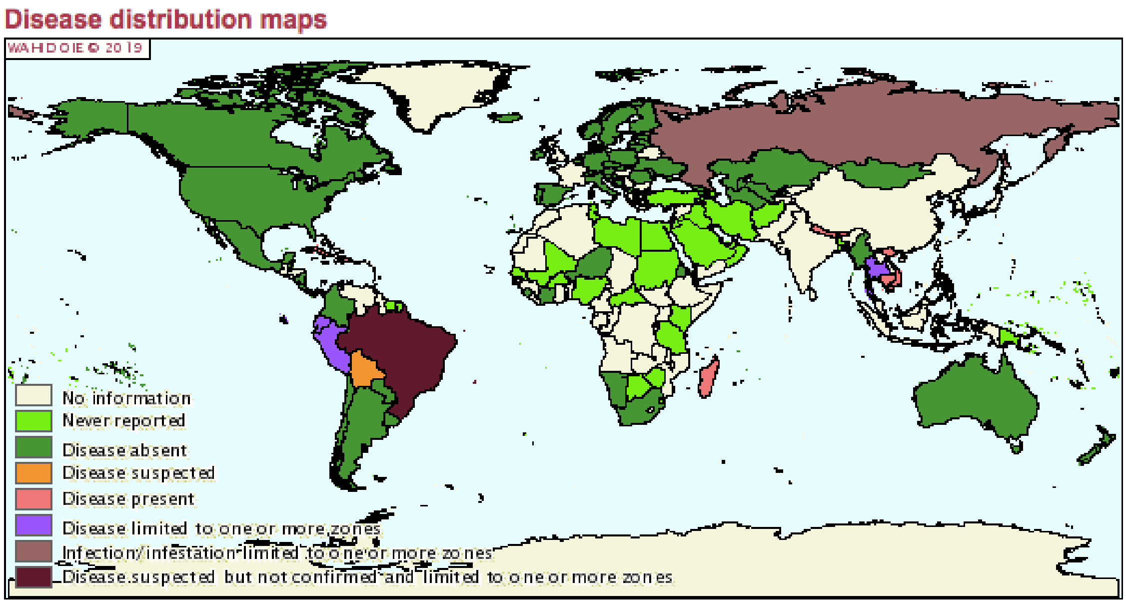

Figure Three: Worldwide distribution of CSF in 2018.*

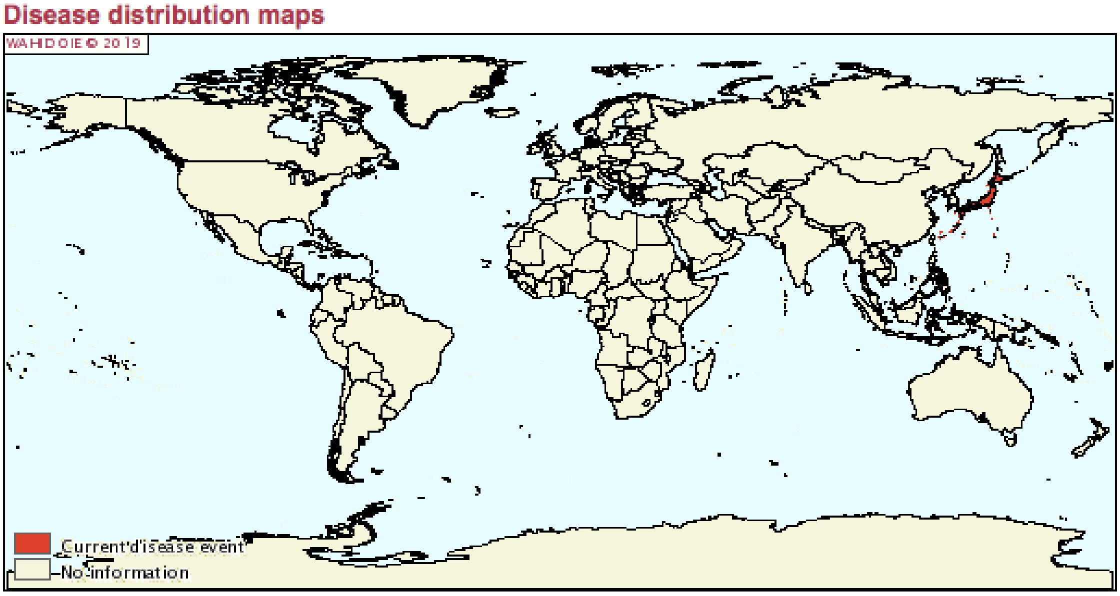

Figure Four: Current CSF outbreaks across the globe; February 2019.*

African Swine Fever: An Update

ASF can be spread by live or dead pigs as well as in pork products, contaminated feed and fomites due to the high environmental resistance of the ASF virus. There is also currently no approved vaccine against ASF which makes prevention and control challenging.[10] Historically, outbreaks have been reported in Africa, parts of Europe, South America and the Caribbean. However, in more recent times it appears the disease is spreading as it has been reported in multiple countries across Africa, Asia and more of Europe with isolations in both domestic and wild pigs. There have also been recent reports in Belgium and China. Figures five and six below illustrate the spread of disease between 2005 and 2018. Further spread throughout these countries and the world is likely as attempts to control the disease have been ineffective to date.

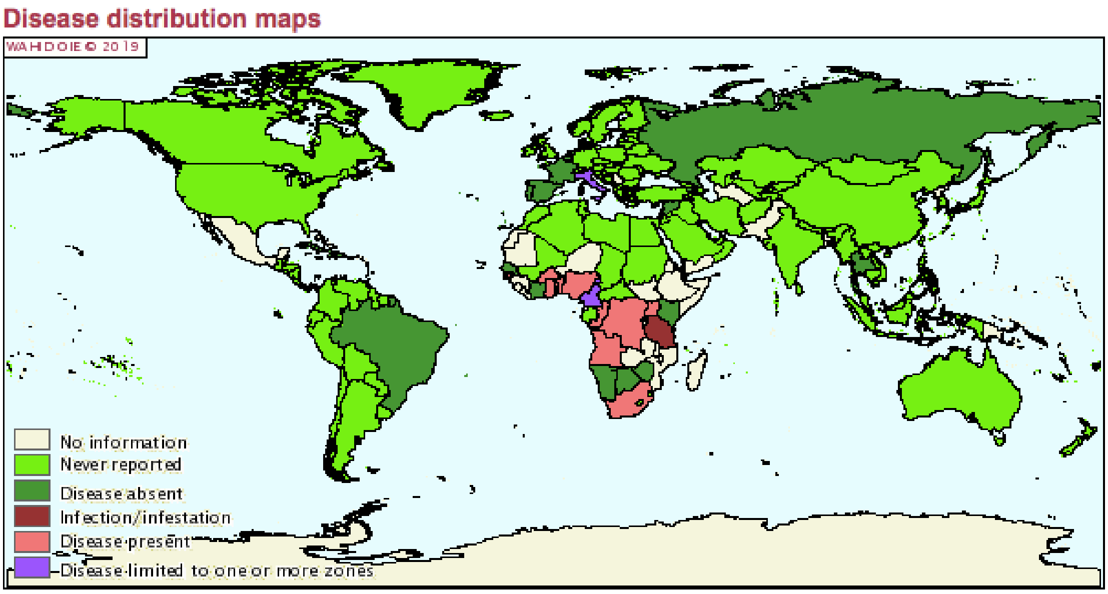

Figure Five: Worldwide distribution of African Swine Fever in 2005.*

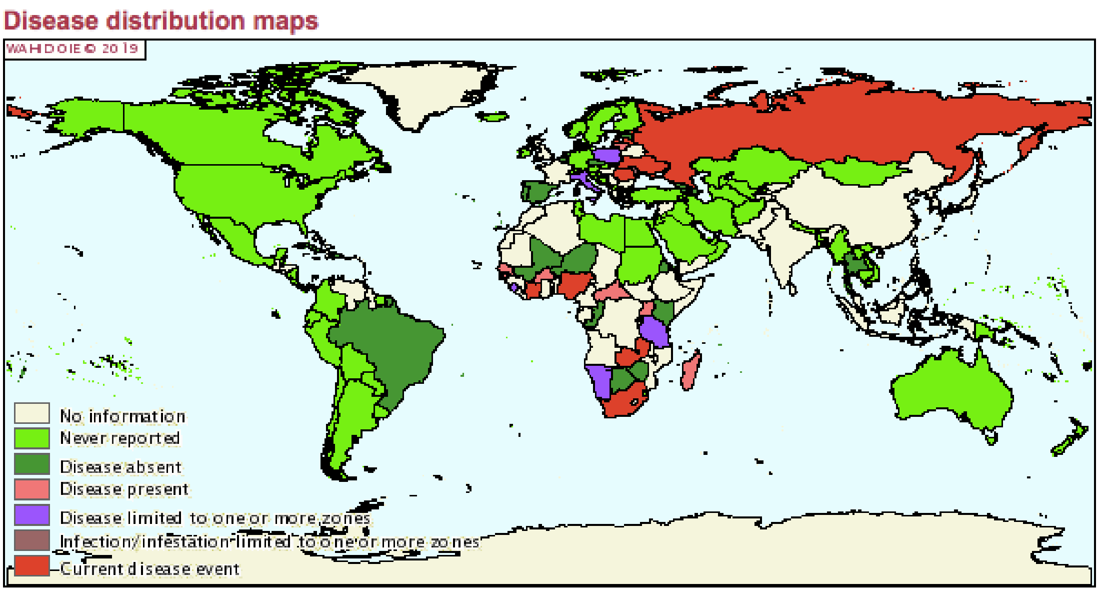

Figure Six: Worldwide distribution of African Swine Fever as of June 2018.*

As of early 2018, P.J.Sánchez-Cordón et al report that ASF virus is present in the Trans-Caucasus, parts of the Russian Federation, Ukraine, Poland, Latvia, Lithuania, Estonia, Moldova, the Czech Republic and Romania.[11] Outbreaks in domestic pigs have also occurred further east in the Russian Federation, including a region close to the Mongolian borders. In Poland and the Baltic countries nearly 8000 wild boar were found to be positive for ASF between 2015 and 2017. In Eastern Europe and the Russian Federation the majority of outbreaks in domestic pigs occurred on small scale or backyard farms where biosecurity standards are generally lower and there is large populations of wild boar close by. ASF outbreaks in 2014 and 2015 in Poland, Lithuania, Latvia and Estonia are estimated to have resulted in a loss of approximately USD$961 million through loss of export markets. In 2011 Russia estimated that ASF cost approximately USD$267million.

In early August 2018 the China Ministry of Agriculture and Rural Affairs (MARA) confirmed the first outbreak of ASF in Liaoning Province of China.[12] Since then over one hundred ASF outbreaks have occurred across China. To date, more than 950,000 pigs have been culled in an effort to slow the spread of disease. Outbreaks were reported in Mongolia in January 2019 and in Vietnam in February 2019. In January 2019 the Australian Animal Health Laboratory detected ASF in six meat products from a batch of 152 products declared and/or seized at airports.[6] The ASF virus is highly resistant and persists for long periods in infected meat products even after cooking. With 8.8million people entering Australia in 2017, an average of 1004 people per hour, breaches like this represent a very real threat the Australian pig industry.

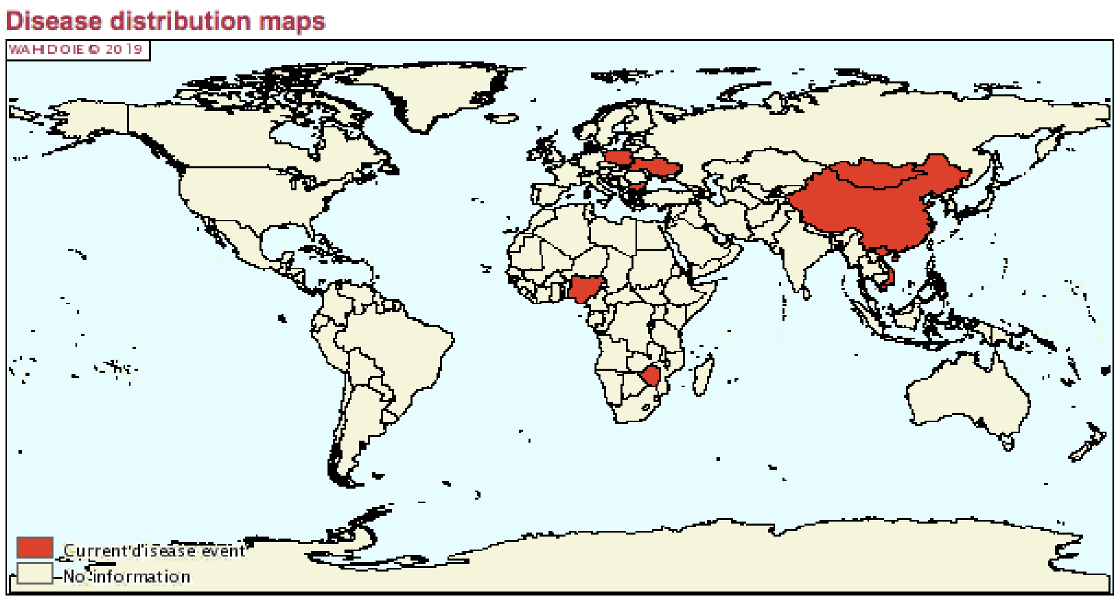

Figure Seven: Current ASF outbreaks across the globe; February 2019.*

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Life skills for kittens: what can owners do to help kittens grow up into confident cats?

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INTRODUCTION

One of the most important reasons for dogs and cats relinquishment and abandonment is behavioural problems. Prevention is the most important tool to avoid this worldwide recognised problem. But a behavioural problem seen from the animal's perspective can only be a way to survive or to copy with the environment. There are cats that immediately run away from visitors, to those that a minimal change in the environment leads to a high stress level that start spraying. The tolerance of the owners is totally different among all, but aggression is one of the less tolerated inadequate behaviour. And aggression is generally not well interpreted. The main reason for aggression in cats is fear and redirected aggression (also linked with fear emotion or stress). So, having a confident cat will bring to the owner the chance to have a good relationship with his animal, having a pet with the adequate skills for a lifetime. But how can we help the owners achieving this task?

TRAINING AND INFORMING

There are many different approaches in this field where we could be useful. We should work with breeders, with petshops, with animal protection associations and especially with the owner. The 3 first mentioned are really important, as we'll see, because they are involved in raising and first experiences of kittens. But is the owner that will have the chance to consolidate the learning for life. So, there are formal training sessions for owners (but all the other mentioned can also attend), called kitten classes. In this training the owner has a chance to learn about cat's behaviour and to avoid problems in the future. But also the kitten starts its training. Kersti Seksel (2004) developed a program of socialization, training and early education for kittens called *Kitten Kindy*®.[1] During this program not only the relation between cat and owner will increase, but there is also an habituation to veterinary environment in a positive way[1]. In these classes the owner can learn about the normal cat's behaviour in order that they can understand and avoid future problems[1]. Generally the classes focus in the appropriate way to interact and play, but also to understand body posture and cat's language[1]. Nevertheless, some colleagues suggest that during these classes, as the socialization period has already passed when they start, the advantages need to be more discussed. But, at least, informative kitten sessions can be organized at the clinic, with or without the kitten. Animals that are raised by people with knowledge show appropriate social behaviours and develop less behavioural problems[2]. Apart from this "formal" training, the vet should also pass adequate information during the first consultations and at the waiting room can have posters and leaflets about cat's behaviour and needs.

SOCIALIZATION

The socialization period (3 to 8 weeks old) in cats is earlier and shorter when compared with puppies. But it is a biological window of opportunities where the kitten can learn how to play, hunt, hygiene and biting/scratching inhibition. This learning procedure is firstly learned with the mother, but the adequate manipulation by humans will be useful

to have a confident cat with humans (20mn/day, with positive experiences with women, men, children). If we don't use this period to socialize than fear reactions can happen in the future.
The separation from mother should never happen before 6-8 weeks, because it is really important what she has to teach to the kittens. It is well studied that cats that are weaned and separated from mother too early are more reactive and more phobic, having a higher predisposition to frustration aggression, but also have no bite/scratch inhibition. The contact with other cats, kittens and other species is also very important during this phase. But this relation with other species is fragile and can be partial (for instance the cat can see the black rat as a "friend", but not the white one). It is important that the contact is maintained along time.

JUVENILE PERIOD

But the kittens come to the clinic about 8 weeks to start the vaccination and the socialization period is almost gone. Nevertheless we still have many things to teach. It is during this period that the cat organizes its territory and develops the affiliative behaviours within the social group (allogrooming). So, we have to create a secure, stable and well organized territory with enough resources (*see proceedings from the author about "Environmental Enrichment"*). At the same time the owner has to find some free time to play, being totally forbidden to do it with hands, feet or parts of the body. Stop playing immediately (negative punishment) if the kitten starts biting or scratching, and then redirect the behaviour toward a toy or something appropriate to present it. Cat's training ("sit", "lay", "give me five", etc) should start now.
Habituation to different type of manipulation is very important. The owner should associate, in a positive way, the manipulation of ears, mouth, hair and teeth brush, cut claws, but also handling and holding in different positions. Everything must be done gradually. The same should be done with different sounds, as the vacuum cleaner. The habituation to the cat carrier should be done the earliest, as possible, as well as the experience at the vet must be very positive.

CONCLUSIONS

At the end, how can owners prevent undesired behaviours and have confident cats? 1. Reinforcing the desired behaviour; 2. Ignoring (negative punishment) undesired behaviour; 3. Guide the undesired behaviour (as predation) to adequate targets (as toys). Finally the cat's initiatives should be respected and reinforced (if desired). This is the reason that the owner needs to understand cat's body posture to clearly interpret and understand his own cat. To have a confident cat, the skills need to be taught early in cat's life and there is a role for each one of us (from owners to vet, but also the important role of the kitten's mother). All together we can work to have more confident adult cats in the future, making their life easier ensuring its welfare.

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Chronic Stress and Renal Disease in Cats

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INTRODUCTION

Stress is considered to be involved in the majority of behavioural problems in cats[1], and currently it is recognised that chronic stress plays a negative role in the health status of an individual[2]. Constant noradrenergic activation leads to an inadequate adrenocortical control that seems be deeply linked with the evolution of chronic disease[3]. Specific medical conditions related to unresolved stress and anxiety are present in almost all veterinary specialties. Therefore, stress and anxiety can increase the risk of various diseases and exacerbate many medical disorders.

THE STRESS RESPONSE AND ITS RELATIONSHIP WITH KIDNEY DAMAGE

When an animal is exposed to a stressing stimulus, this immediately starts a response[4], through the activation of the Sympathetic Nervous System[5]. The continuous activation of this system due a situation of chronic stress leads to a constant release of renin by the juxtaglomerular apparatus. Renin, through the Renin-Angiotensin-Aldosterone System (RAAS), stimulates the release of Angiotensin II that leads not only to an increase in cardiac output but also an increase in peripheral vascular resistence and, consequently, systemic hypertension. The kidneys are a target organ in the regulation of systemic blood pressure and the maintenance of its physiological values. The abundant blood flow that each kidney receives is fully required to maintain an adequate renal function. For this reason the organ is very sensitive to changes in the blood flow[6].

Angiotensin II has several roles, including the release of aldosterone by the adrenal cortex, culminating in sodium and water retention by the kidney. The increase of vasopressin (or antidiuretic hormone) release will also increase water retention[6] in the distal convoluted tubule and collecting tubule. Poor excretion of sodium and regulation of fluids, induced by stress, can lead to a slow recovery of blood pressure values after a stressor stimulus and contributes to lesions in target organs[2] (figure 1).

The action of Angiotensin II in the kidney, causing vasoconstriction of the glomerular arterioles, is more intense in the efferent arterioles when compared with the afferent. So, there is an increase in the capillary pressure at the intraglomerular level. The progression of pathology and the decrease of the glomerular filtration rate is partly due the persistence of intraglomerular hypertension associated with an increase in transit of macromolecules for the kidney mesangium that results in a proliferation of cells and the mesangial matrix and consequent glomerulosclerosis. Simultaneously, the increased processing of Angiotensin I leads to tubulointerstitial nephritis[7]. Therefore, even if the activation of the RAAS has the objective to regulate sodium levels to minimize the effects on blood pressure, the circulating levels of Angiotensin II and Aldosterone can lead to renal and cardiac fibrosis, contributing to the progression of Chronic Kidney Disease (CKD)[8].

Beyond the role of RAAS in renal arterial hypertension and in CKD, the increased catecholamine release also contributes to the pathophysiology of CKD[9] and as we all know, catecholamines are also released as part of stress response.

NEW EVIDENCES AND CONCLUSION

With all these principles in mind the author's PhD Dissertation (data not published) hoped to ascertain whether Multimodal Environmental Modification (MEMO) can influence physiological indicators (cortisol and blood accounts) and CKD symptoms (blood pressure, phosphorus, blood urea nitrogen, serum creatinine, serum alkaline phosphatase) in cats. It was hypothesized that environmental modification and enrichment would promote changes in physiological indicators and reduce CKD symptoms. Twenty-eight cats from different households, with CKD in stage I or II of the International Renal Interest Society enrolled in this study. Every cat was fed with the same diet. Clinical reevaluations were done every 2 months during one year. At the middle of the study a training session for the owners introduced toys, pheromone diffusers and other enrichment techniques for the cats in the study. The physiological indicators and CKD symptoms were statistically analyzed at the end of the study. The most important result of this study was the highly clinically and statistically significant positive alteration of physiological indicators (leucocytes, erythrocytes) and reduction in CKD symptoms (Systolic Blood Pressure and blood urea nitrogen) after the introduction of MEMO. MEMO resulted in significant improvement of CKD symptoms and physiological indicators of stress. This is not to say that stress causes CKD or other cat pathologies, only that stress reduction plays an adjunctive role in the therapy of some chronic feline disorders, as Barrow & Jacobs (2002) suggested in humans[10].

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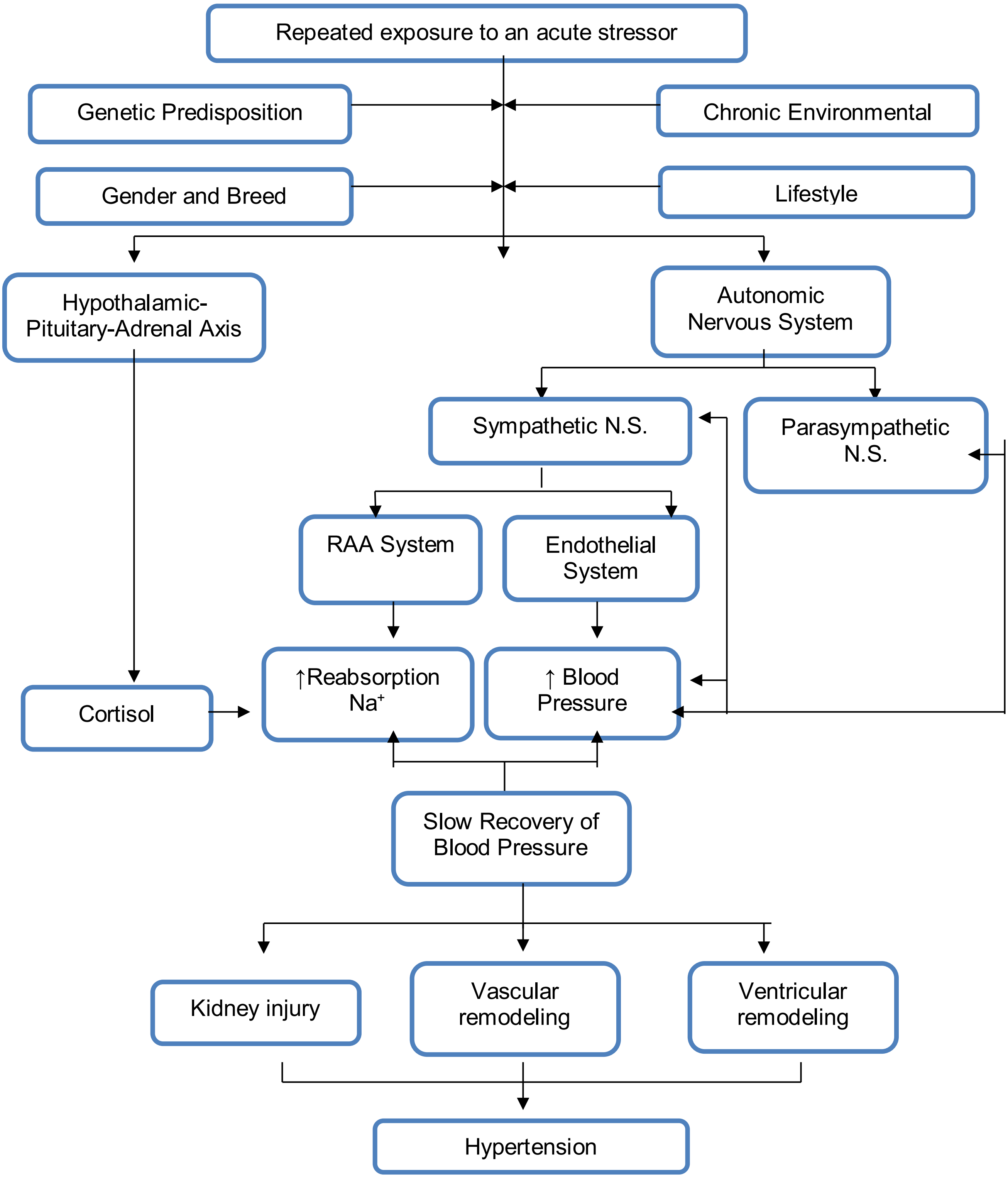

Figure 1. Model of kidney damage and hypertension induced by stress and influence of genetic and environment (adapted from Imumorin *et al*, 2005)[2].

Environmental Enrichment: Can living conditions lead to cats' behavioural problems?

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Cats are now living in a restricted environment inside our houses with partial or complete deprivation of access to the outdoor environment. This outdoor environment is itself a coveted resource that, when limited, can lead to physical and behavioural disturbances. When monotonous, unchanging and unchallenging environments provide insufficient mental stimulation, animals show signs of boredom, often manifesting as abnormal behaviour patterns[1]. Furthermore, cats living in a restricted environment may not have enough physical space to allow an acceptable flight distance thereby reducing the cat's opportunity to retreat. This situation is exacerbated by an increased density of cats living in the same home. In general, the presence of other individuals in its territory is not well tolerated, using displays of aggression to keep the others away[2]. The owner also promotes the creation of social relations incompatible with the behavioural nature of cats, not providing, in most of the cases, conditions to address many of their basic instincts[2]. Cats organise their territory in a way that allows them to hunt, feed, rest and eliminate far from other cats. The environment's balance can be disturbed by a poor distribution of required zones and resources (food, water, litter trays)[3]. The increase of interactions between cats can cause an increase in agonistic encounters and unwanted behaviours (such as spraying, scratching and aggression)[4].

When living and interacting together, cats and humans create a bond[5]. The number of interactions is, on average, low but there is a variability in this and also in the types of interactions presented[6]. Owner's expectations of their cats can cause an increase in pressures, both behaviourally and evolutionary, that can cause welfare issues[7]. Inadequate contact with people and uncontrolled access to other cats have been mentioned as stressors for this species[8].

So, let's now concentrate on ways to prevent behaviour problems! There are 3 effective ways: environmental enrichment, introduction of facial pheromones and pharmacological management with pschycotropics[9] (this last topic will not be covered in this lecture). Environmental enrichment is constituted by all measures that make the environment where the cat lives more stimulating[9] and that allow the manifestation of characteristic behaviours of the species[2]. The goal of environmental enrichment is to decrease the perception of possible threats present in the household, and consequently decrease the activation of stress response[10]. As methods of environmental enrichment, the following guidelines are recommended:

1. Litter tray: the available litter tray should be big enough[3] to allow the cat to turn around inside, getting in and out without any problem. Usually it is recommended to have at least one and a half the cat's size. The majority of cats prefer a fine (like sand) litter[11] and it is recommended to have, at least, one litter tray per cat plus one extra. They should be distributed throughout the territory and not all concentrated in the same room. They should be located in quiet, easy access places. The litter trays should have, if possible, two different access points to avoid the feeling of entrapment[3], and far

from noisy machines such as washing machines. Cats usually prefer open litter trays, with simple litter without any odour. Litter trays must as clean as possible.

2. Food and water: the feeding station should ensure that all cats from the same household have access, with an individual food and water bowl, being available *ad libitum*[3] Cats in the wild hunt between 100 and 150 times per day, over a period of 6 to 8 hours[2]. This behaviour is represented by the typical feline feeding pattern of 10 to 20 small meals over 24 hours. Having one food bowl per cat in different places, helps avoid problems of bullying between cats. The existence of multiple feeding stations, at different heights and in quite locations, allows cats to eat in a calm way without stress[11]. For that reason, the number of food and water bowls should be, at least, the same number as cats in the household. Both food and water bowls should be spread in different rooms of the household, in order to ensure access to these essential resources. Food and water bowls should not be side to side, and never close to the litter tray!

   Wet food is a way to increase a cat's water intake. An electric fountain allows access to running water. The water bowls should be wide and not too deep. The cats should be given the option to choose between dry and wet food in separate bowls, instead of substituting the usual diet with a new one, allowing the animal to express their preferences. If the cat refuses wet food, or the owner prefers not to feed wet food, other ways to increase water intake should be investigated (fountains, drop by drop taps, change the type of water bowl or even the material).

   One very efficient technique of cognitive and environmental enrichment for cats, is hiding or complicating the access to a type of food that they like (with a strong odour). The hiding place should not be so difficult, especially in first uses of this enrichment, otherwise it will cause more frustration and, consequently, more anxiety to the animal. Interactive toys and food dispensers are also recommended[11].

3. Physical Environment, Hiding Places and Flight Spot: every cat needs to have access to a resting place, where it can be isolated from the rest of the group (including from humans). In this resting territory, a hiding place should be available (can be a simple card box, a shelf, a drawer). This is an important mechanism for decreasing stress, allowing the cat to cope with situations that it considers a threat. Cats have a clear preference for vertical height, because this give better control of their territory and, consequently, a greater feeling of security and less anxiety. Usually, cats feel more secure when they are higher than the "predator" or environmental threat[13]. A cat tree with different surfaces levels can be recommended. A shelf, top of a cabinet, beds or sofas, can also be used. Baskets, cloth or blankets are very much appreciated by cats, requiring an individual area for each animal[3].

   We should also consider that every cat always needs to have a flight spot that ensures less confrontation with threats, and from where they can withdraw and flight. Scratching posts allow to the cat not only to use its claws, exteriorising and maintaining them, but is also a way of territory marking, thus a behavioural need. The post should be placed in a visible spot, close to the place where the spend most of their time, especially close to their resting place (generally where cat goes after waking up). In general, the favourite posts are those that cut up and tear easily: ropes, mat, carpet, tree trunk, paperboard[11]. In summary, the physical environment should always provide the possibility for the cat to climb, scratch, hide and rest. We need to keep in mind that cats tend not to aggregate in groups, preferring isolation. Felines that cohabit in the same household must have respective areas that allow them to “flight” from each other.

4. Play and games: Toys available should stimulate their predatory instinct, with fast movements, such as “fishing rods”, “moving” rats, aluminium foil balls, among others[3]. Forming a routine of physical contact with humans when young kittens will be beneficial because it creates a level of tolerance to contact with the owner. The guardian should dedicate some of his daily time to providing play and games with the cat, in order to promote physical exercise (with obvious consequences on decreasing stress). Also, and very importantly, it creates an affiliative link with the rest of the social group, where the owner could be also included. Play should allow cat to express natural predatory behaviour but directed to targets well selected by the owner. Therefore, we should never recommend owners play with their cats with hands or feet.

5. Pheromones: pheromonotherapy is an essential tool for the management of several behavioural problems related to stress and anxiety in cats[4,9]. Pheromones are volatile fatty acids released by different areas of the body that transmit information highly specific between individuals from the same species. Cats naturally use this type of chemical and odoriferous communication in several and varied situation. Facial pheromones are released in appeasing and satisfaction situations, for example during mutual grooming. The F3 fraction from the facial pheromone allows the cat to distinguish what is familiar, being deposited in objects and spaces where they rub their heads[14]. *Ceva Sante Animale* (Libourne, França) created a synthetic analogue of the facial pheromone that is naturally released by cats, called Feliway®. It has reduced the manifestations of behaviour related to anxiety in cats. Its effect and efficacy have been scientifically proven in several published papers. This product also helps the cat to identify the individuals from its social group. It can be used for the arrival of new members to the family (cat, dog, baby, etc).

Owners may also consult the internet, after seeking information from their veterinarian. We should tell the owners that this information needs to always be scientifically validated and properly filtered. One well renowned website and with useful information is www.indoorcat.org. It’s better that we give provide appropriate websites, because we all know that every owner will go and consult Dr. Google.

If we decrease surrounding stress, improving the cat’s quality of life and animal welfare[15] through the implementation of therapeutic strategies that decrease the noradrenergic activation[10], surely we will decrease the risk and manifestation of pathologies (including behaviour and many others)[15].

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Feline Psychogenic Alopecia

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INTRODUCTION

Repetitive behaviour can have various medical causes, but when the problem is not medical it can have several different aetiologies. The behaviour can be a normal part of the animal's behavioural repertoire, it can be environmentally induced (boredom, anxiety, among others) or it could be conditioned. Alternately, the problem might be medical (here the concept of medical, means organic disease, but behaviour which includes brain that is also an organ is also medical or organic. Therefore, the distinction between medical and behavioural helps us to differentiate between the two; but remember that we cannot split the animal into pieces taking off the brain and call behaviour problems as non-medical). For example, we can have a dermatology lesion (focal dermatologic pathology in the paw) that causes pruritus to the animal, which the owner then reinforces when trying to stop. Even when you start treating the dermatologic problem, you'll also likely need a behavioural approach for behavioural modification. But it can happen also the other way around. The cat could be very anxious and start overgrooming (the mechanism will be explained below) leading to development of skin pathology. Therefore, the approach to treat a repetitive behaviour can involve both "medical" and behavioural approaches. Garner (2006) defined repeated motor patterns that are uniform and where the focus appears to be on performance of the motor pattern itself, as stereotypies[1]. Stereotypy describes a repetitive, ritualised, out-of-context locomotor behaviour. A different definition is presented for a compulsive behaviour that results from a fixation on a goal and non-locomotor related. Compulsive behaviours can range from staring at an object to very complicated rituals involving a high level of cognition, that is displayed out of context, exaggerated or sustained. Hewson and Luescher (1996) defined compulsive behaviour as "*behaviours that are usually brought on by conflict, but that are subsequently shown outside of the original context. The behaviours might share a similar pathophysiology (e.g. changes in serotonin, dopamine and beta-endorphin systems)*".[2]

BIOLOGY OF THE CONDITION

Compulsive disorder (CD) arises as a result of a pathological process in the central nervous system. Little is known about the aetiology of obsessive-compulsive disorder (OCD) in humans and the evidence that this is the same disorders in dogs is circumstantial. Most evidence of pathophysiological mechanisms came from pharmacological studies. High dosage of dopaminergic drugs (such as amphetamine) induce stereotyped behaviour in animals, while dopamine antagonists (haloperidol) supress the stereotypical behaviour[3]. Some authors suggest that beta-endorphin antagonists may be at least temporarily effective in suppressing compulsive behaviour[4,5]. There is a positive response in human patients with OCD to treatment with serotonin re-uptake inhibitors (SSRIs), while serotonin receptors agonists can exacerbate symptoms of compulsion. Based on this human evidence, veterinary behavioural medicine has been strongly influenced by the comparison made between compulsive disorders in animals with OCD in man. Nevertheless, even though compulsive disorder in animals and OCD in humans have some similarities, regarding ritualisation and repetition of behaviour, human OCD involves thoughts, preoccupations and attempts at rationalisation. In humans these behaviours can involve cognitive or physical rituals, and there is a discussion among different authors if this condition does have or not have cognitive components that would be considered an obsession. But, to avoid further discussion, we'll consider that animals do not obsess, and that these are compulsive disorders (CD). As seen previously by the definition presented before, a CD predicts that the compulsive behaviour is first shown in a specific conflict situation that can be prolonged and/or repeated. This situation can be generalised to other contexts in which the animal has a high level of arousal. This can lead to cases where the animal presents the compulsive behaviour constantly except when eating, drinking or sleeping and may, in very extreme cases, interfere with survival behaviours. Differences in the presenting signs of CD will vary between individuals. The link to explain why CD develops in animals is related to the coping strategy that a specific animal uses during arousal or anticipation. In some animals, if the outcome from an arousal stimulus or anticipation is less rewarding, or never happens, then it will become a frustration. Frustration and fear are negative emotional experiences that involve anxiety but are essentially the same emotional state in animals. Generally, animals cope with arousal, anticipation and frustration and do not develop problems. Whenever an animal is in a situation of frustration or motivational conflict, it is normal to perform a conflict behaviour, but this behaviour is only shown in conflict situations. In contrast, in CD the conflict behaviour is shown in situations in which there is no outside stimulus inducing it. CD starts when the animal discovers that the repetition of a behaviour produces a reduction in arousal and frustration. This is the reason why usually the ritualised behaviour is one that the animal is already highly motivated to perform. It can correspond to a displacement or self-appeasing behaviour or escape behaviour, arising from species- or breed-specific behaviour, or a behaviour that was learned through previous intermittent reinforcement (for instance by the owners). Once ritualised, the compulsive behaviour is a powerful self-rewarding system. CD in cats are generally associated with the repetition or exaggeration of self-maintenance behaviours such as grooming, sucking or self-mutilation. In this presentation we will concentrate only in overgrooming behaviour that leads to alopecia.

RISK AND UNDERLYING FACTORS

1) Environment Factors

Cats are highly reliant and depend on their ability to control and use the resources in their environment. There is a strong need to hunt, feed and perform self-maintenance behaviours during each day, at specific time intervals. When a cat cannot control its environment (e.g. a social conflict with another cat, or separation related problems), the resources are not available at expected times, or there is a lack of stimuli, then stress, frustration or conflict may lead to the start of a CD. A disease that increases stress or irritability can also contribute to CD. But environmental factors are extremely important for every behavioural problem in cats, especially for CD. This disorder is frequently associated with the lack of ability to perform normal behaviours combined with social stress (secondary to competition and conflict). For instance, grooming can be exacerbated as a substitute for hunting or the territory maintenance.

2) Genetic susceptibility linked with breed

Pure-breed Oriental cats, especially Burmese and Siamese show higher rates of wool-sucking and self-mutilation, but recently created breeds (e.g. Bengal cats) also appear to be at risk. This fact cannot be simply related to genetic predisposition as these cats are often reared in "non-domestic" situation such as a cattery. A possible lack of socialisation and interaction with common domestic stimuli can develop fear and anxiety problems that in adulthood manifest in different ways, one of which could be CD. A lack of space and stimulation is also a risk factor in Bengal cats.

3) Physical stimuli
As mentioned in the introduction, a physical lesion (such as skin) or irritation (such as allergy or neuropathic pain), may trigger CD. Although not proven, it is assumed that the stress associated with a lesion or irritation can be a first trigger for an already susceptible cat.

4) Conditioning
Compulsive behaviours can be reinforced by owner attention or, at least, may condition normal conflict behaviours to a level that appears compulsive. If the compulsive behaviour is only present when the owner is around (or always that he is not around), then this is suggestive of a conditioned behaviour.

DIAGNOSIS

As a syndromic diagnosis, it is essential that other aetiological diagnoses be considered as a first essential step. So, a medical assessment is very relevant to rule out neurological, dermatological or other medical pathologies that produce similar signs or that can co-exist in the same animal with CD. A complete physical examination with a basic neurological examination is required. In addition, prior to use of drugs the medical investigation should include: complete blood cell count, blood chemistry profile and possibly urinalysis. Imaging and other investigations (such as thyroid profile, dermatological tests, allergy testing, a complete neurological examination) may be required depending on the specifics. A trichogram should also be performed. When alopecia is secondary to deliberate grooming, the hairs will feel sharp when compared to normal hair touch. The reason for that feeling is that the tips have been taken off and the microscopic examination on a trichogram will confirm the state of the hair tips. Another sign of psychogenic alopecia is that the hair loss is restricted to parts of the body that can be reached by the tongue. Differential diagnoses should include: parasitism (from fleas to notoedric mange), staphylococcal furunculosis, dermatophytosis, endocrine dysfunction and allergic skin pathology, as well as localised or referred pain. Feline idiopathic cystitis can also be a reason for an alopecia in the perineal area with hair loss in the groin or abdomen. Feline hyperaesthesia may be associated with an acute onset of self-biting and hair pulling but other signs can help to differentiate from compulsive over-grooming. Psychogenic alopecia should only be considered when all the other medical causes have been eliminated. However, we must keep in mind that stress can lead to both exacerbation of auto-immune disease or allergy or cause immune depression, which can lead to a cycle. For instance, licking can worsen many dermatological lesions, resulting in an itch-lick cycle, and licking can persist much longer even after the original dermatological cause has been removed.[9]

TREATMENT
The treatment consists of 3 main steps: a) Environmental enrichment – to change the animal's environment and social interactions, removing (if possible) triggers and giving more consistency; b) Behavioural modification – avoiding every type of interactive punishment and increasing exercise, play and cognitive activities (from training basic commands to cognitive play to fill a greater proportion of his time and energy budget); c) Drug therapy - in most cases.

1) Environmental Enrichment
Since stress and anxiety are inter-related with CD, and particularly to psychogenic alopecia, triggers should be identified (based in the history) and removed (when possible). If it is impossible to remove the trigger, then desensitisation and counterconditioning is required as behavioural modification. Improving the cat's living environment is vital. Increasing the range of activities available for the cat and maintaining cognitive stimulation with interesting toys available and provided on a rotational basis. Food-dispensing toys may satisfy a cat's

motivation to hunt. The relationship between cats from the same household, as well as the relationship with the owners, must be properly assessed, so that underlying psycho-social stress may be removed or at least alleviated. Nevertheless, an adequate interaction with owners is required, as well as opportunities for play.
Dr. Buffington presented an environmental enrichment program referred to as Multimodal Environmental Modification (MEMO) that was used as therapy for cats with idiopathic cystitis[7]. MEMO resulted in a significant decrease of clinical signs, as well as signs of anxiety and a tendency to reduce aggressive episodes. This program provides environmental enrichment tailored to minimize environmental stress that can lead to conditions as feline idiopathic cystitis[7]. So, if for treatment of psychogenic alopecia we need to decrease stress, then MEMO should be recommended. MEMO advocates evaluating and optimising four key resources that can generate stress and anxiety: food, water, litter trays and resting/hiding places. Pheromonatherapy has been suggested as another useful tool to decrease perception of threats and increase the perception of safety within the household.

2) Behavioural modification

Highly structured interactions (as command-response-reward) should be used to avoid casual interactions. Obedience sessions allow for consistent interaction and can be very stimulating for the cat (depending on the reward, motivation, intensity and frequency of each session). This should be provided regularly and consistently throughout the day. The owner should also play with toys and the training sessions can have some tricks, such as retrieving a ball, using clicker-training. Punishment must never be used. It is aversive, unpredictable, leads to more stress and anxiety, and can generate undesired associations. An alternative that could be used is response substitution. When the cat engages in the inappropriate behaviour a noise might be used to stop this behaviour, immediately followed by a command and the animal rewarded for obeying the command.

3) Drug therapy

Pharmacotherapy is indicated in cases of compulsive self-mutilation or severe self-grooming. A reason not to delay the use of pharmacological treatment is that the longer the behaviour has been going on, the less effective its use is[8]. **Fluoxetine** (0.5-1mg/kg PO q24h), **clomipramine** (0.25-0.5-1mg/kg PO q24h – dose increase depending on the result), **sertraline** (0,5mg/kg PO q24h, during 6 to 8 weeks and then could be increased to 1mg/kg PO q24h) and **paroxetine** (0,5mg/kg PO q24h, during 6 to 8 weeks and then could be increased to 1mg/kg PO q24h) have proven effective as supportive treatment to behavioural modification and environmental modification. Tricyclics other than clomipramine are highly unlikely to have sufficient efficacy in psychogenic alopecia, as they have a weaker effect on serotonin reuptake. As compulsive behaviour, when established, can be performed even without a high arousal or high states of anxiety, anxiolytic drugs alone are generally insufficient. Drug results should be assessed after 4 to 8 weeks. The owner should closely track the number and severity of self-grooming bouts (since the beginning). If there is no improvement, the drug therapy may be changed, the dose may be increased, or combined poly pharmacology be considered. A successful drug therapy will produce about 70% decrease of the compulsive behaviour. After a period of 6 to 8 weeks without further over-grooming, a drug discontinuation plan can start. Weaning of the drug should be very gradual, starting with 75% of the dose for 2 weeks, then 50% for 2 weeks and 25% of the dose for 2 more weeks. This dose can be maintained for an extra 3 weeks, every other day, before being completely discontinued. During the weaning plan, if the behaviour reappears, the dose should be increased to the previous level that was effective for more 2 to 4 weeks before restarting the weaning. Be cautious of a possible rebound effect, where the compulsive disorder may reappear worse than before, due a sudden or too fast discontinuation of the drug. Some animals will need the medication for life. This can be seen as a chronic disease. Owner compliance is vital to the success of the treatment, but the outcome of the treatment can be negatively affected by problem duration[9]. The earlier the treatment starts, the better the results.

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Is it simply a toilet or details make the difference? Approaching the main causes of elimination problems

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People can experience a reduction in stress by having cats as part of their social structure and cats can benefit due to the plentiful food, shelter and social interactions involved[1]. Both people and animals can benefit from a social relationship between species[2,3,4,5], but is the actual human lifestyle compromising the cat's welfare and quality of life? The major role of the veterinarians is to assure the well-being of animals "relieving suffering, whether it is related to physical or emotional pain"[6], as stated by Beaver and colleagues (2004). "Does it matter to the animal?" is the first question that should be asked when assessing any animal's quality of life. In 2007, a panel of experts proposed that a first step towards assessing a species quality of life and welfare would be the establishment of an ethogram and definition of the behaviours consistent with optimal quality of life for that individual species[7]. With this in mind, a complete understanding of feline behaviour is important when evaluating the cat's wellbeing. Correct interpretation of behaviour will allow seeing changes in behaviour that can be good indicators of fear, frustration, or pain[8]. Some behaviours may be so strongly motivated as to constitute a "need". Odendaal (1994) defined needs in terms of their effect on quality of life[9]. If a behavioural need arises, then it is important that the environment provided allows for that need to be met. Are this cat's behavioural needs being ensured currently in the majority of households?

Pet cats are now living in a restricted environment inside our houses with partial or complete deprivation of access to the outdoor environment. This outdoor environment is itself a coveted resource that, when limited, can lead to physical and behavioural disturbances. When monotonous, unchanging and unchallenging environments provide insufficient mental stimulation, animals show signs of boredom, often manifesting as abnormal behaviour patterns[10]. The absence of activity and environmental enrichment are major causes of stress. Furthermore, cats living in a restricted environment may not have enough physical space to allow an acceptable flight distance thereby reducing the cat's opportunity to retreat. This situation is exacerbated by an increased density of cats living in the same home. Group housing of cats is seen in both shelter situations and in personal homes. Usually these cats are not related, are neutered and cannot migrate[11]. Wolfle (2000) suggested that for some social species (such as some rodents, dogs and non-human primates) companionship is often considered the most important need to achieve well-being[12]. But, in its wild nature, cats are solitary hunters[13]. In general, the presence of other individuals in a cat's territory is not well tolerated, the cat uses displays of aggression to keep the others away[14]. The owner also promotes the creation of social relations incompatible with the behavioural nature of these animals, not ensuring, in most of the cases, conditions to address many of their basic instincts[14]. Cats organize their territory in a way that allows them to hunt, feed, rest and eliminate far from other cats[15]. The existence of protected isolation and elimination areas is crucial[13]. The space reduction affects each individual in a different way, according to their respective distance and isolation needs. The environment's balance can be disturbed by a poor distribution of required zones and resources (food, water, litter trays)[15]. The increase of interactions between cats can cause an increase in agonistic encounters and unwanted behaviours (as spraying, scratching and aggression)[16].

After identifying the major stressors that influence cat's welfare, one could think that owners are evil. But a possible reason is totally different: owners simply don't know their cat's behavioural needs. Da Graça Pereira and colleagues (2014) in their study showed that owners had a lack of information about their cat's behavioural needs[17]. But, worse than this, another conclusion from the same study was that, when compared with veterinarians in certain categories of cat's behavioural needs, veterinarians and owners were at the same knowledge level[17]. So, there is a long way to change the life-style of cats, as we need to change scholar curricula and also give educative/informative training for owners[17].

One of the major stressors identified in cats are its toilets. And there are many details that make the difference by the cat's perspective. Some of the characteristics of a good litter tray are presented in this abstract, and during the lecture the author will show some signs to identify if the cat is enjoying its toilet or not. The available litter tray should have a size large enough[15], that allows the cat to turn around inside, getting in and out without any problem. Usually is recommended to have at least one and a half the cat's size. The majority of cats prefer a thin (like sand) litter[18] and is recommended to have, at least, one litter tray per cat plus one extra or another possible rule is: "*a cleaned toilet for each cat, in enough number, giving the chance to all cats to use one, if they need it, at the same time*". The litter trays should be dispersed by the territory and not all concentrated in the same room. Each should be located in a place of easy access and quiet, if possible with two diferent accesses to avoid the feeling of closure[15], and far from noisy equipment such as washing machines. Cats usually prefer opened liter trays, with simple litter without any odour, but preference test can be performed. That is, giving the cat the option to choose what it prefers. Hygiene must be guaranteed according to the use of the litter tray, but the tray must be as clean as possible.

If we decrease the surrounding stress, improving quality of life and animal welfare[19] throught the implementation of therapeutic strategies that decrease the noradrenergic activation[20], surely we will decrease the risk and manifestation of pathologies (including behaviour and many others)[19].

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Noise reactivity: Diagnose and Treatment

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Introduction

Anxiety disorders, fear and phobias are among the most common behavioural problems of companion dogs. These problems include generalized anxiety, separation related problems and phobias of specific stimuli such as storms, fireworks, or other noises. In each of these disorders, affected dogs exist in a state of heightened arousal and distress. They may cause damage to their surroundings or themselves as an expression of their anxiety. As such, anxiety disorders represent an important welfare issue for affected dogs and may negatively impact the human-animal bond, becoming one of the major reasons for relinquishment of animals to shelters.

There are many possible causes for anxiety disorders and reactive behaviours to different stimuli. Among other causes, the following are the most common: a) insufficient socialization (including impossibility to express normal behaviour and/or unpredictable social interactions); b) Central Nervous System fear pathways unregulated; c) Traumatic event (including severe or frequent positive punishment); d) Genetic predisposition; e) Pain (or other medical issues) and f) Cognitive decline.

Signs of light fear (subtle and commonly not understood as fear by the owners) to severe uncontrollable fear (know as phobias) are common causes of referral to behavioural practices. The most commonly reported reactivity is to noise (also known as sound phobia), being fireworks and thunderstorms the most common triggers. Sometimes, separation anxiety in dogs, can have its origin in sound phobias.

Assessment and Diagnose

We can consider 2 different phases that each animal can arrive to our consultation. To make it easier, it will be considered phase 1 and phase 2.

Phase 1

The animals show signs of fear only when expose to a specific noise. During this event tries to hide. Usually, predicting the events, can engage in a copying strategy to control the behavioural expression of its emotional state.

Phase 2

Fear becomes more severe and the avoiding or predicting strategies fail. The sound is now experienced in a sensitising context that will lead to more serious complications. From these complications there are 2 that are very important. The first one is the excessive sensitivity to sounds, especially the unexpected ones. The other important complication is the generalisation that can occur in this phase. The generalisation can be related to individual sounds, but also to contexts and predictive cues associated to this trigger. These complications result in disruption of the daily life of the animal (and its owner).

It is really important to keep in mind that any phase 1 can progress to phase 2. Unexpected exposure, altering access to hiding places, impossibility to control the exposure, are some of the factors that can influence this progression.

Management of noise related behaviour problems

The aim of managing this behavioural problem is supporting the animal to develop coping strategies. To make good management it is required a not simply environmental management (guarantee always access to hiding places or refuges), but also alterations in human interaction. Owners should never use positive punishment or even force the animal to the face the sound that develops the fear signs. Instead, owners should act as a role model, as not bothered with the sounds and not worried. Ignoring the fearful animal is also something inadequate, but still being recommended. Giving support and asking for an alternative and incompatible behaviour (previous trained with positive reinforcement) will help the animal to move its emotional state to a positive one.

Treatment

Treatment is based in 3 strategies: behavioural modification, pharmacological intervention and pheromonetherapy.

Behavioural modification

Dessensitization and counter conditioning is the basis to treat this animals. However the knowledge of clients to apply an adequate plan is usually low. Thus, making sure the client understand exactly all the steps and have someone (trustable animal trainer or a behaviourist) to support the behaviroural modification plan. There are many limitations in the treatment, and every step must be well previously thought to guarantee no mistakes can happen.

Pharmacological intervention

Drugs are appropriate when there is generalization, complications, cognitive imparement, affected welfare and the inducing event may cause a relapse or worsening of the problem.

Never use acepromazine as it increases in the sound sensitivity and reduces the escape response, but also can cause disorientation and confusion, including potentially disinhibiting aggression. In the short term medication, benzodiazepines can be recommended (diazepam or alprazolam being the most suitable). There are advantages in its use: amnesic effect, anxiolytic properties and dose related sedation. However some animals can experience a paradoxic effect with an increase of excitability, disinhibition and impair learning. Transmucosal medetomidine (Sileo®) has been used with very good results around the world,and is a great option to use when owners can apply it correctly.

In long term medication, Selegiline or Sertraline can be used. Selegiline can be used when there are patterns of behaviour that are inhibited or avoidant. Seleigline is also useful in cases with profound generalization or high level of sensitivity. Sertraline is indicated also with high level of sensitivity and may be useful when the predominant feature is anxiety or significant panic elements.

Pheromonetherapy

Pheromones can play a long term in long term treatment approaches. Adaptil® or Feliway® can increase the appeasing qualities of the environment supporting the behavioural modification plan.

Conclusions

From welfare perspective and quality of life, these animals deserve appropriate management and/or treatment. When left without intervention, these conditions can get worsen.

Old cats: Behaviour problems and treatment

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INTRODUCTION

Repetitive behaviour can have various medical causes, but when the problem is not medical it can have several different aetiologies. The behaviour can be a normal part of the animal's behavioural repertoire, it can be environmentally induced (boredom, anxiety, among others) or it could be conditioned. Alternately, the problem might be medical (here the concept of medical, means organic disease, but behaviour which includes brain that is also an organ is also medical or organic. Therefore, the distinction between medical and behavioural helps us to differentiate between the two; but remember that we cannot split the animal into pieces taking off the brain and call behaviour problems as non-medical). For example, we can have a dermatology lesion (focal dermatologic pathology in the paw) that causes pruritus to the animal, which the owner then reinforces when trying to stop. Even when you start treating the dermatologic problem, you'll also likely need a behavioural approach for behavioural modification. But it can happen also the other way around. The cat could be very anxious and start overgrooming (the mechanism will be explained below) leading to development of skin pathology. Therefore, the approach to treat a repetitive behaviour can involve both "medical" and behavioural approaches. Garner (2006) defined repeated motor patterns that are uniform and where the focus appears to be on performance of the motor pattern itself, as stereotypies[1]. Stereotypy describes a repetitive, ritualised, out-of-context locomotor behaviour. A different definition is presented for a compulsive behaviour that results from a fixation on a goal and non-locomotor related. Compulsive behaviours can range from staring at an object to very complicated rituals involving a high level of cognition, that is displayed out of context, exaggerated or sustained. Hewson and Luescher (1996) defined compulsive behaviour as "*behaviours that are usually brought on by conflict, but that are subsequently shown outside of the original context. The behaviours might share a similar pathophysiology (e.g. changes in serotonin, dopamine and beta-endorphin systems)*".[2]

BIOLOGY OF THE CONDITION

Compulsive disorder (CD) arises as a result of a pathological process in the central nervous system. Little is known about the aetiology of obsessive-compulsive disorder (OCD) in humans and the evidence that this is the same disorders in dogs is circumstantial. Most evidence of pathophysiological mechanisms came from pharmacological studies. High dosage of dopaminergic drugs (such as amphetamine) induce stereotyped behaviour in animals, while dopamine antagonists (haloperidol) supress the stereotypical behaviour[3]. Some authors suggest that beta-endorphin antagonists may be at least temporarily effective in suppressing compulsive behaviour[4,5]. There is a positive response in human patients with OCD to treatment with serotonin re-uptake inhibitors (SSRIs), while serotonin receptors agonists can exacerbate symptoms of compulsion. Based on this human evidence, veterinary behavioural medicine has been strongly influenced by the comparison made between compulsive disorders in animals with OCD in man. Nevertheless, even though compulsive disorder in animals and OCD in humans have some similarities, regarding ritualisation and repetition of behaviour, human OCD involves thoughts, preoccupations and attempts at rationalisation. In humans these behaviours can involve cognitive or physical rituals, and there is a discussion among different authors if this condition does have or not have cognitive components that would be considered an obsession. But, to avoid further discussion, we'll consider that animals do not obsess, and that these are compulsive disorders (CD). As seen previously by the definition presented before, a CD predicts that the compulsive behaviour is first shown in a specific conflict situation that can be prolonged and/or repeated. This situation can be generalised to other contexts in which the animal has a high level of arousal. This can lead to cases where the animal presents the compulsive behaviour constantly except when eating, drinking or sleeping and may, in very extreme cases, interfere with survival behaviours. Differences in the presenting signs of CD will vary between individuals. The link to explain why CD develops in animals is related to the coping strategy that a specific animal uses during arousal or anticipation. In some animals, if the outcome from an arousal stimulus or anticipation is less rewarding, or never happens, then it will become a frustration. Frustration and fear are negative emotional experiences that involve anxiety but are essentially the same emotional state in animals. Generally, animals cope with arousal, anticipation and frustration and do not develop problems. Whenever an animal is in a situation of frustration or motivational conflict, it is normal to perform a conflict behaviour, but this behaviour is only shown in conflict situations. In contrast, in CD the conflict behaviour is shown in situations in which there is no outside stimulus inducing it. CD starts when the animal discovers that the repetition of a behaviour produces a reduction in arousal and frustration. This is the reason why usually the ritualised behaviour is one that the animal is already highly motivated to perform. It can correspond to a displacement or self-appeasing behaviour or escape behaviour, arising from species- or breed-specific behaviour, or a behaviour that was learned through previous intermittent reinforcement (for instance by the owners). Once ritualised, the compulsive behaviour is a powerful self-rewarding system. CD in cats are generally associated with the repetition or exaggeration of self-maintenance behaviours such as grooming, sucking or self-mutilation. In this presentation we will concentrate only in overgrooming behaviour that leads to alopecia.

RISK AND UNDERLYING FACTORS

1) Environment Factors

Cats are highly reliant and depend on their ability to control and use the resources in their environment. There is a strong need to hunt, feed and perform self-maintenance behaviours during each day, at specific time intervals. When a cat cannot control its environment (e.g. a social conflict with another cat, or separation related problems), the resources are not available at expected times, or there is a lack of stimuli, then stress, frustration or conflict may lead to the start of a CD. A disease that increases stress or irritability can also contribute to CD. But environmental factors are extremely important for every behavioural problem in cats, especially for CD. This disorder is frequently associated with the lack of ability to perform normal behaviours combined with social stress (secondary to competition and conflict). For instance, grooming can be exacerbated as a substitute for hunting or the territory maintenance.

2) Genetic susceptibility linked with breed

Pure-breed Oriental cats, especially Burmese and Siamese show higher rates of wool-sucking and self-mutilation, but recently created breeds (e.g. Bengal cats) also appear to be at risk. This fact cannot be simply related to genetic predisposition as these cats are often reared in "non-domestic" situation such as a cattery. A possible lack of socialisation and interaction with common domestic stimuli can develop fear and anxiety problems that in adulthood manifest in different ways, one of which could be CD. A lack of space and stimulation is also a risk factor in Bengal cats.

3) Physical stimuli
As mentioned in the introduction, a physical lesion (such as skin) or irritation (such as allergy or neuropathic pain), may trigger CD. Although not proven, it is assumed that the stress associated with a lesion or irritation can be a first trigger for an already susceptible cat.

4) Conditioning
Compulsive behaviours can be reinforced by owner attention or, at least, may condition normal conflict behaviours to a level that appears compulsive. If the compulsive behaviour is only present when the owner is around (or always that he is not around), then this is suggestive of a conditioned behaviour.

DIAGNOSIS

As a syndromic diagnosis, it is essential that other aetiological diagnoses be considered as a first essential step. So, a medical assessment is very relevant to rule out neurological, dermatological or other medical pathologies that produce similar signs or that can co-exist in the same animal with CD. A complete physical examination with a basic neurological examination is required. In addition, prior to use of drugs the medical investigation should include: complete blood cell count, blood chemistry profile and possibly urinalysis. Imaging and other investigations (such as thyroid profile, dermatological tests, allergy testing, a complete neurological examination) may be required depending on the specifics. A trichogram should also be performed. When alopecia is secondary to deliberate grooming, the hairs will feel sharp when compared to normal hair touch. The reason for that feeling is that the tips have been taken off and the microscopic examination on a trichogram will confirm the state of the hair tips. Another sign of psychogenic alopecia is that the hair loss is restricted to parts of the body that can be reached by the tongue. Differential diagnoses should include: parasitism (from fleas to notoedric mange), staphylococcal furunculosis, dermatophytosis, endocrine dysfunction and allergic skin pathology, as well as localised or referred pain. Feline idiopathic cystitis can also be a reason for an alopecia in the perineal area with hair loss in the groin or abdomen. Feline hyperaesthesia may be associated with an acute onset of self-biting and hair pulling but other signs can help to differentiate from compulsive over-grooming. Psychogenic alopecia should only be considered when all the other medical causes have been eliminated. However, we must keep in mind that stress can lead to both exacerbation of auto-immune disease or allergy or cause immune depression, which can lead to a cycle. For instance, licking can worsen many dermatological lesions, resulting in an itch-lick cycle, and licking can persist much longer even after the original dermatological cause has been removed.[9]

TREATMENT
The treatment consists of 3 main steps: a) Environmental enrichment – to change the animal's environment and social interactions, removing (if possible) triggers and giving more consistency; b) Behavioural modification – avoiding every type of interactive punishment and increasing exercise, play and cognitive activities (from training basic commands to cognitive play to fill a greater proportion of his time and energy budget); c) Drug therapy - in most cases.

1) Environmental Enrichment
Since stress and anxiety are inter-related with CD, and particularly to psychogenic alopecia, triggers should be identified (based in the history) and removed (when possible). If it is impossible to remove the trigger, then desensitisation and counterconditioning is required as behavioural modification. Improving the cat's living environment is vital. Increasing the range of activities available for the cat and maintaining cognitive stimulation with interesting toys available and provided on a rotational basis. Food-dispensing toys may satisfy a cat's

motivation to hunt. The relationship between cats from the same household, as well as the relationship with the owners, must be properly assessed, so that underlying psycho-social stress may be removed or at least alleviated. Nevertheless, an adequate interaction with owners is required, as well as opportunities for play.
Dr. Buffington presented an environmental enrichment program referred to as Multimodal Environmental Modification (MEMO) that was used as therapy for cats with idiopathic cystitis[7]. MEMO resulted in a significant decrease of clinical signs, as well as signs of anxiety and a tendency to reduce aggressive episodes. This program provides environmental enrichment tailored to minimize environmental stress that can lead to conditions as feline idiopathic cystitis[7]. So, if for treatment of psychogenic alopecia we need to decrease stress, then MEMO should be recommended. MEMO advocates evaluating and optimising four key resources that can generate stress and anxiety: food, water, litter trays and resting/hiding places. Pheromonatherapy has been suggested as another useful tool to decrease perception of threats and increase the perception of safety within the household.

2) Behavioural modification

Highly structured interactions (as command-response-reward) should be used to avoid casual interactions. Obedience sessions allow for consistent interaction and can be very stimulating for the cat (depending on the reward, motivation, intensity and frequency of each session). This should be provided regularly and consistently throughout the day. The owner should also play with toys and the training sessions can have some tricks, such as retrieving a ball, using clicker-training. Punishment must never be used. It is aversive, unpredictable, leads to more stress and anxiety, and can generate undesired associations. An alternative that could be used is response substitution. When the cat engages in the inappropriate behaviour a noise might be used to stop this behaviour, immediately followed by a command and the animal rewarded for obeying the command.

3) Drug therapy

Pharmacotherapy is indicated in cases of compulsive self-mutilation or severe self-grooming. A reason not to delay the use of pharmacological treatment is that the longer the behaviour has been going on, the less effective its use is[8]. **Fluoxetine** (0.5-1mg/kg PO q24h), **clomipramine** (0.25-0.5-1mg/kg PO q24h – dose increase depending on the result), **sertraline** (0,5mg/kg PO q24h, during 6 to 8 weeks and then could be increased to 1mg/kg PO q24h) and **paroxetine** (0,5mg/kg PO q24h, during 6 to 8 weeks and then could be increased to 1mg/kg PO q24h) have proven effective as supportive treatment to behavioural modification and environmental modification. Tricyclics other than clomipramine are highly unlikely to have sufficient efficacy in psychogenic alopecia, as they have a weaker effect on serotonin reuptake. As compulsive behaviour, when established, can be performed even without a high arousal or high states of anxiety, anxiolytic drugs alone are generally insufficient. Drug results should be assessed after 4 to 8 weeks. The owner should closely track the number and severity of self-grooming bouts (since the beginning). If there is no improvement, the drug therapy may be changed, the dose may be increased, or combined poly pharmacology be considered. A successful drug therapy will produce about 70% decrease of the compulsive behaviour. After a period of 6 to 8 weeks without further over-grooming, a drug discontinuation plan can start. Weaning of the drug should be very gradual, starting with 75% of the dose for 2 weeks, then 50% for 2 weeks and 25% of the dose for 2 more weeks. This dose can be maintained for an extra 3 weeks, every other day, before being completely discontinued. During the weaning plan, if the behaviour reappears, the dose should be increased to the previous level that was effective for more 2 to 4 weeks before restarting the weaning. Be cautious of a possible rebound effect, where the compulsive disorder may reappear worse than before, due a sudden or too fast discontinuation of the drug. Some animals will need the medication for life. This can be seen as a chronic disease. Owner compliance is vital to the success of the treatment, but the outcome of the treatment can be negatively affected by problem duration[9]. The earlier the treatment starts, the better the results.

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Who is guilty? Mind? Body? Or both?

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Many "abnormal" behaviours can have various medical causes, but when the problem is not medical it can have several different aetiologies. The behaviour can be a normal part of the animal's behavioural repertoire, it can be environmentally induced (boredom, anxiety, among others) or it could be conditioned. Alternately, the problem might be medical (here the concept of medical, means organic disease, but behaviour which includes brain that is also an organ is also medical or organic. Therefore the distinction between medical and behavioural helps us to differentiate between the two; but remember that we cannot split the animal into pieces taking off the brain and call behaviour problems as non-medical). For example, we can have a dermatology lesion (focal dermatologic pathology in the paw) that causes pruritus to the animal, which the owner then reinforces when trying to stop. Even when you start treating the dermatologic problem you'll also likely need a behavioural approach for behavioural modification. But it can also happen the other way round. The cat could be very anxious and start overgrooming (as grooming can help decrease stress and anxiety due dopamine and endorphine release) leading to development of skin pathology. Therefore, the approach to treat this animal with a repetitive behaviour pattern can involve both "medical" and behavioural approaches.

It is important to recognize the interplay between emotional pressure and physical symptoms. First publications in human medicine presented the evidence that psychological factors influence immune function[1]. These studies initiated 30 years ago demonstrated the role of psychological stressors and their influence on the effects of other diseases. It is now well established that emotional factors play a central role in several medical disorders in veterinary field. It is generally accepted that chronic stress develops an immune depression with subsequent vulnerability to infectious diseases. Prolonged exposure to any stressor can increase an animal's vulnerability to the processes of morbidity and mortality. It is currently recognized that chronic stress plays a negative role in the health status of an individual[2]. Constant noradrenergic activation due to inappropriate adrenocortical control seems to be closely linked with the evolution of chronic pathology. Thus, anxiety is not only crucial in a welfare context but also a question of good medicine. Though different studies present diverse definitions and manifestations of anxiety, all authors agree that anxiety does exist in non-human animals and some animals, especially cats, are very skilled at hiding their level of anxiety with visible signs that may be very subtle. Thus, physiological changes related to stress responses can be also lead to several diseases that have been associated with stressful environmental situations[3,4,5]. Specific medical conditions related to unresolved stress and anxiety are present in almost all veterinary specialties, such as:

- Neurology - feline orofacial pain syndrome[6]
- Endocrinology - obesity and diabetes[7]
- Dermatology – atopy[8], compulsive licking[9] and other dermatologic pathologies[10]
- Gastroenterology - chronic idiopathic gastric pathologies[11]
- Urology - feline idiopathic cystitis[2]

So, stress and anxiety can increase the risk of various diseases and exacerbate many medical disorders. Anxiety can be induced by chronic ambivalent situations and inappropriate conditions of life. Physiological changes related to a stress response are identified in a wide variety of physiological parameters, namely blood level parameters.

Leukocytosis occurs during fear and excitement as a result of adrenaline release. The release of endogenous glucocorticoids during stressful situations or the administration of glucocorticoid drugs can alter the distribution and use of leucocytes leading to neutrophilia, lymphopenia, eosinopenia and perhaps a monocytosis[12,13]. In humans, the cardiac system is particularly susceptible to stress and can manifest as an increase in blood pressure[14]. With chronic stress, mechanisms that normally regulate blood pressure give rise to not only high blood pressure but also hypertension[15].

So, to reach a final diagnosis, it is essential that other aetiological diagnoses be considered as a first essential step. A medical assessment is very relevant to rule out neurological (including pain!!), dermatological or other medical pathologies that produce similar signs or that can co-exist in the same animal with a behavioural problem. A complete physical examination with a basic neurological examination is required. In addition, prior to use of drugs the medical investigation should include: complete blood cell count, blood chemistry profile and possibly urinalysis. Imaging and other investigations (such as thyroid profile, dermatological tests, allergy testing, a complete neurological examination) may be required depending on the specific behavioural alteration.

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Why the Long Face? Sucking the Marrow from your Veterinary Career

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Background

Many have written extensively about strategies to mitigate stress, stave off anxiety and avoid depression. The veterinary profession has, in many ways, led the charge in regards to mental health issue awareness, and has dug deep into the underlying causes of the debilitating illness that manifests itself in so many of us statistically. [1]

What is lacking though is discussion and research on the enhancement of the average veterinarian's wellbeing. We are right to focus on the plight of those suffering with mental health problems, but is it not time, when we now have a much better understanding of the issue and have been so successful in raising awareness of cause, effect and treatment, that we spend some time and resources on helping those who are *not* ill, become even more effective, successful and fulfilled?

The Theory

It is true that we have many who are suffering with mental health issues. The evidence in the literature is compelling. [1] The evidence from informal discussions with colleagues after seminars and workshops or over a glass of red wine is even more so. The underlying causes and contributing factors have been well established and whilst the most significant contributing causal agent may be attributed differently by different authors and mental health advocates, what is beyond dispute is that we are a profession that is more susceptible to mental health disease. Having spent more than 20 years thinking about, talking about and researching the basis and possible solution, I am quite convinced that there is no easy fix. Part of the issue is societal. Our connected, constantly plugged-in lives lead to information overload and challenges finding true down-time. We are constantly exposed to the best parts of everyone else's lives through social media leading to a sense that we are all leading mundane and insignificant lives. We are bombarded with information and have less and less genuine down-time. We are always contactable and/or exposed to all the biggest and smallest news items. But this is no different to any other group of people other than the desperately isolated and poor (who by the way have been reported to have the highest levels of happiness!).

As veterinarians we are thought to be more sensitive than the average person, with a tendency to be introverts. We take failures to heart and are generally perfectionists. Our jobs often expose us to great hopelessness, trauma, stress and sadness. Our access to painless and highly effective methods are undoubtedly a large part of the reason we are over-represented in the suicide statistics.

We have also potentially become victims of our own success in recognising we have a problem early, and being early adopters of mechanisms that raise awareness and start discussing the potential solutions at most of our major continuing professional development sessions. Could we be influencing some of our profession to *expect* they will suffer at some stage from mental health issues? Is this a good thing or are we simply increasing their susceptibility? There is good research that says visualising your goals will make it more likely you achieve them. Does *expecting* to suffer mental health disease make you more likely to? Many of the veterinary students at our Australian Veterinary Teaching institutions lament the constant discussion of suicide. I have personally witnessed a number of well-meaning presenters portray some of their negative personal experiences in order to better prepare the audience for the "reality" of veterinary practice. But that is one person's reality and I would hate to think that our profession is as bad as many make out. Have we overstepped in our attempts to build their resilience?

We also need to remember that, whilst a minority do struggle and do suffer, the majority *do not*. Are we spending all our resources on a problem that may or may not have a viable solution leaving those who have much greater potential to gain enhanced fulfilment and success from their career by the wayside?

Feminisation of our profession is a huge challenge to the retention of the workforce. Starting a family is a fork in the road for veterinary mothers requiring major fundamental changes to their relationship with their careers. Their experiences prior to motherhood will be formative in their decision if, when, how and to what extent they re-establish their working life as a veterinarian. The extra challenges to working mothers pursuing fulfilment through their career are obvious. Much like the issues we face with mental health in society in general, these challenges are society-wide, but there are parts of being a veterinarian that make these challenges a little more significant for those trying to juggle the responsibilities of a career as a vet and their family responsibilities. For some, the poor remuneration, fuzzy shift finish times and intense nature of the work just don't add up to making it worth going back to employment as a vet.

Representative bodies such as the AVA in are constantly looking for ways to improve their relevance to the majority of veterinarians. "I see some of the things the AVA does, but rarely does it relate to me and what I am doing in my professional life". Finding greater fulfilment is clearly of benefit to everyone.

Is there a Solution?

So what can we do practically, professionally and personally to become better veterinarians, better friends, better workmates and better people? We have a really unique segment of the community whose attributes include a high level of practical and emotional intelligence, strong work ethic and unparalleled problem solving skills, and yet we appear to be more often struck by poor mental health than other professions. We spend a lot of time worrying that the busy intense nature of our working lives will eventually take its toll.

This article has been written to challenge our current thinking, and hopefully to free many of the readers minds enough to enable them to find their own personal solutions to these problems.

The first challenge is determining if in fact you are "happy".

Seligman defines "Authentic Happiness" as having 5 elements:[2]

Positive emotion
Engagement
Meaning

Accomplishment/ Achievement
Positive relationships

He goes further in his book *Flourishing*. “To Flourish: an individual must have all the “core features” below and three of the six “additional features””:

Core Features	**Additional Features**
Positive emotions Engagement Interest Meaning Purpose	Self-esteem Optimism Resilience Vitality Self-determination Positive relationships

Within our profession we have spent much time asking ourselves if we are anxious, depressed or suicidal. It is probably more appropriate that we start with are we happy?

Self-love, self-improvement or various reincarnations of this concept is now a burgeoning industry with every indication that social media opportunities will fuel an exponential growth over the coming years. Are we as veterinarians and support staff special enough to need anything different than what is on offer in the broader community?

Maybe, but we do know that we are a different enough subset of the general community to potentially look at some particular strategies that are more likely to resonate with our slightly unique personality types.

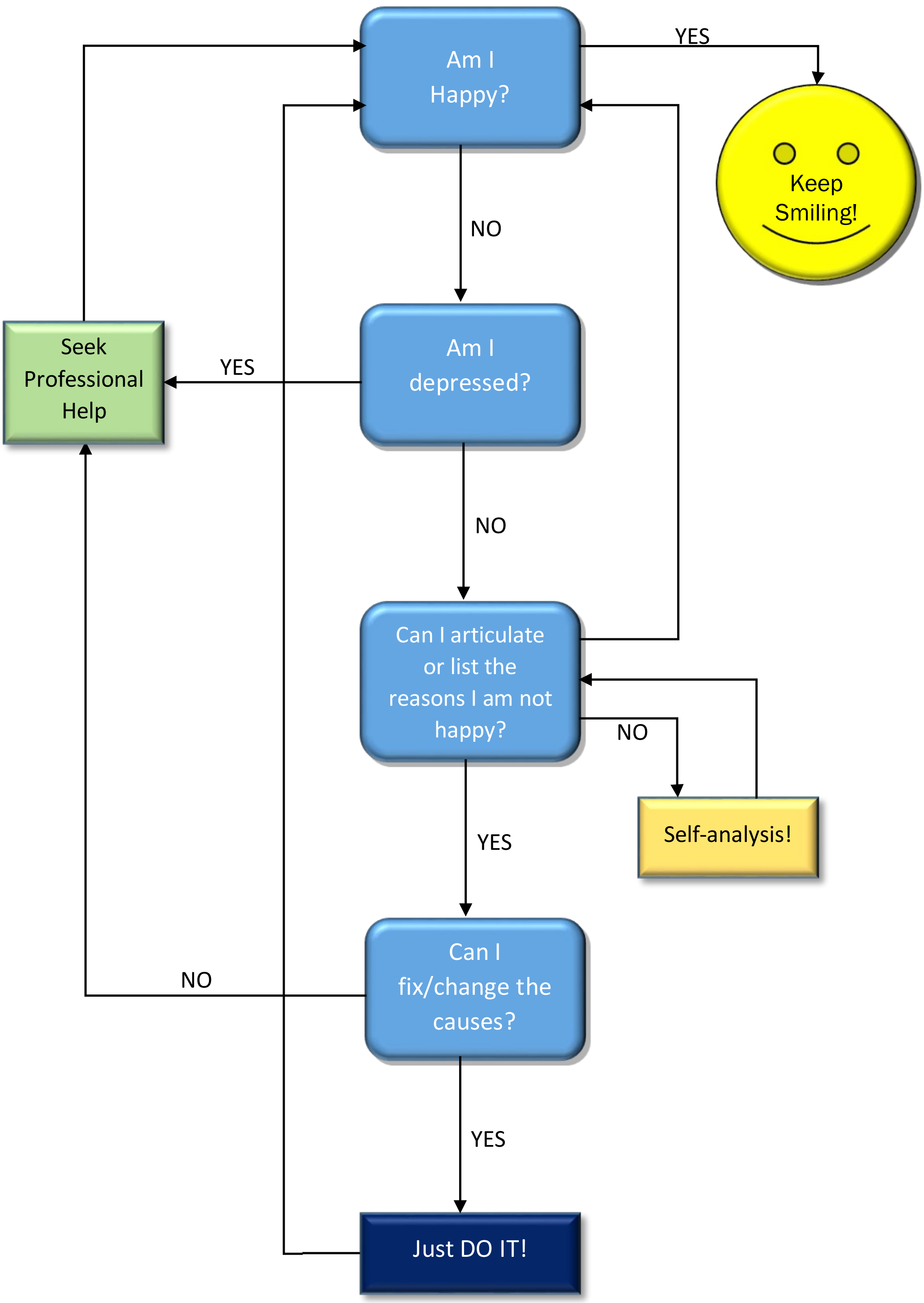
We have all been exposed to mindfulness, meditation, and other relaxation techniques. Most would have heard of cognitive therapy, positive psychology principles and brain elasticity exercises that aim to retrain our default mindset. That already places us ahead of most people who are not familiar with these concepts.

We are a highly trained, highly intelligent, driven and resourceful group of science-oriented professionals. That makes us different again. Importantly, it means we can self-diagnose and make scientifically sensible choices about our health and wellbeing.

We all understand life-balance as a concept, even if we don’t have it. We understand physical and mental health requires maintenance, even if we don’t embrace it. We are generally very sensitive to the needs of our clients, friends, relatives and workmates, even if we don’t act on what we perceive. We also have unprecedented access to tools and resources to educate ourselves further and commence therapy or treatment programs should we think we may benefit from them even if we don’t actually access these resources.

So what is the barrier to us translating all this knowledge and understanding of what we need into action? Here are some suggestions:

Happiness Flowchart



Inertia.

The attached flowchart may be very simplistic, but in the privileged resource and wealth-rich community we live in, many of our real problems are “first world problems”. That is, we are worrying and complaining about things that don’t actually matter in the overall scheme of things. That’s not to say that the problems don’t *feel* very real, or potentially lead to serious outcomes if left to unsolved. It is useful however to cast a critical eye over one’s problems on occasion and place them in proper perspective. If we decide that our problems are not actually that bad, we can probably prescribe ourselves some positive psychology exercises and achieve a higher plane of happiness very simplistically.

However, if we find through self-analysis that we have some very real problems, we need to identify the sources of our unhappiness. We are motivated, driven, and capable of managing incredible workloads and pursuing seemingly impossible academic goals. Why can’t we make the changes that would make us happier?

If we are living beyond our means, live in a smaller home, or sell the Mercedes and buy a Holden; or better still, a bicycle. If we are not getting enough sleep, go to bed earlier, stop drinking coffee and lose some weight. Not getting enough exercise? Get up earlier and set some fitness goals.

This again may seem simplistic, but essentially it is the crux of the problem, and its not just veterinarians. Western cultures are full of people on good incomes, with excellent housing, health care and lifestyles that are simply not “happy”.

It may be a sense of acceptance that this is as good as it gets, or fear of the unknown, but many of us seem unwilling to plan and implement changes to our lives even when we know that they will make things better for us. Of course there are always going to be reasons NOT to act, but we have to stop focussing on the negatives, and start thinking more positively about the future, or we will become our own self-fulfilling prophecy.

Career inertia

It is a highly challenging and expensive pathway to becoming a veterinarian with a very high opportunity attached to it. Many of us feel we are “veterinarians for life”. We are labelled by our degree as such, and many of us have grown up thinking we would *always* be veterinarians. It is incomprehensible that we would be anything else. Some of the unhappiest members of our profession are teetering on the brink of a change in career, but deeply and profoundly adversely affected by what they feel that will mean to their souls. They feel they are letting themselves and their families down, they have wasted so much of their formative and working lives to date and will be somewhat lesser if they pursue a different career. Some of the happier people I have met have been veterinarians that have leapt enthusiastically into this “abyss” and explored a different career pathway. The point is that for some, the grass is very much greener, and it is silly to let pre-conceived expectations of barriers prevent someone from pursuing a potentially happier working environment.

Is it time we called our degree something other than “Veterinary” so that we all have permission to explore the infinite careers that would still wholly or partly utilise our impressive academic credentials but not pigeon-hole us as one very specific type of scientist? At its core, our qualification is a wonderful blend of rich biological science and high level problem solving. It is eminently transferrable to a myriad of other industries. At the very least we should be celebrating and promoting those of us who have taken a different path, particularly to our younger members.

For those that are truly hoping to remain a veterinarian for life, what are you planning to do if you one day wake up and feel you don't like what you are doing anymore? Perhaps its time to think about that, and put some plans around how you reinvigorate your role. CPD is one great way of injecting yourself with a dose of extra enthusiasm, but at some stage you may need a complete reboot. The special interest options within our profession are extensive. Consumerism and deregulation are helping to enable those without Board-certification level credentials offer more specialised care to our patients, and our clients seem to care less and less about those credentials. The opportunities for veterinarians to explore more specific pursuits has never been greater though in some sense it seems we are less and less willing to go there.

There are ample opportunities around for continuing professional development in core topics of veterinary medicine, surgery and management. What there is less of is opportunities to "retool". That is to add non-veterinary skills to your CV. Of course nothing stops us from taking night classes or online courses in theory, but in practice there does not seem to be a lot of veterinarians that actually retrain to a point where they become employable in a different industry or profession. Radiography, physiotherapy, podiatry and occupational therapy for example would all lend themselves to veterinarians as relatively small steps. Do we feel it's too far a step backwards to take on these para-medical roles, or are we still stuck in the delusion that we "love animals" and don't relate well to people?

There is a much touted, but hard to verify statement bouncing around the internet from a Dell study that declares that "85% of the jobs that will exist in 2030 have not been invented yet" [4]

If this is even vaguely true, then we should all be keeping one eye on our chosen career path and the other firmly on any interesting tracks that lead off the beaten path. What exciting opportunities are we potentially missing through our inertia?

Some other concerning quotes relating to career changes include:
"Today's 15 year olds are predicted to have 5 career changes over the next 17 years of fulltime employment" and "91% of millenials expect to stay in their job less than 3 years". [5],[6]

Are you ready for a rapidly changing employment landscape?

Life Balance

Another well-established cause of unhappiness is a lack of balance in our lives. The intense nature of our interpersonal relationships with workmates and clients, as well as the emergency-room equivalent high pressure caseload takes its toll if we do not implement strategies to counteract those stressors. For some the imbalance sneaks up unannounced and we call that a "breakdown". For others it's slower and more insidious, and we believe this may be contributing to burnout and compassion fatigue. Regardless, amongst other self-care strategies, appropriate life-balance is well known to help stave off these potential causes of disengagement, absenteeism, "presenteeism" and lack of career fulfilment.

When we do choose to pursue something to help balance our lives, these attributes usually lead to us being highly successful. I know veterinarians who also have represented the state or Australia at the highest level in Hockey, Volleyball, equestrian, AFL, triathalon, rowing, freediving, yoga and of course cave-diving. We have all met high-achieving veterinarians whose arguably greater achievements lie outside their veterinary career. Then we look beyond athletic endeavour to business achievements and political interests and the message is clear. We can actually do anything if we set our minds to it.

Some barriers to commencing self-help strategies:

We know we should eat well, get plenty of sleep, exercise regularly and have a positive mindset. A few reasons we are less likely to achieve this would include:

1) Commencing a mindfulness or meditation program is to acknowledge that we need it, and/or are in some way not coping, or weak.
2) Dedicating time to something other than work, family or other structured commitments requires one to say “no” to something else or someone else. We are not good at saying “no”.
3) We are used to being a slave to the workload. We are very good at getting through a hectic schedule of cases, but we are actually not great at time-management when we need to sort our own personal schedule out.
4) We are getting confused between being busy and being stressed. We are embracing the concepts of working less and asking for more pay, but we are not necessarily working smarter or becoming more *productive.*
5) There are still some stigmas attached to getting coaching to become better. This is at odds with our perception of our academic development, where we are very willing to pay for further education should we perceive it will help us become better vets. Why the barrier to employing a coach to help us become healthier mentally, more resilient, more functional, more fulfilled?
6) Insurance companies have been watching us. They know we have an issue, and they have started increasing premiums and refusing to offer life insurance and income protection to veterinarians who have sought counselling or psychologist consults. This is a matter that needs urgent attention and action.

<u>Life Goals</u>

Our primary school challenge lasted 7 years (for those of my generation) and high school was a 5 year project. University was another 5 year Everest (for most of us) and then we hit employment. I have had 5 jobs over 25 years since graduating. Each one has been a roughly longer stint than the one prior. As a practice owner, I find that I must reinvent my role every 2-5 years in order to remain enthused and engaged. I don’t think it is easy to remain fulfilled by your employment if you simply do the same thing for extended periods, especially if you don’t have specific goals to work towards.

In business we know that we can’t manage what we don’t measure. We should undertake some sort of SWOT analysis (Strengths, Weaknesses, Opportunities, Threats) or mini strategic plan every few years. I don’t think our personal lives are that different in benefiting from some critical objective analysis and some projections and planning to map out the future. Ask yourself how happy you are, whether your goals are still relevant and appropriate, and if you need to change anything. If you need to change things, exactly what needs changing and how are you going to go about it. New goals should be SMART (Specific, Measurable, Achievable, Realistic and Timely). They do not have to be work-related, but they need to be meaningful, and challenging enough to get the adrenalin flowing. We all know vets who have tackled triathalons, marathons or other extreme feats of athletic endeavour. We probably don't hear as much about the new hobbies, the new leadership role in a local community group or the restoration of an old car, but they are no less valuable as personal goals.

Positivity

"A positive mindset" is the new catchcry of teachers at my daughter's primary school, and it comes from the Positive Psychology movement that has become the mainstay of a lot of corporate and academic psychology theory over the past 10 years or so. Cognitive Therapy is aimed at giving the patient exercises that swing default negative thoughts (or Autonomic Negative Thoughts - ANTs) around into a more positive and helpful attitude.

Much of Seligman's work is designed to help people to rediscover and maintain a positive outlook on life, without changing your job or fundamental life structure. There are many other authors, apps and resources that aim to achieve the same thing. In my experience, veterinarians are slow to embrace these principles, preferring the "hard sciences" discussions relating to neurotransmitters and cortisol levels. Perhaps this is one reason we are so quick to self-medicate rather than seek help from psychologists and counsellors? Regardless, the concepts are well recognised and now well established as being effective in improving the average person's ability to enjoy their lives. In putting this presentation together I have been reminded that it is probably time that I re-read some of Martin Seligman, Peter Singers and others works that I have in my library to reboot my mindset and reinforce my resilience.

I also seem to need reminding to try to surround myself with fun, positive things. There are some theories that suggest we age faster and become less happy when we stop "playing". Competitive sport is not really what they mean, though I challenge you to find a happier image than a bunch of team-mates celebrating a key sporting victory. Look for it and ask yourself what else makes a group of adults jump and fling their arms and heads around screaming and yelling with happiness? Of course there are corresponding scenes of despair from the opposition, but a positive mindset quickly forgets the losses and replays the wins.

Not often enough I *rediscover* what makes me tingle, feel alive, find my "flow" and its an exhilarating feeling. It seems I find it easy to forget what I really like to do. Weird, but we are so busy with things we *have* to do, I guess it makes some sense that we may forget to make time or devalue those things we really enjoy.

Scuba diving is mindfulness, meditation, physical exercise and cognitive therapy rolled into one for me. For some reason I keep forgetting how therapeutic music can also be. And I don't mean listening to Nova 93.7 in the car on the way to work. I mean deliberately creating a playlist and cranking the home stereo to allow yourself to actually *feel* the music and *hear* every instrument. If you don't get shivers down your spine at least once every third track, you need to rethink your playlist and or the quality of your sound-system.

Colouring-in, skiing, painting, knitting, cosplay, cooking…the list of possible hobbies and interests that may be your thing is endless. You are looking for things that make you forget everything else; activities that seem to make time stand still. Csikszentmihalyi calls this "flow" and I absolutely experience this when scuba diving and snorkelling. [3] Its just as well I wear a dive computer that alarms when it's time to surface because I literally lose track of time.

Note: I think the jury is out on any activity that employs screens. The immersive nature of computer games and their various analogues is obvious, and their value for developing/improving brain plasticity, hand-eye coordination and other positive mental and physical wellbeing attributes is well established, but I think we need to acknowledge that many of the games bring out the worst of those of us with addictive personalities, and we are all well familiar with the debates around the potential harm from excess screen exposure on eyes and posture as well as wifi and other radio-wave exposure. Call me a conservative, but its just not a natural state to be staring at a screen for too long. There may be some exceptions, but as a general rule I don't think screen-time actually achieves this state of mindfulness and "flow" that truly reboots, refuels and revitalises the brain.

The adverse effects of social media on our mindset are also well established as images of people's squeaky clean best moments fill our consciousness. In addition, the sheer volume of information is overwhelming, and fills our minds with extraneous noise, leaving less room for creative self-driven thought and mindfulness.

Summary

If we are looking for a short check-list that ensures we are maintaining ourselves in the best possible mental state, the below is a reasonable selection:

Undertake a happiness stock-take regularly
Look beyond your career for additional sources of fulfilment
Set life-goals and reset them regularly
Maintain a healthy life-balance
Look forward to and embrace change.
Surround yourself with things that make you feel good

Veterinarians are one of the luckiest groups of people in the world, blessed with intelligence, the highest level of academic education, infinite job opportunities and security and high public regard for what we do. We can, and should be rated as the *least* likely to suffer from mental health issues. Perhaps it's time to more thoroughly explore our potential as one of the most happy and fulfilled professions, instead of languishing at the bottom of the mental health leader-board.

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Antimicrobial Stewardship – What's the resistance?

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Introduction

Antimicrobial stewardship (AMS) has become a term symbolising the overall movement, including a contribution from the veterinary profession, towards minimising both the growth and effects of antimicrobial resistance (AMR). At its core for the veterinary profession, is the concept of the way in which today's veterinarians pass on the efficacy of the currently available antibiotics. This is both to the next generation of working veterinarians and the 'One health' concept of all medical professions and the global society. Are veterinarians considering their social responsibility of prescribing in relation to AMR? In this paper, we look at the practicalities of implementing an antimicrobial stewardship in practice and what needs to be considered.

Antimicrobial resistance (AMR)

At the very simplest level, AMR is the genetic trait which results in one bacterial phenotype having a survival advantage amongst its cohorts in an environment where the antibiotic is present. Examples of these traits include:

- enzyme detoxification like beta-lactamases,
- efflux pumps in bacterial cell walls to lower intracellular antibiotic concentrations or
- antibiotic target alterations

All of these render the chosen antibiotic treatment less effective.

When AMR is considered as a genetic trait then locally, regionally and globally it becomes an 'all-in or nothing' concept as globalisation and travelling make our world smaller. AMR creation and spread between individuals, species, regions and across country borders is in the simplest terms, just the transfer of genetic material. Everyone needs to be involved to limit the creation of AMR.

Coverage in the media of AMR results is divided between assigning blame and promoting action. It would be simple to point blame towards other veterinarians, other medical professions, the pharmaceutical companies or other countries. There is evidence within the literature that any growth in the consumption of antibiotics is associated with the development of AMR[1]. But in a similar manner to other social issues, such as global warming or single use plastics, AMR has grown its media profile and therefore its importance within the political and corporate environments. The outcome of all these discussions will never be black or white, but currently for AMR there is a transitioning to taking action.

Lastly, AMR consequences are often discussed at the human level, focusing on the financial impact or case outcome consequences. These overshadow an equally important consideration being the demand from agricultural systems to supply a sustainable protein source for a growing world population. A driver of AMR is the use of antibiotics within animal agriculture[2], but there is a growing corporate[3], government[4] and industry[5] policy shift that will focus and balance systems towards ensuring sustainable practices with antibiotic use.

A little theory ….

Although they work in partnership towards the same end goal, it is important to separate AMS from judicious use of antibiotics across a timeframe. AMS is the passing of antibiotics on to future generations in a state where they remain as effective in treating diseases as how they were inherited. AMS is the long term management of AMR and caring for our shared and finite antibiotic resources. Judicious use of antibiotics is a momentary decision on the manner to use antibiotics to deliver the best outcome. It is the clinical decision, repeated frequently each day by clinicians, that considers all the case variables, one being AMR.

Before beginning the practicalities of creating an AMS policy required for a practicing clinician, pharmacology provides some important but technical considerations.

- Prior to prescribing consider the minor variations in manufacturing that vary any dose calculations. Following prescription human error or poor compliance of the end-user result in variations of the dose received.
- During the dose, pharmacokinetics (PK), the way a body interacts with a medicine, (ADME – absorption, distribution, metabolism and excretion) and pharmacodynamics (PD), the way a medicine interacts with a body or pathogen, are different between individuals despite a one size fits all approach to pharmaceutical product labelling. At the moment, individual variations of dosing outside weight are theoretical in practice, but there are known PK and PD effects within the varying breeds of one species[6] and individually, dependent upon health status i.e. obesity, stage of life (paediatric, adult, geriatric), disease effects and in the case of antibiotics, host immunity.

After every dose of antibiotics, unintended consequences are beginning to be investigated and fully understood. Consider

- for oral antibiotic medications, what are the consequences of any interactions prior to absorption between the prescribed treatment and microbiota, the intestinal flora?
- Similarly, for oral antibiotic treatments the portion of a dose that is not bioavailable, not absorbed, or for both parenteral and oral treatments the portion excreted in its active form, what of the interactions between that portion of the dose and any commensal or environmental flora?
- Lastly the minute portion of antibiotics from a treatment that remains as a residue entering into the food chain. Considering the frequency of consumption in humans, are there effects and consequences to the microbiota of the consumer?

These variations, as with many aspects of veterinary science, results in AMS policy around dosing being considered above the individual.

Antimicrobial Stewardship (AMS)

There is no 'one size fits all' AMS policy for veterinary clinics. Individualities of region and veterinary business, in so far as the prescribing requirements, impact on the contents of any policy. Any document created should be fit for purpose and pro formas need to be adjusted accordingly. Individualities could include:

- Species – the influence of animal species treated and the intensity of any agricultural system on prescribing habits
- Demographics – within any animal type age (geriatric, paediatric or adult) influences prescribing requirements
- Regionally prevalent disease and the animal type treated

- The common preventative treatments available for the diseases and their use e.g. vaccines or teat sealants
- The development of uniform treatment plans
- Welfare or ethics considerations to any treatment plan

Using the above drivers that personalise any AMS policy, individual components of an AMR policy include:

1. Clinical guidelines
    - Thorough examples, supported by scientific understanding or literature, defining use against overuse
    - Definition of antibiotics considered as important for human use, as the recent AVA policy[7]. This needs to be applied when choosing first line antibiotics.
2. Infection control – the term for biosecurity
    - Hygiene between patients
    - Protective clothing between patients or farms
    - Equipment sterilisation
3. Clinical microbiology
    - Within treatment plans, defining the necessity for laboratory samples when AMR provides significant risk
4. Resistance and use surveillance
    - Collating use records to feedback and influence policy decisions
    - Using laboratory resistance results to benchmark AMR regionally
5. Pharmacokinetics and pharmacodynamics
    - Being aware of pharmacological advances and how this practically applies to clinical practice and prescribing decisions
6. Compliance
    - Checks and balances to ensure policy is being adhered to and to provide a basis for reviewing outcomes
7. Education
    - Share clinical outcomes from policy decisions or alterations
8. Regulations
    - Formal – driven by industry bodies, corporations or governments
    - Informal – driven by community or personal expectations
9. Leadership
    - Applying government and industry policy suitably within a clinic

AMS policy has clear goals as defined by the 5R's[2].

The 5 R's

- Responsibility – Good clinical decisions about when to use antibiotics
- Reduction – Minimize use with good preventative programs
- Refinement – Use antibiotics which are appropriate for the situation
- Replacement – Consider alternate treatments which may not require antibiotic use
- Review – Continuous improvement in how we use antibiotics

They should be visible where prescriptions occur. An AMS policy is actively practiced, becoming a document that is shared entirely or in part with colleagues, staff and clients. Antibiotics are an important but finite resource and we must care for them as such. Put simply, blaming others for the selection and propagation of AMR traits is not AMS. We are all in this together, after all AMR will not respect geographical borders.

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Ten Top Tips for Avian Surgery

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Introduction

Traditionally surgery is regarded as a difficult and dangerous procedure to perform on the avian patient. Much of this belief stems from the early days of avian medicine when, by comparison to what we do today, procedures and drugs were very basic. With the knowledge that we have now, derived from research, extrapolation and much hard-won experience, surgery on the avian patient is no longer the minefield it once was.

This paper describes ten tips for improving the surgeon's success when taking a bird to surgery.

Prepare your patient

Avian surgery is rarely an emergency – the best outcomes are achieved when the patient has been stabilised prior to the procedure.

The first step is to assess the patient. A thorough physical examination, paying particular attention to the bird's body condition and weight, hydration status, cardiorespiratory function and any abnormalities (e.g. ascites, anaemia), can be supplemented with laboratory diagnostics (especially PCV, total plasma solids, and blood glucose) and imaging (radiology and ultrasound). When all this information is collated and interpreted, the next step is to stabilise the patient.

Stabilisation includes correcting the patient's body temperature, hydration status and nutritional status. A well-hydrated patient that is eating voluntarily is a much better surgical candidate. Adequate analgesia and antimicrobial therapy are also useful adjuncts where appropriate. If the patient is anaemic, consideration should be given whether to delay the surgery until the bone marrow has replaced the deficit or prepare for a blood transfusion.

Plan the procedure

If you have not performed this surgery recently, revise the relevant anatomy and physiology. This information can be found in most avian textbooks[1,2]. Plan your surgical approach (what structures will you encounter, what changes in physiology can you expect) and, sometimes more importantly, plan your exit – will you be able to close the incision with minimal tension?

Expect the unexpected. Things will go wrong – unexpected haemorrhage, respiratory depression, a disease state worse than you had planned for, etc. Have a contingency plan in your mind for dealing with complications and discuss them with your team before you start.

Remember that surgery is a team sport. Other players include an assistant surgeon, your anaesthesia team, and your nurses (often the same people). Everyone must know what you are planning to do, and what their role is. Planning discussions and 'walk throughs' can ensure that minimal time is wasted once the patient is on the table.

Have the right equipment available. Good surgical instrumentation (ophthalmic instruments are great for small patients), vascular clips or appropriate suture materials, and plastic or paper drapes can make a dramatic difference during surgery. Similarly, the use of optical loupes and good lighting can make operating on a small patient much less stressful on the surgeon.

Develop a sound analgesic and anaesthetic plan

In the early days of avian surgery, the maxim was to always keep your surgical procedures to less than 20 minutes. Patient morbidity and mortality rates escalated once this time frame was exceeded. Previously, avian analgesia and anaesthesia were emerging fields, and patient support was limited to keeping the bird as warm as possible without 'drying it out'. The only way to minimise the adverse effects of anaesthesia and surgery was to keep the procedures as short as possible. With improvements in analgesia, anaesthesia and patient support, surgical times can be pushed out well past 20 minutes and procedures taking 1-2 hours are now common.

We now know that pain has a direct physiological effect on wound healing and that recovery rates for animals receiving effective analgesia are faster than those whose pain is not managed well[2]. The use of multimodal analgesia (including opioids, NSAIDs and local nerve blocks) effectively manages pain while simultaneously reducing the doses of anaesthetic drugs required to maintain a surgical plane of anaesthesia.

The rapid metabolic rate of birds dictates an early return to normal appetite. Anaesthesia drugs should therefore have minimal hangover effects, allowing the bird to return to normal function (including appetite) as soon as reasonably possible. Inhalant gases, such as isoflurane or sevoflurane, meet this requirement well. The use of effective pre-medication, including an opioid and a benzodiazepine[2], reduces the MAC of anaesthetic gases, minimising their cardiorespiratory effects, and allow a smoother induction and recovery. The only issue with using inhalant gases is the loss of respiratory tract integrity when the coelom is opened. This allows room air to be inspired, occasionally lightening the plane of anaesthesia. This needs to be monitored by the anaesthetist and may require higher oxygen flow rates and anaesthetic concentration to overcome the effect.

The greatest improvement in anaesthesia safety is the level of patient support and monitoring that can now be employed. This allows lengthier procedures with minimal additional risk to patient safety, and results in better outcomes.

Parameters	Support	Monitor
Body temperature	Radiant heat Hot air warming	Cloacal temperature
Respiratory function	IPPV – mechanical or manual	Manual observation Capnography Pulse oximetry
Cardiovascular function	IV fluids Drugs for cardiovascular support	Stethoscope Doppler ECG Pulse rate and amplitude

Remember Halsted's Principles of Surgery

- **Strict aseptic technique**
  In avian surgery there is often a compromise between asepsis and retaining feather insulation. Avoiding hypothermia during and after surgery is an important consideration when prepping a patient for surgery. Skin preparation should include the following:
  - Pluck feathers rather than cut them. This encourages immediate regrowth
  - Pluck along the line of growth to avoid tearing the skin
  - Only pluck what you need for surgical access. You can use adhesive tape to hold others out of the surgical field
  - Avoid using alcohol for the skin disinfection –alcohol causes evaporative cooling

  Drapes should be used to not only minimise contamination of the surgery site but also to retain the patient's body heat. Plastic or paper drapes may be used, but care should be taken to avoid heavy cloth drapes in small patients, as their weight may compromise respiration.

- **Gentle handling of tissue**
  Gentle handling of tissues will help minimise post-surgical pain and wound self-trauma. The use of fine surgical instruments and minimal cutting and dissection of tissues will help, as will the prevention of desiccation of exposed tissue (especially under a radiant heat source).

- **Meticulous haemostasis**
  The blood volume of most birds is 10% of their bodyweight (comparatively higher than mammals), although non-flighted birds may have a lower blood volume (perhaps reflecting their lower oxygen requirements). Birds are also able to withstand comparatively greater blood loss than mammals. This is thought to be the result of:
  - An increased capillary surface area within skeletal muscle allowing for rapid extravascular fluid resorption to maintain vascular volume.
  - The ability to mobilize large numbers of immature erythrocytes.
  - The absence of the autonomic response to haemorrhage that contributes to haemorrhagic shock.

  Birds, unlike mammals, rely on an extrinsic clotting pathway rather than the intrinsic – in particular they rely on the presence of tissue thromboplastin released by tissue damage. For this reason many surgeons like to clamp the skin with haemostats before incising it.

  Effective haemostasis allows visualisation of the surgical field while preserving the total blood volume. Haemostasis is achieved by ligation, pressure, electrocautery, and avoiding damage to major vessels. Only the vessel to be occluded should be incorporated in a ligature or clip. Electrocautery can be used for minor haemorrhage; large vessels should be ligated.

- **Preservation of blood supply**
  Wherever possible surgery and haemostasis should result in minimal compromise of the circulation in the surgical area. Large blood vessels should be avoided where possible, and only vessels that need to be transected should be ligated or cauterised.

- **Accurate tissue apposition**

Accurate tissue apposition enhances healing and promotes rapid return to normal function. Tissues are replaced to their normal anatomic positions with minimal amount of tension by sutures to prevent tissue devitalisation. The type of suture material and pattern to be used will be dictated by the tissue involved and forces applied to those tissues.

- **Obliterate dead space**
  "Dead space" refers to the soft tissue and bony defects left behind after a surgical wound is closed, such as after the removal of a space-occupying mass or evacuation of fluid. Appropriate management of this space is necessary to reduce the risk of persistent infection and delayed wound healing. In mammals this is often achieved through the use of drains until the dead space fills with fibrin. In birds, however, the use of drains is complicated by the lack of lysozymes in heterophils, meaning that avian pus is usually caseated and therefore does not drain readily. The use of drains in these wounds may not be of benefit unless combined with a flushing effect.

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  The inflammatory response is less marked in birds than in mammals and so sutures can be tied tighter. However, care must be taken to prevent sutures tearing through the thin skin. The use of monofilament absorbable sutures such as Polydioxanone (PDS) is often appropriate.

  Incisions that cannot be closed primarily, or contaminated wounds, may be left to heal by secondary intention. Care should be taken in this situation to keep the wound clean and avoid desiccation of the exposed muscle or subcutis by use of wet dressings or hydrocolloid dressings such as Duoderm®.

Conclusion

Avian surgery should not be regarded as something to be avoided. By understanding the patient's anatomy and physiology and then following the principles of surgery as discussed in this paper, surgical outcomes can be improved.

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Anecdotal, long accepted as a fact

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VETTRAIN 3 - - the latest phase of a landmark training program

Ian H. Douglas BVM&S, MACVS
VETTRAIN Coordinator
Vets Beyond Borders

Veterinary training institutions and animal welfare projects in the Asia-Pacific region and many other parts of the world are not uncommonly impacted by a dearth of staff with advanced teaching and clinical skills. A likely contributing factor is a historical tendency in some countries for veterinary graduates to pursue careers in administration and research, rather than the clinical disciplines.

However, the veterinary clinician has a major role to play in efforts to address global challenges to the health and welfare of domestic pets, livestock, wildlife, humans and the environment. *VETTRAIN 3* has been devised by Australian-based charity, Vets Beyond Borders (VBB), to demonstrate the demand for, and benefits of, clearly focused clinical training in refining the skills of veterinary personnel in parts of the world where continuing education in clinical disciplines is not readily available.

VBB's involvement in the delivery of clinical training had its beginnings in 2006, in the Himalayan state of Sikkim in northeast India, shortly after commencement of the Sikkim Anti-Rabies & Animal Health (SARAH) Program. The SARAH Program is a collaboration between VBB and the Government of Sikkim and was the first statewide animal birth control and anti-rabies project in India. In response to the requests of veterinary staff working with the SARAH Program, a training course was devised and delivered by VBB volunteers, covering aspects of small animal anaesthesia and surgery of relevance to animal birth control/anti-rabies (ABC/AR) work.

As a result of the success of this initiative, in 2008, VBB received a request from the Government of Bhutan to provide training to government veterinarians and paraveterinarians, in preparation for commencement of their nationwide dog population management and rabies control project. A team of VBB volunteers, including SARAH Program staff, travelled to the regional centre of Paro to deliver a sixteen-day long ABC/AR course, based on that delivered in Sikkim.

Soon after, VBB was approached by the Animal Welfare Board of India to develop a "train the trainers" program, with the aim of refining and standardising the skills of staff working with ABC/AR projects across India. *VETTRAIN* was devised in response to this request. Its pilot phase, held at the National Institute of Animal welfare in Haryana, was a collaboration between VBB, the Animal Welfare Board of India and the Ministry of Environment & Forests (Government of India).

Between February and June 2009, six, three-week long, training sessions were delivered. Each session involved three simultaneous streams (for veterinarians, paraveterinarians and humane animal handlers), delivered by a visiting faculty of veterinarians, veterinary nurses and humane animal handling trainers appointed by VBB. In addition to provision of clinical training, faculty members were charged with refining and developing the curricula and training materials previously compiled by VBB.

A subsequent collaboration in 2011 and 2012, involving VBB, the Animal Welfare Board of India, the University of Queensland, Humane Society International and AusAID, increased access to *VETTRAIN* within India, with multiple courses held at selected regional training centres.

Since its inception, hundreds of participants have completed *VETTRAIN* ABC/AR courses in India and other countries in the region and has resulted in measurable improvements to the clinical standards and effectiveness of ABC/AR initiatives. As a result of its success, the *VETTRAIN* model has been adopted by other organisations active in India.

Continuing its emphasis on delivery of free clinical training, in 2012, VBB released its ABC/AR clinical handbook, "The Essentials of ABC Surgery". This was followed in 2015 by completion of "The Art and Practice of Humane Animal Handling", a comprehensive manual and video training package, produced at the request of the Animal Welfare Board of India, for use throughout India, to address concerns regarding the quality and consistency of capture and handling of street dogs. This much-needed resource was produced in collaboration with respected animal handling trainer and veterinarian, Dr Mark Johnson of Global Wildlife Resources, USA.

In 2018, VBB decided to broaden the scope of *VETTRAIN* and seek to offer training courses on a variety of clinical topics. A further development was that, in addition to supporting clinical staff working with animal welfare organisations, under the new phase of the program, *VETTRAIN 3*, clinical training would be made available to veterinary associations in countries where continuing education opportunities are few, and also to the significant number of veterinary colleges, in many parts of the world, which have difficulty sourcing and/or retaining teaching staff, leading to deficiencies in undergraduate and postgraduate education.

To this end, with the support of the Australian and New Zealand College of Veterinary Scientists, the British College of Veterinary Specialists, the American Board of Veterinary Specialities, the European Board of Veterinary Specialisation, the Australian Veterinary Association and the Commonwealth.

Veterinary Association, VBB has created of a pool of volunteer teaching staff: veterinary specialists and veterinarians with other relevant postgraduate qualifications and/or teaching experience, willing to travel to animal welfare projects run by VBB Partner Organisations or centres of veterinary education, to deliver *VETTRAIN* courses. Veterinary nurses with advanced certification and teaching experience are also encouraged to join the ever-growing community of registered VBB instructors.

The list of Partner Organisations is constantly increasing, providing VBB- registered instructors with volunteering options in several countries. All Partner Organisations have undergone rigorous appraisal in terms of facilities, clinical protocols, capacity to host volunteers and volunteer safety.

VETTRAIN 3 offers instruction in a range of small animal and large/production animal subjects, including anaesthesiology, surgery, internal medicine, dermatology, neurology, diagnostic imaging, emergency/critical care, epidemiology and veterinary nursing. Since late 2018, courses have been delivered in Cambodia and India. Topics have included small animal orthopedics, fundamentals of clinical neurology and surgical analgesia. Courses are of variable duration but typically five days to three weeks, dependent on local logistics and the availability of VBB volunteer instructors.

VBB is also promoting the Veterinary Education Twinning Programme, recently established by the World Organisation for Animal Health (OIE) and views *VETTRAIN 3* as a highly complementary initiative.

VBB has always placed a strong emphasis on evaluating the effectiveness of *VETTRAIN*. This entails use of pre- and post-training test papers, clinical competency assessment and distribution of questionnaires for completion by participants, teaching staff and employers of participants. This process is continually reviewed and refined.

As demand for *VETTRAIN* continues to grow, VBB welcomes expressions of interest from veterinarians and veterinary nurses with appropriate credentials and/or teaching experience, to assist in this much-needed program, in the knowledge that their generous donation of skill and time will be greatly appreciated and that VBB will do its upmost to ensure a rewarding *VETTRAIN* experience - for all involved.

Reproductive disease: a problem of the chicken or the egg?

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Introduction

Reproductive disease is a broad, though common, reason for a pet bird, typically a chicken or species of parrot, to present to a veterinary clinic for assessment and treatment. A clinicians' confidence in their ability to accurately differentiate, for example, between the egg-bound individual and the case of coelomitis of reproductive origin, is something that will immediately improve the likely outcome for avian patients. The challenge arises in that the clinical signs associated with the various disease ailments of reproductive origin in birds are very similar.

This paper briefly documents some of the common disease processes in female bird species. Any predisposing factors, including species traits, have been noted. The main aim is to provide clinicians with knowledge they can utilise within their initial consultation, when reproductive pathology is a potential contributor for a patients' presentation. The classical distraction points from physical examination findings will be identified, to lend clinicians the upper hand against potential red herrings and mis-diagnoses. Diagnostic modalities that can assist in narrowing the window of differentials, will be briefly explored. Therapies that can be invoked for these cases will be discussed. Ultimately, the presentation aims to add to the knowledge, skill and confidence of clinicians, when faced with cases of reproductive disease in birds, resulting in improved patient outcomes and development of strong veterinarian-client bonds.

Anatomy and Physiology Refresher

For the majority of bird species, the female bird has a single left sided ovary and oviduct. The ovary lies caudal to the lung and cranial to the kidney, and has a very complex vascular arrangement, supplying and draining the organ. Once a bird is mature, and able to start ovulating, the oocytes develop into follicles. There are multiple developing follicles on an active ovary at any one time; typically four or five undergoing growth and readying for ovulation[1]. The primary, or F1 follicle[2], ovulates a secondary oocyte into the oviduct[1].

The oviduct is made up of five histologically distinct segments; infundibulum, magnum, isthmus, uterus and vagina. The infundibulum receives the secondary oocyte, and is surrounded by air sac to reduce the chance of ectopic ovulation[1]. The passage of the ovum through the infundibulum is quick, requiring fertilization to occur here, before moving in to the magnum where albumen is deposited over a few hours. In the isthmus, the shell membranes are formed, and calcium deposition begins. The developing egg spends the majority of time within the uterus (or shell gland) undergoing calcification[3]. The utero-vaginal sphincter separates the uterus from the vagina. Prostaglandin E, along with Prostaglandin F2α and vasotocin contribute to utero-vaginal sphincter relaxation and uterine contraction respectively, allowing oviposition (lay) to occur[4].

Laying intervals vary between species, with the chicken being known to produce an egg daily, parrots producing one approximately every 48hours[3] and the kiwi producing an egg every 44 days[1]. The number of eggs produced in a clutch is variable, with some birds being determinant layers having a fixed clutch size (eg, budgies) and others that will continually replace lost eggs, which are indeterminant layers (eg, chickens and cockatiels)[1]. Lay is initiated by a range of endogenous and exogenous factors, including increasing day length, other environmental cues (eg, rainfall) and the presence of a mate and/or nesting material.

Hormones such as gonadotrophin releasing hormone (GnRH), luteinizing hormone (LH), progesterones, corticosterone, androgens, oestrogens, progestins all have some role in the onset of lay and the process of ovulation[1,5,6]. At the end of lay, the prostaglandin ratios change, calcium levels become limited and negative feedback loops result in down regulation of the laying process. Complete regression of the reproductive tract, and end of lay, is typically signified by a molt. Incubation of eggs has yet another set of triggers, though is beyond the scope of this presentation. Given the complicated physiological parameters contributing to the production of an egg, it is no wonder there are a multitude of potential disease conditions that are unique to female birds.

A summary of some common reproductive diseases

Chronic egg laying and "egg-binding"

Chronic egg laying is a problem commonly occurring in cockatiels, lovebirds and finches[5]. Eggs are continually produced, often without the presence of a natural mate or normal breeding season[4,5]. In the extreme circumstance, a state of metabolic exhaustion ensues and blood ionized calcium levels will be critically reduced[3]. The depleted calcium levels result in reduced muscular contraction of the oviduct and compromise to the normal peristaltic activity required to enable lay. Pathological fractures can also be observed[4,7].

The term "egg binding", refers to the failure of an egg to pass through the oviduct in the normal time frame[3,6]. Thus, it is not simply that the egg is static somewhere within the oviduct, but may be progressing through the tract at a slowed pace[5]. This differs from a true dystocia, which is the mechanical obstruction of the passage of an egg to oviposition, typically within the caudal uterus or cloaca (for example, a malpositioned egg)[2,5]. The multi-factorial nature of the risk factors for both terms results in them being interchangeably utilised, though they have the potential for independence.

Egg binding events are very unlikely to occur in chickens bred for lay and are overrepresented in a variety of small parrots[6,7]. This is likely due to the size of these animals, especially when considering the inverse relationship between the size of the egg and the size of the bird. Other maternal predisposing factors include[3,4]:

- obesity;
- loss of muscle tone due to lack of exercise;
- the presence of a coelomic space occupying mass;
- oviductal damage;
- immaturity;
- nutritional deficiencies;
- hereditary factors;
- temperature fluctuations;
- systemic illness; and/or
- traumatic pelvic injury, being acute or chronic.

A bird with a version of egg binding will typically present to the clinic in a compromised state and require immediate stabilisation.

Ovarian and oviductal cysts

Cystic structures in birds can occur in the form or a persistent right oviduct, within the left oviduct, or be present on the ovary itself. Ovarian cysts are common in cockatiels, budgies, canaries, pheasants and waterfowl[5,6]. Birds with cystic disease, will typically have ceased egg production, though remain exhibiting reproductive behaviours (eg. nesting, regurgitating, masturbating)[5]. The size and location of the cystic structure will determine the resultant clinical signs.

Oophoritis, Salpingitis and Metritis

An inflammatory condition, of any type, of the ovary is termed oophoritis, and of the oviduct, is termed salpingitis. If this condition can be further narrowed to the uterus (shell gland), it is termed metritis. It is commonly of a bacterial aetiology[5], and can be the result of ascending infection, haematogenous spread, egg yolk peritonitis or an obstructive disorder (eg. prolapse, dystocia, oviduct impaction)[3]. An early indicator of metritis can be abnormal shell formation[3]. In severe cases, the affected reproductive tract can rupture, resulting in a septic coelomitis.

Oviductal impaction and rupture

A birds oviduct can become impacted due to an egg binding or dystocia event, metritis, or the presence of cystic hyperplasia[2,5]. Oviductal impaction will often present quite vague clinical signs, though should be ruled out in the case of broody behaviour in the absence of lay. Oviductal rupture is most common secondary to a dystocia event[5]. Should an oviductal rupture occur, bacterial coelomitis and septicaemia can ensue.

Egg yolk coelomitis

With presentation of an unwell, female, mature chicken to the clinic, egg yolk coelomitis is high on the differential diagnoses list. Egg yolk coelomitis is one of the most common clinical presentations for a pet chicken in practice[6]. This disease process is very common in layer breeds, purposefully bred for high egg production. The highest risk period for this occurring is at the start and end of lay[1]. Essentially, ovulation occurs into the coelom, rather than the infundibulum. It has been postulated to occur in response to a stressful event, as a result of reverse peristalsis, oviductal rupture, or secondary to oviductal disease preventing normal passage of the developing egg through the oviduct[4].

At times, ectopic yolks can occur and resorb[5], without appreciable change or incidence to the bird[2]. Birds with incidental ectopic yolks are at risk of yolk emboli formation during the resorption phase, and sudden death. The cases that present to a clinic, are usually those that are non-resorbing. The presence of multiple ectopic yolks does cause a significant inflammatory reaction, and is typically sterile[4]. In the case of oviductal disease being responsible for the ectopic yolk/egg, a septic coelomitis may be present.

Prolapse

Reproductive tract prolapse is rare in chickens, and more commonly seen in parrots. It can be secondary to all the reproductive diseases aforementioned, along with any disease process that causes straining or increased coelomic pressure, such as gastrointestinal diseases, cloacal papillomas, or coelomic space occupying masses[2,3]. The prolapsed tissue can include the egg itself.

Oviductal and ovarian neoplasia

Chickens are highly represented[3,6] when it comes to neoplasia of the reproductive tract, most commonly being afflicted by ovarian adenocarcinoma. Uterine adenocarcinoma in chickens are typically highly malignant and metastasise to the lungs and other organs haematogenously, whereas ovarian adenocarcinomas develop more slowly, and undergo transcoelomic implantation on serosal surfaces[6]. Budgies are another species prone to ovarian cancers. These birds are typically older females[7], and may present with a colour change to the cere[2].

Practical Consultation and Workup

Signalment and history

The patients signalment is one of the key points to pay attention to when faced with a sick female bird. For example, a chicken is unlikely to present for a true dystocia secondary to egg over production[6]. A cockatiel, however, is highly likely to have this problem. Age and breed is similarly important; Isa Brown chickens, having been bred for their high productive output, are much more likely to be presenting with reproductive disease from 18months of age or older.

When obtaining a history, the process is not dissimilar to that of a mammalian consultation. Ascertain information about the birds' demeanour and behaviour both in the immediate and longer term history. Owners perceptions of these often gives useful insight into the possible disease processes. Assess if the diet is adequately balanced[5]. Birds fed a seed only diet can be assumed to be deficient in essential nutrients, thus predisposing to some of these conditions. Ensure to specifically ask about laying and reproductive behaviours[3] in every consult with a female bird. In the case of birds whose sex is unknown or even sometimes with birds presenting as males, such as, an aged budgie with a blue cere, it is prudent to still query egg production; this may be the female budgie with ovarian neoplasia.

Clinical presentation

The challenge for birds presenting to a veterinary clinic often lies in the vague clinical signs, common to a range of disease processes; reproductive diseases are no exception. Most birds will have a classical sick bird look (SBL); be fluffed, reluctant to move, and closing its eyes. These are all very non-specific signs, but do alert you to a very unwell individual, as it is now unable to fill its innate evolutionary response of hiding illness.

Birds presenting in such a state, will likely be dehydrated, evidenced by mucous strings in the oropharynx and increased skin turgor and dryness over the spine or keel. For the most part, an anaemia of chronic disease is observed as pale mucous membranes in the case of parrots, or pale combs and wattles in chickens. Body condition is likely to be poor in a majority of these individuals, as reproductive disease may present as a chronic condition. An exception for poor body condition would be the acutely egg-bound parrot, who is experiencing dystocia due to a conformational inadequacy (obesity, pelvic damage, hereditary condition, etc).

Often, clinical signs are vague when reproductive disease is present. Specific clinical signs common of reproductive disease include a wide based stance, waddling walk, coelomic distension, tail bobbing and dyspnoea[3,6]. Birds may occasionally exhibit straining behaviour, and be producing droppings of variable consistencies. In the case of parrots with coelomic distension, an egg may be directly palpable. Such cases may also present with a limb paresis or paralysis, as a result of the egg causing secondary nerve impingement[4,5]. This differs from chickens, in which case, coelomic distension will be more likely due to fluid accumulation, inspissated yolk, and/or caseous change.

Physical examination distractors

There are many ways in which clinicians can be misled during an avian consult. Inappropriate emphasis on signalment is one of the first traps; for example, a chicken that is unwell must be "egg bound", or the fluffed cockatiel must have psittacosis because it's a parrot. Not completing the physical examination is the second trap. In the authors' experience, there are two main distraction points encountered during the physical examination of a bird with reproductive disease: body weight and crop distension or stasis.

As already mentioned, the body condition of these birds is likely to be low due to the chronicity of the disease. However, body weight can mislead this finding, if a thorough examination of the birds' muscle mass is not conducted. Fluid, cystic structures, eggs, or a space occupying mass, within the coelomic cavity can artificially elevate body weight, and give a false impression of health. Palpate the pectorals, examine the musculature around the spine and the legs. The pectorals, especially in flighted birds and meat producing poultry breeds, should be highly developed muscles. If you can feel every bony prominence, the musculature is profoundly depleted, and the birds body condition is poor, regardless of the measurement on the scales.

Crop distension and stasis is a common finding, especially in chickens with reproductive disease. Any systemic disease has the potential to slow the gastrointestinal transit time in birds, resulting in a functional ileus as a secondary problem. On palpation, the crop may feel distended with a fluid or doughy consistency. A foul odour may erupt from the mouth with palpation of the crop; the bird may even have presented for vomiting. Diagnosing the colloquial "sour crop" at this point, and missing the severely distended coelomic cavity, is a common mistake.

Establish a diagnosis

The common physical presentations of reproductive disease often require additional diagnostic tests to narrow the field of differentials. As abdominal distension is most often present with reproductive diseases, this becomes the focal point for establishing the diagnosis. A majority of this assessment can be completed in the initial consultation, enabling the client to be provided with an immediate plan for treatment.

In the case of chickens, coelomocentesis is a relatively non-invasive first step. This is often performed in conscious patients, with or without ultrasound guidance, on the midline, a few centimetres ventral to the cloaca. Be prepared to aspirate large volumes; it is not uncommon to aspirate 500-1000mL from some chickens. Aspiration simultaneously offers temporary therapeutic benefit, especially if dyspnoea is present secondary to the fluid accumulation[6]. A sample of the fluid can then be subjected to fluid analysis, sediment examination and culture and sensitivity. Pending the findings, the differential list may be profoundly narrowed at this point, to enable treatment to be initiated, or further diagnostics may be required. Radiographs will be helpful once the fluid has been drained, to be able to assess the gonads, or, alternatively coelomic ultrasound may be useful in the assessment of reproductive disease.

In the case of parrots, the presence of a shelled egg, either palpably or identified radiographically, increases the likelihood the parrot is experiencing egg binding. Radiographs in a parrot with a fluid filled coelomic cavity are often unrewarding. Should a shelled egg not be confirmed, and a loss of serosal detail present on radiographs, ultrasound guided coelomocentesis is advised. Ultrasound can be of diagnostic value[3], though there are potential limitations due to the presence of highly developed air sacs in parrots, and small patient size.

A common finding in female bird species with reproductive activity or disease, is the presence of polyostotic hyperostosis[2]. The value of this finding in disease assessment lies in being able to confirm reproductive activity in the absence of lay[5], and resolution of this process with successful reproductive disease treatment[7]. Serum biochemistries will be quite similar for a majority of the reproductive processes; with both normal reproductively active birds and diseased birds typically having a hyperglobulinaemia and hypercholesterolaemia[3]. There is a large variability of blood calcium levels in reproductively active or diseased female birds. A severely depleted ionized calcium measurement can confirm the presence of metabolic exhaustion[2], but tends not to alter the treatment plan. Packed cell volume is useful for assessing the degree of associated anaemia of chronic disease, and can assist in monitoring the patients response to therapy. With many processes of reproductive disease, a heterophilia or monocytosis can be present, depending on the chronicity of the underlying condition. The presence of toxic change and degenerative left shift add to evidence of sepsis and poorer prognoses.

Treatment plan

Clinicians can easily be daunted by the prospect of treating a sick bird, regardless of its presenting complaint. Keeping it simple and applying first principles, should produce appropriate treatment decisions. Determining which body system needs treatment first, based on degree of compromise, is a very good place to start.

Should you have had the chicken with the fluid filled coelom, you have likely already incidentally started therapy for this chicken by draining its coelom. Focus now turns to correcting metabolic disturbances. Likewise, for the egg-bound cockatiel, provided it is still able to perch and is not deemed a surgical emergency, the next step is to start to treat the metabolic exhaustion it is likely experiencing.

As is the case with almost any severely sick and compromised patient supportive care is paramount. The high surface area to volume ratio means birds loose heat quickly, and therefore, should be provided supplemental heat to a temperature of approximately 30°C[6]. Fluid therapy should also be considered; birds respond surprisingly well to even subcutaneous fluid therapy if IV access is not available. Providing warmed (approximately 38-39°C) Lactated Ringers Solution, accounting for a maintenance rate of 50mL/kg/day[6], with replacement of estimated losses over 12-24hours, will start to correct underlying dehydration. Suspected calcium deficits can be accounted for with parenteral dosing[5] of calcium gluconate every 3-6 hours[4]. Crop feeds with highly digestible, sugary substances will provide nutrition[4]. Analgesia should be provided, and antibiosis if indicated by the clinical picture.

It is worth noting some points for caution, especially when treating a suspected case of egg binding. As this is typically a case of metabolic exhaustion, correcting these deficits is more often than not, sufficient for allowing oviposition to occur[2]. Clinicians will often also opt for oxytocin use. Whilst not contra-indicated, there is some question over its clinical efficacy in all bird species[4]. If efficacious, oxytocin, and PGF2α for that matter, induce oviductal contractions without relaxing the uterovaginal sphincter[3,5,6]. Utilising these hormone therapies when the utero-vaginal sphincter is closed, can result in a uterine rupture[2]. Thus, these should only be utilised, if the egg shell is visible per cloaca and calcium deficits have been corrected, and/or if an appropriate utero-vaginal sphincter relaxing agent (PGE2) has already been applied topically, without successful oviposition.

Given the appropriate supportive and hormonal therapies, oviposition usually results within a few hours. If this is not the case, more drastic measures, such as digital manipulation and massage of the egg, ovocentesis (transcloacal being preferred over transcoelomic), or salpingohysterectomy may be required[4,6]. These all require the patient to have a general anaesthesia, and the client to be aware of the potential associated risks.

Once the initial crisis or insult has been controlled, longer term control of the reproductive disorder can be attempted. This too, is multifactorial, and a combination of dietary modification, husbandry changes and medical therapies can be invoked[4]. Gonadotrophin releasing hormone agonists (eg, deslorelin implants or leuprolide injections) are being commonly used in avian medicine to assist in down regulation of hormonal pathways through negative feedback[3,7]. These products are given as off label products, have very limited or no recognisable side effects, though are known to have a variable effect and duration of action. Other therapies, like medroxyprogesterone, have fallen out of favour due to their notable side effects[4].

Conclusion

Reproductive disease is a common cause of presentation of female birds in veterinary practice. Despite the vague clinical signs, obtaining a full history and performing a thorough physical exam can direct clinicians to the treatments required to stabilise the patient. Given the overlap of many of the disease conditions, empirical therapies prior to establishing a definitive diagnosis are usually beneficial. Even in the cases of limited finances and diagnostic modalities, prognostic indicators for the potential treatment plans and outcomes for such birds can be ascertained, all the while, developing a strong rapport between the veterinarian and the client.

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Altruism – professional or pathological and fit for purpose?

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Altruistic behaviour is increasingly associated with health issues of caring professionals[1] while, in contrast, society values altruism in its health professionals[1-3] and applicants for the caring and health professions are motivated by altruism.[4, 5] The continued value attributed to altruism either by professional themselves or by society, in the context of a paid health care career such as veterinary science, could be considered to be contradictory, misguided[6] and not 'fit for purpose' for the sustainability of the profession.

Altruism is '*an individual's enduring tendency to enact self-less behaviours promoting other's welfare such as sharing, helping, caring, and (expressing) empathy'…even at a (physical, mental or financial) cost to themselves'.*[7-9] This requirement of personal sacrifice, risk or cost to the involved person[1, 9-11] is what differentiates altruism from other forms of pro-sociality and humanism.[12]

How does altruism compare and relate to empathy, sympathy and compassion? We summarise the relationships of empathy, compassion and altruism, including evidence from neuroscience to support the empathy-altruism hypothesis [8] that empathic concern drives costly altruism.[13, 14] By doing so we hope to build understanding of the concept of altruism – its pros and cons for the veterinary profession, and its place in professionalism.

Altruism is often regarded as a virtuous trait[15] however for some people, their altruism can become pathological and cause serious problems for them, while intended to benefit another being.[16] We present a number of altruism types from the biological, psychoanalytic and recent neuroscience literature e.g. normal altruism types (proto- and generative-),[16] costly altruism,[13] pathological altruism types (e.g. psychotic- and pseudo-, accompanied by underlying masochism)[16, 17] and induced altruism.[16]

Costly altruism is manifested in altruistic health care professionals who may over respond or under respond in their day to day work with suboptimal discernment and judgement, and this may lead to apathy, burn out and/or compassion fatigue,[1, 6] Compassion fatigue, burn out and other mental health problems are highly represented in the veterinary profession[18-20] and veterinary science students.[21-23]

Induced altruism[16] interests us as it may be present in some veterinary workplaces and is a serious HR issue. Many graduates work in micro and small enterprises, owned by people with no management training, and which may be more prone to less than satisfactory management. Induced altruism occurs when one (self-centred) being manipulates, exploits and/or generally manoeuvres another into self-sacrificing behaviour.[16] Highly other-centred employed veterinarians might find themselves in 'toxic' workplace environments, vicariously undertaking induced altruism, in addition to normal altruism.

For caring professions such as medicine, nursing and veterinary science with high levels of empathy and altruism, development of skills of discernment and judgement become even more important if not critical. Educators for these professions are responding by including in curricula the ability to practice self-awareness as a defence[16] and to provide other forms care such as empathetic care rather than altruistic care[6] but there is still a lot of room for improvement.

We wonder if altruism is an appropriate trait or behaviour for veterinary science graduates at all, given the potential for self-sacrifice to the point of morbidity, the context of being a paid health care career that also boasts graduate salaries amongst the lowest in Australia.[24, 25]

We support a move away from valuing altruistic behaviour towards 'pro-social behaviour' for veterinary graduates, thus placing importance on the benefits of an action without encouraging self-sacrifice. This may continue to provide graduates with motivation associated with empathy, whilst encouraging self-care and work-life balance.[26] Herein lies the challenge for admissions, formal and hidden curricula.

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Knights or knaves – are veterinary science graduands different to others?

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Other-(pro-social) and self-concern (pro-self) orientation of individuals have implications for individuals in the workplace in performance evaluation, work-place behaviour, personal initiative in the workplace[1], job-satisfaction[2] and self-rating of performance.[3] Other-orientation is an individual characteristic that refers to the basic prosocial tendency to be concerned with and be helpful to others[3]. For more highly other-concerned persons, differences in perceived job attribute beliefs (e.g. job richness, autonomy) are less associated with differences in job satisfaction.[2] Other-oriented employees show greater agreement with ratings provided by their supervisors and less leniency relative to their supervisors' evaluations, and vice versa for self-concerned employees.[3] leading to conflict and lowered job performance.[3]

Altruism, a particular form of other-orientation or pro-sociality, is differentiated by the enduring tendency and motivation of the individual to help others even at a risk or cost to themselves.[4-6,7, 8] Driven by empathy,[9] altruism is a known motivator for individuals studying for the human health caring professions[4, 10,11, 12] Without discernment and judgement, empathetic and/or altruistic health care professionals may over or under respond in their day to day work, and this may lead to apathy, burn out and/or compassion fatigue.[6, 13] Wellbeing is an issue for veterinary[14-17] and other caring professionals.[13, 18, 19] Burn out, compassion fatigue and other mental health problems are highly represented in the veterinary profession.[20, 21]

Are veterinary science graduands (final years) similar or different to those of other caring professions terms of the extent they find altruism opportunity in the use of their discipline attractive?

"…I want to be a veterinarian as I want to help animals…" (Anon)

De Dreu and Nauta (2009) propose that other-orientation and self-concern can co-exist in individuals, while Meglino and Korsgaard (2004) consider these to be either ends of a continuum. Drawing on Le Grand (2003)'s taxonomy, we explore the degree to which individuals from different professions segment into types such as 'knights' (those with dominant pro-social altruistic orientation) or 'knaves' (those with dominant self-interest).[22]

Women and men final year students from five disciplines - engineering, science, non-STEMM, nursing and veterinary science of University A, and from veterinary science from two other universities, participated in a paper based choice experiment in the years 2015 and 2016. The 'experiment' involved the participant rating the attractiveness of each job to them of eight job scenarios (using their disciplinary skills), each with high or low levels of three components in an 'orthogonal design' (opportunity for altruism, income level and work effort required) requiring participants to utilise trade-off/decision rule theory based thinking in their ratings.[23] Multiple regression analysis (*a posteriori* decomposition) of the interval-scaled rating of the attractiveness of the eight potential job scenarios, 'revealed' respondents altruistic and self-interest dispositions.[24] Hierarchical clustering (Wards method, squared Euclidean distance) using IBM SPSS version 24.0 (SPSS Inc) was undertaken on the conjoint analysis output data.

Four distinct clusters were revealed: Knight (altruistic), balanced (mixed), self-concerned and Knave (selfish) profile (ANOVA & post hoc, $p<0.05$), supporting existence of other-centred or self-interested taxonomies.[22, 25,3]

Proceedings of AVA Annual Conference, Brisbane, 2018.
Feakes, A – Altruism in the next gen: a professional trait?

Frequency analysis highlighted differences between disciplines. Veterinary science students of University A were found to have the largest proportion of altruists, while veterinary science students of University C had a similar percentage of knaves to Engineering students of University A. Nursing students and then veterinary students of University A overall were the most altruistic considering the combined proportion of altruists and balanced (mixed) categories. The proportional representation of each profile differed for veterinary science of University A, B and C and this was not expected.

Factors that may contribute to this difference include differences in culture(s) of the participants,[1, 2] marketing, admission procedures, curricula design, and role-modelling/vicarious learning. At the time of entry of participants to their respective programs, admissions processes differed. Academic score was the basis of acceptance into veterinary, engineering, entrepreneurship, nursing and science programs of University A. Conversely, entry into the veterinary programs at University B and C each involved a mix of questionnaire submission, evidence of knowledge and experience with animals/veterinary profession in addition to academic score.

The sample population may not be representative of the Australian veterinary final year cohort of the years 2015 and 2016 being from three of the seven Australian veterinary schools. Preliminary analyses indicate differences for international students, and this is not surprising due to other-orientation being higher in individuals from collectivist cultures.[26]

This study contributes to educational practice via utilising a non-Likert response measurement tool. Researchers in veterinary education looking to avoid reliance on 'self-reporting' using Likert scaled items with associated risk of acquiescence or social desirability bias[27, 28] may be interested in the metric choice experiment (MCE) as a quantitative tool. The MCE reveals actual cognitive maps (personally held decision rules) and beliefs used by an individual to arrive at their decisions.

Employers of veterinary graduates may be interested in our findings that while most veterinary graduands are concerned about income level and work effort, there are others that value the opportunity for altruism in their work well above income/work effort. Understanding other-orientation and self-concern in veterinary graduates may help contribute towards more nuanced understanding of job-fit, feedback effectiveness, job-satisfaction and psychological and financial wellbeing of graduates.

Further research is called for to determine if marketing, recruitment and admissions processes and/or curricular design of veterinary programs are associated with differences in levels of altruism in veterinary graduands.

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Legal frameworks for wild animal welfare

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Introduction

The development of legal frameworks for wild animal welfare remains in its early stages in Australia. As yet, no Australian State or Territory has created a legislative scheme that is expressly intended to promote positive welfare states and minimise negative welfare states for individual wild animals. However, each State and Territory does have a patchwork of statutory provisions, located within nature conservation and animal cruelty legislation, that operate to make some forms of harm to individual wild animals “legally cognisable” and to regulate some human actions that cause those harms.

This paper describes features of these patchwork legal frameworks for wild animal welfare in Australia. It deals with the legal frameworks for free-ranging wild animals (i.e. animals in a condition of “natural liberty”) and does not address legal frameworks for wild animals who are in the care, custody or possession of a person (e.g. for rehabilitation).

Foundations for legal frameworks in Australia

Legal frameworks for wild animal welfare in Australia are largely founded largely in State and Territory nature conservation and animal cruelty legislation. However, Commonwealth legislation, and specifically the *Environment Protection and Biodiversity Conservation Act 1999,* also provides a legal framework for wild animal welfare, particularly to fill gaps where State and Territory legislation does not extend to or has a more problematic application, e.g. offshore environments.

State parliaments have, subject to the *Australian Constitution*, plenary legislative power to make laws for the peace, welfare and good government of the State. It is a long-standing legal principle that native animals belong to the people of the State. The notion that native animals belong to the people underpins the statutory vesting of property in fauna in the State (or “Crown”) under nature conservation legislation. In *Yanner v Eaton* (1999) 201 CLR 351, the High Court of Australia observed that such vesting represents “a fiction expressive in legal shorthand of the importance to its people that a State have power to preserve and regulate the exploitation of an important resource” which imposes upon a State a “sort of guardianship for social purposes” in relation to that resource.

How laws work – legal effect on natural and corporate persons

Legal systems focus upon the regulation of human conduct. The laws which comprise a legal system operate through their legal effect on “natural persons” (human beings) and “corporate persons” (corporations and incorporated associations). Laws create rights, duties, powers, liabilities, liberties, and liabilities for natural and corporate persons. Some of these can only apply to natural persons – e.g. you can’t sentence a corporation to prison.

Criminal laws operate by creating a norm of conduct and attaching a penalty if a person or organisation breaches that norm. An offence is an act or omission (or a series of acts and omissions) that makes a person liable for punishment. The effect of a creating an offence is to impose a “public duty” on persons which is owed to society generally and not to any particular person. For example, the legal effect of a prohibition on cruelty to animals is to create a duty not to be cruel to individual animals.

Laws may also operate by creating legal relationships between persons. Such relationships are vital for the operation of the civil law in Australia. For example, entering into a contract creates legal rights and correlative legal duties for the contracting parties. Similarly, the law of negligence recognises that some persons may have a duty of care that is owed to other persons.

At least three other features of Australian legal systems are relevant to understanding legal frameworks for wild animal welfare in Australia.

First, although these legal frameworks are largely founded in statutory law, native title and common law rights relating to wild animals are also important, to the extent that these rights have not been extinguished or rendered inoperative because of the imposition of statutory law.

Second, persons may obtain or be granted a proprietary interest in a wild living natural resource (e.g. a stock of fish). The effect of these proprietary interests is to give that person a “bundle” of rights in relation to that resource.

Finally, legal frameworks for wild animal welfare also include legislative schemes for authorising actions and activities that harm individual wild animals, either as a purpose of the action or activity (e.g. harvesting of wildlife) or as consequence of it (e.g. clearing of native vegetation). The legal framework regulating the exercise of a statutory power to approve an action or activity may require or prohibit a decision-maker to take wild animal welfare into account. More commonly, however, the legal frameworks for administrative decision-making will make wild animal welfare a permissible, but not a mandatory or prohibited, consideration for decision-makers.

The legal status of wild animals in Australia is complex. The effect of the legal frameworks for wild animal welfare is that individual animals may be the beneficiary of laws that operate to restrict or prevent human actions. Similarly, it is appropriate to talk about individuals as being the victim of certain offences. And, at least conceptually, we can talk about individual animals having a liability to being harmed or an immunity from being harmed. However, Australian legal systems have not yet developed to the point where wild animals have legal personhood in the sense of possessing liabilities or immunities (or powers, liberties, or positive rights/claims over other persons) <u>because of</u> their status as wild animals. In other words, wild animals have not yet been integrated into legal systems as animal “persons” capable of holding and exercising rights, powers, liabilities, immunities, etc. in the way that natural and corporate persons do, or of participating in the web of legal relationships that exists among natural and corporate persons.

Making harm legally cognisable

Human actions may harm the welfare of individual animals by causing some immediate or longer-term adverse change to the animal’s physical or mental state involving physical injuries, other pathological conditions, pain, and psychological distress to individual animals.[1, 2] Human actions may also affect wild animal welfare by depriving animals of positive welfare states (e.g. actions which remove animals from the wild or prevent animals from engaging in natural behaviours).[3]

To influence wild animal welfare, laws must make a form of harm to individual wild animals "legally cognisable" and allow for a causal link to be made between this harm and a specific human action (or activity).

In broad terms, a harm is legally cognisable if it falls within the meaning of a statutory word or phrase, either expressly (the harm is stated in the text of the statute) or as a matter of statutory construction (i.e. the interpretation of the meaning of the statute and its application to particular factual circumstances).

Definitions of "harm" in animal cruelty statutes have expressly recognised a broad range of harms. For example, section 3 of the South Australian *Animal Welfare Act 1985* defines 'harm' to mean any form of damage, pain, suffering or distress (including unconsciousness), whether arising from injury, disease or any other condition, and section 5 of the Western Australian *Animal Welfare Act 2002* defines 'harm' to include injury, pain and distress evidenced by severe, abnormal physiological or behavioural reactions.

The statutory text may also indicate whether wild animal welfare is a permissible consideration for an administrative decision-maker. For example, section 3(1) of the Western Australian *Environmental Protection Act 1986* (WA) defines "environment" to mean: "living things, their physical, biological and social surroundings, and interactions between all of these". Thus, the harming or killing of a 'living thing' falls within the concept of an "effect" on the "environment" that a decision-maker could have regard to in assessing the environmental impact of a proposed development.

Offence provisions relevant to wild animal welfare come in two basic forms. The first uses "action" verbs to describe the offending conduct, e.g. "kill", "injure", "harm", "disturb". These verbs often combine the human act and the result of the act – e.g. "kill" and "injure" would encompass both the relevant physical conduct and the harm that an animal suffered as a result of that action. Legal questions may arise as to whether <u>action/activity X</u>, which results in <u>effect Y</u> to <u>wild animal Z</u>, falls within the meaning of a particular verb, e.g. did exposure to high concentrations of an agricultural pesticide in a farm pond poison birds and therefore "kill" them? The efficacy of legal frameworks for wild animal welfare will often turn on legal questions about whether a particular harm, and the specific human action that causes that harm, fall within the meaning of a word or phrase in a statutory provision.

The second form that an offence provision may take is to use evaluative words, such as "unnecessary", "unreasonable", or "unjustifiable", in relation to the "harm" that an animal sustains because of a human action or activity. Animal cruelty provisions often include these evaluative expressions, usually in conjunction with other provisions that prescribe unlawful human actions to more precise terms.

Evidentiary considerations must also be evaluated when determining whether a particular harm is "legally cognisable" in a practical sense. For example, a prosecution for a taking offence can only succeed if it is possible to adduce evidence by which a court (e.g. a Magistrate) could be satisfied that an animal had suffered a particular harm. This may present challenges as to the availability of physical evidence (e.g. possession of a animal body for post-mortem examination) and witness evidence (e.g. persons who observed the animal being harmed), the quality of the evidence (e.g. post-mortem changes in an animal body), and whether evidence of the harm could be given at all (e.g. there are no reliable indicators for a particular harm, psychological distress that does not manifest in an observable behavioural response).

How laws can affect wild animal welfare
Laws can influence wild animal welfare in several ways.

1. Laws may prohibit certain acts that harm wild animals.

For example, all jurisdictions in Australia have some form of statutory prohibition on the killing or injuring of wild animals without lawful authority. These are frequently referred to as prohibitions on the "taking" of wild animals or "taking" offences. For example, the Western Australian *Biodiversity Conservation Act 2016* defines "take" to mean, in relation to fauna, to mean "to kill, injure, harvest or capture fauna by any means". Statutory prohibitions on cruelty to animals may also extend to wild animals. The legal effect of taking and animal cruelty offences is to impose a liability to be punished on persons if they perform acts that "take" wild animals or are "cruel" to an animal.

2. Laws may confer a person with an immunity from a legal liability for an act or omission that harms wild animals.

Statutes often exempt certain activities from taking prohibitions or allow persons to apply for a licence to "take" wild animals for a particular purpose. The exemption or licence provides an immunity from prosecution for a taking offence, as long as the person complies with circumstances of the exemption or the conditions of the licence. Similar exemptions or provisions for licensing exist for animal cruelty offences.

3. Laws may fail to create liability for persons for certain harms that they cause, either because of their content or because of the manner in which they are enforced.

(a) Statutory construction: The harm itself may not fall within the meaning of a statutory word or phrase. For example, the meaning of "injure" in the definition of a taking offence might not extend to include psychological distress or pathological conditions other than physical injuries
(b) Legal causation: A court may, as a matter of policy, find that there is no causal basis (i.e. no chain of causation) to connect a human act, omission or activity to a legally cognisable harm to a wild animal. For example, a court might refuse to find that the unlawful clearing of native vegetation caused an animal to be severely injured by a car on an adjacent road, two days after the vegetation was cleared.
(c) Prosecutorial discretion: Prosecutorial authorities may refuse to lay a charge for an offence in particular factual circumstances, even if liability in the circumstances is at least arguable. Prosecutorial discretion can thus lead to under-enforcement of laws that might otherwise protect wild animals.

4. Laws may require persons to perform acts that harm wild animals.

For example, biosecurity legislation may create a statutory duty requiring landowners to undertake prescribed measures to control declared pest species.

5. Laws may grant a person a positive right to harm wild animals.

For example, at common law, persons had a right to fish in the sea and in tidal waters and to access intertidal areas. Native title rights include rights to hunt and fish. In Australia, the principle of parliamentary sovereignty means that statutory laws can abrogate or extinguish native title and common law rights.

6. Laws may create positive duties towards individual wild animals.

For example, animal cruelty provisions may impose a legal duty on owners or carers of wildlife who are in their care or custody (e.g. pets or animals held temporarily in captivity) to provide for their basic needs. Persons may have positive duties towards free-ranging wild animals if a legal agreement (e.g. a conservation covenant), a condition attached to a regulatory authorisation (e.g. a licence or development approval, or a court order (e.g. an injunction) requires them to take some positive measure (e.g. to create and maintain a habitat feature) or prevents them from removing or degrading something that already exists (e.g. native vegetation).

7. Laws may require public authorities, government officers and other persons to take wild animal welfare into account when performing or exercising a statutory duty or power (undertaking a statutory function) or when making a decision under legislation (engaging in administrative decision-making).

Examples of statutory function include a government department using a statutory power to manage Crown land under its care, control, and management to conduct prescribed burning.

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Reconstructing transmission networks FMD outbreaks

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Introduction

Several transmission network models are available that combine genomic and epidemiological data to reconstruct networks of who infected whom during foot-and-mouth disease (FMD) outbreaks. Early in the response to large-scale outbreaks of FMD and other infectious diseases of animals, when contact-tracing is just getting underway, the implementation of appropriate interventions is time-critical. Central questions at this stage for the outbreak investigation team are 'who infected whom?', 'who will be infected next?' and 'how and where should we intervene?'. Answering these questions in time to inform decision-makers can lead to large potential savings through better targeting who to investigate and which farms to quarantine, and/or depopulate or vaccinate.

For models to reliably inform such decision-making they must be transparently validated, robust, and capable of producing accurate predictions within the short data collection and inference timeframes typical of outbreak responses. This series of studies follow on from a comparison of fitness for purpose of ten transmission network models based on a set of foot-and-mouth disease outbreaks simulated in Australia, with corresponding simulated phylogenies and genomic samples from animals on infected premises.

The aim of the present analyses was to extend the best-performing models and apply them to real outbreak datasets.

Methods

Ten algorithms for integrating genomic and epidemiological data compared (Firestone et al., provisionally accepted for publication). The best-performing model (Lau et al., 2015) was extended to (a) incorporate farm-level covariates with verification based on simulated outbreaks then application to the real outbreak dataset from FMD in Japan in 2010, and (b) inclusion of animal movement data with verification again based on simulated datasets in anticipation of application to further outbreaks.

Results

The modified model that incorporated farm-level covariates achieved improvements in overall accuracy of between 5 and 6% when tested on the simulated outbreaks in Australia and Japan. When implemented on the actual outbreak data from Japan, infected farms that held predominantly pigs were estimated to have higher transmissibility than infected cattle farms and to be less susceptible. The farm-level incubation period was estimated to be shorter than the latent period, the timing of the primary seeding event that initiated the outbreak in Japan was inferred and all key linkages between clusters and features characterising important farms in widespread dissemination of this outbreak were elucidated. The modified model that incorporated animal movement data achieved improvements in overall accuracy of between 6 and 24% when tested on the simulated outbreaks in Japan.

Discussion and conclusions

Extending Lau's systematic Bayesian inference to incorporate additional parameters representing predominant species and numbers of animals held on a farm resulted in improvements in overall accuracy across a series of varied simulated outbreaks, so too did incorporating inferred parameters representing the likelihood of transmission given traced animal movements from infected farms.

Biologically plausible estimates of the relative transmissibility and susceptibility of farms that held predominantly pigs compared to those that predominantly held cattle and sheep farms were achieved. Inferences of key epidemiological parameters included the farm-level incubation, latent and infectious periods, the number of infected premises at the point of detection, the time back to the seeding event into the population and the number of primary sources.

To improve accessibility the modified Lau model has been implemented as an R package and will be available for use to inform decision-making in future outbreaks of FMD and other infectious diseases of livestock such as *Mycoplasma bovis*.

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Water quality impacts on the health of freshwater turtles

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Introduction

Australian freshwater turtles, such as the Eastern long-neck turtle and Murray river short neck turtle are both commonly kept as pets in Australia. They are found in the wild in a variety of watercourses, although research has not defined preferred water quality parameters in wild turtles. Pet turtles are often kept in glass aquarium kept inside the house and are presented for vet examination due to anorexia, shell and skin abnormalities. As part of the clinical workup of these patients, blood tests and water quality tests are recommended. Often despite the age of the turtle, most turtle tanks are suffering from 'new tank' syndrome due to poor tank maintenance. From basic testing done in consults over about 50 – 70 cases, an association between high ammonia and nitrite levels and the health of the turtles can be made. The result of poor-quality water results in anaemia, reduction in vitamin A and E levels compared to wild and healthy captive turtles and systemic infection with possible sepsis. Except for one study on the effect on ammonia and nitrate on the hatchability of turtle eggs, there are no studies that exist demonstrating the association of individual water quality parameters with any particular syndrome. However, every text on turtles does talk about the importance of water quality while providing no detail on what particular values are seen with disease.

As part of the diagnostic work-up for the sick turtle, a review of tank husbandry, water quality testing and some basic blood tests such as PCV (packed cell volume) and TS (total solids), blood glucose and an estimated total white blood cell count are recommended to the client and performed when consent is obtained. This presentation covers some of the observations from physical examination, blood and water testing of chelonian patients seen at the Adelaide Bird and Exotics Vet Centre in the last two years.

Understanding water quality parameters

During the consultation, the client is asked questions about tank husbandry. Water quality parameters are ideally tested during the consult. The most commonly determined water quality parameters tested are water pH, ammonia (NH3 or NH4), nitrite (N02-) and nitrate (N03-) levels. These are all reactive forms of inorganic nitrogen. Ammonia is broken down into nitrite by aerobic bacteria such as *Nitrobacter* and *Nitrososomas*. Nitrate may be broken down by some bacteria but often requires physical removal by performing water changes. Water hardness could also be measured, but is not readily available in water quality testing kits for freshwater tanks.

Water Ph. It is thought that turtles prefer a pH that is neutral to mildly alkaline (pH 7 – 7.4), although in the wild, they are found in watercourses with variable alkalinity. However, acid water with a pH of 6 does appear to be associated with a higher likelihood of damage to skin and shell.

Ammonia. Ammonia levels higher than 8ppm (parts per million) have resulted in eyelid swelling (blepharitis) in three Long-neck turtles. This has not been seen in turtles who live in tanks where the ammonia is lower than this value. Acceptable levels of ammonia are 0ppm. Increasing tank temperature, in the case of turtles to 26°C, and increasing pH increase the availability and thus toxicity of ammonia. Treatment of high ammonia levels can be targeted at commercial products (which have a variable success), addition of bacteria to the tank (which may help with maturation of the filter), quarantining the turtle from the tank by dry docking and waiting for maturation of the filter bacteria.

Nitrite. Acceptable nitrite levels are less than 0.25ppm. Both elevated ammonia and nitrite levels are seen in 'new tank' syndrome. This is a misnomer, as it truly refers to the age of the filter system rather than the tank itself. A tank can be ten years old but with aggressive, frequent filter cleaning in tap water, the bacterial population in the filter does not mature and thus the levels of bacteria that consume ammonia and nitrite remain low, with the result being elevated levels. Many clients' perception is to equate a dirty filter with dirty, unsafe water.

Nitrate. Nitrate is not broken down by the nitrite-eating bacteria in the filter system. Acceptable levels are less than 40ppm. Toxic levels appear to be at around 160ppm when this is present for several weeks. It is not as toxic as nitrite, but can cause gastroenteritis in some species. Methemoglobinemia, anaemia and nephritis are associated with chronic exposure to low levels (3). The anorexia seen in pet turtles presented for illness may reflect the damage to the gastrointestinal tract from nitrate. Nitrate is removed from the tank by regular water changes. It is likely that nitrate is converted to nitrite by anaerobic gut bacteria in turtles, as seen in other species.

The current recommendation is 20-25% of water is changed weekly to remove the nitrate. The tank water being replaced by tap water treated with a commercial water ager/conditioner which has a chemical to remove chlorine products (such as chloramine) present in the tap water. Some clients refuse to use tap water, siting its lack of suitability for turtles. However, testing of filtered water and rainwater in water samples supplied by clients has demonstrated that both of these water sources are usually acidic with a pH of 6 or possibly lower.

Nitrite and nitrate toxicity

Nitrite is more toxic at lower concentrations than nitrate. However chronic exposure to high levels of nitrate has been associated with toxicity. Nitrate toxicity in humans and rats causes methaemoglobinaemia due to its conversion to nitrite by anaerobic gut bacteria. This then blocks the ability of blood cells to exchange haemoglobin. In many sick turtles where the nitrate level remains high due to infrequent water changes, anaemia (a PCV of less than 25%) has been observed, suggesting that a similar mechanism is taking place in turtles. In severe cases, PCV of less than 10% has been seen. When the PCV is less than 10%, it is unlikely that the turtle will survive. Many of these turtles will also have hypoglycaemia as well.

Nitrate toxicity in cattle creates clinical signs of ill-thrift, poor appetite and poor growth, similar to the clinical signs seen in sick turtles.

Methylene blue has been used historically in the treatment of nitrite toxicity in cattle. This treatment has not been attempted in pet turtles at this stage, and due to the chronic nature of exposure, may not prove to efficacious.

Tank care

As part of the history collection in the consult, questions about how the tank is managed must be asked to determine if water quality is appropriate. These questions can provide information on the tank temperature; water changes (volume, frequency and use of conditioners to remove chloramine); filter care including frequency of cleaning and how, as well as frequency and food item offered as the diet.

In a survey of our clientele, there are few clients who understand the nitrogen cycle and few who maintain their tanks well. Their most common source of information on tank care are pet shop staff. Few clients are using water conditioners or agers, often being told by pet shop staff that it is unnecessary for turtles. The purpose of these products is to remove the chlorine from tap water. Chlorine is added to tap water for its antibacterial properties. Failing to remove it results in the chlorine affecting the nitrogen-eating bacteria required by the filter to remove nitrogenous waste produced by the turtle.

Water changes were rarely performed at the correct frequency or amount. The most common response was water changes of 50% volume every 1-3 months. This regime results in significant change to the filter bacteria population and appears to recreate new tank syndrome.

Physical examination findings

Clients most commonly present turtles with either a syndrome of anorexia/lethargy or skin/shell lesions. Although turtles can survive without eating for at least 40 days, clients present turtles within 10 – 21 days of the onset of anorexia. The majority of pet turtles are fed daily, irrespective of age. Despite regular feeding, the majority of sick freshwater turtles present in moderate to poor body condition as assessed by the size of the masseter/temporalis muscle on the head of the turtle. Although not validated, it provides a consistent measure of muscle condition in both of these species. Nitrate toxicity in cattle creates clinical signs of ill thrift, poor appetite and poor growth, similar to the clinical signs seen in sick turtles. Many of the sick turtles that have presented are about 6- 8 years of age but are only 200 – 300g, rather than an expected 500g or more.

Turtles are usually reactive to handling but more advanced cases are physically weak and do not paddle with their legs when handled. Many sick turtles will struggle to raise themselves up to walk when placed on the ground although they may paddle their legs weakly. This may reflect low levels of oxygenated blood being present.

Ulcerative skin lesions involving the extremities such as nail beds or webbing between the feet may be seen. Inflammation of the peri-cloacal skin is common. In severe cases, petechiation of the skin particularly of the pre-femoral fossa may be observed. This occurs in association with leucocytosis.

Aural abscessation, commonly seen in American pet turtles in association with low dietary vitamin A levels has not been seen, despite low levels of Vitamin A in the blood. However, many of these sick turtles have a pale pink tongue, indicating anaemia likely due to methaemoglobinaemia. In severe cases of anaemia, the tongue and interior of the mouth appear white.

Haematological findings

Blood is collected from the jugular vein of the turtle. In many cases, less than 0.1ml is collected, due to size of the patient and difficulty in collecting a larger volume. The blood sample is then used to provide a PCV/TS (Packed Cell Volume/Total Solids). A hand-held glucometer (Accu-chek®, Accu-chek, NZ) is used to measure blood glucose. A blood smear is made and stained with Diff-quik to perform an estimated white blood cell count, similar to how this is performed in birds. A monolayer is found, and the average number of white blood cells is calculated over 10 fields using the 40x objective (high and dry) (3). Evaluation of the presence of a left shift, monocytosis or lymphocytosis is made either visually or by a leucocyte differential performed after the consultation.

Vitamin studies

Blood levels of vitamin A, C and E have been shown to reduce in nitrite/nitrate toxicity as their production is impaired. To confirm that reduced vitamin levels were occurring in the sick turtles and thus may explain both the skin lesions seen and the poor response to antibiotics alone due to impaired immune function without these antioxidants, a comparison was made between three groups of turtles. Adelaide Zoo has determined vitamin A, E and C levels in their captive population of Long and Murray river short neck turtles. Wild turtles of both species that presented to ABEVC for shell fracture repair were also sampled on the day of arrival as part of their health screening prior to anaesthesia for shell repair. Pet turtles owned by clients which were presented for illness were also sampled to determine PCV/TS, glucose and ETWBCC were also sampled where sufficient blood (0.5-1ml of whole blood) could be collected based on blood volume being 1% of bodyweight.

Vitamin A, E and C levels were significantly lower in the sick client-owned turtles compared to both captive and wild turtles. The difficulty lies in determining which is a truly abnormal result in a patient as the normal range of these vitamins in these turtle species is yet to be established. Vitamin E levels are influenced by dietary levels. Many turtles are on frozen turtle dinner blocks (Fish Fuel Co, Thebarton SA), a diet based on published diet composition in these species (4). Interestingly, in the few pet turtles presenting for dystocia, low vitamin levels were not seen.

Vitamin E supplementation has been demonstrated to reduce the effect of nitrate toxicity in rat (2) and calf (6) studies. So, supplementation with monthly injections of ADEC (ADEC injection, Vetafarm®, Ltd, Wagga Wagga, NSW) at 0.1ml/150g has been initiated and appears to be helpful. Vitamin E acts as an antioxidant to reduce haemolysis in erythrocytes suffering oxidative stressors, such as nitrate (6). It may be that a more frequent dosing schedule or different dose rates may be required for pet turtles. A future proposed project will be to take serial samples after injection to determine whether the dose rate and formulation being used are effective at increasing vitamin A, E and C in freshwater turtles. Determination of the normal ranges of other oxidative scavengers such as uric acid, TEAC (Trolox equivalent antioxidant capacity) and FRAP (ferric reducing ability of plasma) will allow evaluation of what constitutes an elevated level to confirm the degree of oxidative damage from nitrates that is present.

Treatment of sick turtles

A multifactorial approach has been used to address the condition of these sick turtles. Hospitalisation was offered based on PCV <15%, glucose < 1mmol/L, presence of petechiae. As turtles respond slowly, a minimum of five days hospitalisation was routinely recommended. The owner was requested to undergo tank maintenance, such as daily water changes of 20% for five days to reduce nitrate levels, or to purchase a large out-of-tank filter.

If treated as an outpatient, monthly visits were performed to reweigh, repeat blood tests and redispense antibiotics. As mentioned above, an injection of vitamin ADEC is given at monthly rechecks. The owner was requested to undergo tank maintenance based on water testing results and to dry dock the turtle as described below.

Antibiotics are given in the presence of infection, which is determined by an estimated white blood cell count over 15 x 10(9). Septic turtles often had ETWBCC greater than 20 x 10(9). Those with ETWBCC greater than 40 x 10(9) were more likely to die. Antibiotics were continued until the white blood cell count returned to the normal range. Ceftazidime (Fortaz®, Teligent) 20mg/kg IM q 72h was the most commonly used antibiotic. It was selected due to its low dosing frequency and broad-spectrum activity making it suitable for septic patients. It has activity against Pseudomonas-related species that may be found on turtle skin. In severe hospitalised cases, clindamycin (Dalacin®, Pfizer) at 11mg/kg IM q 24h was administered during hospitalisation. The antibiotic treatment period averaged 60 days, with sicker turtles requiring longer course. When it is considered that ‘new tank’ syndrome takes at least 42 days resolve, the longer courses are likely to be necessary as the environment that the turtle is housed does not alter for most of this treatment time. If a client did not address the tank issues, there was an increased likelihood that the treatment time extended beyond 60 days.

In pet turtles with blood glucose values of less than 1mmol/L, hospitalisation for intra-coelomic glucose was recommended. 1% bodyweight of the fluid composed of 2.5% glucose, 0.45% saline (Baxter) was administered once daily. Supervised swims were done daily, but when the glucose is this low, these turtles are unable to swim longer than 10 – 15 minutes at a time before fatiguing.

In anaemic turtles, with PCV of less than 15%, human erythropoietin (EPO) (Aresnep, darbepoeitin, Amgen®) has been administered. EPO has been used in Green sea turtles (*Chelonia mydas*) to stimulate erythrocyte production. Using this published dose rate of 10 000 IU q7d for 14 days of 72h dosing has been trialled with increased PCV at the following monthly visit.

Management of hospitalised turtles involved dry-docking with daily swims. Dry docking involves a turtle being housed in a plastic tub with a wet (i.e. dripping water) towel, replaced daily. Half of the tub is placed on a Wombaroo Cosy pad (Wombaroo, Mt Barker, SA) between the tub and the floor, which is to provide heat and humidity by evaporation of the water from the towel. This can maintain the turtle at 22- 24% and 70% humidity. This system appears to avoid dehydration and weights are maintained using this approach. Daily swimming for 10 minutes to one hour or more based upon the turtle's behaviour. Food is offered daily during the swim.

Where skin lesions such as ulceration or wounds are present, topical sulfasalazine (Flamazine®, Smith & Nephew) is applied daily at the commencement of the dry dock period. Although other products such as 1% iodine (often done as Betadine® mouth gargle from the chemist) have historically been used for turtle skin, Flamazine in my hands has reduced the healing time from 90 days to less than 60 days for many lesions. This is possibly due to its ability to penetrate an eschar and is not cytotoxic to living cells, unlike iodine.

Conclusion

At this early stage, there appears to be some evidence for nitrite/nitrate toxicity in turtles. Good water quality must be maintained in the tank to ensure the health of the turtle. Both haematological tests and water quality testing can be used to determine the severity of illness in the sick turtle. Treatment is aimed at addressing the infection, stimulating bone marrow to produce erythrocytes and vitamin supplementation.

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Examination and diagnosis of oral diseases in rabbits and rodents

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Summary
Good anatomic knowledge, a well performed clinical assessment and interpretation of correctly exposed radiographs are the keys to diagnose problems and establish their treatment.

Notes
Despite the Order of rodents being the largest mammalian order, this presentation will include only guinea pigs, chinchilla, degu, rats, hamsters and prairie dogs which represents three suborders: Porcupine-like, Rat-like and squirrel-like rodents. These representatives are kept as pets and may present in general practitioner's waiting room. More over in all these species, primary and secondary dental diseases are very often diagnosed. Dental diseases are accompanied by decreased food intake, weight loss, problems with appetite, drooling, digestive disturbances, facial deformities, exophthalmos and poor coat condition.
With their continuously growing teeth, these pets are predisposed to dental disorders every time when mistakes in nutrition occur. They suffer from periodontal disease, malocclusions, and fractured teeth that can all result in the formation of abscesses. Some dental problems as in all edentulous creatures can cause systemic complications.

Diagnosis of dental disease in rodents.
To make an accurate diagnosis it helps to have sufficient data regarding the patient, its history and the signs observed. In most cases the history and an initial external examination of a sick animal will give indications as to any probable dental involvement.
Although it is possible to perform oral examination of awaken patient with the use of an otosccope as an oral endoscope in some species, the restricted field of view means that many lesions will be missed. Even when examining a patient under anaesthesia using mouth gags, cheek dilators, mouth mirrors and magnification, it is common for experienced clinicians to miss significant oral lesions.

Therefore, the complementary and very important part of examination is radiography. Positioning techniques vary from the ones used in dogs and cats: extra-oral views are most common instead of intra-oral techniques however both can be employed. For extraoral radiographs four views are standard: lateral, dorso-ventral, diagonal, and rostro-caudal. The first three are similar to the ones used in horses; they allow a good look at the alignment of the teeth and at the anatomy of the radicular ends. The last position allows a good view of the sinuses and of the temporomandibular joints.In lateral and dorsoventral projection it is possible to use the reference lines. Using the reference lines, the extent of malocclusion guinea pigs and chinchillas can be acquired more exactly and the results are reproducible by different examiners (staging). In addition to this these special lines facilitate to accurately monitor the progress of dental changes and thus to predict a probable long-term prognosis. The reference lines are absolutely applicable for daily use in practice.

Intraoral techniques providing full mouth radiography in a rabbit have been described in step by step manner and in a series of ten images one can appreciate tooth resorption, lysis, ankylosis, dysplasia, periapical lesions and cortical perforations. The images captured by the intraoral technique can be used in combination with extraoral views to evaluate other skull structures and complement occlusal assessment.

There is undoubt superiority of 3 dimensional imaging over standard 2D radiography. In terms of the range of details as well as number of informations Computed Tomography or Cone Beam Computed Tomography significantly exceeds diagnostic value obtained from 2D imaging. The recent studies shows that CBCT images are superior to CT.

The most frequently recognised dental problem in rabbits and rodents is malocclusion of their easily examined elodont (aradicular hypsodont) incisor teeth. Without the normal regular wear from chewing or gnawing activity, these non-functional maloccluded teeth continue growing, further impeding function.

The second most common dental problem in these small herbivores is cheek tooth overgrowth. This often accompanies incisor malocclusion; either as a part of the primary condition, or as a purely secondary problem; but it is also seen as a primary problem in its own right. Cheek tooth overgrowth may result from lack of wear due to malocclusion or an insufficiently abrasive diet. Whether a malocclusion is primary or secondary, abnormal tooth wear of the naturally curved cheek teeth tends to cause development of sharp enamel spikes/spurs which irritate the cheeks and tongue leading to wounds and ulcerations associated with pain.

The close proximity of the root apex of the mesial cheek teeth to the lacrimal duct accounts for the occurrence of epiphora and the frequent spread of periodontal infection from these teeth to the tear duct, producing the typical purulent ocular discharge.
Root extension of the mandibular cheek teeth leads to remodelling or thinning of the adjacent cortical bone with development of palpable swellings along the ventral surface of the mandible. Suspected mandibular or maxillary root extension is best confirmed radiographically.

Facial and mandibular abscesses are generally caused by dental problems, though infection of external wounds does also occur.

Pseudo-odontoma in praire dogs. The constant, concussive force on the teeth of these prairie dogs causes the roots (or apices) to react, become irregular and thicken. As the roots increase in size, they begin to fill the nasal area. Since prairie dogs breathe only through their noses (called obligate nasal breathers) this increase in tooth size makes breathing difficult. The condition is worsened by obesity, which is very common in captive prairie dogs. The cause of this is theorized to be the unnatural materials (metal, wire, hard plastic) of which their enclosures are made. These materials are much harder than the grasses, clay, sand and stems on which they would normally chew in the wild.
Prevention seems to be the best solution as surgical treatment brings relatively high risk and guarded prognosis.

Management:

The incisor tooth overgrowth problem has traditionally been managed by repeated tooth shortening (preferably trimming using a diamond bur in a high speed dental handpiece), without investigation of the underlying problem. In cases with recurrent overgrowth, extraction of the offending teeth has been suggested as a more permanent solution. In rodents correction of incisor teeth by burring a reverse bevel on the mandibular incisors to create an incline plane like interlock with the maxillary first incisors, cannot be accomplished. In the majority of cases, occlusal equilibration of the cheek teeth is also required.
Treatment of the cheek teeth by removing enamel spikes and occlusal equilibration is sometimes possible, though the long term results are poor in most cases. As with the correction of rabbit incisor malocclusion, the treatment is most successful if performed at an early stage, in mild cases. Dietary alteration (providing hay as the largest component of the diet) to increase the rate of attrition is highly beneficial in these cases. In more advanced cases the provision of a more

abrasive diet is helpful, but it is usually necessary to repeat occlusal equilibration at regular intervals.

For dental procedures in small herbivores the series of dedicated tools are available. operating table with head and jaws positioners, examination instruments, dedicated low speed handpicks and burs are necessary to perform safely and efficiently oral surgery. Nail clippers are not recommended, they are even viewed as detrimental as they can cause diagonal fractures causing pulp exposure, pulpitis, pulp necrosis, periapical granuloma, and even osteomyelitis and sepsis. Rasps are not on the list either, as they are inefficient and often cause trauma to the soft tissues of the oral cavity.

The most frequently recognised problems in rabbits are: malocclusion, cheek teeth overgrowth and facial and/or mandibular abscesses. Repeated tooth crown shortening, removing enamel spikes, extraction of infected teeth are the most common procedures applied for maintenance of oral health and occlusal equilibration. Dietary alteration (providing hay as the largest component of the diet) to increase the rate of attrition is highly beneficial in these cases.
For dental procedures in small herbivores the series of dedicated tools are available. Operating table with head and jaws positioners, examination instruments, dedicated low speed handpieces and burs are necessary to perform safely and efficiently oral surgery. Nail clippers are not recommended, they are even viewed as detrimental as they can cause diagonal fractures causing pulp exposure, pulpitis, pulp necrosis, periapical granuloma, and even osteomyelitis and sepsis. Rasps are not on the list either, as they are inefficient and often cause trauma to the soft tissues of the oral cavity.

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Treatment options for linguoverted canine teeth in dogs.

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Summary
This malocclusion is associated with pain and thus requires treatment. Different treatment options are available.

Notes
This malocclusion can be observed in deciduous and permanent dentition. In most cases this problem causes traumatic relation of the mandibular canine teeth which are impinging the palatal soft tissue thus are associated with pain, discomfort and malfunction of dental apparatus. Chronic neglected cases can lead to palatal bone infection, oro-nasal communication, teeth attrition and involved teeth pulp diseases. In deciduous dentition the best solution is interceptive orthodontic procedure: extraction of the teeth causing trauma. This procedure must be done with caution and radiographic control to avoid damage of the permanent teeth.

There are numerous options available for treatment of permanent mandibular canine linguoversion. Each choice has benefits and drawbacks which must be considered when planning a treatment protocol. The first step is to determine whether the tooth should be orthodontically treated or not. There are many alternatives to corrective orthodontics including: extraction, crown reduction and vital pulpectomy, rubber ball therapy, surgical tooth repositioning and gingival contouring. With class I linguoversed mandibular canine malocclusions, orthodontics is usually the best choice as it is relatively straight forward, effective, and the tooth is preserved.

Interceptive extraction of the mandibular canine will address the malocclusion however it irreversibly change the rostral mandible structure.

Crown height reduction is generally considered a better option to extraction as it alleviates the malocclusion, is much less painful, and retains enough crown to provide some function. Crown reduction is not without risk however, and can lead to restoration failure, infection, and pulpitis which can potentially occur years later. Consequently, annual radiographic follow-ups are essential, requiring numerous subsequent anesthesias

Rubber ball technique. This can be effective if only a short tooth movement of the mandibular canines is required and the interdental space between the maxillary third incisor and the canine tooth is wide enough to allow mandibular canines to come out.

Gingival contouring involves removing a section of gingival tissue from the maxillary diastema, between the canine and third incisor, to allow the mandibular canine to occupy that space and the jaws to fully close. This procedure requires only one anesthesia, but is efficacious only in mild cases.

There are a number of different devices that have been successfully used to correct mandibular canine linguoversion. Temporary canine teeth extension material can be placed on the tips of mandibular canines which elongate and guide them into correct position when the jaws are closed.

The fixed acrylic incline plane is a common choice for orthodontic correction of linguoverted mandibular canines because it is fabricated chair-side by the dentist, and therefore saves money and multiple anaesthesia events. There are a few variations on the construction of the apparatus. The device may consist of two separate units bonded to the maxillary canine and third incisors, or a span of material placed across the palate, bridging the two sides in order to strengthen the whole unit.

The laboratory manufactured modality of incline plane is the **fixed cast metal incline plane.** This modality of treatment requires series of anaesthesia and is more expensive than custom-made acrylic one.

Removable incline planes involve using a sequence of removable, vacuum-formed, semi elastic plastic aligners to place an intermittent, lateral tipping force on the mandibular canine crowns when the patient closes its mouth. One should have access either to manufactured devices ready to use in different sizes or produced in laboratory specifically for exact patient. Both are available in limited areas of the world.

There are many ways to address the problem of mandibular canine linguoversion. The clinician must determine which method will most likely succeed depending on the patient's signalment, health and temperament, the owner's compliance and financial means, and the practitioner's ability and armamentarium. This malocclusion is painful and should be corrected. Each patient deserves a healthy, comfortable and functional occlusion. Clients should be advised about the heritability of the malocclusion. Orthodontics provides a relatively straightforward fixation for this malocclusion, saving teeth from extraction or disfigurement. As long as there is a reasonable chance of success, especially with class I malocclusions, orthodontics should usually be the treatment of choice.

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Challenging periodontal disease case management

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Summary

Periodontal disease is generally described in two stages, gingivitis and periodontitis. Both are manageable although often require very complex approach. Lecture presents more complex periodontal pathology which treatment can be challenging for the operator and the pet-owner.

Notes

Apically repositioned flap

Periodontal flaps can also be sutured at different levels on the tooth. Apical repositioned flaps are utilized to move the gingival attachment apically. This reduces pocket depth while maintaining the vital attached gingiva. This results in easier care of the diseased areas and decreased infection. In general apically repositioned flap refers to mandibular or maxillary incisors. These flaps are generally used in areas with horizontal bone loss where regeneration of the lost bone is not currently possible. This is most common in the mandibular incisor area. However, an additional indication is in areas of grade III furcational defects. This procedure lowers the gingival height allowing for superior homecare in the furcational area. These flaps can also be used to widen the zone of attached gingiva. This technique requires a technical and time consuming split thickness flap to be created in order to maintain the periosteum over the bone to be exposed. This allows for the regeneration of gingival tissues above the repositioned flap.

Periodontal regeneration

Periodontal regenerative surgery is designed to reconstruct the periodontal attachment (periodontal ligament, cementum, and alveolar bone). Only periodontal ligament cells have the ability to regenerate the periodontal attachment apparatus. Gingival soft tissues recolonize faster than these periodontal structures, resulting in a long junctional epithelium. While this does temporarily resolve the infection, it is general not a lasting solution. Therefore, it is necessary to retard the downward growth of the gingival tissues and to allow the slower periodontal ligament/alveolar bone to regrow. Thus, this procedure is called "guided tissue regeneration", in that we are selecting the tissue which repopulates the site. Guided tissue regeneration (GTR) has been shown to achieve greater attachment gains for infrabony pockets when compared to straight open flap debridement. However there is only strong

GTR is performed by placing a barrier over the bony defect to allow for the alveolar bone and periodontal ligament to populate the defect rather than gingival tissues[1]. [2] While there has been significant advances and research in the area of bone augmentation/grafting materials,

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in most cases, the barrier is the key therapeutic modality (as opposed to the selected graft, if any), especially three wall pockets, where grafting does not necessarily increase attachment gains

Periodontal splinting

Periodontal splinting is designed to provide temporary stability of significantly diseased and mobile teeth during the healing period. This is because mobility will decrease the effectiveness of guided tissue regeneration. It does **not** directly lead to any long term stabilization.

It is best utilized on teeth with significant angular bone loss which may respond to GTR. It may also be utilized to stabilize slightly mobile incisors which are not significantly infected. However, it should be noted that stabilizing the mandibular incisors across the symphysis is not recommended, especially if it is expected to be in place for a significant period of time. . In some instances it is utilized for long term maintenance of a tooth (particularly show dogs). This is not a recommended technique for several reasons including:

1) It is very difficult to maintain the splint and associated teeth as the splint interferes with homecare
2) It keeps diseased teeth in the oral cavity, which allows for continued infection
3) It has the potential to damage the anchor teeth

Furthermore, it should not be used in cases of periodontic- endodontic involvement of non-strategic teeth, teeth with less than 20% bone remaining, and non-complaint clients.

Conclusion

Overall, autografts are still considered the gold standard for repair of bony defects. However, several substitutes have come a long way, and when used in combination are showing very positive results as an alternative or perhaps even superior to autografts. The major purpose of advanced periodontal techniques is to change the periodontal environment and reduce the likelihood of abnormal depth of the gingival sulcus (periodontal pocket). Additionally selective removal of the affected periodontium should provide chance to recreate normal architecture either through regenerative technique or by enhanced healing.

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Introducing endodontics: pulp capping in practice

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Summary

Preserving a vital immature or mature tooth within the periodontium is the goal of the veterinary dentist whenever possible.

Notes

Vital pulp therapy has been an accepted practice for preserving the vitality of the pulp in complicated fractures of immature permanent teeth (under 11-18 months of age) and in the treatment of malocclusion

After exposure of the pulpal tissue, immediate treatment is necessary in order to avoid complete pulpal infection. No chemically caustic or physically aggressive means of stopping the haemorrhage should be used in order to avoid pulpal necrosis. Injectable antibiotic (amoxicillin) should be given to limit pulpal infection and dental treatment must be performed as soon as possible. Adequate dental treatment in this case is partial vital pulpectomy with direct pulp capping. The purpose of the procedure is to save vital pulpal tissue. This is particularly useful when dealing with young dogs (usually less than 16 months of age) which still have an immature tooth (thin dentinal wall and more or less open apex). When the pulpal tissue dies, maturation of the tooth stops, leaving a very fragile tooth in place. This treatment can be attempted if the fracture is less than about 5 days old in a young dog. Treating older dogs or older fractures is much less likely to be successful. The teeth are scaled, and the oral cavity is rinsed with a chlorhexidine solution. The procedure is performed under aseptic conditions. The coronal part of the pulpal tissue is amputated at a depth of about 8-10 mm with a sterile round diamond bur or with a small dental excavator if the size of the cavity permits it. Hemostasis is performed on the pulpal stump with sterile cotton pellets moistened with sterile saline. Once achieved, a biologic material called MTA™ is placed over the amputated pulpal tissue. A glass ionomer cavity base is placed over it. The final obturation of the cavity is performed with composite material. Control radiographic examination is scheduled at 4 to 12 months. If the procedure is successful, the radiograph should show thickening of the dentinal wall with decrease of the size of the root canal and closure of the apex if the tooth was immature. No periapical radiolucency should be seen.

At this time, the most common indication for vital pulp therapy is treatment of traumatic malocclusions.[5] Crown reduction and vital pulp therapy of mandibular canine teeth is a common technique for treatment of linguoversion of mandibular canine teeth, especially so in the presence of class II malocclusions. The owner choice and compliance can dictate the use of crown reduction and vital pulp therapy to relieve traumatic malocclusions. Crown reduction and vital pulp therapy for the treatment of mesioversion of maxillary canine teeth (lance) is also performed.

Disarming procedures to lower canine height in aggressive animals (both canine and feline) is another use of vital pulp therapy. Crown reduction and vital pulp therapy of the mandibular canine teeth is used to treat buccal trauma in feline patients with a history of bilateral maxillary canine exodontia.

Radiographic evidence of pulpal death (such as widened root canals or periapical rarefaction), root fracture or severe periodontal disease rendering the tooth non-salvageable are all contraindications for this procedure.

Vital Pulp Therapy Technique:

a. Preparation: appropriate pre-operative work-up, anesthesia, and perioperative care

b. Dental prophylaxis (scaling, polishing, and full mouth evaluation) before initiation of vital pulp therapy should decrease intraoral bacterial load.

c. Flush the mouth with 0.12% Chlorhexidine solution

d. Utilise appropriate regional anaesthesia

e. Place a rubber dam a sterile surgical glove may be used as a dam

f. Use a sterile surgical pack and a sterilized handpiece with sterile NaCL coolant or assistant-dripped sterile NaCL flush is recommended as this is vital tissue.

g. Crown reduction is performed to appropriate height of tooth, usually to height of adjacent incisor with a taper-fissure (e.g #701) carbide bur.

h. Pulp amputation is performed with a straight or round coarse diamond bur. A coarse diamond bur may decrease hemorrhage by creating a small amount of mechanical cautery.The depth of amputation is ideally 5-7 mm to allow placement of MTA[f], intermediate layer of glass ionomer and final restoration.

i.Hemostasis is a key step. Hemostasis is achieved by placing inverted sterile saline soaked paper points onto the pulp.

j. MTA (mineral trioxide aggregate)[f] is the gold standard due to its low cytotoxicity, biocompatability, good production of dentinal bridge, and maintenance of pulp viability. Place the MTA with an amalgam carrier to appropriate width of pulp canal, making a layer 1-2 mm thick.

k. Clean walls with slightly moistened paper points. Care should be taken not to liquefy the MTA so as to not loosen the attachment of the MTA to the pulp canal wall.

l. Place glass ionomer[g] over the MTA and light cure according to manufacturer's directions. Ideally, this is 2 mm thick.

m. Prepare access for final restoration.

n. Place final restoration

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Malignant oral tumors - preparing for the worst

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Summary
Each proliferative or ulcerative lesion should be considered a potential tumor. How to proceed when the oral mass is found and what to do if it is malignancy will be the subject of the lecture.

Notes
Oral tumours represent the fourth most common malignancy in dogs and cats. Unfortunately, many of oral malignancies go unnoticed and at the moment of diagnosis reach fairly advanced stage. Review of the most common oral and maxillofacial neoplasms will be discussed, as well as available resective and non-resective treatment protocols.
Malignant oral neoplasms typically invade bone early in the course of disease, resulting in irregular, ragged bone destruction. Initially, the bone will have a mottled “moth eaten” appearance, but radiographs late in the disease course will reveal a complete loss of bone (the teeth will appear to float in space). If the cortex is involved, an irregular periosteal reaction will be seen.

Histopathologic testing is always necessary for accurate diagnosis of oral masses since a variety of benign or malignant tumours appear radiographically similar. In addition, osteomyelitis can create the same radiographic findings as malignant tumors. Finally, aggressive tumours will often show no bone involvement early in the course of disease. The prudent practitioner will note the type and extent of bony involvement (if any) on the histopathology request form (and may include copies of the radiographs and pictures) to aid the pathologist. It is key to interpret the histopathology result in light of the radiographic findings. A diagnosis of a malignancy without bony involvement should be questioned prior to initiating definitive therapy such as aggressive surgery, radiation therapy, or chemotherapy. Conversely, a benign tumor diagnosis with significant bony reaction should be further investigated prior to assuming that the patient is safe.
Additional diagnostic tests in questionable cases include complete blood panel, urinalysis, bacterial and/or fungal culture, as well as fungal serology. General physical examination may give indications of distant metastasis. Three-view thoracic radiographs are indicated in all cases of suspected malignancy. Radiographic examination (preferably dental) of the affected jaw is mandatory. With the increased availability of computed tomography (CT) and magnetic resonance imaging (MRI) these advanced imaging techniques are now more frequently used at referral institutions.

The goal of curative intent therapy is to achieve a long-term remission or tumor control. Palliative therapy is indicated for patients where a curative intent therapy cannot be performed, either because of concurrent medical conditions, advanced clinical stage, or financial concerns. Complete en bloc surgical excision with adequate margins can be proposed when definite diagnosis and prognosis are available.

Adjuvant therapy methods include immune stimulation either targeted (vaccine) or non-specific. A variety of adjunctive therapies are employed in controlling the clinical signs encountered in dogs and cats that are treated for cancer. A treatment goal for any oncology patient is to maintain quality of life by limiting treatment side effects, pain, and discomfort. This direction of treatment also includes pain management and appropriate nutrition. Recognition and alleviation of pain in oncology patients is essential for maintaining quality of life. Providing a complete and balanced diet, whether commercially available or homemade, is imperative.

Cytotoxic therapy is indicated for patients with a diagnosis of oral lymphoma, melanoma, osteosarcoma, or other high-grade malignancies which carry a metastatic potential. Conventional chemotherapy is also known as maximally tolerated dose (MTD) chemotherapy. This refers to administration of chemotherapeutic agents at the maximum recommended dose followed by a recovery period for drug-sensitive cells, such as those of the bone marrow and gastrointestinal tract. Although this approach maximizes tumor cell death and is associated with a low chance of serious side effects, the periods between treatments may also allow for tumor regrowth.

Radiation therapy should be considered in cases where complete excision is not feasible. Orthovoltage machines capable of delivering low energy external beam radiation are not optimal for treating oral malignancies in most cases. Megavoltage radiation therapy is currently the standard of care for dogs with oral tumors. Acute effects of oral radiation such as oral mucositis, alopecia, local moist dermatitis, and desquamation occur in almost every case. However, these effects are not dose limiting. These effects are reversible, medically manageable, and resolve over 2–3 weeks after completion of radiation therapy. Despite its proven efficiency the access to this treatment modality is still difficult.

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Managing oral fractures - the dentists' way!

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Summary

In the case of head trauma multiple injuries of maxillofacial structures may occur so the patient require thorough assessment prior to implementation of reparative procedures. Apart from surgical solutions there exist also less invasive "dental" methods to manage oral fractures.

Notes

Oromaxillofacial fractures can provide unique challenges for treatment. In the case of trauma, multiple injuries may be present at the same time, and may be compounded by serious complications such as neurologic damage. Treatment of traumatic oromaxillofacial fractures should be performed once the patient has been fully assessed for existing diseases and is stable to undergo anaesthesia and surgical repair.

Maxillofacial fractures in small animals are a common occurrence in veterinary practice. The frequency varies between the species. Surveys conducted showed that mandibular fractures in dogs make up 1.5 to 2.5 % of all fractures, whereas in the cat this figure varies between 11.5% and 23.1%. The higher incidence in cats is related to the fact that they present with a high percentage of symphyseal separation (fracture), usually due to "High Rise Syndrome". A high percentage of iatrogenic mandibular fractures also occur during dental procedures.

Most mandibular fractures are open to either the oral cavity or the skin. The anatomical location of fractures differs between the species. In the dog, fractures are most common in the premolar region followed by the molar region and then the symphysis. In cats they occur most common in the symphysis and then the premolar/molar region. Favorable mandibular fractures are those with fracture lines that run caudodorsally because the masseter, temporal, and pterygoid muscles insert on the caudal end of the mandible, and therefore cause compression of the fracture fragments. Conversely, unfavorable fracture lines run caudoventrally.

Prior to application the operative methods there is possibility to use non-invasive technique: a tape muzzle. Tape muzzles be used as temporary, definitive or adjunctive therapy, this is a good method in cases of pathological fractures or where the bone is very porous and will not support a fixative device. Where fractures are stable this is also a good technique.

Possible "dental" stabilisation techniques for maxillofacial fractures include: acrylic intraoral splinting, interdental wiring with composite splint or maxillo-mandibular fixation. Extracting periodontally healthy and/or stable teeth in the fracture line at the time of fracture is not currently recommended. Teeth may provide stability to the fracture repair and can always be extracted at a later date if causing a problem.

Reinforced by acrylic or composite material interdental wiring techniques are the top „dental" fracture stabilisation options. It is inexpensive technique that acts at the tension surface and can combine with other techniques. It does not disturb local blood supply.

Comminuted fractures or complex injuries can be managed with maxillo-mandibular fixation. With this method there is no disruption of blood supply to fracture segments. With proper application the acrylic device does not interfere with occlusion. Acrylic/composite can be applied directly to the teeth (bonded) or indirect by stabilising interdental wires or as a mould that can be fixed perimandibularly.

Maxillary fractures often do not require stabilization and may be more difficult to detect because displacement of the fractured parts is often minimal. Loose nylon muzzles can be used for conservative treatment. The more severely comminuted fractures can be treated with interdental fixation and acrylic splints.
Management of multiple fractures can be performed with maxillo/mandibular composite fixation.

Where these devices have been placed one can use a chlorhexidine containing oral rinse to flush into the oral cavity daily. Owners are also motivated to improve oral hygiene (toothbrushing) in order to remove plaque efficiently. Any intraoral device will increase the retentive areas for plaque and can increase the risk for periodontitis.

Once the desired effect has been reached extreme should be taken when removing the intraoral devices. Since they are attached to underlying teeth, sections should be made interdental to prevent cutting healthy teeth when the device is being removed. Composite that is attached to the teeth after removal can be removed using extraction forceps and any remainder should be ultrasonically removed or smoothed with a white Arkansas stone.

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Rabbit dentistry - common treatments

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Summary
Case based presentation of the most frequently recognised problems in rabbits: malocclusion, cheek teeth overgrowth and facial and/or mandibular abscesses.

Notes
Tooth elongation – eruption rate exceeding wear rate. This is the probably the commonest cause of dental disease in pet rabbits and presents as a progressive pattern of abnormalities. Rabbits on a low fibre and high carbohydrate diet have reduced tooth wear or attrition, resulting in elongation of the crown. It is noticeable that rabbits consuming a low fibre mixed grain or pelleted diet tend to crush these items with an “up and down” motion rather than the lateral grinding motion employed when eating a highly fibrous diet.

Periodontal disease is common in rabbits, especially as the weak structure of periodontal ligament renders it more likely to injury and food impaction. Elongation is a significant factor, especially with the cheek teeth, as this causes disruption of the tightly packed occlusal surface and the opening up of gaps (diastemas) between the teeth. Periodontal infection, often with anaerobic oral bacteria such as Fusobacterium species, or Staphylococcus. or Streptococus spp. may spread to the tooth apex, leading to endodontic lesions as the infection affects the pulp. Abscesses frequently result from periodontal infection, or mucosal damage caused by dental ‘spikes’. Unfortunately most dental abscesses result in gross changes in the surrounding tissues including the alveolar bone, so that there are residual problems even if the abscess is successfully treated. If not treated early, abscesses tend to behave as expansile masses, and they can displace teeth.

Management
When detected in its very earliest stages, uncomplicated tooth elongation can be corrected simply by dietary change. Established tooth overgrowth may be helped by repeating burring at 4 to 6 week intervals. Radiographic assessment of tooth roots is essential in all cases before undertaking treatment.
A high speed handpiece rotating at 2-400,000 times a second will cut the teeth with minimal effort, but care should be taken to avoid overheating. Clippers should never be used as they leave sharp edges and longitudinal cracks in the teeth and will often expose the pulp.

Regular crown reduction or, preferably incisor extraction, is indicated for affected animals by mandibular prognathism/maxillary brachygnathism.

Coronal reduction of cheek teeth requires general anaesthesia, and specialist mouth gags and cheek dilators. A straight slow speed dental handpiece with a long-shanked taper fissure burr is recommended. The aim of coronal reduction is to shorten the crown and attempt to restore the normal occlusal pattern.

Early caries may be eliminated by burring away the affected tissue. However, they often re-form unless the diet is corrected and the coronal reduction may result in abnormal wear of opposing teeth. Periodontal pockets deeper than 3mm are difficult to clean in rabbits.

Standard subgingival curettes may be used but small dental excavators are often more effective.

Irreversibly diseased teeth must be extracted. Incisors extraction is to be performed gently with the use of dedicated extraction kit and slim luxators. Once loosened, the tooth should be gently rotated and pressed back into the socket to destroy apical germinal tissue – failure to do this will result in tooth regrowth, and even when this is done incisors will occasionally regrow. Cheek tooth extraction can be very difficult unless the tooth is already loosened by periodontal disease. Their extraction is mostly associated with treatment of facial abscesses.

Abscesses carry a guarded prognosis in some reports, and the chance of cure can be improved by being more surgically aggressive and by packing antibiotics directly into the affected sites. The goal is to first thoroughly debride the infected area and then to maintain a constantly high level of antibiotics at the local level. Debridement consists of flushing the area with saline solution and cutting out diseased tissues. Local antibiotic treatment may be achieved in several ways. Installation of antibiotic-impregnated polymethylmethacrylate (AIPMMA) beads into the defect created by surgical removal is a common technique that allows locally high antibiotic levels with little systemic absorption. Systemic administration of antimicrobials is generally not necessary for more than 2-3 weeks post-operatively in case surgery causes a bacteraemia. However, in cases where complete excision is not possible, long term systemic antibiosis may be necessary

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What went wrong? Mistakes and malpractice in small animal dentistry

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Summary
In medicine, errors occur but they may occur after intentional or unintentional actions. The first group is classified as malpractice the second is medical error.

Notes
Small animal dentistry is a very technically challenging discipline. At each stage of a procedure there are steps, which must not be neglected. For the purposes of this presentation, the author focuses on three of the most popular procedures in veterinary dentistry: diagnostics, prophylactic procedures and extractions. These three procedures statistically comprise 75% of daily dentistry regardless of whether they are performed in general or specialist practice. Since all dental procedures are performed under general anaesthesia and many of them may cause postoperative pain, very strict standards of anaesthesia and appropriate pain management are essential.

An anaesthesia and pain management protocol requires an initial preoperative laboratory workup. The dental procedure always requires intubation, protection of the respiratory tract by insertion of a pharyngeal pack, monitoring of vital functions (pulse, blood pressure, capnography, temperature maintenance) and fluid therapy. Depending on the extent and severity of the procedure, preoperative pain management is used and an adequate nerve block should be performed. Intraoperatively, the most important way to minimise pain is to use a minimally invasive approach. Post operative pain management depends on the kind of procedure as well as on the individual needs and conditions of the patient.

Diagnostics. This heading includes clinical assessment and radiography. A complete oral examination and charting is necessary to make a treatment plan for discussion with the client. A initial thorough examination is made in the conscious patient as a reliable assessment of occlusion can be made in this way. After sedation, the oral examination is continued with the use of a periodontal probe, dental explorer, mirror and if necessary retractors. All oral structures must be thoroughly examined and the measurements taken must be charted. It is important during periodontal probing to measure the probe depth in at least six different places around each tooth. Oral imaging is a complementary part of the dental assessment and can include entire head radiography as well as dental radiography. The value of the radiographic evaluation in veterinary patients has been proven in studies, which found 27.8 % of clinically important lesions in dogs and 41.7% in cats would be missed without full mouth radiography. Currently, three advanced diagnostic techniques can be used to obtain optimal information for diagnostic consideration. These are: MRI (Magnetic Resonance Imaging), CT (Computed Tomography) and the newest one which is proven to be excellent for dental purposes: CBCT (Cone Beam Computed Tomography). In patients evaluated after maxillofacial trauma, CT scans demonstrated 1,6 times more injuries in dogs and 2 times more in cats than conventional radiographs.

Prophylactic procedure. Prior to scaling, an oral rinse with chlorhexidine solution reduces contamination and decreases bacterial load. Mechanical scaling requires the use of appropriate length tips. The application of the tip to an individual tooth must not last for longer than 15 seconds at any one time. The working part of the tip is the distal 1/3 where the area of maximum vibration occurs. The working tip is applied gently to the enamel in a featherlike parallel, not perpendicular, style. For subgingival scaling, only the use of periodontal tips is allowed. Periodontal hand instruments are effective only when sharp. Even careful scaling may result in enamel abrasions and a rough surface. Polishing smoothes the tooth surface. It is performed with a rubber prophy cup with a rotation speed not faster than 3000 rpm and for not longer than five seconds per tooth. Flour of pumice with or without the addition of chlorhexidine is the most commonly used medium for polishing. It is important to use either disposal containers or a dappen dish for each patient, which can be sterilised and reused. Possible complications which may occur during the prophylactic procedure are: overheating of the vital structures which can result in pulpitis or alveolar bone necrosis as well as incomplete removal of dental deposits or incomplete smoothing of a rough surface. The latter allows faster accumulation of plaque immediately after the prophylaxis and worsening of the situation. Such a situation always occurs after anaesthesia free dentistry. According to an AVDC EVDC AVD statement, so-called anaesthesia free dentistry is harmful to the patient and is not acceptable. The final part of the prophylactic procedure is gingival sulcus lavage with saline or 0,12% chlorhexidine solution and optionally fluoridation and/or the application of a barrier sealant. Every client, must receive after-treatment home care recommendations every time and be given a date for a recheck.

Extractions. An extraction is the most common surgical procedure in the oral cavity and statistically results in the largest number of possible complications due to medical errors. The list of standards to be met is as follows: adequate nerve block; preoperative radiography, gingival attachment cutting, surgical access to the root and periodontal space, cutting and tearing of the periodontal ligaments with the proper use of luxators and elevators; removal of the tooth, postoperative radiography, alveolar debridement (if required); smoothing of any sharp edges; preparation for closure, tension-free suturing. Neglecting any one of these steps can be interpreted as an error although it may not always cause complications. The list of possible complications following extraction are: incomplete removal of the tooth structure, iatrogenic fracture of the surrounding bone, iatrogenic damage to the surrounding soft tissues, osteomyelitis following excessive tissue damage, dry socket due to contamination left in the alveolus, a non-healing wound or wound dehiscence, oronasal fistula, traumatic malocclusion, infection, emphysema, malfunction of the mouth or tongue. This list does not include the iatrogenic complications like penetration of eye, jaw fracture or damage of the adjacent tooth. A good example showing the difference between a genuine mistake and malpractice is the situation when an infected root is fractured during extraction. Most if not all dentists have experienced this problem. It is a mistake but very common and not intentional. The key is to evaluate radiographically after extraction and if there is visible root remnant, it should be removed. If it is left in place, it is undoubtedly malpractice, which will be inevitably associated with pain and infection.

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One Health System Strengthening

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Introduction

This paper describes a group of 6 One Health research projects being conducted in South East Asia and the Pacific that aim to strengthen connections between animal and human health agencies and research organisations, address country-specific priorities and inform policy decisions around antimicrobial resistance and specific zoonotic diseases. This program will be of interest to Australian veterinarians because of the wider implications it has in terms of informing the discussion around the costs and benefits of One Heath collaborative work and the implication for policy decisions both within our region and at home.

Program Overview

Australia's Health for Development Strategy, 2015-2020, emphasises the role of strong health systems, innovative research and regional collaboration and linkages in improving health security. This is in line with Sustainable Development Goal Target 3.d: to "strengthen the capacity of all countries, in particular developing countries, for early warning, risk reduction and management of national and global health risks". Health risks include zoonotic diseases with epidemic and pandemic potential such as highly pathogenic avian influenza and neglected tropical diseases such as rabies.

Whilst One Health has strong in-principle support from the World Health Organization (WHO), Food and Agriculture Organization (FAO) and World Organisation for Animal Health (OIE) and major international donors, serious questions remain around its operationalisation, particularly in low- and middle-income countries faced with a range of health priorities. It is for this reason that applied One Health research for development - showcasing how best to develop One Health approaches within existing policy and governance frameworks in countries with many competing health and development priorities – has the potential for Australia to positively influence regional health security.

Research areas

1. Antimicrobial stewardship in the Pacific
2. Zoonotic vector-borne disease control in Australia's near neighbours
3. Community-level health literacy and risk perception in the Greater Mekong Region
4. Policy process analysis for collaboration between health and agricultural sectors in the Greater Mekong Region
5. The contribution of *Mycobacterium bovis* to human tuberculosis in the Pacific.

These projects will inform a longer-term program by facilitating dialogue, and promoting partnerships, between Australian public and animal health institutes and their international research partners.

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Practical approach to the Neurological rabbit

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Introduction

The neurological rabbit is a common presentation to exotic, emergency and small animal clinics. Often neurological signs occur acutely, and owners will present to their closest veterinary clinic for urgent assessment and treatment.

There are many causes of neurological disease in rabbits and this paper will aim to address the most commonly encountered conditions. This will include otitis media, *Encephalitozoon cuniculi* and 'Floppy Rabbit Syndrome'.

Symptoms of Neurological disease

Rabbits show very similar signs of neurological disease to cats and dogs, such as:

1. Head tilt
2. Unilateral facial paralysis
3. Nystagmus*
4. Flaccid paresis and paralysis of multiple limbs
5. Loss of the swallowing and gag reflexes

*In the author's opinion, horizontal, vertical and multidirectional/circling forms of nystagmus are not reliable locators of a neurological lesion.

What is Floppy Rabbit Syndrome?

Floppy rabbit syndrome (FRS) describes the presentation of flaccid paresis or paralysis in rabbits and is **not** itself a diagnosis. The syndrome is not synonymous with any one condition, despite often been used interchangeably with *E. cuniculi* infection in rabbits. The flaccidity may affect both hindlimbs, all four limbs or in severe cases, affect all limbs and the cervical muscles. Most rabbits with FRS still have a normal demeanour and will show interest in eating and drinking. However, as rabbits with FRS are very reliant on supportive care, they can decline rapidly due to hypoglycaemia, gastrointestinal ileus, dehydration or hypothermia.

There are many suspected causes of flaccid paralysis in rabbits and often the exact cause is unknown. Potential causes include:

1. Toxicity – heavy metals, certain plants (see the Victoria state government's website for a list)
2. Infection – *E. cuniculi,* coccidiosis, *toxoplasma gondii* and *clostridium botulinum*
3. Trauma – spinal fractures or compression
4. Metabolic causes – hypocalcaemia, hypokalaemia, low vitamin E/selenium or hypoglycaemia in young rabbits
5. Immune-mediated causes – meningitis, myasthenia gravis or polyradiculoneuritis
6. Neoplasia – brain or spinal cord
7. Vascular accidents

Encephalitozoon cuniculi

E. cuniculi is a microsporidian parasite that can infect the ocular, brain and renal tissues in rabbits. In Australia, it is estimated that approximately 40-50% of rabbits carry E. cuniculi and in many rabbits it will stay latent in the brain tissues lifelong. However, in the immunosuppressed rabbit, E. cuniculi can reproduce and cause a granulomatous meningitis. Other forms of E. cuniculi-related disease include renal deficiency and in the case of a foetal in-utero infection, E. cuniculi can also cause uveitis and cataracts to develop.

Management of E. cuniculi-related disease depends on the organ affected. Fenbendazole is widely used at 20mg/kg SID PO x 28 days to suppress the E. cuniculi parasite but is not curative treatment. Ongoing treatment with fenbendazole in E. cuniculi-positive animals is recommended lifelong by the author, with the initial course being for 28 days, and subsequently for 9 days every 6 months. Fenbendazole has been associated with bone marrow suppression, though in the author's experience at this dosing frequency the suppressive effects are typically limited to mild anaemia.

More specific treatment depends on the presenting signs. For renal cases, fluid therapy, pain relief and management of any associated gastrointestinal stasis is often required. In ocular forms of the disease, treatment of cataracts is rarely indicated, though uveitis cases will require pain relief and in some refractory cases, enucleation.

Finally, in the neurological presentations of E. cuniculi, the importance of supportive care should not be underestimated, particularly in the cases of flaccid paralysis. Regular assisted feeding, rehydration, physiotherapy and management of faecal and urinary staining is essential to the patient's recovery. In the case of head tilts or facial paralysis, prochlorperazine has been successfully used by the author to improve quality of life during the acute to subacute stages of recovery (refer to Table 1 for doses).

Varying combinations of meloxicam, ranitidine and simethicone may be required depending on the patient's presenting condition.

It should be noted that the prognosis in suspected cases of E. cuniculi is highly variable. For rabbits that present with only hindlimb paresis, the prognosis is often good for a full return of neurological functions. However, in cases of cervical and/or tetraparalysis, the prognosis is guarded. The prognosis can be improved will intensive supportive care and early medical intervention.

Cases with a head tilt or facial paralysis often improve with fenbendazole and analgesia, however, some degree of these signs may remain after the acute signs of disease are treated.

Floppy Rabbit Syndrome and E. cuniculi

The relationship between FRS and E. cuniculi is still under investigation and is not well understood. Many rabbits that present with FRS are also found to have antibodies against E. cuniculi on Immunofluorescent assays (IFA). However, it should be noted that many rabbits with FRS have also repeatedly tested negative to E. cuniculi, whilst others that test positive may never develop any clinical signs (refer to Table 3).

Given this, it is important to consider E. cuniculi as a potential cause of FRS, though it is by no means the only cause of this syndrome.

Otitis media

Otitis media is a common cause of unilateral facial paralysis, nystagmus, ataxia and head tilting in rabbits, due to the presence of the peripheral vestibular nerve in the tympanic bulla. In cases where there is no obvious trauma, otitis media and E. cuniculi should both be considered, along with less common conditions such as cystic or neoplastic compression of the vestibular nerve and/or nuclei.
The presence of otitis media can be evaluated on imaging. In the author's experience, whilst radiographic assessment is widely available, it is not as sensitive or accurate as CT, and otitis media should not be ruled out with the use of x-rays alone.
Radiographic findings consistent with otitis media include:

1. Replacement of the gaseous appearance of the tympanic bulla's lumen with soft tissue
2. Asymmetry of the tympanic bullae
3. Lysis or thickening of the temporal bone

Treatment of otitis media can be attempted medically or surgically. Medical management is often unrewarding but can be offered pre-operatively to prevent progression of the infection or instead of surgery where this option is not available (e.g. financial constraints or a poor surgical candidate). Medical management may include long term parental penicilins, meloxicam and in some cases gabapentin. Surgical treatment is recommended via a Partial Ear Canal Ablation and Bulla Osteotomy (PECABO) procedure, though post-operative head tilts and facial contracture are commonly cited complications. This procedure also does not prevent the recurrence of otitis media and often extended courses of antibiotics are offered post-operatively to reduce the chance of this occurring.
Strong multimodal analgesia such as buprenorphine and fentanyl should also be provided peri- and post-operatively to patient's undergoing this surgery.

Table 1. Medical approach to the Neurological rabbit

Specific medications	Fenbendazole	20mg/kg PO SID x 28d
Pain relief	Meloxicam	0.5mg/kg PO BID x 5-7d
	Tramadol*	4-11mg/kg PO BID x 3d *Variable analgesia with moderate sedation
	Gabapentin	2-5mg/kg PO TID x14d+
	Buprenorphine	0.03-0.06mg/kg SC, IM q4-6hrs
	Midazolam	0.2-1mg/kg IM
Gastrointestinal support	Simethicone	0.4ml PO BID-TID
	Ranitidine	2-5mg/kg PO BID
	Cisapride*	0.5mg/kg PO BID-TID *Avoid in cases of GIT obstruction
	Critical care*	10ml/kg/4hrs *Mix 1:4 to make up solution
Neurological support	Prochlorperazine (stemetil)	0.2-0.5mg/kg PO q8hrs
Fluid therapy	Maintenance	100-150ml/kg/24hrs
	Surgical rates	10ml/kg/hr
Antibiotics	Procaine Penicillin	0.5ml/kg SC *Never give orally- contraindicated
Other considerations	Bladder Care	Express 2-3 times a day
	Temperature management	<37.0°C active warming is required
	Physiotherapy	Extend and flex any paralysed limbs and alternate sides of lateral recumbency to prevent GIT issues and ulcer formation.

Table 2. Medications to avoid in rabbits

Metoclopramide, cisapride	Used for cases of gastrointestinal ileus. Should be avoided in cases of suspected gastrointestinal obstruction. NB: the effectiveness of metoclopramide as a prokinetic in rabbits is questionable, in the author's opinion.
Corticosteroids	Rabbits are a corticosteroid-sensitive species that develop gastrointestinal side effects more readily as well as long-lasting endogenous suppression of their immune system.
Oral penicillins, β-lactams	Due to the largely gram positive, anaerobic nature of the rabbit's gastrointestinal microflora and their heavy dependence on these organisms, oral penicillins and beta-lactams are considered contraindicated in the species.

Table 3. Diagnostic approach to the Neurological rabbit

Blood smear	Assess for signs of infection (leucocytosis or toxic changes). E. cuniculi is often associated with a monocytosis.
Biochemistry	Rule out hepatoencephalopathy, hypoglycaemia, hypokalaemia and acute renal failure.
E. cuniculi Immuno-fluorescent assay	Assess for E. cuniculi infection status – note that this test does not differentiate between carriers and actively diseased individuals.
Imaging	Radiographs and CT are useful to assess for spinal fractures or trauma. CT can be used to assess for middle ear infections, whilst MRI is useful to assess for intracranial lesions.
Electrolyte panel	Useful for assessing for hypocalcaemia through ionised calcium levels

Table 4. Example of a 'typical' Floppy Rabbit case

Presenting complaint	Not moving
Signalment	Mini lop, castrated male, 1.5 years old, approximately 1.3kg
History	The owner reports the patient was normal yesterday (was eating, drinking, urinating and defaecating normally), but this morning the patient isn't able to move. When the owner placed vegetables in front of the patient, he seemed interested in this and ate a few leaves. The other rabbit in the household appears normal. The owner reports that she did have some friends and their kids over for dinner last night, but otherwise no other environmental changes.
Physical exam	• Temperature: 37.1°C – mild-moderate hypothermia • Pulse and respiratory rate normal • Thoracic auscultation normal • Gut sounds moderately reduced and no faecal pellets palpable in the abdomen • Approximately 7-8% dehydrated and moderate moult • Flaccid paralysis of the all four limbs, however, still able to support his head and swallow. No pain on spinal palpation and no withdrawal reflexes in the hindlimbs. Still some tone present in the forelimbs – moderate paresis.
Diagnostics	Recommended full bloods with a E.cuniculi IFA and blood glucose
Results	• E.cuniculi IFA pending (takes 2-4 days) • Blood glucose 3.8mmol/L (low) • Blood smear shows mild monocytosis with some toxic changes, otherwise is normal • Biochemistry shows a moderate hyperglobulinaemia
Treatment	Advised 24-48 hours of hospitalisation to correct the dehydration, with assisted feeding to address the hypoglycaemia and reduced gut sounds. Recommended symptomatic management for E.cuniculi-related neurological disease given the reasonably normal blood results and history of environmental stress (new people in the household). Advised fair to poor prognosis given the presence of neurological signs in all four limbs. <u>Started the patient on:</u> • 5mg/kg ranitidine PO BID • 0.5mg/kg meloxicam PO BID – to be given after the first two hours of hydration • 20mg/kg fenbendazole PO SID • 10ml/kg IVFT Hartmanns fluids through the marginal auricular vein • 10ml of Critical Care (herbivore supplementary food) every 2-3hours, and assessing for improvement through gut sounds and faecal production • Active warming through ambient heat cage (set to 30°C), with rechecking the temperature every 2 hours and stopping active warming when the patient reaches 37.8-38.0°C.

International Perspectives on One Health

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Global One Health

One Health (OH) is a broad movement that recognises the fact that human, animal and ecosystem health are interdependent and that multidisciplinary collaborations are often necessary in order to attain optimum health solutions. OH is endorsed by global standard makers, namely, the World Organisation for Animal Health (OIE), the World Health Organization (WHO) and the Food and Agriculture Organization of the United Nations (FAO), as well as being supported by the World Bank.

The last two decades have seen a large number of conferences and a series of international inter-ministerial meetings with a focus on emerging infectious disease, and, increasingly on antimicrobial resistance. There are currently around 20 universities offering OH graduate degree courses, including institutes in America, Africa, Europe and Asia. The OH Initiative acts as a clearinghouse for information on OH (http://www.onehealthinitiative.com), the One Health Global Network operates a web-portal (http://www.onehealthglobal.net/) to facilitate communication and the One Health Commission also shares information (https://www.onehealthcommission.org/). The EcoHealth Journal publishes on EH and articles on both OH and EH have appeared in other major epidemiology and infection journals.

However, despite this large and growing body of evidence supporting the usefulness of OH, the great majority of medical education, clinical practice, ancillary services, development programmes and research continue to operate within disciplinary boundaries.

The International Livestock Research Institute (ILRI) is based in Nairobi, Kenya and has offices in several countries in Africa and Asia. Its mandate is to conduct livestock research with the objectives of reducing poverty, improving human nutrition and health, and safeguarding natural resources in developing countries. ILRI has been involved in OH and Ecohealth for decades and this paper summarizes research, findings and impact for four important emerging diseases (highly pathogenic avian influenza (HPAI), middle eastern respiratory syndrome (MERS), Ebola and Rift Valley fever).

Highly pathogenic avian influenza

Highly pathogenic avian influenza (HPAI) represents a threat to poultry industries worldwide and to people's livelihoods, and a potential threat to human health. HPAI Asian H5N1 is especially deadly for poultry. The virus was first detected in 1996 in geese in China and human cases were first detected in 1997. Because of its ability to cause human cases, and the possibility it could evolve to cause a pandemic of human disease, the re-emergence in 2003 was of great concern. ILRI is not a first response agency, but as the crisis deepened, started to develop an institutional strategy for research support which included the following.

1. Supporting surveillance and response in Africa: ILRI provided training in laboratory techniques, participatory epidemiology, GIS-based risk mapping and use of risk maps. A suite of risk maps was developed for Africa. A trained team diagnosed peste des petits ruminants (PPR) in Nigeria, which led to an effective emergency disease control program. In 2008, a participatory disease surveillance PDS program was introduced in Egypt, with technical support from ILRI. By 2014, it was covering 30% of Egypt districts, again demonstrating potential to deliver impact at scale. In 2011,

when the overall HPAI surveillance slowed down due to the socio-political situation associated with the "Arab Spring," the PDS program proved resilient contributing over 50% of the confirmed cases in 2012.

2. Develop approaches to inform response in Indonesia: By 2009, the PDS programme was operating in 27 out of 33 provinces of Indonesia. About 20,000 villages (30% of all villages in Indonesia) and two and a half million backyard poultry producers were covered by surveillance, control and prevention activities. The longitudinal study found vaccination was effective in backyard poultry (Bett et al., 2015). Incidence of HPAI declined by 12% in the HPAI-vaccinated group and by 24% in the HPAI + Newcastle disease-vaccinated group. However, vaccination appeared not to be feasible as an open-needed program, because the cost of avoiding one poultry death (USUS$ 8-22) was far greater than the value of a bird (Lapar et al., 2012).
3. Evaluation of HPAI control in Nigeria: When highly pathogenic avian influenza (HPAI) of the subtype H5N1 hit Nigeria as the first African country in early 2006 the Federal Government of Nigeria requested a World Bank (WB) International Development Association credit of USUS$50 million-equivalent, provided under the Global Program for Avian Influenza and Human Pandemic Preparedness and Response (GPAI) to fight ongoing outbreaks. ILRI was also asked to conduct an independent evaluation of avian influenza response in Nigeria. A broad-reaching multi-disciplinary evaluation found overall positive impacts but drew attention to areas that needed to be addressed to ensure benefits were sustainable

Middle East Respiratory Syndrome (MERS-CoV)

Middle East Respiratory Syndrome coronavirus (MERS-CoV) is an emerging virus first identified in 2012, in Saudi Arabia. The case fatality for infected humans with overt respiratory symptoms is in the region of 30%. As with many coronaviruses, MERS is thought to originate in bats, though camels appear to play a significant role in maintaining the virus and in transmitting it to humans. While the virus appears to be poorly transmissible to humans, it can cause human-human infections. Indeed, the largest outbreak of the disease was in South Korea in 2015 when human-to-human transmission resulted in hundreds of infections.

ILRI and partners have explored the role of camels in the epidemiology and transmission of MERS-CoV, with a focus on Kenya. The first important work was to mine biobanks of historical camel samples (Corman et al., 2014), as far back as the early 1990s, which confirmed extensive exposure of camels to MERS or MERS-like viruses. High seroprevalences (up to 50%) were identified in herds in central Kenya (Deem et al., 2015) and in herds from locations around the whole country. Liljander et al. (2016) identified one human seropositive in Kenya.

Ebolavirus

Ebola was first described in 1976 with two simultaneous outbreaks in the Democratic Republic of Congo (DRC; Zaire at the time) and South Sudan. It has since been responsible for several deadly outbreaks, most recently in 2013-2016 in West and central Africa and presently in eastern DRC. Many aspects of Ebola epidemiology remain unknown, such as the virus reservoir and the role of wildlife and domestic animals in its transmission. However, there has been long-standing concern that domestic livestock, in particular swine, might have a role in disease epidemiology (Atherstone et al. 2014). Pigs in the Philippines were shown to be infected with Ebola Reston, a type of Ebola which is related to the Ebola found in Africa but which does not affect people. Moreover, pigs have been experimentally infected with Ebola Zaire and antibodies to Ebola have been found in pigs in Africa.

In Uganda, ILRI is supporting a gradual intensification of pork production and pork value chain as a way of offering new forms of livelihoods to livestock farmers and value chain agents, capitalizing on the increased demand for pork in the country. Uganda has also experienced several Ebola outbreaks, with four outbreaks reported since the disease was first identified accounting for 606 cases (probable and confirmed) and 283 deaths. An Ebola outbreak would be very harmful to pig value chain development, and in Kenya, motivated by improving risk management, ILRI has conducted ex-ante assessments of the potential health risks associated with the pig value chain in the country (Atherstone et al. 2017). This work is pioneer in the country and does not only inform a “healthy” or “risk-free” development of the pig production and pork value chain in the country but is giving an important basis to understand the missing links in the complex epidemiology of Ebola disease.

Rift Valley fever

Rift Valley fever (RVF) is a mosquito-borne viral zoonosis that mainly affects sheep, goats, cattle, buffaloes and camels. Humans become infected following a bite from an infected mosquito, or after close contact with acutely infected animals or infected tissues. In people, the disease manifests as a mild influenza-like syndrome in a majority of cases (> 80 per cent) or a severe disease with haemorrhagic fever, encephalitis, or retinitis in a few cases. In livestock, the disease manifests as extensive abortions and perinatal mortality. RVF outbreaks occur after periods of above-normal precipitation associated with the warm phase of El Nino/Southern Oscillation (ENSO) phenomenon (Bett et al., 2017).

Data from studies identifying rainfall patterns associated with increased risk of RVF provided the basis to develop dynamic models for evaluating RVF transmission dynamics. The model allowed the definition of alternative vaccination strategies for RVF. ILRI also conducted extensive research into the economic and health burden of RVF. On a macroeconomic basis, we estimated that RVF induced losses of over Ksh 2.1 billion (USUS$32 million) on the Kenyan economy, based on its negative impacts on agriculture and other sectors (transport, services, etc.) alike (Rich and Wanyoike, 2010).

ILRI, in partnership with the Kenya Department of Veterinary Services and US Centres for Disease Control, Food and Agriculture Organization of the United Nations (FAO) and African Union Interafrican Bureau for Animal Resources (AU-IBAR) has developed an RVF risk map, contingency plans and a decision model to be used jointly to determine interventions during an RVF outbreak.

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No food security without food safety: lessons from low and middle income countries

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Introduction

Meat, milk, eggs and fish are important sources of the micro-nutrients and high quality proteins essential for growth and health. In poor countries, livestock and fish feed billions. In developing countries, most of the meat, milk, eggs and fish produced are sold in traditional, domestic markets, lacking modern infrastructure and without effective food safety regulation and inspection. Concerns over food sold through these informal markets has been heightened by the landmark Global Burden of Disease studies which found that diarrhoea is among the most common causes of sickness and death in poor countries; as much as half is linked to animal pathogens or animal-source foods.

Food-borne illness and animal disease are of growing concern to consumers and policymakers alike. Consumers respond to scares by stopping or reducing purchases, with knock-on effects on smallholder production and informal market sellers. Policymakers often respond to health risk by promoting industrialisation and reducing smallholder access to markets. These changes are often based on fear not facts. Safer food can generate both health and wealth for the poor but attaining safe food production in developing countries requires a radical change in food safety assessment, management and communication.

Food safety and nutrition:

Stunting, or extreme shortness (very low height-for-age), is the result of a combination of long-term (chronic) poor dietary intake in terms of quality as well as quantity of food and repeated infectious disease episodes. Both wasting (extreme thinness, or low weight-for-age) and stunting are associated with increased mortality as well as poor health and longer-term development outcomes. FBD and hazards may contribute to both wasting and stunting through additional pathways, summarised in Grace (2017) and Grace et al. (2018):

- Diarrhoea is associated with malnutrition, but a causal link is hard to demonstrate; a 9-country study found that 25% of stunting could be attributed to experiencing more than four episodes of diarrhoea before the age of 24 months. Studies find a strong peak in diarrhoea after the introduction of supplementary foods, and find that weaning foods often have high levels of microbial contamination and adulteration.

- Aflatoxins may directly contribute to stunting, and there are demonstrated associations between higher toxin levels and poorer growth in several contexts, although a causal relation, while plausible, is as yet unproven.

- Ingestion of animal faecal material through food or from the environment may contribute to environmental enteric dysfunction.

Cost of foodborne disease

Only recently has systematic and comprehensive evidence on the health burden of FBD in developing countries started to become available. The landmark first assessment of the global burden of FBD, conducted by the World Health Organization (WHO), considering 31 hazards for which there was enough information to allow global burden estimates, was published in 2015 (Havelaar et al. 2015). This shows that FBD has a health burden comparable to malaria, HIV/AIDS and tuberculosis. Most (98%) falls on developing countries and 40% on children less than five years of age. The global burden of FBD caused by the 31 known hazards considered in 2010 was 33 million DALYs: children under five years bore 40% of this burden.

More recently, a World Bank study estimated the economic costs associated with foodborne disease. This was based on “productivity losses,” as measured by gross national income per capita and associated with disability or premature death captured in DALYs. The total productivity loss associated with FBD in developing countries was estimated at US$95.2 billion a year. By region, LMICs in Asia account for US$63.1 billion, and those in Sub-Saharan Africa for US$16.7 billion. The cost of treating foodborne illnesses should be added to this. These are estimated at US$15 billion a year in LMICs (Jaffee et al., 2018).
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Key messages from ILRI research on food safety in informal markets

ILRI and partners have been conducting research on food borne disease in informal markets for over a decade. Some of the key lessons are summarised in Roesel & Grace (2014).

- Informal markets are highly preferred and likely to persist

Informal markets are the most important source of meat, milk and eggs for poor people in Africa and Asia and will continue to be so for at least the next decade. Informal markets often sell food at lower prices, but they have other desired attributes including food freshness, food taste, livestock products from local breeds, trust in the vendor, credit or other services.

Moreover, research in West Africa and India suggest that as value chains become longer and more complex, so hazards increase. However, a series of studies in informal milk and meat markets showed that although hazards are always common in informal markets, risk to human health is not necessarily high. For example, data from East Africa showed that milk had many hazards but less risk (mainly because of consumer practices in boiling). In Nigeria, however, there was a clear link between consumption of beef and increased illness.

The take-home message is that risk to human health cannot be assumed for informal markets: evidence is required.

- Perception is a poor guide for risk managers

Proper risk assessment is needed to understand the source of risk. For example, dairy cattle are the reservoir of cryptosporidiosis, a serious disease for infants and people with HIV. Yet in Nairobi, risk was associated with vegetable consumption and not milk. Similarly in Vietnam, although pork meat in live animal markets had high microbial loads, increased diarrhoea was associated with consumption of vegetables, not meat. Furthermore, studies in East Africa, North-East India and Vietnam came to the surprising conclusion that food sold in formal markets, though commonly perceived to be safer, may have lower compliance with standards than informally marketed food. This emphasizes that food safety policy should be based on evidence and not perception, and failure to do this may be prejudicial to the poor who dominate and rely upon informal value chains.

- Draconian food safety policy makes things worse

Analyses of food safety in six countries revealed that stakeholders often blame insufficient legislation or lack of strict implementation for poor food safety. Paradoxically, legislation can increase the level of risk. Work in Kampala showed the importance of poor dairy farmers as risk managers and the paradoxical effects of conventional policy. Thirty practices were described which were used spontaneously by farmers that reduced risk. Moreover, farmers who had experienced harassment by authorities or who believed urban farming to be illegal used significantly fewer risk managing practices.

- Food safety is a fixable problem

Studies on milk in Kenya and India, and meat in Nigeria, have shown that simple interventions can lead to substantial improvements in food safety. These interventions involved training, simple technologies (such as use of wide-necked vessels for milk which are easier to clean), social approval, tests for food safety which can be applied by traders and consumers (e.g. lactometers to check for added water) and certification of trained vendors. In Nigeria, butchers were trained in hygienic meat handling and given hats, aprons and stickers to signal their training. After the training, 20% more meat samples met standards. The intervention cost US$9/butcher, but resulted in savings of $780/butcher/year from reduced cost of human illness.

Acronyms
FBD, Food Based Disease; DALY's, Disability Life Adjusted Years; ILRI, International Livestock Research Institute

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Ins and Outs of Bull Insurance

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Introduction

There is always pressure to claim the breakdown of a bull or a loss of use under insurance. This can readily place the veterinarian in an ethical dilemma.
Attention to detail in both an insurance proposal and a future claim should allow for a hassle free claim process.

Purpose of Insurance

Definitions

Wikipedia: Insurance is a means of protection from financial loss. It is a form of risk management primarily used to hedge against the risk of a contingent, uncertain loss.

John Lehman: Livestock insurance is a contract by which the insurer agrees to indemnify the insured against such lossor damage as he may sustain by reason of injury to, or the death of, livestock by the happenings of the perils specified, (*see terms of contract*), or a contract to pay a certain sum of money on the death of an animal from disease or accident".

Ethics

The ethical question is always: Is it a genuine insurance claim or is it a claim back against the vendor for faulty goods? What is the long term effect of a bogus claim and not placing the onus back on the breeder/vendor?
All contracts have clauses on pre-existing conditions. With livestock, when structural weakness and predisposition lead to breakdown or death, is it ethical to claim against the insurance to protect the vendor against financial loss even if the client is paid out in full?

Insurance Proposal

Most livestock insurance is handled at stud stock sales following the fall of the hammer. This is done on the assumption that the animal/s are presented in a sound and healthy condition as breeding animals. BULLCHECK is a credible Insurance Proposal document and carries much weight with the insurers as a guide to the suitability of the animal as an insurance subject when completed comprehensively

Insurance Claim

A positive and accurate identification of the animal must be made. The history of the animal may be difficult to establish particularly in extensive areas but all salient points should be recorded. In the event of a dead animal in a decomposed state, the cause of death may not be possible but a credible hypothesis is essential.

Cardiovascular derangements in the sick horse

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Introduction

Cardiovascular derangements are a common finding in the critically ill horse, largely seen in conjunction with gastro-intestinal disease and sepsis. The aims of this talk are to evaluate the causes and manifestations of cardiovascular derangements in the critically ill horse caused by both SIRS and sepsis, but also when the underlying disease process is cardiac in origin.

SIRS and the cardiovascular system

Although there are many causes of critical illness in horses and foals, many present with similar clinical signs. Most are tachycardic and hypotensive, the latter partly due to vasodilatation. These animals however often have a normal to increased cardiac output[1, 2] and are in hyperdynamic hypovolaemic shock with some degree of distributive shock. There are also primary cardiac conditions that are much rarer to identify in horses compared with human patients and small animals. These can mimic SIRS and in the adult horse present with signs consistent with abdominal pain or with oesophageal obstruction.

Causes of cardiovascular dysfunction in SIRS and sepsis

Cardiac dysfunction in sepsis is characterised by decreased contractility, impaired ventricular response to fluid therapy, and in some human patients', ventricular dilatation. Current data support a complex underlying physiopathology with a host of potential pathways leading to myocardial depression. Circulating factors such as cytokines (TNF-α, IL-1β), lysozyme c and endothelin-1 have direct inhibitory actions on myocyte contractility. Nitric oxide plays a complex role in sepsis-induced cardiac dysfunction. Current data suggest it has both deleterious and positive effects on the myocardium determined by the nitric oxide sub-type expressed. Recent studies have shown that mitochondrial dysfunction and apoptosis also play a role in the development of sepsis-induced cardiac dysfunction. Current treatment for sepsis-induced cardiac dysfunction is based on appropriate treatment for the primary focus (antibiotics and source control) and hemodynamic support (fluids, vasopressors, and inotropes).[3] In several small studies in human patients[4] with sepsis and dogs with gastric dilatation-volvulus,[5] cardiac troponin I concentrations has been shown to be predictive of the likelihood of survival and predictor of the severity of dysrhythmias present. It has been suggested that increased cardiac troponin concentrations may help to identify high-risk patients, who should undergo echocardiography, more invasive hemodynamic monitoring, and optimised therapy.[4] Results in horses are less clear and one study undertaken in horses with strangulating small intestinal lesions did not find an association with cardiac troponin I concentrations and mortality, but due to high survival rates was likely underpowered to evaluate this.[5] A more recent study in horses found 53% of horses had an abnormal cardiac troponin concentration at one time point and was correlated with surgery, identification of ventricular dysrhythmias and outcome,[6] but another study did not find an association with outcome.[7] An experimental study in horses found that an endotoxin infusion resulted in increases in cardiac troponin I concentrations, but would have only been slightly outside the normal reference range for horses.[8]

Types of cardiac dysfunction identified in SIRS and sepsis

Experimentally in horses, it has been shown in a hypovolaemia model that left ventricular internal dimensions reduce and free wall thickness increases.[9] However echocardiographic changes in either experimental models of sepsis or in clinical cases have not been undertaken to the authors' knowledge. However, in human patients, myocardial dysfunction, characterised by transient biventricular impairment of intrinsic myocardial contractility, is a common complication in patients with sepsis. Left ventricular systolic dysfunction is reflected by a reduced left ventricular stroke work index or, less accurately, by an impaired left ventricular ejection fraction (LVEF).[4] It is not predictable which patients will have prolonged or severe myocardial depression, but lack of improvement over time has been associated with a poorer prognosis.[10] This can be assessed echocardiographically and newer echocardiographic tools examining strain and strain rate and tissue Doppler imaging may be of value for myocardial assessment.[11] In human patients, it is not absolute numbers that are important, but in fact changes and improvement in response to therapy over time.

Based on the pathophysiology of myocardial dysfunction and mitochondrial dysfunction, it is not surprising that rhythm disturbances can be seen in our critically ill patients. Ventricular premature depolarisations and ventricular tachycardia are a common finding in dogs in gastric dilatation-volvulus.[12] In a study of 111 horses that presented with abdominal pain, twenty-four (27.5%) horses had cardiac rhythm disturbances detected with seventeen horses having ventricular dysrhythmias, 6 supraventricular dysrhythmias, and 1 had both.[6] However other studies have failed to show a difference between the prevalence of dysrhythmias between normal horses and those that have presented with abdominal pain,[13, 14] and this may relate to the high prevalence of dysrhythmias in normal horses[15, 16] and the fact that these study are underpowered. In one study there was an association between the presence of dysrhythmias in horses with abdominal pain and a change in sodium concentration suggesting that hypovolaemia may be contributing to this finding.[14] Very few horses however develop rhythm abnormalities that require therapeutic intervention, which is usually only required if the rate and rhythm significantly affects cardiac output or an R on T phenomenon is observed.[11] Therapy may have limited success if the underlying SIRS or sepsis that has triggered the derangement is still ongoing.

In SIRS and sepsis, due to underlying disease and concurrent electrolyte derangements can lead to cardiac arrest. Cardiac resuscitation can be a challenge in the adult horse and requires a large number of staff in order to try and perform effectively. It is easier to effectively perform in the foal, but success depends upon the underlying cause of the cardiac arrest.

Of all of the electrolyte derangements seen in the horse, hyperkalaemia is the most likely to lead to severe cardiac derangements in the horse. There are many causes of this electrolyte derangement, but the one most likely to result in the severe, acute increase in potassium concentration is post-renal disease, specifically ruptured bladders. These animals have characteristic electrocardiographic changes and bradycardia. Management focuses on reducing the potassium concentration and providing mechanisms to try and stabilise cell membranes.

Primary cardiac derangements

Although SIRS and sepsis are the most common cause of cardiac derangements in the critically ill horse, it is important to be alert for horses with primary cardiac disease that may often present with clinical signs consistent with gastro-intestinal disease, particularly abdominal pain and oesophageal obstruction.

The horse that presents with a cardiac murmur and is otherwise critically ill may have a haemic murmur secondary to a primary disease process caused by a change in blood viscosity (in the horse usually due to hypovolaemia and haemoconcentration rather than anaemia as would be more common in other species) or cardiac dilatation. They may also have an incidental murmur and primary non-cardiac disease or alternatively have endocarditis. Hemic murmurs are usually meso-, telo- or holosystolic murmurs with a point of maximal intensity left fifth intercostal space. Endocarditis is usually diagnosed in young horses and generally affects the mitral or aortic valves leading to a left-sided systolic or left or right sided diastolic murmur, although up to 40% of animals with endocarditis do not have an auscultatable murmur.[17, 18] Other signs associated with endocarditis are fairly non-specific and include tachycardia, lameness and synovial distention, pyrexia, leukocytosis, hyperfibrinogenaemia, hyperglobulinaemia, hypoalbuminaemia and anaemia.[17, 18]

Horses rarely present in heart failure, but when they do it is usually secondary to mitral regurgitation with signs consistent with biventricular failure. These horses will often have tachycardia and concurrent atrial fibrillation. Pulmonary oedema can be confused with oesophageal obstruction in some cases.

Ionophore toxicosis cases will usually present with the non-specific signs of depression and inappetence as well as diarrhoea and ataxia.[19, 20] These animals in moderate to severe cases will have significantly reduced myocardial contractility, resulting in tachycardia in order to maintain cardiac output. These animals are also reported to have an increased prevalence of ventricular dysrhythmias.[19]

Ventricular tachycardia presents similar to abdominal pain. Potential underlying causes include aortic regurgitation, aorto-pulmonary fistula, which are commonly seen in Friesians, aortocardiac fistulas often in older horses, pheochromocytomas, cardiac haematomas, cardiac neoplasia, heart failure, myocarditis and myocardial necrosis, SIRS and electrolyte derangements. Likely success of treatment is dependent upon the underlying cause, which is not always easy to ascertain. Treatment options would include intravenous magnesium sulphate, lignocaine, procainamide, amiodarone and oral sotalol.

Third degree atrio-ventricular blockade is a rare cause of collapse in the horse and appears that donkeys are over-represented. This can be transitional, associated with electrocardioversion of atrial fibrillation.[21] More commonly it is persistently present and in many cases the cause is unknown, although has been associated with pregnancy and rattlesnake envenomation.[22] Treatment requires a fixed or variable rate pacemaker.[23, 24]

Summary

In summary, there are many causes of cardiac derangement in the critically ill horse. Some are triggered by the inflammatory cascade in SIRS and sepsis and others are due to primary cardiac disease. Primary cardiac disease often mimics more frequent presentations and should be considered when cases don’t completely fit the picture. Establishing the cause of the cardiac derangement will allow an appropriate treatment and prognosis to be established. In equine critical care, we are probably underdiagnosing cardiac dysfunction in our equine patients and could use some of the techniques used serially in human patients to identify abnormalities and address them.

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Case discussion on equine emergency and trauma cases

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Triage and assessment of the emergency patient

In busy practice, triage becomes the mainstay of management of critical cases in order to achieve the optimal outcome. Triage is the process of determining the priority of patients' treatments based on the severity of their condition. This rations patient treatment efficiently when resources are insufficient for all to be treated immediately. The cases that require immediate attention are those with impending or absolute cardiopulmonary arrest. To identify those in the most need requires detailed AND REPEATED cardiac and respiratory examinations assessing airway, breathing, circulation and catastrophic haemorrhage. Animals struggling to breathe, animals with severe blood loss and colic are to name a few are likely to need to be seen as a priority.

Excellent assessment of critically ill animals involves attention to detail and is often involves both art, experience and science. It is more than taking a heart and respiratory rate, but considering rhythm disturbances, respiratory patterns and respiratory noise too. The focus on the critically ill patient needs to initially centre on the cardiorespiratory system (a major body system assessment) and then consider other key organs. For example, an animal with traumatic brain injury may well need monitoring and therapeutics of its neurologic status, but this will follow management and monitoring of hypovolaemic and/or haemorrhagic shock.

Identification and subsequent treatment of shock becomes the mainstay of initial management of many emergency patients. Majority of patients that present are hypovolaemic and fewer are dehydrated. Quantification of hypovolaemia is relatively easy to achieve using a variety of clinico-pathological samples (heart rate, mucous membrane colour, capillary refill time, urine specific gravity, packed cell volume, total protein/solids, blood lactate concentrations to name some), whereas this is very challenging for dehydration (increased skin tent, dry mucous membranes, sunken eyes). It is depletion of circulating volume (hypovolaemia) however that will be life-threatening in the short term. Differentiation is important for choice of certain therapies, however.

Traumatic brain injury

Despite the relatively high prevalence of horses hitting their heads and developing clinical signs consistent with traumatic brain injury, there is a paucity of information in the literature regarding management of this condition in the horse and publications are limited to reviews, single case reports as well as one retrospective study of 34 horses.[1]

As clinicians, we can have no control over the initial cerebral insult, so we need to focus management on reducing the extent of secondary brain injury, which is largely due to increased activity of excitatory neurotransmitters, generation of reactive oxygen species and production of inflammatory cytokines, which all contribute to neuronal damage. This damage to neuronal tissue then results in cerebral oedema, increased intracranial pressure, damage to the blood-brain barrier and alterations in the cerebral vascular automaticity.[2]

Traumatic brain injury (TBI) in humans and small animals is associated with high mortality,[3] although it is reported dogs and cats can successfully compensate for loss of cerebral tissue. This is also likely true for horses. In the retrospective study in horses, survival was 62%; this is probably higher than overall survival of these injuries in the population, where the most severe or those that quickly died would not have been referred.

Severity of TBI is categorised in humans using the Glasgow Coma Scale (GCS) and in small animals using the modified GCS (MGCS). In humans this drives management. No such severity score is available in the horse and too little published for this to guide our therapeutic approach. Signs most commonly identified in horses with acute brain injury include ataxia, nystagmus, abnormal mentation and abnormal pupil size, symmetry and PLR.[1]

Guidelines are available for the management of traumatic brain injury in humans and the focus is on maintaining adequate cerebral perfusion. Guidelines created for small animal traumatic brain injury are still controversial and due to lack of published clinical data are based on experimental investigations, human head trauma studies or personal experience.[4]

As with all trauma patients, assessment and correction of imminently life-threatening abnormalities is essential. In humans, 60% of TBI patients have concurrent injuries to other organs[5] and this would also be true for small animals, whom have often been involved in road traffic accidents. However, this is perhaps less common in horses where causes of head trauma often relate to rearing over backwards, hitting their poll whilst rearing or hitting their heads (on fences, trees, during recovery from anesthesia).[1]

One of the focuses of improved care of traumatic brain injury is on pre-hospital care.[6] This is an area that has started to be developed for small animals, although is plagued with challenges as to how to successfully achieve this in many areas beyond offering excellent advice to clients remotely.[7] No such guidelines are available for horses and there are unique challenges to delivery of these, not least relating to caregiver safety.

The mainstay in the assessment of TBI cases is humans is performing a computed tomography (CT) scan within 8 hours of injury[8] as even in mild TBI cases that did not lose consciousness or have amnesia, can still have intracranial bleeds. CT is extremely sensitive for identifying bleeds.[9] More and more small animal hospitals have access to CT scanners, but animals often require general anaesthesia, where risk may outweigh benefits. Fewer equine hospitals have CT scanners, thus routine assessment using this modality may involve transport, which may be either unfeasible or unsafe. There are now more, and better quality, CT scanners that would allow head CT to be performed in a standing, sedated equine patient, although some patients would be too ataxic for this to be feasible. A recent study in horses suggested that radiography could identify majority of skull fractures when compared with CT as the gold standard, but different interpretation and perception of fractures were observed.[10]

Intra-cranial pressure monitoring has become a common method for making management decisions in human TBI cases rather than simply relying on subjective changes to neurological function and is now a standard procedure. This is however a relatively expensive technique and carries the risk of infection, particularly in a stall environment in a recumbent horse.

Management of TBI cases

Commonly reported management of equine TBI cases from a single centre in the 90's and 2000's included administration of dimethyl sulfoxide (DMSO), NSAID's, corticosteroids, antimicrobials, mannitol and intravenous crystalloids. Other treatments administered less frequently included hypertonic saline, furosemide, diazepam, vitamin B complex or thiamine and vitamin E.[1]

Fluid resuscitation is essential to maintain cerebral perfusion pressure. Types and volumes of fluids are controversial in human and small animal guidelines. Aliquots of 10-20ml/kg of isotonic crystalloids enable correction of hypovolemia whilst minimising overhydration and worsening of cerebral oedema.

Hyperosmolar therapy is also a requirement in cases of TBI. There is also controversy regarding the best agent for use. Traditionally mannitol has been used, but there is a substantial body of evidence in humans and animal models that hypertonic saline may be superior to mannitol, regarding increases in cerebral perfusion pressure and outcome.[3]

Analgesia is always required in these patients. In equine practice, NSAID's are usually the first line drug. This contrasts to management of human patients where ketamine and/or opioids would be first line and any detrimental consequences of opioids on respiration would be managed. In small animal patients' opioids would be most commonly administered[3] and reported side effects are managed either by short-action opioids via CRI (fentanyl) or using reversal agents.

Seizure medication is used as required. Ideally seizures should be pre-empted as they increase cerebral oxygen demand and exacerbate increased intracranial pressure due to hyperthermia, hypoxaemia and cerebral oedema. In human and small animal patients, traditionally barbiturates have been used as a first line, although have detrimental effects due to cardiorespiratory depression. Leviteracetam has gained popularity in small animal TBI cases as it may selectively prevent hypersynchronisation of epileptiform burst firing and propagation of seizure activity and has fewer side effects than the barbiturates, particularly regarding respiratory depression and sedation.

Supplemental oxygen and maintenance of a normal $PaCO_2$ is desirable. The former is achievable in the horse, but the latter more challenging for prolonged periods except in foals or ponies.

Hyperglycaemia is associated with increased mortality or worsened neurologic function in human patients with head trauma and in animals with experimentally induced head trauma. It is challenging to know whether this is a marker of severity of the head injury or a cause of worsening secondary injury. It is thus unclear whether we should maintain blood glucose concentrations within the normal reference range using insulin or if this a futile, However, in humans and small animal patients, it is felt iatrogenic hyperglycaemia should not be induced with the administration of corticosteroids.

Corticosteroids, the main anti-inflammatory used in equine practice, has become a controversial treatment in TBI cases as they have been associated with increased mortality in human patients. They have thus largely fallen out of favour in the management of small animal TBI cases. It is perhaps a treatment modality we need to consider removing from our armoury in equine TBI cases. There is minimal to no evidence that vitamins B, C and E (or DMSO) have any positive benefits in TBI cases.

Hypothermia is another ancillary treatment that has become controversial in the management of human TBI and is challenging to accomplish in veterinary medicine. Mechanism of action of hypothermia is not clearly understood. It was initially thought to reduce brain metabolic demands, but other theories include reduction in release of excitatory neurotransmitters such as glutamate and reduction in release of inflammatory cytokines such that the blood brain barrier is maintained. Total body cooling to 32-34^{o}C is unlikely to be possible in our equine patients, but focal cooling may be and could be considered.

Gastrointestinal ‘ulceration’ secondary to stress has been reported in human trauma patients, particularly those maintained on the ventilator and management with proton-pump inhibitors has become common practice, which has also been adopted in small animal patients. No need has thus far been identified in equine patients. Similarly, motility modification is often required in human and small animal patients with traumatic brain injury, but again no need has been described in the horse. However, ensuring appropriate nutritional support and intake is essential and may require esophagostomy tubes in animals with fractured jaws or sore necks. Parenteral nutrition may also be required and should be used judiciously to avoid precipitation of hyperglycaemia.

Sequelae to TBI in humans are common and can be life-changing.[11] Complications include delayed seizures, which can be seen in 4 to 42% of patients with severe TBI, stroke, neurodegenerative disorders and central diabetes insipidus. This prevalence of seizures is uncommon in small animal patients and equine patients.

One interesting sequelae to TBI is cerebral-cardiac syndrome, which is reported in human patients and small animals and has been identified in horses. TBI is thought to induce general adaptation syndrome, which results in myocardial dysfunction, damage, ischemia and heart failure.[12] The mechanism underlying this phenomenon is thought to occur secondary to cerebral oedema, which results in reduced cerebral perfusion. This can lead to hypothalamic dysfunction-induced autonomic dysfunction, which results in increased catecholamine release from the adrenal gland. These catecholamines affect myocyte function, can induce necrosis and influence contractility.[12] As signs can be mild, they may be missed or altered cardiac findings assumed to be secondary to hypovolemia or caused by other injuries. Thus, echocardiography and Holter electrocardiography may be prudent in TBI patients that have abnormal or inappropriate cardiovascular parameters. In the retrospective case series of TBI in horses, many were reported to be tachycardic.[1]

Conclusions- TBI
In summary, there is still controversy regarding optimal treatment of TBI in human and small animal patients, but some of the newer advancements may be of benefit for implementation in our equine cases.

Ophthalmic emergencies seen in practice
Despite protection of the eye by eyelids and eyelashes, a complete bony orbit and cushioning by extra-ocular muscles, fat and fascia, the horse with its prominent eyes, is still prone to marked ophthalmic damage.[13] Although eyelid lacerations and fractures are common and important equine ophthalmic emergencies, the focus of this talk with be emergencies of the globe itself using case examples and unpublished data.

Trauma to the eye is an emergency due to the predicted and purported degree of pain associated with ocular trauma, the likelihood of development of uveitis and risk to vision. The sooner appropriate treatment is instigated, the more likely vision can be saved.

Examination of the whole horse, head, globe and eye is essential to ensure other co-morbidities are not missed when animals present with head trauma. Frequently, the eye is extremely painful and analgesia, sedation and local blockade are essential to facilitate a thorough examination. In many cases, corneal opacification or eyelid or conjunctival swelling precludes examination of the internal structure of the eye with ophthalmoscopy and slit lamp examination. Ultrasonographic examination using a 10-15MHz linear probe can provide significant information regarding the internal structure of the eye and facilitating the development of an appropriate treatment plan. For example, if a retinal detachment is present, implementing a treatment plan to attempt to save the eye is unnecessary. Transpalpebral ultrasonography is extremely well tolerated and although a few additional artefacts are evident when compared with a transcorneal technique, it reduces the risk of further damage to the corneal surface.[14,15] Its disadvantage is that it cannot provide information about whether the eye is visual or provide detailed information about corneal or retinochoroidal disease. The exception to this is when very high frequency ultrasound (35-50MHz) or ultrasound bio-microscopy (UBM) is used. Use of such a high frequency probe, which is sadly not routinely available in veterinary medicine, allows evaluation of the anterior segment pathologies like glaucoma, foreign bodies, trauma, cysts and neoplasms.[16,17]

Some emergency ocular conditions seen in the horse

Head trauma, particularly trauma to the poll, which occurs when horses rear, flip over backwards or on recovery from anesthesia can result in damage to the optic nerve resulting in atrophy. This can be uni- or bilateral and is permanent. Initially horses will present with dilated and fixed pupils and no menace response and then over weeks the optic nerve loses the paurangiotic vessels and becomes pale.[18]

Ocular trauma can also result in retinal detachment. Classically this is seen as a seagull sign on ultrasonography, but they are not always that obvious. Differentials for retinal detachment include thickened posterior membranes, hyaloid remnants, linear mobile echoes, posterior vitreal detachment, choroidal detachment and potentially posterior uveitis.[19] It is important to successfully differentiate these differentials and they have very different outcomes.

Lens dislocation, luxation and subluxation can all be seen secondary to trauma in the horse. Surgical removal of the lens is usually required if retinal detachment is not present.

Blunt force trauma to the eye can result in recognition of fibrin (or more rarely blood) in the anterior segment. Fibrin can be successfully and rapidly treated using tissue plasminogen activator administered into the anterior chamber within 48 to 72 hours of the traumatic event. This is however contra-indicated in the face of haemorrhage.

Damage to the corneal surface commonly occurs and can be secondary to infection (bacteria or fungi) or from physical trauma, which can result in deposition of foreign bodies within the cornea, penetration and tearing of the cornea and less acutely development of stromal abscesses. Melting corneal ulcers are certainly ophthalmic emergencies as they can penetrate Descemet's membrane in 8 to 24 hours. Management may be possible with aggressive medical therapy or may require surgical management which can be achieved using a variety of techniques including corneal grafts, pedicle flaps and use of amnion.[20]

Iris prolapse can be seen following full thickness corneal lacerations (as well as being associated with perforated corneal ulcers and stromal abscesses). These can be managed both medically and surgically, although surgical repair is most commonly undertaken. Vision was retained in one study between 33 and 40% of cases[21] and in a later study from the same institute remained visual in 65% of cases when corneal grafts with conjunctival flaps and/or amnion were utilised.[22]

Lastly, for any traumatic condition it is essential to successfully manage uveitis to minimise sequelae that may be sight threatening. Unlike many of the conditions listed above, where there are a range of treatments that can be implemented which will depend on clinician experience and combination of signs seen, most people approach uveitis in a similar manner: systemic NSAID's, topical atropine and in refractory cases topical or systemic corticosteroids.

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Practical Fluid Therapy for Use in the Field and Clinic

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Intravenous fluid therapy

In humans and animals, fluid therapy is a core element for the resuscitation of critically ill patients, since fluid therapy is essential to decrease mortality and accelerate recovery. In equids fluid therapy is a fundamental component of the treatment for diseases, such as SIRS, sepsis, colic, hypovolaemia and shock. The purpose of fluid therapy is to restore effective blood volume, correct hypotension, improve cardiac output, normalise tissue oxygenation and correct electrolyte and acid base abnormalities. The ultimate aim is to prevent organ dysfunction. Thus, fluid therapy is the mainstay in the treatment of SIRS and sepsis induced hypovolaemia.

Sepsis is a complication of critical illness with high degree of mortality. In horses with gastrointestinal disease SIRS is a common complication, and as in man, it carries a high mortality rate. In adult horses SIRS commonly originates secondary to gastrointestinal disease (i.e. colitis) due to bacterial and endotoxin translocation through the gut wall. The most current Surviving Sepsis Campaign guidelines recommend crystalloids over hydroxyethyl starches (HES) as the preferred fluid for resuscitation. The most commonly used type of intravenous fluid therapy for resuscitation in humans and equids are the polyionic isotonic crystalloid fluids, Lactated Ringers and 0.9% sodium chloride.

Intravenous fluid therapy effects and side effects

Intravenous fluids expand the intravascular space. However, depending on the fluid type, leakage into the extravascular space varies in speed and degree. Crystalloids fluids counteract the movement into the extravascular space due to the osmotic pressure exerted by its solutes. Whilst colloids create oncotic pressure gradients to keep fluids within the intravascular space. Thus, in theory the blood volume expansion may be proportional to the solute tonicity or oncotic power. Colloid fluids remain in the intravascular space longer than crystalloids, thus less fluids are needed to achieve similar hemodynamic effects, as shown by the recent CHEST trial. Colloids have been used for rapid and long-lasting circulatory stabilisation, although data supporting this practice is lacking.

The major complications of fluid resuscitation are pulmonary and interstitial oedema. There are concerns with the use of colloids with regards to immune effects in critical illness, acute kidney injury, coagulopathy, increased risk of death and higher costs. However, the administration of large volumes of 0.9% sodium chloride has been associated with hyperchloraemic metabolic acidosis due to increased plasma chloride and decreased strong ion difference. Furthermore, hyperchloraemia may cause renal vasoconstriction and decrease glomerular filtration rate, leading to acute kidney injury and higher mortality.

Enteral Fluid therapy

Where enteral fluids can be used, they have many advantages, not least from a financial point of view, particularly in the management of impactions. Enteral fluids can provide oral supplementation of electrolytes, provision of nutrition and treatment of dehydration when present without hypovolemia. Interestingly NICE guidelines recommend that justification is required as to why intravenous fluids are chosen over enteral fluids and that, if the former is used, should be stopped as soon as practical.

Water absorption and blood flow
Water absorption from the gastrointestinal tract, in the normal horse, primarily occurs in the large intestine, and more specifically the large colon. In an adult horse a volume of up to 100L of fluid and associated secretions is absorbed during the course of the day. In the hypovolaemic horse, in order to protect the vital organs, blood flow is diverted from the gastrointestinal tract. Once blood flow is reduced, so too are gastrointestinal motility and absorption. In addition, obtunded, hypovolaemic horses have a reduced thirst drive. It is for this reason that using oral fluid therapy in hypovolaemic animals is unsuccessful at best and detrimental in certain scenarios.

Enteral fluids can be delivered in the form of water from a bucket, via an indwelling narrow nasogastric tube (continuous administration of oral fluids), via a conventional nasogastric tube (intermittent fluid administration) and when data is extrapolated from other species instilled per rectum.

Proctoclysis
There are various descriptions in the literature of the use of fluids per rectum in a variety of veterinary species and in man. They appear to be an effective way of providing maintenance requirements or provision of enough fluids to allow other vascular access to be obtained. They have been reported to be used for over 100 years in man on the battlefield. They are commonly reported for use in elephants and have been partially used successfully in a small number of horses. In humans, the maximum amount of fluid that can be absorbed in a 70Kg adult is 150ml/hr with a maximum installation rate of 500ml/hr via a foley catheter. Similar fluid instillation rates proportionate to body size have been used in elephants and horses and significantly higher rates experimentally in rabbits. The suggestion from one study is that absorption of fluids per rectum will still occur in the face of hypovolaemic shock.

Fluids used enterally
These include water, hypo- or isotonic fluids supplemented with electrolytes (e.g. sodium chloride and potassium chloride) and also hypertonic magnesium sulphate. Hypotonic fluids given enterally are likely to be absorbed from the gastrointestinal tract, whereas isotonic fluids are more likely to remain the gastrointestinal lumen.

Magnesium sulphate is used enterally as a cathartic agent as an initial treatment for large colonic impactions. Initial use of 0.5g/kg is recommended diluted in water. Experimentally magnesium sulphate is not as effective in increasing colon water content when compared with a balanced electrolyte solution but does increase the water content of faeces in the small colon. Balanced electrolyte solutions can be made from sodium chloride and combined sodium and potassium chloride (LoSalt®) with approximately 5g of each per 1L of water. If only using sodium chloride, add a maximum of 9g per 1L of water. In horses that have severe hypokalaemia, potassium chloride can be used orally (0.1-0.2g/kg/day) in addition to intravenous fluid supplementation.

Products not recommended for use enterally
Mineral oil can be used as a marker of gastrointestinal transit (18 hours to anus if transit time is normal). For impactions, it works its way around without penetrating it as well as hindering water penetrating. Based on this observation, increased cost over water and the fatality if this product ends up in a horse's lungs it cannot be recommended for treatment of pelvic flexure impactions.

Dioctyl sodium sulphosuccinate is a detergent that should penetrate impacted faecal material by affecting surface tension, thus allowing water to enter the faeces. Care should be taken as a 3-fold overdose is fatal and also increases absorption of mineral oil so should not be administered with it. There is no benefit of this over water based on an experimental study.

Sodium sulphate is an even more potent cathartic than magnesium sulphate, but consistently causes hypernatremia and hypocalcaemia, so is not recommended.

Oral glucose in fluids does not provide sufficient nutrition to be valuable in horses and glucose and glycine containing fluids in an experimental diarrhea model resulted in incomplete fluid absorption.

Case discussion of when a colic is not a colic

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Introduction

For many of us, we are familiar with the presenting signs of colic as they are a bread and butter emergency seen in all types of equine practice. The presenting signs are those that we usually associate with abdominal pain. These would include inappetence or selective eating, flank-watching, rolling, sweating, kicking at the abdomen, and lying down. We know from epidemiological studies, that approximately 80% of these cases will be successfully managed medically with analgesia and anti-spasmodic drugs. Another 8-10% may require surgery or euthanasia due to strangulating lesions of the small or large intestine. The differentiation between medical and surgical colics can largely be achieved based on physical examination, rectal examination, findings from nasogastric intubation and sometimes from peritoneocentesis or basic blood work. We are then left with the 'pseudo-colics' that don't fit the normal patterns of what we expect and require very different management.

All of the other body systems can have disease processes that can manifest as colic. These include other gastrointestinal causes, musculoskeletal, genito-urinary, cardiac and respiratory.

Other gastrointestinal causes

These gastrointestinal causes would include diarrhoea, which usually manifests as colic due to ischaemia to the bowel wall, peritonitis, secondary to serosal inflammation, or ileus secondary to serosal inflammation, and neoplasia. Neoplasia may cause signs consistent with abdominal pain for several reasons, which would include large masses that temporarily obstruct advancement of intestinal contents, intra-luminal masses causing partial or total obstruction of the intestinal flow and masses that result in stretching of organ capsules as may happen with splenic or hepatic neoplasms. Choleliths can also cause severe, intermittent pain whilst being passed down the biliary tree.

Musculoskeletal causes

The obvious cause of 'pseudo-colic' in this class is laminitis, where pain may cause horses to become recumbent and not eat as it is too painful to get to the food. However, generalised myopathies and acute rhabdomyolysis can result in quite marked signs of pain and manifest as recumbency, rolling, sweating and kicking at the abdomen. The pain is caused by both muscle breakdown and re-perfusion injury.

Genito-urinary causes

For many people, especially those that manage breeding mares, the most common cause of 'pseudo-colic' are animals that rupture their middle uterine arteries and bleed into their broad ligament. This causes pain from distention of the broad ligament and pain from hypoxia and distress from the drastic reduction in cardiac output and blood flow.

Renal disease, particularly pyelonephritis and nephroliths, can result in severe, unrelenting or more insidious and intermittent pain. In addition, ureteroliths and uroliths can result in signs of colic – again severe and persistent or insidious and intermittent.

Cardiovascular causes

Cardiac dysrhythmias, particularly tachydysrhythmias and ventricular tachycardia will often present similar to true abdominal pain. These rhythm abnormalities can be primary or secondary to electrolyte derangements, other systemic disease, phaeochromocytomas or other forms of cardiac disease that would include aorto-cardiac fistulas and severe aortic regurgitation. Other cardiac causes that often present as 'pseudo-colic' include any cause of heart failure, which is often secondary to severe mitral regurgitation, although can have a multitude of causes and where the underlying rhythm is often fast atrial fibrillation. The last cardiac cause that present mimicking abdominal pain is pericarditis, which again causes pain due to increased pressure in the pericardial space and results in reduced filling of the heart and reduced cardiac output, once again leading to a tachycardia. One of the vascular causes of 'pseudo-colic' is the formation of thrombi proximally in the arteries. The most common one recognised are thrombi that lodge in the iliac arteries, which are thought to occur secondary to aberrant large strongyle migration.

Respiratory causes

Lastly, there are several respiratory conditions that can mimic colic. These include diaphragmatic ruptures and any accumulations within the pleural space (air, blood, pus, chyle). Diaphragmatic ruptures often result in temporary or permanent obstruction of intestinal flow and also inflammation in the pleural cavity, which is extremely painful. The same applies for pneumo-, pyo and chylothorax. The air or fluid accumulation results in discomfort to breathe and also results in pleural inflammation, known to be a very severe form of pain that is difficult to localise. Where haemothorax and haemoabdomen are concerned, as well as the space-occupying effects and the inflammation, there is likely additional pain from resulting haemorrhagic shock and ischaemia.

Conclusions

In summary, there are many causes of pain that can manifest as colic. Some are more common than others and seen in particular horse types (e.g. bleeding into the broad ligament in mares post-foaling), whereas others are relatively rare and need to ruled in or out. These causes of 'pseudo'colic' should be suspected in animals that have normal borborygmi or are passing faeces and have normal clinic-pathological variables suggesting that they are not hypovolaemic (although some of the above examples will result in hypovolaemia) such are normal lactate concentrations and normal packed cell volume and total solids.

Case discussion on management of the sick foal

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Introduction

The key to success with the newborn foal is to understand what is normal and to immediately act upon anything that deviates from this, as time is of the essence if sick foals are to be saved. The key therefore revolves around ATTENTION TO DETAIL. Minor changes in behaviour can be warning signs of impending illness and the sooner treatment is started, the more likely there will be a positive outcome. The normal gestation of a horse is between 320-340 days but can be significantly more or slightly less than this. Immediately after birth, the cardiorespiratory system must adapt to extra-uterine life.

Breathing

Spontaneous breathing should begin within 1 minute of birth, although many foals attempt to breath as soon as the chest has passed through the pelvis. Whilst adaptations to extra-uterine life occur, respiratory rates are normally >60 breaths per minute but should decrease to 30–40 breaths per minute within a few hours. The foal may appear slightly blue at birth but this should resolve within a few minutes.

Cardiovascular system

The heart of a normal newborn foal should have a regular rhythm and the rate should be greater than 60 beats per minute after the first minute. Occasionally rhythm disturbances can be auscultated but should resolve within 15 minutes after birth. Murmurs may be heard in the first week of life but should resolve beyond this time. A continuous or systolic murmur may be heard for the first few days after birth in the third or fourth left intercostal space and is due to a patent ductus arteriosus. Other systolic murmurs, thought to be flow murmurs, may also be auscultated. Murmurs that persist beyond the first week of age, those that are loud (>3/6) or associated with clinical signs should be further investigated. Mucous membranes should become moist and pink within a few minutes of birth.

Normal responsiveness

Foals are usually unresponsive whilst in the birth canal. As the foal is born, it should possess a righting and withdrawal reflex. Menace response is absent at birth and has a learned component and as such may not be present until 7-10 days. Thus, it should not be used to assess vision. Foals should have a strong suck reflex within 10 minutes of birth. However, try not to allow foals to suck fingers as they ingest organisms present on human skin, which can be pathogenic. Within 1 hour of birth, foals show evidence of being aware of auditory stimulation evidenced by movement of their ears. Eyes are angled slightly ventromedially at birth (similar to what would be seen in lightly anaesthetised small animals) and the eye angle gradually becomes more dorsomedial over the next month.

Foals should attempt to stand within 20 minutes of birth, and most can stand unaided by 1 hour. They should then be steady enough and driven to nurse by 2 hours. Some foals will pass their meconium (first faeces) shortly after standing, but other will not defaecate until they have consumed colostrum approximately 3-4 hours after birth. Urination is more variable - fillies usually urinate before colts.

Foal resuscitation

For foals with difficulties, it is important to recognise and institute appropriate resuscitation procedures speedily. A scoring system (modified Apgar) has been developed as a guide to help effectively implement resuscitation and estimate the level of foetal compromise. A combined score of the four parameters of less than 3 should initiate cardiopulmonary resuscitation. If the score is between 4-6, then the foal should be closely monitored as it is suggestive of some degree of asphyxia.

The priority in resuscitation of the newborn foal, assuming that there are no other life-threatening abnormalities present, is to establish an airway (A) and breathing (B) pattern. The upper airway should be cleared of membranes or any fluid. If the airway is suctioned, it should be performed with care. Tactile stimulation (stimulating the nose or ear) can be attempted initially. If this is not successful, then ventilation should begin. The main option available at birth is the use of an Ambu bag. Evidence suggests that room air is as good, if not preferred to, 100% oxygen. Respiratory rate should be 10-15 breaths per minute.

If the foal's heart rate does not increase with ventilatory support, then chest compressions should be initiated. The recommended rate is 100-120 beats per minute for a minimum of 2-minute cycles. The foal should be placed in right lateral recumbency on a hard surface. The clinician should then place the hands over the widest point of the thorax dorsally and aim to compress the chest to about 1/3rd of the chest depth with each compression. As a small proportion of foals are born with rib fractures, it is prudent to try and rule-out their presence prior to beginning chest compressions. The key to the success of chest compressions is to not keep pausing to assess efficacy. If possible, efficacy of chest compressions generating cardiac output can be assessed with the presence of pupillary light responses. If the animal is intubated in a hospital, adequate cardiac output generated by chest compressions can be identified if end-tidal carbon dioxide is >20mmHg.

Drugs should be administered if a cardiac rhythm is not present within one minute of initiating chest compressions. Intravenous epinephrine is the drug of choice and should be administered at a 'low dose' (0.01–0.02 mg/kg). It can be repeated every 2–3 min during compressions. Atropine and doxapram are not recommended.

Foals at birth should be dried and placed in dry bedding. They ideally should not be in very cold environments, but equally should NOT be actively warmed. Being cool can have advantages as it reduces the metabolic requirements of the brain, heart and other tissues.

These foals may also need fluid therapy and intravenous nutrition. Fluids need to be given cautiously in these scenarios as these foals are not hypovolaemic (unless they have bled). Fluid boluses may be required if the foal is obtunded, has poor peripheral pulse quality, has cold distal extremities or has other clinical signs consistent with septic or haemorrhagic shock.

Practical management of sick neonates

All sick foals are septic until proven otherwise. If these foals are treated appropriately early on in the disease process, they can have a good prognosis. The first step with the critically ill foal is to ensure the client appreciates the severity of the condition and is emotionally and financially behind a decision to treat the foal. The next step with a moderately to severely ill foal is to move or refer the foal to an appropriate facility. This should be done promptly and is often easiest without the mare. Foals can easily be transported in cars and the mare can be transported later.

Initial tests and samples that should be considered in the foal include a major body system assessment, haematology, biochemistry, urine specific gravity, blood lactate concentration, arterial blood gas assessment and IgG concentration. A central venous cannula should be placed aseptically and then three samples should be taken for blood culture over the next 4 hours. Plasma should be defrosted as required. The foal's fluid deficit should be assessed; most recumbent foals have a fluid deficit of 8% or greater. There are various ways to approach correction of a fluid deficit in the hypovolaemic patient which we will discuss. The foal should be maintained in sternal recumbency to make the work of breathing less and minimise the risk of atelectasis and intra-nasal oxygen should be provided at 5-10L/min. Intra-nasal tubes should be used with the tip of the catheter being inserted up to the level of the medial canthus. A urinary catheter and urine collection bag should be used in recumbent foals. These not only prevent urine scalding but allow renal function and perfusion to be assessed and aid titration of fluids. Foals should produce 1-2 ml/kg/hr of urine and due to their milk-based diet as this stage should produce hyposthenuric urine (USG<1.008). Once initial fluid boluses have been administered, a more conservative fluid plan can be implemented that includes the inclusion of plasma and parenteral nutrition (glucose and amino acids or glucose, amino acids and lipid with vitamins and minerals).

Recumbent collapsed foals are unlikely to have an effective gastrointestinal tract, predisposing these animals to necrotising enterocolitis and abdominal pain due to ileus if enteral nutrition is used. In addition, recumbency will promote the risk of aspiration if milk is administered. Once all blood cultures are collected, intravenous bacteriocidal broad-spectrum antimicrobials should be administered. Choices would include gentamicin and benzyl penicillin, trimethoprim sulphadiazine, ceftiofur or cefquinome (if available). Non-steroidal anti-inflammatory drugs may also be administered; COX-2 selective drugs such as meloxicam or carprofen may be associated with fewer side effects. However renal function should be monitored when nephrotoxic drugs are administered. If the foal is hypotensive, pressor agents or inotropes such as dobutamine or norepinephrine, may be administered by constant-rate infusion to improve blood pressure and organ function. The use of sucralfate may be beneficial in conjunction with fluids and inotropes to help improve gastric mucosal blood flow and prevent (or treat) gastric disease. Hyperglycaemia is common in these foals secondary to the disease process and provision of parenteral nutrition. As such it becomes important to control blood glucose at least below the renal threshold or the osmotic diuresis can make it challenging to maintain hydration status.

It may be prudent early in the management of these critically ill foals to perform thoracic and carpal and/or tarsal radiographs. Thoracic radiographs allow any thoracic pathology to be identified and treated as appropriate. Some foals have such severe pulmonary disease that they need mechanical ventilation, which would require referral to a specialist centre. If foals are premature or dysmature, lack of or very poor ossification of the small carpal or tarsal bones significantly reduces the likelihood that the foal will become an athlete and as such clients may choose to stop treatment.

The key to success is frequent reassessment and adaptation of the treatment plan. Foals should be kept dry and warm, but not actively heated and turned frequently. Ideally foals should be weighed daily to ensure that they are not losing body weight. Foals are at risk of thrombophlebitis, and the development of septic joints, so should be checked regularly for the development of these conditions as well as for decubital and corneal ulcers. Physical therapy or passive range of motion exercises should be provided.

As these foals hopefully improve, it becomes possible to reduce fluid rates and gradually wean them off parenteral nutrition and onto enteral nutrition. Whilst the foal is ill and recumbent it is helpful for the mare to be near and able to see the foal but physically separated from it. Milk can be stripped from the mare and stored until the foal is ready for its re-introduction. As the foal becomes stronger and copes with enteral nutrition, in many cases it is possible to re-unite the mare and foal and the foal will learn to obtain milk from the teat with help. If the mare rejects the foal, then bucket training the foal is warranted. Bottle feeding foals increases the risk of aspiration pneumonia, but also often results in the development of an unmanageable orphan foal when it reaches adulthood.

Conclusions

In conclusion, management of the sick, equine neonate is both challenging and rewarding. Success is dependent upon prompt recognition of often very subtle abnormal clinical signs with a swift and aggressive response and treatment plan to these signs. If treated appropriately, prognosis can be good.

Getting the most from clinical pathology in the sick horse – what's useful and what's not

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Introduction

Repeated major body system assessments are the mainstay for the assessment of sick horses and for monitoring effectiveness of therapy in these patients. However, selective diagnostic tests can be extremely valuable in order to assess the severity of disease, allow assessment of where the case can be managed (in the field, at the practice or require referral), can guide appropriateness of treatment, and in some instances provide information on the likely prognosis for that patient. From a practical point of view, these tests don't have to be expensive, many can be done horse-side and thus can provide instant results but do require a move away from thoughts that all answers will be gleaned from a full haematology and biochemistry. This includes urinalysis, PCV, total solids and blood smear, blood lactate concentrations, blood gas analysis and electrolyte concentrations, fibrinogen and serum amyloid A concentrations and targeted biochemical testing (for example creatinine, glucose, cardiac troponin I, ammonia concentrations and bile acids).

Urinalysis

Although horses rarely suffer from intrinsic renal disease, measurement of urine specific gravity and performing urine dipstick analysis (glucose, protein, blood, Hb, Mb) can be a very useful and cheap method for assessing appropriate renal function and likely degree of hypovolaemia when compared with the USG of plasma (1.008-1.012) and PCV, TS and blood lactate concentrations. Microscopy can be used to identify renal casts, cellularity and presence of bacteria (if collected aseptically). In addition, more sensitive tests can be performed to assess for renal tubular damage (eg. measurement of urine activity of gamma glutamyl transferase).

Assessment of fluid deficit

Packed cell volume and total solids have traditionally been used to assess hydration status in horses. However, they can be misleading in many of the clinical scenarios with sick horses. Reference ranges for PCV and many other haematological and biochemical markers are set using groups of healthy Thoroughbreds. For many of our native and heavy breeds, a normal PCV will be much lower than the reference range, thus meaning that we miss the severity of hypovolaemia based on this parameter. In addition, when faced with acute haemorrhage, splenic contraction can artefactually increase the PCV thus underestimating both blood loss and degree of hypovolaemia. Total solids can also cause us some issues when trying to use this parameter to quantify hydration status in animals with protein-losing disease. Although protein-losing nephropathy is uncommon, concurrent gastrointestinal disease with hypovolaemia is extremely common especially in our sick adult equine patients. In hypoproteinaemic hypovolaemic animals, the total solids may appear in the reference range and therefore underestimate the severity of hypovolaemia in these cases. Both of these parameters can be misleading when assessing severity of hypovolaemia.

Lactate concentrations

Lactate is produced in anaerobically respiring tissues and is therefore a valuable marker of poor perfusion to tissues. This parameter not only provides information regarding severity of hypovolaemia, but in many studies has been shown to be a valuable prognostic indicator due to what it measures. It can now easily be analysed using hand-held portable machines so results can be quickly obtained horse side. Many of them are more reliable using plasma or serum, but even on whole blood will provide broad trends. The only practical comment about this is that it is often measured prior to the administration of intravenous fluids and then after a period of administration. Despite clinical improvement, the lactate concentration may increase. When animals become moderately to severely hypovolaemic, they conserve blood supply to essential organs. This means that lactate becomes trapped within the capillary beds of many organs. Following fluid administration, these capillaries are reperfused and lactate is washed back into the circulation.

Blood smears

Although these should be performed in conjunction with all abnormal haematology, when money is short or you don't have access to a haematology machine in the practice, these can provide fast, pertinent information in the sick horse. These include the presence of bands and percentage of bands, percentage of neutrophils and whether any of these are toxic. Cell counters can be purchased in order that more accurate cell counts can be obtained using the microscope.

Blood gas analysis and electrolyte concentrations

Assessment of acid-base in any depth is outside of the remit of this lecture. However, valuable information can now be obtained relatively cheaply using portable machines about the severity of acid-base disturbances and concurrent electrolyte abnormalities that are present. Majority of the adult horses that present with colic have a metabolic acidosis primarily due to a lactic acidosis and require intravenous fluids. Bicarbonate is contra-indicated in animals when the acidosis is due to increased lactate concentrations. These horses also are or become hypokalaemic and hypomagnesaemic, due to low concentrations in resuscitation fluids and/or these electrolytes not being absorbed from the gastrointestinal tract or are not being ingested. Colics are variably and usually mildly hypocalcaemic. For other sick horses

Fibrinogen and Serum Amyloid A concentrations

Both of these are markers of acute inflammation. Serum Amyloid A (SAA) is the more acute, acute phase protein of the two listed here and is now more frequently available on biochemistry machines and at specialist laboratories. Fibrinogen can also be obtained on many commercial biochemistry machines but can also be estimated easily and cheaply in any small practice laboratory. This can be done either by measuring the difference in total solids between spun EDTA and serum samples or by using a heat precipitation test. In this test, take plasma (EDTA or heparin). Have one microhaematocrit tube that remains unheated and one that is heated to 58^{o}C for 3 minutes. Measure TS on both tubes and subtract the heated from the unheated to provide an estimate of fibrinogen. These tests are not completely accurate in terms of values in g/L but will provide an idea about whether fibrinogen is normal, increased or very increased.

Facial action units, activity and time spent with dam are effective measures of pain in response to mulesing of Merino lambs

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Introduction

Pain is an essential function of the nervous system and is defined as an unpleasant sensory and emotional experience associated with actual or potential damage to the body.[1] Pain can be quantified by physiological, neuroendocrine and behavioural changes. However, measuring physiological or neuroendocrine changes, such as plasma cortisol concentrations, is not practical on–farm. Furthermore, handling of the animal itself creates stress which can generate misleading results.[2,3] Recent studies have shown that pain can be identified in lambs following husbandry procedures through behavioural observations such as posture, gait and the way an animal lies down.[3-8] However, these measures of behaviour are often not repeatable in quantifying pain in ruminants.[9-13] There is now emerging evidence that pain can be measured using assessments such as facial expression, activity, time spent with dam and time taken for lambs to identify their dam after lamb marking. A combination of these assessments may be more robust given the subjective nature of measuring pain in animals.

Pain can alter facial expressions which is likely to be an involuntary response and hence is may be a more sensitive assessment of pain.[14] McLennan *et al.*[15] developed a facial expression scale to model pain in sheep and identified orbital tightening and ear posture as key facial action units appropriate for sheep. 'Statue standing' is observed in mulesed lambs and classified as a behaviour that is indicative of an animal in pain and as such is perceived as a reduction in activity.[7] However, Grant found lambs castrated with rubber rings, mulesed and tail docked with a hot iron displayed increased active pain behaviour (restlessness, kicking/foot stamping and rolling) compared to lambs that were tail docked, mulesed and not castrated.[7] These differences observed between mulesing and the inclusion of other husbandry procedures are likely to be attributed to the type of pain experienced. Studies have shown that surgical procedures often result in abnormal postures and a reduction in activity whereas the use of rubber rings results in ischaemic pain and an increase in abnormal behaviours (kicking, biting, foot stamping) and postures.[16-18] Standing still may not provide an accurate assessment of an animal in pain and therefore measuring actual activity, movement, and distance travelled by the lamb could be more useful. The distance between lamb and dam may also be of use when measuring pain in lambs.[7] Further investigation is warranted to determine if distance between dam and lamb, time spent with dam and activity are comparable with other behavioural measures of pain.

A recent study by Inglis *et al.*[8] identified that a combination of meloxicam (Non-Steroidal Anti-Inflammatory Drug) and a topical analgesic (Tri-Solfen®) reduced pain in response to mulesing in Merino lambs when assessed via behavioural observations such as posture, gait and the way an animal lies down. Lambs that were mulesed without pain relief displayed significantly more pain-related behaviours during the 30 hours post-mulesing compared with lambs that were not mulesed. Lambs that received a combination of meloxicam and Tri-Solven® displayed significantly less pain-related behaviour during the 6 hours post-mulesing than lambs that were mulesed without pain relief, but still more pain than lambs that were not mulesed. The results of this experiment support the use of pain-related behaviour to measure the efficacy of analgesics and the use of multimodal analgesia during mulesing of lambs. However, pain relief was not evident from behavioural observations 24-30 hours post-mulesing. Further assessments such as facial expression, activity and/or time spent with dam could provide evidence that pain relief was effective for a longer duration than determined using behavioural measures.

The current study tested the hypotheses that; (i) facial action units can be used to assess the efficacy of treatments to mitigate pain following mulesing, (ii) activity of lambs and time spent with dam can be used to assess the efficacy of treatments to mitigate pain following mulesing and (iii) measurements of facial action units and activity complement behavioural observations of posture, gait and the way an animal lies down in the assessment of pain in lambs following mulesing.

Materials and methods

This experiment was approved by Murdoch University's Animal Ethics Committee. All procedures were performed in accordance with the guidelines of the Australian Code of Practice for the Use of Animals for Scientific purposes.

Research site, experimental design and animals

Details of the experiment have been described previously by Inglis *et al.*[8]. Briefly, 178 pregnant Merino ewes (122 single bearing and 57 twin bearing) from two sources lambed between May and June 2017 in Mundijong, Western Australia. Merino lambs (n = 140) were allocated to one of six treatments which were implemented at mulesing (Day 0) and consisted of (1) lambs that were not mulesed (Control), (2) lambs that were mulesed and administered a saline injection as opposed to pain relief (Placebo), (3) lambs mulesed and administered meloxicam (Metacam® 20, Boehringer Ingelheim, Australia) 15 minutes prior to mulesing (MC-15), (4) lambs mulesed and administerd Tri-Solfen® (Bayer, Australia) only (TS), (5) lambs mulesed and administered a combination of meloxicam 15 minutes prior to mulesing and Tri-Solfen® (MC-15+TS) and (6) lambs mulesed and administered meloxicam in the cradle (MC0). Dose, route and timing of adminstration for Tri-Solfen® and meloxicam is described by Inglis *et al.*[8]. At marking, all lambs were placed in dorsal recumbency into a cradle that restrained the animal around the hocks of all four limbs. All lambs except for those in the Control treatment were ear marked, vaccinated, tail docked and mulesed. Male lambs were also castrated using rubber rings. Lambs allocated to the Control treatment were placed in the cradle for 60 seconds (average time of marking) before being released to their allocated plots.

Facial Action units

Facial action units were measured at 1 hour, 5 hours and 26 hours post-mulesing. Videos of up to 20 seconds were taken from 5–25 m using a high definition camera. Each video attempted to capture side and front angles of the lamb's face. The videos were then analysed retrospectively. The scorers (n=3) were blinded to the treatment administered to the lamb and three screenshots were taken from each video to assess the dominant facial expression for that time point. Ear position and orbital tightening was scored as per McLennan *et al.*.[15]. Lambs in pain show 'squeezing' of the eye or closing of the eye described as orbital tightening. Lambs in pain have ears that are tense and point backwards or downwards so that the inner part of the ear is not visible. Each facial action unit was scored from 0 to 2 where 0 is no pain present, 1 is pain partially present and 2 is pain present. A total pain score out of 6 was determined by adding the individual scores for each of the three action units for each set of photographs.

Activity and proximity to dam

Activity was measured using activity monitors that were attached to a collar that was fitted to the ewes at allocation and the lambs when they were in the cradle at mulesing (ActiGraph wGT3X-BT; Actigraph, LLC, Pensacola, FL). The activity monitors capture movement of the lambs which is a defined signature on the output from the monitor. Time spent between the lambs and their dams was also assessed using the Bluetooth function of the activity monitor. For the purpose of recording proximity, the activity monitors fitted to the ewes were programmed as a beacon and those fitted to the lambs were programmed as a receiver as described by Sohi *et al.*[20]. The activity monitors on the lamb receive a 'hit' when in proximity of a beacon and these 'hits' are accumulated over time to identify the dam of the lamb.

Statistical Analysis

All statistical analyses were performed using GENSTAT (18th Edition: VSN International 2017, Hemel Hempstead, UK). Total facial action units, activity, time spent with dam and time to mother up post-mulesing were analysed using Restricted Maximum Likelihood. For time spent with dam the method of restricted maximum likelihood was used with a variance-covariance structure selected in order to model repeated measures over time. The selection was via a sequence of likelihood ratio tests. A heterogeneous power model in which the correlations between observations from the same plot decays as the time delay between the observations increases and the variances can be different at each time used. The fixed effects were treatment, time of measurement (where applicable), rear type, sex and interactions thereof. Random effects included plot, lamb (nested within plot) and measurement period (where applicable; nested within lamb within plot) along with observer and ewe source. Activity and time spent with dam data were analysed for two time periods which only included daylight hours (Day 0; 1-8 hours post-mulesing and Day 1; 24-32 hours post-mulesing). Activity was log transformed plus 100 added to value. Main effects and interactions have been considered significant at the 5% level. Difference between means were assessed using least significant differences, LSD (P = 0.05).

Results

Effect of mulesing and pain relief on Facial Action Units

Lambs that were not mulesed had significantly lower total facial action pain scores at 1-hour and 5 hours post-mulesing compared to lambs that were mulesed (Table 1). Total facial action units showed no difference in pain at 26 hours post-mulesing between lambs that were not mulesed and lambs that were mulesed and offered no pain relief. There were no differences in total facial action units at 1, 5 and 26 hours post-mulesing between lambs mulesed with or without pain relief. There was no effect of sex or rear type on total facial action units between treatments.

Table 1 Average total facial action pain score at 1, 5 and 26 hours post-mulesing for lambs that were not mulesed (Control), lambs that were mulesed and administered no pain relief (Placebo), lambs mulesed and administered Metacam 20 15 minutes prior to marking (MC-15), lambs mulesed and administerd Tri-Solfen® only (TS), lambs mulesed and administered a combination of Metacam 20 15 minutes prior to marking and Tri-Solfen® (MC-15+TS) and lambs mulesed and administered Metacam 20 in the cradle (MC0). Values respresent observations scored out of six.

	Total facial action pain score							
Time post-mulesing (hr)	Control	Placebo	MC-15	TS	MC-15 + TS	MC0	*P*-value	LSD
1	0.73^{A}	2.20^{B}	3.07^{B}	2.23^{B}	2.62^{B}	1.97^{B}	0.005	1.242
5	0.22^{A}	1.66^{B}	2.45^{B}	2.14^{B}	2.68^{B}	1.53^{B}	<0.001	1.317
26	1.28	2.39	2.91	2.46	3.35	2.72	0.063	1.359

^{A,B} values with different superscripts are statistically significant

Effect of mulesing and pain relief on lamb activity

There was a significant interaction between time and treatment effects on lamb activity on the day of mulesing (Table 2; *P* <0.001). During the first 2 hours post-mulesing there was no significant difference in activity between lambs that were not mulesed and lambs that were mulesed with or without pain relief. Over the period of 3 to 8 hours post mulesing, the activity of lambs in the control group was 70% to 180% higher during each hour compared to the activity of lambs that were mulesed with no pain relief. However, there was no difference in activity between lambs that were mulesed with or without pain relief (Table 2). There was no rear type effect on day of mulesing however there was a significant interaction between sex and time on lamb activity. Over the period of 3 to 8 hours post mulesing, the activity of the female lambs was 10% to 45% higher during each hour compared to the activity of the male lambs (*P* <0.001).

On average, activity was approximately 70% lower on day 1 post-mulesing in lambs that were mulesed with or without pain relief compared with lambs that were not mulesed (153 vs 255; *P* <0.001). There was no effect of pain relief on activity of lambs on the day following mulesing through to day 10. Furthermore, there was no difference in activity between mulesed and non mulesed lambs from day 2 through to day 10 of the experimental period. There was no sex or rear type effects between lambs in each treatment for activity from day 1 to the end of the experiment.

Table 2 Average activity 3 to 8 hours post-mulesing for lambs that were not mulesed (Control), lambs that were mulesed and administered no pain relief (Placebo), lambs mulesed and administered Metacam 20 15 minutes prior to marking (MC-15) and lambs administered combination of Metacam 20 15 minutes prior to marking and Tri-Solfen® (MC-15+TS). Values are presented as transformed data with back transformed data in brackets.

Time post-mulesing (hr)	3	4	5	6	7	8	*P*-value	LSD
Control	5.71^{A} (202)	5.93^{A} (276)	5.82^{A} (236)	6.18^{A} (382)	6.12^{A} (357)	6.42^{A} (511)		
Placebo	5.39^{B} (120)	5.35^{B} (110)	5.31^{B} (102)	5.46^{B} (135)	5.47^{B} (138)	5.66^{B} (188)	<0.001	0.258
MC-15 + TS	5.47^{B} (137)	5.43^{B} (127)	5.49^{B} (143)	5.72^{B} (204)	5.73^{B} (208)	5.84^{B} (245)		

^{A,B} values with different superscripts are statistically significant within each hour

Effect of mulesing and pain relief on time to mother up and time spent with dam

There was no significant difference in time to mother up after mulesing between lambs that were not mulesed and lambs that were mulesed with or without pain relief. The percentage of lambs that mothered up in the first hour ranged from 72% to 95% across treatments. On average the total time taken to mother up ranged from 1.2 hours to 2.5 hours between treatments (*P*>0.05). There was no effect on sex or rear type on the time to mother up.

The time spent with dam over the first 8 hours post-mulesing was greater for lambs that were not mulesed compared to lambs that were mulesed, with or without pain relief (36 min/hr vs 27 mins/hr; *P*<0.001). The was a significant difference between sex and rear type where females spent more time with the dam than males (35 vs 30 mins/hr; *P*<0.001) and singles spent more time with the dam than twins (35 vs 31 mins/hr; *P*<0.001).

Discussion

Facial action units, activity of the lamb and time spent with dam were effective measures of pain in response to mulesing in Merino lambs. Facial action units showed that lambs mulesed without pain relief experienced 4-fold more pain during the first 5 hours post-mulesing than lambs that were not mulesed. This is consistent with behavioural measures reported by Inglis *et al.*[8] who reported a 3-fold increase in pain measured by posture and gait. Both methods therefore had a similar capacity to detect pain differences on day 0 between mulesed and non-mulesed lambs, and hence the third hypothesis was accepted. The use of facial actions units to detect pain is supported by Guesgen *et al.*[22] where facial expression was used to identify pain in lambs that had been tail docked compared to control animals. McLennan *et al.*[15] further showed that diseased animals changed their facial expression after treatment and during the recovery phase whilst healthy animals did not. Mulesing causes significant pain and the magnitude and duration of this pain can differ depending on the method of assessment for pain.

Facial expression did not detect any difference in pain between lambs that were mulesed with or without pain relief. Given previous results reported by Inglis *et al.*[8] it was expected that facial action units would show a reduction in pain for lambs administered a combination of meloxicam and Tri-Solfen®, however this was not the case and hence the first hypothesis is rejected. The inconsistent findings between the current study and Inglis *et al.* [8], together with the inability of facial expression to detect differences in pain between mulesed and Control lambs one day following mulesing, suggest that facial expression may be less sensitive than behavioural measures of posture and gait. It is clear that until pain relief treatments used by the sheep industry can provide repeatable and significant reductions in pain, such that mulesed lambs with pain relief resemble lambs that are not mulesed, the sheep industry should continue to pursue non-mulesed options for control of flystrike.

Total facial action units for lambs that were not mulesed indicated that the lambs were experiencing some pain when measured at 1, 5- and 26-hours post-mulesing. This is similar to previous findings in both sheep and other species.[15,22-24] Facial expression may change due to influences such as fear and stress. Furthermore, climatic conditions may also play a role. The Control lambs in this study were handled and separated from their dam as per the other treatments, which was likely to create stress that was reflected in their facial expression. The impact of climatic conditions was also evident as assessment for pain via facial expression and behaviour increased for all groups 26 hours post-mulesing after heavy rain and lower ambient temperatures (15.5mm and 15.2°C).

Activity and time spent with dam were found to be complementary measures. Lambs that were not mulesed displayed greater activity which was mimicked by spending more time with the dam compared to lambs that were mulesed with or without pain relief. Results from Inglis *et al.*[8] suggest that the decreased activity is not associated with the lamb lying down but rather standing still. This is consistent with previous evidence that mulesing is associated with statue standing[7]. Previous information about time spent with dam or distance between lamb and dam is limited. Grant[7] suggests that greater distances between dam and lamb would signal alterations in normal behaviour and on a paddock scale could result in mis-mothering. However, no results were presented and as such it is only a suggestion of the potential usefulness of the measure of pain. Conversely, Futro *et al.*[19] found that ewes direct attention towards lambs that show the greatest pain related behavioural response. Further work is therefore required to determine if the time lambs spend with their dam is a valid measure of pain assessment for lambs. In addition, further work is currently underway to characterise the activity data reported in the paper to include lying, standing, walking and grazing.

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Small lungworm- could it be affecting production in heavily infected flocks?

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Three species of lungworm occur in sheep in Australia, including the small lungworms *Muellerius capillaris and Protostongylus rufescens*. These infections of sheep are often detected at post-mortem examinations and at abattoirs but thought unimportant because few obvious clinical signs or production losses can be directly attributed to them on farms. However, heavy lungworm infections may cause production loss, either directly or by potentiating other respiratory diseases, such as pneumonia.[1] The objective of this study was to quantify production losses associated with small lungworm infection in sheep.

Three farms from the southeast of South Australia were identified with a high risk of lungworm infections (results from Farms A and B are included in this abstract). Live weight, growth rate and carcase weights were compared to the presence and severity of lungworm infection based on visual assessment of lungworm nodules. Lungs were scored on a scale of 0-3, with 0 indicating no nodules and 3 being the most severely affected, with extensive nodules throughout the lungs.[2,3] The presence of *M.capillaris* was verified on a subsection using histology and morphological identification.

On Farm A, 100% of lambs monitored were infected with small lungworm based on visual assessment of the lungs (total n=217; lung score 1 n=120; lung score 2 n=63; lung score 3 n=34). The average growth rate over 15 days in May 2018 was 200g/day (±100 standard deviation) and approached a statistically significant difference between lung scores (P= 0.05). The average carcase weight was 21.4kg (±1.7 standard deviation) which was statistically similar between lung scores (P=0.4).

From Farm B, 71% of lambs were infected with small lungworm (total n=123; lung score 1 n=118; lung score 2 n=5; lung score 3 n= 0). The average growth rate over 42 day from May to June 2018 was 100kg/day (±100 standard deviation) and was statistically similar between lung scores (P=0.4). Average carcase weight was 22.0kg (±1.7 standard deviation) and was statistically similar between lung scores (P=0.5).

These preliminary results are not conclusive but suggest that production was not affected by the presence of small lungworm lesions. All lambs from Farm A were infected, and so these could not be compared to uninfected lambs. Most infected lambs from Farm B had only mild (score 1) lesions and these could not be compared to severely infected lambs.

Further comparison between other farms in the southeast of South Australia and other age classes is being undertaken. Additional investigations aim to better describe where small lungworm commonly occurs in South Australia, develop improved diagnosis methods using molecular techniques and describe the seasonal pattern of small lungworm infections.

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Rectal Tears

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Nearly all rectal tears encountered in equine veterinary practice are a result of iatrogenic injury. Non-iatrogenic rectal tears have occurred during dystocia and during breeding when the stallion's penis accidentally enters the mare's rectum. Spontaneous rupture of the rectum has been reported to result from neurogenic faecal retention and from ischaemic necrosis caused by thrombosis of the caudal mesenteric artery. Many may assume iatrogenic rectal tears are the result of inappropriate actions of the veterinarian – rough technique and inadequate restraint – and, although these may contribute, in the vast majority of cases rectal tears are the result of rectal wall contraction around the veterinarian's arm or hand resulting in tearing of the wall. Inherently it is a numbers game, with the more rectal examinations a veterinarian performs, the closer they become to the first rectal tear they may induce, or second etc. This should reiterate the importance of informing owners of the risks associated with rectal examination prior to the examination rather than shrugging off a rectal examination as a routine investigative procedure. Routine measures should always be in place to prevent rectal tears – such as liberal lubrication to the veterinarian's forearm and hand and appropriate chemical and physical (in a crush) restraint. Gentle technique rather than forceful palpation against peristaltic waves and passage of the hand past intra-abdominal structures of interest and then palpation during retraction will also lessen the risk of the horse sustaining a rectal tear.

Extra precaution is recommended in horses reported to be at greater risk - Arabian horses, ponies and small breeds, horses displaying signs of colic, horses that have sustained a previous rectal tear and stallions/geldings which are unaccustomed to rectal palpation. Tong Lou et al showed N-butylscopolammonium bromide (Buscopan) can decrease rectal pressure by 68%.[1] This chemical has a very rapid onset of action; it significantly reduces rectal straining and should be considered before palpation of higher risk horses. It will cause a transient increase in heart rate, so it should be given after the physical examination, particularly in horses with abdominal pain. However, care is indicated as the use of N-butylscopolammonium bromide and other non-steriodal anti-inflammatories used concurrently is contraindicated due to the risk of nonsteroidal associated complications. Alternatively, propantheline bromide can be used to induce gut wall relaxation, it will also increase heart rate. The gastro-intestinal and cardiac side-effects have a significantly longer duration then that with N-butylscopolammonium bromide. Lignocaine administered rectally prior to examination is commonly undertaken, however the lignocaine must be absorbed through the mucosa to obstruct nerve impulse conduction and prevent activation of receptors involved in rectal wall. There is no evidence to suggest the value of rectally administered lignocaine in relaxation of rectal smooth muscle.

As far as true equine veterinary emergencies are concerned, rectal tears are one of them. When the veterinarian feels the sudden loss of compression against the arm during a rectal examination and then withdraws to see the blood-stained rectal glove the two most important actions come next:

- Immediate first aid for the horse to minimise faecal contamination and optimise chance of survival for the horse
- Communicate openly and thoroughly with the owner

The promptness and quality of the immediate first aid provided to the horse after a rectal tear is sustained will not only contribute greatly to the horses' chance of survival but also to the outcome of any potential claims of malpractice. Immediate actions following concern that a rectal tear has been sustained should be to ensure appropriate chemical and physical restraint of the animal, and then assess the rectal tear and determine its grade. The use of acepromazine in chemical restraint is contraindicated given the propensity for systemic hypotension which, if septic shock ensues, may compound the systemic physiologic instability. Caudal epidural anesthesia should be undertaken immediately, as the onset of action will be delayed, by how much will depend on the agent of choice. I routinely use a combination of xylazine (0.1 mg/kg) and mepivicaine (0.2mg/kg) as this will result in longer duration of action and will minimise the degree of hindlimb ataxia. In thinner mares an 18g 3.5in hypodermic needle will be sufficient, however in mares that are in heavy body condition a spinal needle should be used. Elevation and depression of the tail whilst palpating the rump/tail base region will improve identification of the first mobile intervertebral space. The more cranial the anaesthesia is deposited the more of the rectum and small colon will be desensitised, however the risk of ataxia with cranial migration also increases. Local anaesthesia and aseptic preparation of the site identified for epidural placement should be undertaken. I routinely use the hanging drop technique to ensure correct placement of the anaesthetic agent. Loss of anal tone and a floppy tail indicates the epidural anaesthesia has occurred. This will then allow for grading of the rectal tear with a liberally lubricated arm to gently palpate the rectum and determine the extent of the tear. Anatomically the rectum consists of peritoneal and retroperitoneal portions, with most rectal tears reported to occur around 25-30cm from the anus in the peritoneal portion in close proximity to the junction of the rectum and small colon. Most tears are longitudinal and occur dorsal third of the rectal wall – in the 10-2 o'clock region of the wall.

Grade 1 tears: only the mucosa and submucosa are torn.

Grade 2 tears: only the muscular layer is disrupted, causing the mucosa and submucosa to prolapse through the defect and create a site for fecal impaction.

Grade 3a tears: involve all layers except the serosal.

Grade 3b tears: involve the mesorectum and retroperitoneal tissues.

Grade 4: tears involve all layers and cause fecal contamination of the peritoneal cavity.

The goals of immediate first aid are to reduce feacal contamination and pre-empt septic shock. It is not the tear that will kill the horse, rather it is the feacal contamination of the abdomen. Broad spectrum antimicrobial therapy should be initiated - penicillin (22,000iu/kg IM BID), gentamicin (6.6 mg/kg IV SID) metronidazole (15mg/kg PO TID) are recommended. Flunixin meglumine (1.1mg/kg IV BID) should also be administered not only for the anti-inflammatory properties but also for the anti-endotoxic activity. In an effort to prevent progression of rectal tears in to higher grades, or expansion of rectal tears, due to feacal packing, rectal evacuation and placement of a "tampon" should be undertaken. The tampon should be constructed with 3inch stockinette adequately long enough to extend from the anal sphincter to approximately 5cm cranial to the tear. The stockinette should be lightly packed with povidone iodine-soaked cotton wool at the tied end, well lubricated and placed in the rectum extending beyond the tear, and then once in place filled with further soaked cotton wool, before securing at the external anal sphincter with a purse string suture or towel clamps. Care should be taken not to overfill the tampon and place excessive tension at the location of the rectal tear, rather fill just enough to cease passage of fecal balls. In circumstances in the field where stockinette is not readily available then use of panty hose packed with cotton wool or cotton wool alone can be instituted.

In most cases, rectal tears are encountered infrequently and, as such, so is the management of such cases in the field. This, together with a not uncommonly emotionally charged environment, warrants prompt referral for ongoing management. If prolonged travel is required, then catheterisation and institution of IV fluid therapy may be warranted. The options for management are dependent on the grading of the tear, facilities available and financial constraints of the client. Contacting the nearest referral hospital and gaining further guidance from local specialist practitioners is recommended to help guide clients in their decision making on options for ongoing management of the horse. Grade one and two rectal tears rarely require surgical management rather ongoing medical management of broad-spectrum antimicrobial therapy, flunixin meglumine, nasogastric intubation with mineral oil and dietary modification with reduced fibre content to reduce feacal load on the rectum – small bran mashes, fresh green grass. Grade three tears can be managed medically or surgically. Medical therapy can be prolonged and intensive. It involves medication as above along with ongoing repeated manual rectal evacuation occurring initially very frequently throughout the day and reducing in frequency over days to weeks. Septic peritonitis may ensue and be managed with peritoneal lavage. The cost of which in some cases may be no less then that of surgical management. Surgical management consists of direct suturing, endoscopically guided suturing, blind suture closure, placement of a temporary rectal liner, temporary colostomy and permanent colostomy. Survival of horses with rectal tears reduces with increasing severity of the tear - Grade one and two tears have a very good prognosis with survival rates in the 90%'s reported. Garde 3 tears hospitalised for ongoing management are reported to have around 30-75% chance of survival, with progression to a grade 4 tear being the demise of most non-survivors. Grade 4 tears have a poor prognosis with only around 2-5% of horses surviving.

Self-blame by the attending veterinarian not uncommonly ensues in cases where a rectal tear has been sustained by a horse. In most cases, when the procedure has been undertaken as valid diagnostic procedure and immediate first aid is instituted then, there is no cause for negligence or self-blame. Rather it is the unfortunate outcome of a procedure which carries risk, and although may not occur commonly, is a risk which is present every time a rectal examination is undertaken. A risk all clients need be aware of.

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When Gelding Goes Wrong

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Sarmatians and Scythians are reported to have had geldings in the times of the early romans. The ''peculiarity'' of gelding to make their horses easy to manage was noted by Roman armies, who themselves much preferred males' horses entire – an angry man on an angry stallion was thought of as a formidable weapon in ancient battles. These recordings give us insight into just how long we have been performing castration in the horse and although the recordings of techniques used and complications from these times are not easily obtained, if there are any, they are assumed to be similar to modern times. Unfortunately, despite the major advances in veterinary medicine, including surgical technique, equipment, perioperative medication and analgesia for the most part, routine equine castration remains a rudimentary and brutal surgical procedure. This in the most part is due to financial constraints of clientele and, in some cases, tradition.

The reported rate of surgical complications associated with equine castration varies greatly (5-60%) depending on the study design, population observed, and location. Reports of incidence of complications is limited, in the most part, to those studies which have very close and consistent post-operative assessment of the animal. Although hard to compare to the human field (thankfully castration is not a wide spread routine procedure!), the Gratton institute in 2018 reported a 3% rate of at least one complication in patients undergoing same day procedures. This is far superior to that of equine vets performing routine castration procedures.

There have been a number of reports in the recent literature which can hopefully be instituted routinely to lessen the rate of complications. Abass and colleagues[1] conclusively showed a reduction in the inflammatory cytokine release associated with the castration procedure when local anaesthetic was infiltrated into testis or spermatic cord. Reduced inflammation means reduced swelling, pain and reluctance to exercise. Alternative surgical techniques involving primary wound closure, use of the Henderson castration instrument, and laparoscopic techniques have all been reported and proposed to reduce the incidence of complications.

Complications of equine castration include: swelling, fever, infection, haemorrhage, hydroceole, peritonitis, eventration, evisceration, penile injury, and death!

Swelling: Oedema of the prepuce and scrotum is the most commonly reported complication of the routine gelding procedure. Some swelling is expected, however ongoing swelling beyond the first week or excessive swelling, especially coupled with stiff gait, reduced appetite or reluctance to move, is abnormal. This is usually the result of inadequate surgical drainage and/or inadequate exercise postoperatively. Untreated, excessive swelling can result in the development of paraphimosis, surgical site infection, and dysuria. Management of such cases, not complicated by surgical site infection, can be managed on farm. The horse should be appropriately restrained (sedated, twitch, crush) and the site aseptically prepared to allow for creating surgical drainage. Anti-inflammatory medication and instituting appropriate exercise to prevent premature sealing of the surgical site is imperative.

Fever and infection: The use of perioperative antimicrobials in Australia has recently been reported to be significantly higher than previous reports from studies in the USA. Interestingly it has not changed the incidence in which we are seeing surgical site infections following castration. Horses can present with infected surgical sites days, months and even years following castration. In the more acute instance horses commonly present with swelling, fever, incisional drainage, and stiff gait/lameness. Management on farm of such cases should comprise appropriate restraint, aseptic preparation of the site, creating appropriate drainage, deep sampling for culture, profuse lavage with sterile isotonic solution, anti-inflammatories, broad spectrum antimicrobials and exercise. Sites may require repeat surgical drainage to resolve. Rosanowski et al[2] recently reported on complications of horses undergoing castration in Hong Kong and found a potential pattern of antimicrobial resistance in horses with postoperative infection to commonly instituted first line antimicrobials trimethoprim sulphur and oxytetracyclines, and greater sensitivity to penicillin-gentamicin combinations. Thus reinforcing the need for more sampling to ensure appropriate therapy has been instituted, and to provide greater insight to the antimicrobial sensitivity of the local bacterial populations.

In the more chronic forms, horses may present with a draining sinus and variable external swelling, just swelling of the scrotal/inguinal region and stiff gait or may have no external localising signs rather weight loss, variable fevers and stiffness of gait. In cases of inguinal abscessation they may only be localised on rectal examination. Ultrasound examination preoperatively to assist in surgical planning is recommended; if possible, sampling of any exudate at that time for bacterial culture and antimicrobial sensitivity testing. It is strongly recommended such cases undergo management in the hospital setting. Not uncommonly, intraoperative haemorrhage can be significant, and the surgery can be tedious and unexpectedly prolonged. En bloc surgical resection under general anaesthesia of draining sinuses and abscessed tissue is recommended where possible. Alternatively, surgically creating drainage and profuse lavage of infected tissue where complete resection is not possible. Postoperative management will depend on the surgical management undertaken, however in cases where ongoing drainage is required management should be as above.

Haemorrhage: Comino et al 2018[3] compared the leakage pressure of the testicular artery between the *serra* and the *reimer* emasculators, and concluded the *reimer* emasculator to be superior for open castrations, yet no statistical difference between leakage pressures in closed castrations. A small amount of bleeding should be expected during the immediate postoperative period following castration in which scrotal incisions remain open for drainage but when a slow drip becomes a fast drip or continuous stream then identification of the source of the bleeding and haemostasis should be undertaken. Haemorrhage can arise from the testicular artery, the cremaster muscle and branches of the external pudendal vessels. Immediate management in the field should involve appropriate restraint of the standing horse and examination of the spermatic cord stump. Excessive tension on the stump may occlude the offending vessel, as such, gentle traction is recommended. If the bleeding is coming from the stump, then emasculation and application of a transfixation suture with heavy absorbable suture is recommended (#2 vicryl). In cases where re-emasculation is not possible then crushing forceps should be applied and left in place for 12-24 hours. If a scrotal or cremaster muscle vessel is the source of bleeding, then application of crushing forceps and transfixing ligation should be undertaken. In cases where the source of the haemorrhage cannot be identified in the standing horse, examination under general anesthesia may be warranted (however not always fruitful!!!). In cases in which the elusive vessel cannot be identified packing of the scrotum with laparotomy sponges is recommended. If ongoing haemorrhage or localisation of the source of the heamorrhage cannot be determined, then referral is recommended.

Intra-abdominal haemorrhage can occur, no matter which technique is employed – open, closed, partially closed, Henderson technique, laparoscopic. Horses can present with low grade colic to hypovolaemic shock. Horses which present with hypovolemic shock may display tachycardia, tachypnea, pale mucous membranes, dull mentation, weak pulses, cold extremities, and generalized weakness. Measurement of PCV and TS in theses may provide a false sense of security acutely as they often remain normal for 6-12 hours post haemorrhage (6hr TS; 12-24hrs

PCV). Ultrasound examination of the abdomen should reveal the characteristic echogenic swirling pattern of haemabdomen. Stabilisation with intravenous fluids and then surgical ligation of the vessel should be undertaken. In all cases of post-castration haemorrhage broad spectrum antimicrobial therapy should be instituted as they are all at greater risk of surgical site infection.

Hydrocoeles are an accumulation of sterile serous fluid within the vaginal cavity of the redundant scrotal tissue. They generally occur months to years following castration, and present as flocculant, non-painful fluid swellings in the scrotal area. They are of little clinical consequence and do not require treatment apart from cosmetic reasons. Simple drainage usually results in limited short-term improvement with fluid accumulation recurring soon after. Surgical removal is the treatment of choice if resolution is desired.

Peritonitis: Although uncommon, given the communication between the cavity of the vaginal tunic and the abdomen, ascending infection can and does occur following castration. Most horses referred to our hospital following castration due to septic peritonitis have been castrated in the field in the preceding 2 weeks. They have presented with weight loss, fever, depression, tachycardia, dehydration, variable colic, and anorexia. Abdominal ultrasound can be very helpful in identifying increased abdominal fluid, in most cases with significant echogenicity, however, does not display the swirling pattern presented with haemabdomen. Abdominocentesis should be performed and the fluid should be submitted for cytological evaluation, bacterial culture, and sensitivity testing. Relying on cell counts alone can be misleading as it is not uncommon for total nucleated cell counts of clinically normal horses to be elevated as far as 100,000cells/DL. This is thought to be due to a chemical peritonitis associated with small amounts of heamorrhage in to the abdominal cavity. However, in cases of septic peritonitis the cytology should reflect sepsis – marked predominance of degenerate neutrophils and the presence of bacteria. Immediate management on farm involves stabilisation of the patient for transport – non-steriodal anti-inflammatories, broad spectrum antibiotic therapy (after sample collection!!!) and bolus of IV fluids may be required depending on distance required to travel for referral. Referral to a hospital for ongoing management is recommended.

Evisceration: Although uncommon it is the complication that we all fear as it is the most likely to be catastrophic! The incidence of evisceration is variable depending on the population studied and the castration techniques used. Owens et al 2018[4] in their study on the Australian equine vet population found evisceration to occur in 0.11% of castrations. Risk factors include breed (Standardbreds and draft breeds are over-represented), pre-existing inguinal hernias, presence of an inguinal hernia as a foal, and an internal inguinal ring that 2 or more fingers can fit into on rectal palpation. Undertaking a closed castration, inguinal castration with closure of the inguinal rings or modified open castration with primary closure can reduce risk of eventration (and evisceration or does it only reduce eventration???). Cases in which only omentum has protruded (eventration) can be managed on farm with traction, ligation and transection or emasculation. Rectal palpation may be performed to ensure no intestinal protrusion into the inguinal ring. Ongoing management with broad spectrum antibiotics is recommended. Evisceration with small intestine usually occurs soon after surgery but has been reported to occur well past a week after surgery. The priority of on farm management should be directed at preserving bowel viability - cleaning the bowel of gross contamination and preventing further trauma to the exposed bowel. Ideally profuse lavage with sterile isotonic solution and containing it in the scrotum with sutures or towel clamps. If the intestine cannot be contained in the scrotum then slinging in clean moist towels, sheets, drapes to prevent further trauma and contamination to the bowel during transport to a referral hospital. Horses should immediately commence broad spectrum antimicrobials, nonsteroidal anti-inflammatories, and sedated if systemically stable. Survival rates are widely variable, with intestinal viability, amount of intestine involved, degree of contamination and surgical approach all having a large influence on the outcome.

Penile Injury: Direct iatrogenic injury to the penis occurs uncommonly during castration procedures, and a thorough understanding of the regional anatomy cannot be overemphasised. In most cases the penis is mistakenly identified as a testis and is dissected from the body wall. Fortunately, in most cases the error has been identified prior to emasculation. If the suspensory ligament/connective tissue suspending the penile shaft to the body wall has been disrupted then surgical reconstruction will be required, followed by broad spectrum antibiotics, anti-inflammatories, cold therapy and slinging of the penis to prevent paraphimosis. If the penis is transected, then referral for phallectomy or peroneal urethrostomy is recommended.

Death: Usually a complication of the anaesthetic rather than the surgery. Although in most cases a pre-anaesthetic clinical examination does not yield an abnormality or clinical concern, it will contribute greatly to the management of the aftermath.

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Abdominal ultrasound in the colicky horse

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Introduction

Transcutaneous abdominal ultrasonography is a readily accessible and non-invasive diagnostic procedure used to investigate abdominal pain in the horse. It provides structural and functional information on the state of the abdominal tract. Abdominal ultrasonography is most useful when combined with other clinical and diagnostic findings. Abdominal ultrasonography can be used to localise the lesion, determine if the condition requires medial or surgical management, allow monitoring of the response to treatment and assist with prognostication. Abdominal contents imaged using percutaneous abdominal ultrasonography include: stomach, small intestine, dorsal and ventral large colons, kidneys, small colon and the presence and character of peritoneal fluid.

When to perform abdominal ultrasound in the colic

Investigative methods used to support clinical examination findings in the colicking horse include abdominal palpation per rectum, passing a nasogastric tube, judging the response to analgesics, abdominocentesis and abdominal radiography. Abdominal ultrasonography, in conjunction with the above methods, helps to localise the lesion, narrow the differential list and optimise management. Abdominal ultrasonography is particularly useful in differentiating small intestinal strangulating lesions from non-strangulating lesions, identifying stomach distention, identifying left dorsal displacements of the large colon, identifying cases of colitis and peritonitis.

Equipment and patient preparation

A low frequency (2.0- to 5.0-MHz) curvilinear transducer with penetration to a maximal depth range of at least 25cm is ideal for detecting the majority of abdominal abnormalities in the equine colic patient. If a curvilinear probe is unavailable a 5.0-MHz linear rectal probe may be used instead, but this will give a much smaller window of view compared to the curved pie-shaped image produced by the curvilinear transducer. The patient should be examined standing and ideally unsedated. If sedation is required note that alpha-2 agonists (xylazine, detomidine) cause a transient state of ileus and mild dilation of the small intestine. Clipping hair using a number 40 clipper blade, cleaning the skin and applying ultrasound coupling gel will maximise the quality of the image, however in many thin haired horses clipping is unnecessary and can be avoided by applying copious amount of alcohol or water to the area.

Probe orientation, depth and spatial anatomy

The examiner needs to be aware of the orientation of the probe relative to the patient and understand which way the image is displayed on the screen. Ultrasound transducers have an identifying mark or light that is used to orientate transducer placement on the patient relative to the image projected onto the viewing screen. The transducer mark corresponds with the probe orientation mark on the screen (usually the left side of the image). In abdominal sonography the long axis of the probe is orientated within an intercostal space (ICS), therefore a slightly obliqued transverse image is produced (slice across the long axis of body). If the probe marker is positioned towards the dorsal side of the horse the left side of the image represents the dorsal aspect. If the transducer is flipped 180° in the transverse plane, the left side of the image on-screen will represent the ventral aspect of the slice. The depth of the field of view is also important, as structures may be missed if the depth is too shallow.

The depth of view is displayed on the centimetre scale on each image. When examining deeper structures the focal zones should be positioned in the far field to enhance visualisation of deeper structures. It is important that the examiner understands normal abdominal anatomy, especially the spatial relationship of the viscera, as this holds the key to distinguishing normal from abnormal findings.

Generation of an ultrasound image

Ultrasound images (known as echoes) are only produced when ultrasound waves are reflected back to the transducer. If adjacent tissues have the same acoustic impedance, no sound is reflected and the ultrasound waves penetrate into deeper tissues. Waves are reflected when the sound wave reaches an interface between two tissues with different acoustic impedances and a portion of that wave is bounced back. The greater the difference in acoustic impedance between tissues, the greater the proportion of sound waves that get reflected back. Reflection is not dependant on the acoustic impedance of a single tissue. This is the reason bone-soft tissue interfaces and soft-tissue-air interfaces are both highly reflective and produce an indistinct hyperechoic line on the image, despite the fact that the bone and air are at different ends of the acoustic impedance spectrum. Highly reflective interfaces also prevent penetration of the ultrasound wave to deeper tissues, which is a major limiting factor in equine abdominal ultrasonography.

Complete abdominal ultrasonography technique

In cases with chronic colic, or the stabilised acute colic, complete sonographic examination of the abdominal contents is recommended. Complete transcutaneous evaluation of the abdomen requires both a low frequency transducer (2.0- to 5.0-MHz) for examining deeper structures and a higher frequency transducer (5.0- to 13.0-MHz) for examining superficial structures (e.g. the intestinal wall). Perform the examination systematically and consistently to minimise any abnormalities being overlooked. One technique is to start the sonographic examination on the right side of the abdomen in the caudodorsal paralumbar fossa scanning in a dorsal to ventral manner, moving cranially once ICS at a time up to the 6th ICS. This is followed by imaging the left side of the abdomen starting at the caudodorsal paralumbar fossa and examining in the same dorsal to ventral and caudal to cranial manner. Finally, the ventral abdomen is imaged in a caudal to cranial direction finishing at the costochondral junction.

Fast localised abdominal sonography of horses (FLASH)[1]

Rapid evaluation of the equine abdomen may be necessary in cases of acute colic, therefore a group of researchers have developed a protocol describing seven specific acoustic windows to allow rapid transcutaneous ultrasonography in the colic. The entire FLASH exam can be performed with a 3.0- to 3.5-MHz transducer. This method is considered useful in detecting major intra-abdominal abnormalities even by inexperienced operators. The seven topographical locations are the ventral abdomen, gastric window, spleno-renal window, left middle third of abdomen, duodenal window, right middle third of abdomen and cranial thorax. This allows the abdomen to be evaluated for the presence of free peritoneal fluid, ability to see the left kidney, small intestinal motility, distention, wall thickness and contents, large intestinal distention, wall thickness and contents.

Stomach

The stomach is imaged medial to the spleen on the left side of the abdomen between the 9th-13th ICS, at approximately the level of the shoulder. It typically spans 3-5 ICS and is not seen on the ventral or right side of the abdomen. The stomach is imaged as a curved hyperechoic line adjacent to the spleen and gastrosplenic vein, which is created by the large difference in acoustic impedance at the soft tissue-gas interface between the wall of the greater curvature and the gas in the stomach. This highly reflective interface usually precludes visualisation of deeper structures. However, if gastric fluid is present ventrally a distinct gas-fluid interface may be apparent in the lumen. In the fasted horse, a gastric fold is visible on the surface of the stomach. In cases of gastric distention, the stomach loses its curved shape and extends more than 5 ICS. Note that the sonographic appearance of the stomach will alter depending on its contents (gas, fluid or ingesta) and due to recent gastric lavage or gastroscopy.

Small intestine

The duodenum is identified ventral to the right kidney and can be followed cranially along the right abdominal wall, where it is located between the right dorsal colon and the left liver lobe. Duodenal distention can occur with distal small intestinal obstruction, post-operative ileus and proximal enteritis. Duodenal wall thickening is present in cases of duodenitis or proximal enteritis. Normal jejunum can usually be identified in the caudal left portion of the abdomen ventral and medial to the spleen. It should have a nondescript appearance on ultrasound and wall thickness should not exceed 3mm. Abdominal ultrasonography can be used to differentiate strangulating from non-strangulating small intestinal obstructions. Strangulating small intestine may be turgid, immotile, with thickened walls, whereas non-strangulating lesions are likely to have hypomotile, distended loops without increased wall thickness. The presence of free peritoneal fluid and collapsed but normal loops jejunum, with a second section containing turgid, immotile and thick walled loops increases the suspicion of a strangulating lesion.

Large intestine

The large colon and caecum occupy much of the ventral aspect of the abdomen. They are identified by their anatomical location and surface appearance. When orientated normally the ventral colon is most ventral in the abdomen and its sacculations are sometimes visible, whereas the dorsal colon is positioned dorsally and is non-sacculated.

The caecum is imaged from its base in the upper right paralumbar fossa, along the costal arch, to its apex in the ventral abdomen. The lateral caecal vessels are usually detectable. The normal right dorsal colon is imaged as a curved hyperechoic line located on the right side of the abdomen ventral and medial to the spleen.

Gas distended colon will produce a series of horizontal parallel reverberations on ultrasound and may be found with displacements and other simple obstructions of the large colon (i.e. impactions). In cases of large colon or caecal impaction, hyperechoic intraluminal material may be detected that casts a strong acoustic shadow. The wall may be flattened with normal-to-thinned wall thickness.

Right dorsal displacement of the colon may be suspected if the normally medially positioned colonic vasculature is visible, and engorged, adjacent to the right body wall just ventral to the level of the ribs.

In cases of right dorsal colitis secondary to non-steroidal anti-inflammatory drugs, imaging can demonstrate oedema with a markedly thickened wall along the right side of the abdomen. Normal thickness of large colon should not exceed 3mm. Other clinical findings in cases of right dorsal colitis include hypoproteinaemia, hypoalbuminaemia and pyrexia.

Cases of colitis are usually identified by their clinical findings such as a milder degree of colic, the presence of diarrhoea, fever and leukopaenia. Widespread thickening and oedema of the entire colon wall may be identified sonographically.

The left kidney is found in the left dorsal abdomen in the 16th or 17th ICS or paralumbar fossa, where it sits medial to the spleen. In cases with a nephrosplenic entrapment, or left dorsal displacement of the large colon, colon will be identified medial to the spleen and the view of the left kidney obscured. The spleen may also be displaced from the body wall. Sonographic findings should be confirmed with abdominal palpation per rectum, as a false positive

diagnosis may occur if the small colon has migrated between the spleen and kidney precluding transabdominal viewing of the left kidney.
Torsion or volvulus of the colon are accompanied by acute onset, severe abdominal pain and the post-partum mare is at an increased risk of this type of colic. Colon torsion may result in vascular compromise, resulting in a thickened and oedematous colon wall.

Peritoneal fluid

Peritoneal fluid can usually be detected in the cranio-ventral abdomen between the spleen and the caecum. It is best to use a high frequency transducer to characterise the type of fluid. In a normal horse there is usually only a small amount of anechoic fluid. In cases with peritonitis there will be an increased amount of fluid of variable echogenicity. Fibrin strands may be visible.

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How to manage the non-weight bearing horse

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Introduction

Non-weight bearing lameness in the horse can be a stressful situation for animal, owner and veterinarian alike. The aim of this talk is to provide veterinarians with a systematic approach to managing severe lameness in the horse, including making a diagnosis, formulation of a treatment plan and administration of first aid. Causes of non-weight bearing lameness include subsolar abscesses; solar penetrations; septic synovial structures; fractures; laminitis; cellulitis.

History

Taking a thorough and complete history is important. Age, breed, sex and discipline can predispose horses to certain injuries, therefore knowing these facts may help to focus the differential list and shorten the time to diagnosis. Pertinent questions to ascertain from the client include the onset of lameness (acute, chronic or insidious onset); the duration of lameness; and whether the inciting case is known. Has the horse received any treatment or medications prior to onset of lameness and since the onset of lameness? Is there any other relevant veterinary history?

Clinical examination

Assess the horse's behaviour and posture prior to touching the horse. Perform a short but thorough clinical exam, including rectal temperature, heart rate and respiratory rate, mucous membrane colour and fill. In the neonate, check for any umbilical abnormalities or other joint effusions. Identify the affected limb(s) and observe the horse at the walk, even if only for a few strides. Take note of foot placement, the cranial phase of the stride and any other gait abnormalities. Visual inspection and digital palpation of the affected limb from the hoof to the proximal limb should be performed in a methodical manner. Commencing distally assess for the presence of digital pulses, increased heat in the hoof wall or coronary band swelling. Clean and pare the hoof if necessary and apply hoof testers across the entire sole. Inspect the limb for any haemorrhage, wounds, joint effusion, soft tissue swellings or asymmetry. Clipping and cleansing of the area may be required if the area is hairy or contaminated. Passively flex and extend the distal limb and upper limb and evaluate for a pain response, crepitus, or instability. Compare findings to that of the contralateral limb.

Diagnosis:

Clinical examination will focus the differential list and indicate if further diagnostic tests are required. Prompt diagnosis of a non-weight bearing lameness is of upmost importance, to ensure appropriate management of the condition.

Diagnostic imaging

Radiography and ultrasonography are important diagnostic tools and they are readily available to the practitioner. Advanced imaging may also be indicated: nuclear scintigraphy (particularly for suspected stress fractures), CT and MRI.

Diagnostic anaesthesia

Perineural anaesthesia can successfully facilitate a diagnosis in the non-weight bearing horse, however caution and examination at the walk only is recommended. Radiography may be required to rule out a bony lesion prior to embarking on perineural anaesthesia. Nerve blocks can also facilitate examination and diagnostic imaging procedures. Chronic pain, acidotic tissues and altered tissue blood flow can affect the efficacy of anaesthesia.

Synoviocentesis

In cases of suspected synovial sepsis, obtain a synovial fluid sample as soon as possible using strict aseptic technique. Use an approach that is distant to any wound or area of compromised skin. Submit synovial fluid for both cytology and culture and sensitivity testing, prior to medication with antimicrobials (systemic and local therapy). A positive culture is more likely if a blood culture medium is used and a large volume of synovial fluid submitted. Gross examination findings of septic synovial fluid include normal yellow to dark orange or red colour and may sometimes be an opaque pink or yellow. A septic synovial structure can be suspected on gross examination by colour, turbidity and viscosity and cytological parameters used to definitely define sepsis are a NCC $\geq$ 30 x10^9/L; TP $\geq$ 40 g/L and degenerate neutrophil differential $\geq$ 90%. However, lower counts do not preclude infection, particularly in per-acute cases, or those with significant amounts of fibrin as the cells aggregate within the fibrin clot.

Conditions: Subsolar abscessation

Hoof abscesses generally present as an acute 'fracture lameness', however beware the clinical signs can vary. Potential aetiologies include solar bruising, placement of a shoe nail too close to the sensitive laminae and solar puncture. Infection develops in the region of the sensitive laminae and accumulation of purulent material inside the hoof capsule creates intense pain. Examination may reveal increased digital pulses, heat in the hoof wall, distal limb or coronet band swelling, pain on hoof testers or digital pressure over the sole, coronet band and/or heel bulbs. Lameness usually improves significantly after local perineural anaesthesia, however due to upregulated pain pathways a more proximal nerve block may be required to abolish the lameness. Treatment involves creation of drainage and removal of infected tissue. Remove the shoe and pare the sole. Pay close attention to any nail holes for evidence of moisture, solar defects and debris. Re-apply hoof testers gently across the entire sole and identify the site of the suspected abscess. Use a hoof knife or curette to create a small hole to allow drainage, remove necrotic and underrun horn, clean the area with iodine solution and apply a hoof bandage or poultice. Poulticing of the foot should continue until drainage has ceased and the horse is walking comfortably. Light walking exercise may facilitate drainage. Non-steroidal anti-inflammatory drugs (NSAIDs) are indicated if the animal is distressed. Administration of antimicrobials is only recommended if involvement of deeper structures is suspected. Complicated hoof abscesses may develop into septic pedal osteitis or septic arthritis of the distal interphalangeal joint.

Solar penetrations

Nails, metal debris or other sharp foreign bodies can penetrate the sole resulting in severe lameness. The site, depth and direction of the puncture will determine which structures are involved. Puncture of the distal phalanx may result in septic pedal osteitis or a bone sequestrum; synovial sepsis may occur following involvement of the navicular bursa, distal interphalangeal joint or digital flexor tendon sheath; or septic tendonitis following puncture of the deep digital flexor tendon. Owners should be advised to leave any foreign body in situ until veterinary examination, as this will aid identification of the penetrating tract. Radiograph the foot containing the foreign body, or if removed aseptically prepare the sole and use a sterile probe to delineate the tract. Multiple radiographic views are required. Synoviocentesis is required if synovial sepsis is a possibility. Treatment of septic structures involves debridement, lavage with a large volume of crystalloid isotonic saline solution, local and systemic antimicrobials and NSAIDs. Prompt referral to a surgical facility is recommended in cases of synovial sepsis and/or septic tendonitis.

Septic synovial structures

Neonates are more susceptible to infection and septicaemia than adults and consequently are at an increased risk of developing septic arthritis (with or without osteomyelitis) via haematogenous spread. Septic synovial structures in adults generally occurs secondary to a penetrating traumatic injury, or iatrogenically following surgery or intrasynovial injection. Clinical signs include severe lameness, synovial effusion, heat and pain on palpation and flexion. Diagnosis requires synovial fluid sampling (see synoviocentesis above). Radiography of the area to detect for bone involvement is recommended, and they can also be very useful for comparison purposes at a later date if necessary. Treatment involves extensive synovial lavage and debridement of the structure (arthroscopy, needle lavage), plus local and systemic antimicrobial therapy. Broad spectrum empirical antimicrobial therapy should be amended according to the culture and sensitivity results.

Fractures

Fractures may occur due to a traumatic incident such as a kick or fall, as a consequence of repetitive bone strain sustained during high speed exercise, or rarely due to pathological bone failure. Physeal fractures are a common cause of non-weightbearing lameness in the foal. Horse sustaining fractures can be highly stressed, therefore sedation and analgesia may be required to facilitate a thorough veterinary examination and ensure safety of the handler and veterinarian. A combination of a short acting alpha-2 agonist (0.5mg/kg xylazine) and opioid (0.1mg/kg butorphanol) administered intravenously are the author's drugs of choice. Clinical examination findings will vary depending on the location and classification of the type of fracture. Severe lameness may be accompanied by soft tissue swelling, pain on palpation of the area, joint effusion and pain on flexion (if articular), crepitus (if comminuted) or limb instability (if areas of major weight bearing support are involved). Radiography is an invaluable tool to diagnose and classify the character of a fracture and portable digital x-ray machines allow fractures to diagnosed and stabilised appropriately prior to transportation (see below). Ultrasonography can detect disruption of the normal bony contour and is particularly useful for diagnosing pelvic fractures. If surgical intervention and stabilisation of the fracture is possible, prompt referral to a specialist is recommended. Open fractures carry a poorer prognosis than closed fractures.

Stress fractures, or fissure fractures, are most common in racehorses and occur due to repetitive bone loading. These fractures generally present with a severe, transient lameness that improves after a few days of stall rest. Stress fractures are usually undetectable on radiographs for several weeks; therefore, nuclear scintigraphy is the diagnostic method of choice. Common sites of stress fractures include the tibia, pelvis, scapula and humerus.

Emergency treatment and stabilisation of the equine fracture patient

The objective of first-aid treatment in equine fracture patients is to minimise further damage to the fractured limb. Treatment should involve the following: sedation, wound management, administration of analgesia and anti-inflammatory medication, antimicrobial and tetanus prophylaxis, fluid therapy and fracture stabilisation. Stabilisation of the fracture should aim to reduce the horse's pain and anxiety by facilitating partial weight bearing, immobilise the joints above and below the fracture and minimise further compromise of the fracture site and surrounding soft tissues. Types of stabilisation include a Robert-Jones bandage, application of splints and limb casts. Optimal stabilisation of fracture depends on the location of the fracture and each limb can be divided into four biomechanically important regions to assist with appropriate immobilisation techniques.

Laminitis

Laminitis can occur due to endotoxaemia, endocrine disorders, carbohydrate overload, iatrogenic treatment and in the contralateral limb in a horse with severe lameness. Supporting limb laminitis (SLL) is a devastating condition that carries a high mortality rate and it tends to be associated with sinking of the distal phalanx within the hoof capsule. The development of SLL can be difficult to predict, although the severity and duration of the primary lameness are risk factors. Clinical findings include the development of a palpable subcoronary depression, increased digital pulses, extended periods of lying down and reluctance to move around on the support limb. Treatment of the primary cause of the severe lameness should be combined with prevention methods to minimise support limb laminitis, including adequate analgesia, cryotherapy, padded support boots that decrease tension on the DDFT, thick bedding and slinging of the horse to provide some relief.

Cellulitis

Cellulitis is infection and inflammation of the subcutaneous tissues and can be extremely painful condition in the acute phases. The pathogenesis may be idiopathic, related to a known trauma, wound or skin barrier disruption. The condition is usually unilateral and the hind limbs are more commonly affected. Horses can present with full or partial limb swelling, pitting oedema, marked pain on palpation of the area, increased heat, pyrexia, tachypnoea and tachycardia. In the author's experience the swelling and pain are usually localised to the peritarsal region, with secondary non-septic effusion of the underlying tibiotarsal joint. Ultrasonography may reveal widespread subcutaneous oedema and identify pockets of fluid from which can be aspirated for culture and sensitivity testing. The presence of a septic synovial structure should be ruled out. Treatment involves systemic broad-spectrum antimicrobial therapy, antimicrobial intravenous regional limb perfusions, cold hosing of the limb, NSAIDs, cryotherapy, support bandaging and controlled exercise once the patient is able to bear weight.

When are non-technical skills important to me? Earlier or later.

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Non-technical skills and capabilities are increasingly incorporated into veterinary science curricula[4], their importance having been established for graduates future career and personal success[1,2,4,5]. Employers state strong desire for non-technical capabilities in new graduate employees, but recent graduates express a lack of knowledge in these areas, business capabilities being a particular example[2].

Delivery of learning at a time when students' perceived importance of these skills is relatively low may impact the depth and retention of learnings important for students' professional future[4]. A preliminary cross-sectional study of Australian veterinary students revealed the importance attributed to various non-technical skills differed for entry level, mid program and final year veterinary students[3].

Gaps still exist in the literature despite a number of studies on students' attitudes or views of non-technical skills[2,3,4,5]. Importance of self-care, compared to other professional capabilities, has not been reported in a longitudinally conducted survey. Literature is also limited regarding whether the importance of these skills for veterinary students actually changes with time in their program. There also remains a literature gap in how factors such as demographic and human capital might influence the importance of different non-technical skills to veterinary students. This study aims to contribute to the literature with respect to these identified gaps.

Questionnaire based surveys of veterinary students between 2011 and 2018 attending a number of Australian veterinary schools were undertaken. This study uses responses of participants who participated longitudinally (i.e. at two or three time-points in their veterinary programs: early (e.g. orientation week one), mid program, and final year (pre-exit). The questionnaire included demographic and self-report response items, with the latter asking participants to rate (via Likert scale) and also rank how important to them personally, eight non-technical aspects would be in their later professional life. These aspects were animal welfare, communication, ethics of animal use, financial knowledge, leadership, professional and business ethics, self-care and teamwork.

Preliminary findings to date support differences ($p<0.05$) in importance to respondents of several non-technical skills between time points, and between the eight non-technical items. Overall, the most important non-technical skills to students were animal welfare, communication and self-care. Financial knowledge and leadership were least important. Students' view of the importance of communication, self-care and teamwork increased over the three time points while their view of the importance of animal welfare, ethics of animal use, and professional/business ethics decreased.

Prior tertiary education, business background (parent or self) and animal handling experience, attitudes and other self-report items are being included as covariates in growth curve modelling. We aim to see how and why these might predict the level of importance of non-technical skills for veterinary science students as this may be important for admissions or course design.

It is anticipated that our findings from this study will identify potential areas for innovation in veterinary science admissions or curricula design that might lead to an increase in the level of perceived importance and learning motivation of veterinary students for the less popular but none-the-less important non-technical skills for their career.

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Emerging animal welfare considerations for Australian abattoirs

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Introduction

Recent advances in animal welfare standards, animal welfare assessment and humane slaughter techniques have implications for the welfare of livestock during processing. This presentation provides a simple overview of the challenges and opportunities facing the Australian red meat and poultry industries. Two examples of emerging issues are summarised below:

Animal welfare standards

Animal welfare is a growing concern, especially in Europe (European Commission 2007). This public pressure has led to the development and implementation of a range of animal welfare standards, guidelines and assurance schemes across all aspects of livestock production and trade (FAWC 2001, Sundrum 2001, Blaha 2002, Webster et al. 2004, Edge and Barnett 2008, Mench 2008, Veissier et al. 2008, Main 2009, Webster 2009, Rushen et al. 2011), and this trend is set to continue.

Fundamental to the development of an animal welfare standard is the very definition of 'animal welfare'. Many attempts have been made to agree on a concept, however, there is such a large variety of views and perceptions which makes this very difficult. Welfare is multidimensional, comprising of health, comfort, nutrition and expression of behaviour (Mason and Mendl 1993). This understanding is well illustrated by the six general principles, for animal welfare in production, formulated by the World Organisation of Animal Health (OIE). The OIE, which has over 180 signatory countries, defines an animal as having good welfare if it is "*healthy, comfortable, well nourished, safe, able to express innate behaviour… is not suffering from unpleasant states such as pain, fear and distress*" (OIE 2017). The OIE has developed International standards, which are presented as a series of guidelines covering animal production, transport (land and sea), euthanasia and commercial slaughter. Many private animal welfare standards use the OIE standards as a basis for their animal welfare requirements; an approach recommended by OIE themselves.

The processing industry standard, developed by The Australian Meat Industry Association (AMIC) through Australian Meat Processor Corporation (AMPC), is designed to enable industry to meet customer and market requirements, at a level deemed to be higher than regulatory requirements. Verification of conformance with the standards involves the use monitoring activities, the routine collection of Key Performance Indicator (KPI) data and third party audits. In addition to the voluntary meat industry standard, RSPCA Australia have also released standards for pigs and meat chickens. The meat chicken standard has recently been revised and now contains more detailed provisions covering the stunning and slaughter of poultry.

Embedding scientifically supported animal welfare outcome assessment into animal welfare standards is an essential component of a credible scheme. Contemporary animal welfare standards often include resource, outcome and continuous improvement aspects. The verification of animal welfare outcomes in a processing environment can be a challenge and there is a need to develop and improve assessment techniques.

Stunning and slaughter of poultry

The methods available for stunning and slaughtering poultry have often featured in the public domain and have frequently come under scrutiny, with the main concerns identified as poor stunning, birds regaining consciousness before death and their ability to feel pain during slaughter. In Australian poultry processing plants, electrical waterbath stunning and controlled atmosphere (CAS) stunning are the methods most widely used.

Conventional electrical waterbath stunning, although currently the most applied stunning method for chickens, is associated with the greatest number of welfare issues. Although several measurements can be taken to increase the bird's welfare before and during stunning, it has been proven difficult to control all parameters necessary to result in an acceptable animal welfare outcome. The main points of criticism being:

1. Pre-stun stress associated with the removal from transport crates, inversion and shackling
2. Exposure to pre-stun shocks prior to effective stunning
3. Low stunning effectiveness, as evidenced by:
    - the high number of birds that miss the waterbath and are slaughtered/decapitated whilst conscious
    - the use of electrical parameters that have not been demonstrated to induce a state of unconsciousness
    - the use of multi-bird constant voltage systems that cannot deliver consistent current to each bird
4. Meat quality defects associated with the electrical parameters required to give an effective stun
5. Reliance on an effective slaughter method to ensure the bird dies before it recovers from the stun
6. Lack of practical, meaningful indicators of effective stunning

Alternatives to waterbath stunning are being developed and implemented in the poultry industry, particularly in the larger processing plants. Any viable alternative stunning method for chickens will need to eliminate both the animal welfare and product quality issues associated with conventional electrical waterbath stunning.

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Do skills translate from project to practice?

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Introduction

There is a general consensus around the benefits of building research skills as a prominent aspect of veterinary training. Internationally-recognised competency frameworks include research literacy, and research training is an accreditation requirement of the curriculum. While there is evidence of the value of research training in professional degrees in the educational literature, there is little which focuses specifically on veterinary curricula. In contrast, there is ample evidence of the tendency for curriculum overload(1, 2), with explicit recommendations to manage this included in best-practice guidelines(3). In the context of the awareness of the negative impact of curriculum overload on student engagement, the lack of evidence to support the value of research in veterinary curricula relative to other content is notable. Therefore, the aim of this study was to study the effects of a curricular reform which included the introduction of a research-based project into a veterinary degree.

Methods

This paper reports on the comparative performance and attitudes of two cohorts of students doing different veterinary degrees simultaneously in the same institution. Key differences between the curricula were the duration of 5 years following 1 year of tertiary study prior to admission for BSc-BVMS students compared to a total of 5 years for the BSc-DVM, and the different qualification levels, with the 6-year course being at AQF level 7 and the new 5-year DVM program being at level 9. The latter mandated the inclusion of a greater amount of research training and the completion of research-based projects. The ultimate aim of the project is to investigate to what extent this supports the development of transferable skills relevant to the day 1 competencies of veterinary graduates.

Academic performance, survey responses and focus groups were used. Trends in academic performance over the final two years of the degrees during which the projects were conducted and students' perceptions of achieving day 1 competencies were analysed. Attitudes towards the inclusion of research in the degree and towards their own experience of this were also analysed.

Results

When compared to other content in the veterinary degree, students do not value research training or the completion of a project highly in comparison to training in clinical skills, which they perceive to be more directly relevant.

Academic performance of the two classes was comparable. This supports the prevalent view of teaching staff that students in the two classes were largely indistinguishable in their performance during clinical rotations.

Discussion and conclusion

The number of other differences between the two degrees besides the research training and project mean that the effects seen cannot be ascribed solely to the research component of the DVM degree. Conversely, the exposure to the same staff, facilities and a host of other inputs which form part of the 'hidden' curriculum may have obscured potential effects of the curricular reforms, given that both groups were similarly exposed to these. Another limitation of the work is the fact that it was conducted in the context of a double cohort, impacting on outcomes and student perceptions independently of curricular structure and content.

Long-term data collection is envisaged in the form of a graduate survey to further characterise the effects of the curricular reform.

Based on analyses performed to date, there is no discernible difference between the outcomes of the two degrees. This supports the view that it is feasible to achieve the outcomes of an AQF 9 level veterinary degree in 5 years.

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Slings & surgery: A clinical perspective on bull sheaths

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Introduction

The mating apparatus of the bull is highly specialised and complex. The fibroelastic penis is folded in an 'S' (or sigmoid) shape at rest. It straightens and increases in length by approximately 30% at full erection, propelling the tip of the penis forward from its resting position between the back legs to somewhere below the bull's sternum; a considerable distance of travel. The ejaculatory thrust generates yet further extension and forward propulsion, semen is deposited in the cranial vagina of the female, after which the erection subsides, the bull dismounts and the penis returns to the resting position. The role of the sheath in this quick yet well-orchestrated sequence of events is not generally appreciated, but the fact that the sheath is prone to a range of pathologies which commonly render bulls unable to mate illustrates the importance of this organ.

Normal structure and function

The sheath consists of an inner lining, the lamina interna, and an outer covering of skin. The lamina interna is tubular and connects to the penis at the fornix and to the skin at the preputial opening. Between the lamina interna and the abdominal wall dorsally and the skin ventrally are many layers of loose and highly elastic connective tissue which enable the range of movement described above. The blood vessels and nerves traversing these tissues are also convoluted and elastic. The lamina itself is also very pliable and has many folds, easily everting and stretching to cover the extruded penis, at which point it becomes cylindrical in shape.

The direct muscular support to the sheath consists of two main muscles. The cranial preputial muscle or 'protractor' is a bilaterally symmetrical muscle coming down from the sides of the abdomen and meeting just caudal to the preputial opening in most bulls. It forms a sling around the bottom of the preputium, serving the function of the 'sphincter' found at other body openings and can be seen as a distinct upward 'notch' or angle in the skin when it is contracted. The caudal muscle or 'retractor', also bilaterally symmetrical, arises in the region of the external inguinal opening and reaches forward to attach to the lamina interna close to the fornix. It helps to pull the lamina into the preputial cavity when contracted, together with the retractor penis muscles which attach to the penis itself from their origin at the base of the tail. All of these muscles are under autonomic nervous control.

The umbilicus also plays a role in preputial positioning. It consists of a firm fibrous band between the abdominal wall and skin just cranial to the preputium. As such it serves to anchor the skin to the abdominal wall, and can often be observed as an upward notch in front of the preputium.

There is considerable individual variation in these structures, primarily genetic as indicated by the significant breed-based variations. These variations are with regard to five main anatomical points as follows:

i. <u>Looseness of the skin:</u>
The greater amount of loose skin in Bos indicus breeds and synthetic breeds with a high Bos indicus component results in the preputial skin being looser and the preputium being generally positioned further from the body wall. The skin cranial to the umbilical stump is also often pendulous forming a prominent skin fold in front of the umbilicus.

ii. <u>Distance between the cranial preputial muscles and preputial opening:</u>
In bulls where this distance is significant the front portion of the preputium hangs vertically. The amount of sheath in front of the muscles predisposes to eversion of the lamina.

iii. <u>Length of lamina interna:</u>
The above variations are often associated with a greater amount of lamina interna, with profuse folding and a tendency for the excess to evert.

iv. <u>Prominence of the umbilicus:</u>
The length and size of the umbilical remnant accounts for some variation in the distance between the body and the sheath opening and the position of the skin in front of the sheath opening relative to the opening itself.

v. <u>Preputial muscle development:</u>
Both the cranial and caudal muscles vary in their development. Weakness of either or both has the common consequence of ready eversion of the lamina interna; in the case of the cranial muscle due to a weak sphincter action at the opening and in the case of the caudal muscle due to weak retraction.

Clinical examination

The main components of a clinical examination of a breeding bull with a view to determining his breeding ability and the purpose of each is outlined. Visual observation of the bull is aimed at observing the shape of the sheath and its positioning in relation to the body wall and an imaginary line drawn between the hock and fetlock joints. The presence of eversion of the lamina interna should also be noted, as well as the condition of any visible lamina.

Palpation should be performed from the back and sides, with due consideration of operator safety. Careful points to note are the ability of the bull to lift the preputium and retract the lamina, the mobility of the lamina and penis relative to each other and to other surrounding structures, the thin, supple state of the lamina all the way from the opening to the fornix, and the position of the caudal reflection of the lamina (approximately 10-12cm behind the tip of the penis in the average bull). The length of the lamina interna should be estimated, either by passing a probe (eg. An AI pipette) up the cavity all around the penis or by pushing the tip of the penis back until the lamina is under tension and the opening is seen to pull inwards. In particular, the amount of healthy lamina should be estimated in cases where there are lesions, as an indicator of the viability of removal of part of the lamina. The penis should also be palpated to establish the position, mobility, sensation and presence of any lesions.

The extent and shape of the preputial cavity should be investigated in cases where preputial trauma, inflammation or a combination of the two are suspected. This can be done by digital probing and passing a probe up the cavity. In addition, filling the cavity with water under moderate pressure and ultrasound examination is useful in showing any narrowing, adhesion to or lesions on the free portion of the penis, or other irregularities in the lamina. Cool pre-boiled water gives the best image clarity.

Extrusion of the penis should be attempted to confirm the absence of impairment of the free movement of the penis. In cases where semen evaluation is indicated this can be achieved with an electro-ejaculator. If this is unsuccessful or not indicated, a phenothiazine tranquilliser can be given, which usually results in relaxation of the retaining musculature. Topical application of local anaesthetic further facilitates handling of the penis without any response from the bull. A pudendal nerve block is another viable option to facilitate penile extrusion, examination and minor procedures.

Although more crucial in the workup of penile abnormalities, a serving ability test may be useful in demonstrating normal preputial appearance and function, should the logistics and condition of the bull allow. Video footage of several mating attempts enhances the ability to clarify the exact point of breakdown in the mating process.

Preputial pathology

The sheath, particularly the lamina interna, is prone to injuries and inflammation as a consequence of insults to itself or surrounding structures such as the penis. The risk of injury is increased when the anatomical factors listed above are present but morbidity is not limited to bulls with poor conformation.

The common pathologies are:

i. **Infection**
The sheath is prone to infection as a sequel to injuries, haemorrhage or other insults. The commonest causes are penetrating injuries at the sheath opening as a result of eversion, penetrating injuries along the length of the lamina as it is stretched over the erect penis during breeding, and tearing away of the remnants of the attachment between the penis and lamina interna during breeding attempts, most commonly associated with semen collection using an artificial vagina.

Unless identified early and treated as outlined above, infection commonly results in chronic-active inflammation which can result in excessive scar tissue formation, adhesions or abscessation.

ii. **Prolapse**
Lamina interna prolapse is a common sequel to infection, injuries and urethral rupture. Unless treated effectively as outlined above, fibrous tissue formation can result in permanent damage, requiring surgery.

iii. **Stenosis**
Narrowing of the cavity can result from injuries and uncontrolled inflammation, preventing extrusion of the penis and, when severe, causing damming back or urine and further inflammation. By the time this is diagnosed surgery is generally inevitable.

iv. **Adhesions**
Inflammation stretching across layers of loose connective tissue underlying the lamina interna can result in the formation of inflexible scar tissue which limits the mobility of the penis and sheath. Once formed, the prognosis is poor.

Regardless of the aetiology, the main therapeutic objectives should be to control inflammation and the associated oedema and, if necessary, to remove any lesions which impede the mating process.

Inflammation is best controlled by a combination of antimicrobials and anti-inflammatories and topical and physical therapy. The latter consists of placement of a sling to lift the sheath, daily cleansing and vigorous hosing down and the application of topical products with disinfectant, hygroscopic and wound-healing properties. This is time-consuming and requires good facilities and a compliant patient, thus often not practical in the on-farm situation.

Surgical intervention is often necessary to return the bull to functionality.

The poor prognosis associated with adhesions as outlined above justifies an aggressive approach to treatment of even apparently minor sheath pathology in high-value seedstock.

Preputial conformation

Given the increased risk of morbidity in bulls with poor preputial conformation the evaluation of this trait is recognised as an important part of bull selection and of a bull breeding soundness evaluation (BSE). These two procedures are driven by breeders and breed societies in the case of bull selection, and veterinarians and other operators in the case of the BSE. Scoring systems are available for each of these, the most relevant for this paper being that of the Australian Cattle Vets(1).

Breed society policies vary with regard to their adoption of sheath scoring. In breeds with a recognised genetic predisposition to conformational traits associated with higher morbidity the application of scoring in both bull selection and pre-sale BSE certification is strongly recommended.

Proposals for revisions to the ACV scoring system were made in 2018(2). This system is more comprehensive than predecessors, addressing a wider range of anatomical factors as outlined in this paper. It is recommended that this tool be further refined in consultation with relevant experts and practitioners, and that it then be formally adopted, applied in the field and validated through data demonstrating close associations to morbidity and longevity.

Conclusion

A good understanding of functional anatomy, risk factors and pathophysiology of the sheath will stand the rural practitioner in good stead for clinical management of bulls with pathology of this under-rated part of the reproductive system.

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Research Update: Pathways for practitioners to get involved in research

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Summary
This presentation will provide an update on current sheep health and production projects, and explain some of the pathways for clinicians and animal health advisors to get good ideas onto the research table.

A role for practitioners in research, development and extension
Practicing veterinarians and livestock advisors commonly encounter issues impacting animal health and production that could benefit from further research or improved extension (education) to address the problem. Participating in the research, development and extension process can be rewarding for the veterinarian, and ultimately deliver good outcomes for their clients and the livestock industry more widely.

The process to develop and deliver quality research, development and extension is often complex. Here we discuss some considerations that will help veterinarians engage effectively in the planning and delivery of livestock research, development and extension projects.

1. Understand the problem and clarify the current gaps
If you think you have identified an issue that would benefit from research and development, the first step is to establish what research has already done that addresses the problem.

Searching existing literature will help in defining what is and isn't known about the problem. Online scientific databases are helpful for searching content in scientific journals. Examples are listed at end of this paper (resources). Some databases and journals are free to access online, but others require subscriptions. It is worth checking whether you qualify for access to a university library (e.g. through the alumni association) to allow access to databases and journals under the university subscription. If you are unsure where to start with a review, university library websites often provide useful information and training on searching scientific literature and accessing materials. Science librarians are often able to provide helpful advice on how to use data bases.

It is often helpful to contact researchers working in a similar or related field to the topic you are interested in investigating. They may be able to advise you if work to address the problem has already been done, or is currently underway. Researchers familiar with the issue may be able to direct you to materials that are not searchable with online databases, such as industry reports, conference papers or student research theses.

At this point, clarifying the 'researchable question' will inform the best way to proceed in progressing a proposal:

- If the problem has not been investigated already, it is worth asking, "Why not?" Is this a new problem? Has it been attempted and failed, and if so what could be done differently?
- If some work has been done, but this fails to fully address the current issue, it is important to clarify the specific gaps that need to be addressed.

- If you find that work has been done that addresses the problem, then consider if there is a need for extension to make the findings and recommendations more accessible to veterinarians, advisors or farmers.

Regional livestock research councils (e.g. Western Australian Livestock Research Council, Southern Australia Meat Research Council) may be able to provide advice on current and proposed work, potential funding sources, and contacts for potential collaborators.

2. Decide early on what role you would like to play if work does proceed

Once you have established if new research or improved extension is required, carefully consider what role you might want to play in the project. Clear expectations on what you want to achieve from being involved will inform how you engage with funding bodies and collaborators.

3. Understand the options for supporting and funding the work

There are no hard and fast rules for securing support and funding for research, development and extension projects. The most appropriate sources of funding will depend on the main stakeholders for the work (i.e. who are the beneficiaries). Important issues to consider are whether the project is targeting short-term commercial gain, or longer-term benefits at industry level.

The rural research and development corporations (e.g. Meat and Livestock Australia, Australian Wool Innovation, Grains Research and Development Corporation) fund on-farm research and development. They also fund demonstration sites – these usually have a strong extension focus. Programme managers at the research and development organisations can often help identify funding options available based on scope and target beneficiaries of the proposed project.

State or federal government funds may be available.

Private companies and private equity firms may provide funding and/or in-kind collaboration for projects if there is likely to be direct commercial benefit from the company in doing so. If there is the option to secure cash and in-kind contributions from private companies, then there are options available to further leverage the cash contribution with government funds. Examples of the are CRC-P scheme and the MLA Donor Company.

4. Understand the funding body before submitting a proposal

It is important to understand how your proposal aligns with the priorities and strategic plan of the funding body before submitting a project application.

Depending on the organisation, you may need to show how your proposal addresses issues of local, regional or national significance. Taking time to understand the research priorities for the organisation and showing how your project aligns to these will improve your chance of success.

Many organisations have key performance indicators within their strategic plan, and it is important to be able to show how the proposed project will contribute to achieving those goals.

If it appears that there is limited alignment of your ideas with any terms of reference, then try to identify a pathway to have your concept road-tested. For example, Meat and Livestock Australia use the regional livestock research councils to prioritise issues. Priorities identified by the councils are included in terms of reference for the annual call for proposals for on-farm research and development. Whilst significant lead time and effort is required to brief relevant members of these councils, it does provide a pathway for raising issues that could be included in the terms of reference for on-farm research and development. Regional councils relevant to sheep and goat research are listed with the resources at the end of this paper.

5. Understand the process used for assessing project proposals

A clear understanding how project proposals are going to be assessed will help in preparing your application and will improve chances of success. It is important to pay close attention to the application instructions. In particular, check for terms of reference (i.e. description of what types of projects they will be funding), and ensure that your application shows that your proposal is clearly aligned with these.

Some funding bodies will require you to show benefit-cost analyses or return on investment estimates in the application, or to supply sufficient information that staff at the funding body can do this. The assumptions used to underpin these analyses should be supported with some evidence in your application. If you are unfamiliar with the process, it is worth seeking some advice.

It is worth discussing expectations with the funding body in advance to ensure your proposal has a reasonable chance for success for that particular scheme. Some project funding schemes have expectations that applicants (either individuals or the lead organisation for the project) have a strong track record in delivering research, development or extension in a related field. This is particularly important for competitive grants. In some cases, it may be worth considering collaborating with an individual or organisation with strong track record to strengthen your application. Project funding schemes also have different expectations on the number and type of collaborating parties (e.g. single or multiple university, government or private collaborators) or geographical spread (e.g. local vs state vs national collaboration). Some schemes will have requirements for cash or in-kind contributions from the lead organisation and collaborators.

6. Get advice on contracts and intellectual property

Contracts for research projects can be complex, and it is worth sourcing independent advice to make sure you understand your rights and responsibilities. Clauses around intellectual property can be particularly challenging, and it is worth getting advice on this in advance if there are aspects to the project likely to be commercialised. It is also important to understand clauses on confidentiality and force majeure, and how these could impact the project and your business.

Should your idea be unique and have potential for personal commercial gain, think very carefully before you tell the world. Prior disclosure is difficult to 'undisclose', and potentially damaging if representations about such are later found to be inaccurate.

Resources for sheep and goat research, development and extension

Scientific databases relevant for animal and veterinary science

ABARES: http://www.agriculture.gov.au/abares

Livestock Library: http://www.livestocklibrary.com.au/

Proquest (subscription access): https://search.proquest.com/

Pubmed: https://www.ncbi.nlm.nih.gov/pubmed/

Google Scholar: https://scholar.google.com.au/

Science Direct: https://www.sciencedirect.com

Scopus: https://www.scopus.com

Web of Science (subscription access only): https://www.webofknowledge.com/

Livestock Research Councils

Western Australian Livestock Research Council: http://www.walrc.com.au/

Southern Australian Livestock Research Council: https://www.samrc.com.au/

Research and Development Corporations - funding

Meat and Livestock Australia: https://www.mla.com.au/research-and-development/funding-opportunities/

Australian Wool Innovation: https://www.wool.com/about-awi/where-we-invest/call-for-proposals/

AgriFutures Australia: https://www.agrifutures.com.au/researchers/

Industry collaboration schemes

Cooperative Research Centres Projects Scheme:
https://www.business.gov.au/assistance/cooperative-research-centres-programme/cooperative-research-centres-projects-crc-ps

Meat and Livestock Australia Donor Company: https://www.mla.com.au/about-mla/what-we-do/mla-donor-company/

Australian Research Council Linkage Programmes: https://www.arc.gov.au/grants/linkage-program

Rural R&D for Profit: http://www.agriculture.gov.au/ag-farm-food/innovation/rural-research-development-for-profit

Summary of recent and current sheep production and health R&D (Murdoch University)

Project	Outline	Status	Collaborators	Contact
Merino Lifetime Productivity (MLP)	Increase understanding of genetics and economic interactions over a range of Merino types delivering high quality wool, lambs and meat through life	In progress at Pingelly plus 4 interstate sites. Preliminary data available.	Australian Wool Innovation Australian Merino Sire Evaluation Association Merino ram breeders Murdoch University University of Western Australia	Bronwyn Clarke Bronwyn.Clarke@murdoch.edu.au 0418 957 293 Andrew Thompson Andrew.thompson@murdoch.edu.au 0437 316 117
Genetic Evaluation – Productivity, Efficiency and Profit (GEPEP)	Quantifying differences in appetite, efficiency and whole-body energy between sire groups. The project will improve the estimation of profitability per hectare of the genotypes represented in MLP.	In progress at Katanning Research Station. Preliminary data available.	Australian Wool Innovation Murdoch University	Sarah Blumer s.blumer@murdoch.edu.au 0433 010 764 John Young john@farmingsystems.com.au 0428 336 206
Management guidelines and feeding standard for maternal ewes	Developing recommendations for managing maternal ewes to optimise productivity per hectare. Outcomes to be extended through Bred Well Fed Well, Lifetime Ewe Management and other programs.	Experimentation complete and economic modelling in progress.	Meat & Livestock Australia Murdoch University South Australia Research and Development Institute Agriculture Victoria	Andrew Thompson Andrew.thompson@murdoch.edu.au 0437 316 117 Sarah Blumer s.blumer@murdoch.edu.au 0433 010 764
Reducing mortality and reproductive wastage in ewe lambs	Investigating factors that impact reproductive wastage and mortalities in ewe lambs and their offspring, and identify opportunities for improvement.	Farm survey in progress. Recruiting farms for research sites in 2020.	Meat & Livestock Australia WA Sheep Business Innovation Murdoch University	Elise Bowen Elise.Bowen@murdoch.edu.au Caroline Jacobson c.jacobson@murdoch.edu.au 0418 953 173
Improving the reproductive performance of ewe lambs – economic analysis and decision support tools	Developing decision support tools for producers to make informed decisions about joining ewe lambs	Recently completed	Meat & Livestock Australia Murdoch University	Andrew Thompson Andrew.thompson@murdoch.edu.au 0437 316 117 John Young john@farmingsystems.com.au 0428 336 206
Grazing cereals – lamb survival	Investigating impact of mineral supplements for pregnant ewes grazing cereal crops on lamb survival	In progress	Murdoch University	Serina Hancock s.hancock@murdoch.edu.au 0403 570 823

Project	Outline	Status	Collaborators	Contact
Reducing foetal and lamb losses in maiden ewes	Determine if infectious diseases are contributing to reproductive wastage and identify opportunities to increase marking rates for maiden ewes	Fieldwork in progress. Recruiting farms for 2020.	Meat & Livestock Australia Murdoch University University of Adelaide University of Melbourne Livestock Logic	Tom Clune T.Clune@murdoch.edu.au Amy Lockwood a.lockwood@murdoch.edu.au 0429 976 483 Caroline Jacobson c.jacobson@murdoch.edu.au 0418 953 173
Mums with multiples	Developing recommendations for managing triplet ewes and their lambs	Recruiting farms with high fecund flocks for 2019 & 2020	Meat & Livestock Australia Murdoch University South Australia Research & Development Institute NSW Department of Primary Industries Agriculture Victoria JT Agri Source	Andrew Thompson Andrew.thompson@murdoch.edu.au 0437 316 167 Travis Allington travis.allington@murdoch.edu.au 0497 413 125
Changes in lamb and ewe behaviour and physiology related to the shearing of pregnant Merino ewes	Determining whether mid-pregnancy shearing aids lamb survival behaviours at birth in Merino sheep	In progress	Murdoch University DPIRD	Khama Kelman k.kelman@murdoch.edu.au Dave Miller d.miller@murdoch.edu.au
Lambing density	Development of guidelines for the mob size and stocking rate of ewes at lambing to optimise lamb survival	Recently completed	Australian Wool Innovation Meat & Livestock Australia Murdoch University Agriculture Victoria NSW Department of Primary Industries Landmark Elders	Amy Lockwood a.lockwood@murdoch.edu.au 0429 976 483 Serina Hancock s.hancock@murdoch.edu.au 0403 570 823

Project	Outline	Status	Collaborators	Contact
Unlocking the keys to ewe survival	Quantifying mortality for non-merino ewes over lambing, and determining the primary causes of ewe wastage	In progress. Recruiting farms for 2019 and 2020	Meat & Livestock Australia Livestock Logic Murdoch University Macquarie Franklin University of Melbourne	Caroline Jacobson c.jacobson@murdoch.edu.au 0418 953 173
Grazing cereals – ewe survival	Investigating impact of mineral nutrition for survival of pregnant ewes grazing cereal crops	Completed	Meat & Livestock Australia Murdoch University Charles Sturt University NSW Department of Primary Industries	Serina Hancock s.hancock@murdoch.edu.au 0403 570 823
Weaner survival genetics	Determining impact of genetic fat on survival of Merino weaners and genetic correlations between survival and production traits	Completed	Australian Wool Innovation Murdoch University AGBU	Andrew Thompson Andrew.thompson@murdoch.edu.au 0437 316 167
Fat, efficiency and robustness	Determining impact of genetic fat on efficiency and robustness of Merinos.	In progress	Murdoch University	Sarah Blumer s.blumer@murdoch.edu.au 0433 010 764
Sensor technologies – Grazing Bytes	Use of sensor technologies for animal monitoring and pasture management	In progress. Preliminary data available	Australian Wool Innovation Murdoch University Muresk (DTWD) NextGen Agri (NZ)	Andrew Thompson Andrew.thompson@murdoch.edu.au 0437 316 117 Mark Ferguson mark@nextgenagri.com +64 21 496 656
Bred well Fed well workshop – sheep and beef	Bred well Fed well is a practical, one-day workshops which highlights the key production benefits of genetics, plus feed management for improved reproduction	Bred well Fed well has delivered a total of 213 workshops (183 sheep and 30 Beef) to a total of 5088 participants.	Meat & Livestock Australia Murdoch University	Serina Hancock s.hancock@murdoch.edu.au 0403 570 823

Project	Outline	Status	Collaborators	Contact
Low rainfall legume pasture systems	Project aims to characterize via participatory research the performance of sheep grazing Serradella and Biserulla legume pastures to enable their use for sheep production to be optimised in mixed farming systems	Recently commenced & recruiting farms for 2019/20.	Meat & Livestock Australia Australian Wool Innovation Murdoch University DPIRD	Andrew Thompson Andrew.thompson@murdoch.edu.au 0437 316 117 Colin Byrne Colin.Byrne@murdoch.edu.au 0433 678 172
Waterborne diseases in sheep	Determine if farm dam water represents an important source of transmission for infections that impact sheep health and productivity	In progress	WA Sheep Business Innovation Murdoch University	Caroline Jacobson c.jacobson@murdoch.edu.au 0418 953 173
Pain-mitigating effects of meloxicam in combination with Tri-Solfen® in mulesed Merino lambs	The objective of this experiment was to determine the effectiveness of Tri-Solfen® and meloxicam (Metacam® 20, Boerhinger Ingleheim) at reducing pain-related behavioural responses to mulesing in Merino lambs.	Completed	Boerhinger Ingleheim Murdoch University	Serina Hancock s.hancock@murdoch.edu.au 0403 570 823 Andrew Thompson Andrew.thompson@murdoch.edu.au 0437 316 167
Automated data capture to monitor sheep wellbeing	On-farm remote sensing of sheep behaviours as indicators of health and welfare	In progress	Murdoch University Sheep CRC	David Miller d.miller@murdoch.edu.au Fiona Anderson f.anderson@murdoch.edu.au
Welfare Indicator Dashboard: a pilot study for the live export supply chain	Implementing and testing robust and efficient monitoring tools for the live export industry	In progress	Murdoch University Meat & Livestock Australia Livecorp	Teresa Collins t.collins@murdoch.edu.au Anne Barnes a.barnes@murdoch.edu.au Trish Fleming t.fleming@murdoch.edu.au David Miller d.miller@murdoch.edu.au
A stockperson survey on attitudes to animals in the live export chain	Project investigating beliefs and behaviours around managing animals in the live export process	In progress	Murdoch University	Teresa Collins t.collins@murdoch.edu.au

Summary of recent and current goat production and health R&D (Murdoch University)

Project	Outline	Status	Collaborators	Contact
Goatmeat quality – pathway to the future	Developing producer and processor standards to improve goatmeat eating quality	In progress Recruiting producers & processors	Murdoch University University of New England Meat & Livestock Australia	Liselotte Pannier l.pannier@murdoch.edu.au David Miller d.miller@murdoch.edu.au David Pethick d.pethick@murdoch.edu.au
Parasites in rangeland goats	Investigated animal production and public health impacts of parasites in captured rangeland goats	Completed	Meat & Livestock Australia Murdoch University	Caroline Jacobson c.jacobson@murdoch.edu.au 0418 953 173

Protozoan infections in sheep and goats

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Summary
This presentation will provide an update on the impact of protozoan infections on health and productivity of sheep and goats. Current options for diagnosis and treatment, plus the role of vets in addressing potential public health impacts will be addressed.

Coccidiosis
Coccidiosis is caused by protozoan (single-celled) parasites of the *Eimeria* genus. At least 11 species have been reported in Australian sheep and 10 species have been reported in Australian goats. Improved understanding of *Eimeria* epidemiology has shown *Eimeria* species are host specific and cross infection between sheep and goats does not occur.[1] *Eimeria* species vary in pathogenicity. *Eimeria ovinoidalis* and *E. crandallis* are associated with more severe disease in sheep.[1] *Eimeria ninakohlyakimovae* and *E. arloingi* are considered most pathogenic in goats.[1] *Eimeria* species in sheep and goats are not considered zoonotic.

Sheep and goats are infected by the faecal-oral route. Infective oocysts may remain viable for more than a year under favourable conditions resulting in environmental contamination.[2] Almost all sheep and goats encounter coccidia as lambs/kids, typically via contaminated pasture, feed, bedding or udder of ewes/does.

Clinical disease
Asymptomatic *Eimeria* infections are common in sheep and goats. Gut damage and inflammation may cause scouring within a few weeks of birth. Lambs typically develop a strong immunity whether or not clinical signs occur, although chronic infections with shedding can occur in sheep of any age that act as carriers.

Severe coccidiosis outbreaks with scouring, inappetence, weight loss and deaths of weaker animals can occur. Disease outbreaks are typically sporadic. Young sheep and goats (less than 6 months old) are most susceptible. Other risk factors include high stocking densities and other stressors including prolonged cold wet conditions, poor nutrition, stressful management procedures (such as weaning) or concurrent disease.[1,2] Disease outbreaks may occur in extensively managed unweaned lambs where ewes are in poor condition and lambs are forced to graze earlier than usual. Clinical coccidiosis can occur in older sheep and goats when there is overwhelming infection pressure (usually associated with overcrowding) and concurrent stressors.

Diagnosis
Coccidiosis is usually diagnosed using a combination of history of predisposing factors, presence of clinical signs, high faecal oocyst counts, and characteristic gross or histopathological changes. The site and appearance of gross or histopathological intestinal lesions varies between *Eimeria* species and different life cycle stages.

Oocyst counts should be interpreted with caution. Oocyst shedding may be intermittent, and oocyst shedding in the absence of clinical disease is common. Diarrhoea may precede oocyst shedding in acute disease. Oocyst speciation is recommended to differentiate non-pathogenic species, and aids in interpretation of oocyst counts as shedding varies between species. However, distinguishing *Eimeria* species is challenging, even in research settings.

Traditional methods based on oocyst morphology, pre-patent period, site of infection, or minimum sporulation time are labour intensive, time consuming and lack specificity due to overlapping morphological characteristics between species. Molecular diagnostic methods can be used for detection, quantitation and species identification in sheep[3] and goats,[4] but are not widely available in diagnostic laboratories.

Investigations for suspected coccidiosis should consider concurrent infections because this may increase disease severity. *Eimeria* infection area also associated with increased susceptibility to other infections.[2,5]

Treatment
Clinical coccidiosis is usually considered a self-limiting disease.[1] Treatment is often not warranted. The decision to treat is complicated by limited treatments registered for use in sheep and goats.

Studies have demonstrated efficacy for treatment of coccidiosis for triazones (toltrazuril, diclazuril) and amprolium,[1,5] but these are not registered for use in sheep or goats in Australia.

Sulphonamide antibiotics are sometimes used for managing outbreaks, but do not have a label claim for treatment of coccidiosis in sheep or goats in Australia. Treatment should be given over 3-5 days, and this limits practicality for treatment of outbreaks.[1] Sulphonamide antibiotics only have activity against the last stage of the lifecycle[1], and response to treatment may be related to controlling secondary infections rather than direct effect on *Eimeria*.

Use of in-feed coccidiostats (monensin, lasolacid, decoquinate) may be warranted where heavy environmental contamination with infective oocysts is likely (such as feedlots) and on-going prevention is justified.[1] Of these, monensin and lasolacid sodium are currently registered for use in sheep in Australia with a label claim for prevention of coccidiosis.

Cryptosporidiosis
Cryptosporidiosis is caused by protozoan (single-celled) parasites of the *Cryptosporidium* genus. Seven *Cryptosporidium* species have been reported in sheep worldwide, of which *C. xiaoi, C. parvum* and *C. ubiquitum* are most commonly reported in Australian sheep and goats.[6-12] Zoonotic *Cryptosporidium parvum* and *C. ubiquitum* subtypes with public health significance have been isolated from Australian sheep and goats. *Cryptosporidium xiaoi* infections have been recently identified in immunocompromised humans, and this species is now considered potentially zoonotic.

Infections are transmitted by faecal-oral route through ingestion of oocysts, usually via feed or water contaminated with faeces, or licking contaminated surfaces. Managing environmental contamination is challenging. Oocysts survive up to 6 months in favourable conditions, and the parasite can reproduce without a host in aquatic environments which may increase environmental contamination. The infectious dose for lambs is as low as one oocyst.

Cryptosporidium shedding in faeces is common and widespread in Australian sheep with mean shedding detection ranging 6-68% sheep for studies conducted in asymptomatic flocks sampled on a single occasion.[7-11] As with *Eimeria*, *Cryptosporidium* shedding is more common in younger sheep.[7,10,11] *Cryptosporidium* epidemiology in Australian goats is not well described, but a recent study in captured rangeland goats showed shedding detection ranged 3-14%, with highest prevalence in the period immediately after capture and transport.[12] A peri parturient rise in faecal oocyst shedding acts as source of transmission to lambs.

Clinical disease

Cryptosporidiosis is widely recognised as an important cause of diarrhoea in lambs and kids. Cryptosporidiosis is an important differential diagnosis for scouring in lambs and kids that are too young to have acquired significant nematode burdens.

Cryptosporidiosis has been long thought of as a self-limiting disease that is only of importance for young lambs and kids, and specifically for those raised in intensive production systems. Evidence is emerging to suggest disease impacts are more complex than previously thought for sheep and goats in Australian extensive production systems.

Infections in older sheep and goats have generally been considered asymptomatic based on detection of parasites in older animals (post weaning) with no evidence of scouring. However, most studies have not assessed impact on measures of sheep productivity such as weight gain, wool growth, carcass or reproductive performance. Recent Australian studies have identified associations between *Cryptosporidium* shedding and reduced live weight, reduced carcass weight and reduced dressing percentage in lambs post-weaning,[6,13,14] and reduced live weight and diarrhoea in rangeland goats post weaning.[15] This suggests potential for impacts on health and productivity apart from scouring.

Most studies have not investigated impacts of different species and genotypes on sheep and goat productivity. A recent study demonstrated that *C. xiaoi* shedding was associated with reduced growth rate (but not diarrhoea) in 9-12 month old Australian goats.[15] *Cryptosporidium xiaoi* is the most common species identified in Australian sheep and goats, therefore this finding suggests impacts on production are likely to be underestimated for studies that rely only on scouring as an indicator of disease. As such, cryptosporidiosis should be considered as a differential diagnosis for scouring and sub optimal growth or carcass production in sheep and goats, even after weaning and in animals without evidence of scouring.

Recent studies have also shown that shedding can be detected in some animals on repeated occasions.[7,12,13] It's not clear whether this represents persistent infections or increased susceptibility to reinfection in some animals, but repeated detection has been associated with greater impacts on carcass weight.[13] As such, more work is needed to determine whether cryptosporidiosis is indeed a self-limiting disease with respect to impacts other than scouring.

Detection of a greater number of pathogens (nematodes, protozoans, bacteria) has also been associated with greater risk of scouring.[15] As such, *Cryptosporidium* should be considered as a component of mixed infections in sheep or goats with scouring outbreaks where other infections are identified, but animals do not respond well to treatment.

Diagnosis

Diagnosis of cryptosporidiosis in field investigations for sheep and goats is challenging. Oocysts can be detected in faeces using microscopy with specific staining techniques, indirect immunofluorescence and enzyme immunoassays. These techniques lack sensitivity and generally can't differentiate species. Molecular techniques are used in epidemiological studies to quantify shedding and determine species and subtypes,[10] but these are not widely available in diagnostic labs.

Post-mortem examinations can be used to demonstrate characteristic histopathological changes and confirm diagnosis in outbreaks with high morbidity and mortality. Catarrhal enteritis, distension of caecum and colon, congestion and hemorrhagic inflammation in the last third of the ileum, and/or hypertrophy of the mesenteric lymph nodes may be observed on gross post-mortem examination, but are not pathognomonic for cryptosporidiosis.

Treatment

Options for treatment or prevention of cryptosporidiosis outbreaks are limited. Treatments including halofuginone lactate, paromomycin and β-cyclodextrin have been evaluated for management of naturally-acquired coccidiosis in young lambs in European studies, but these are not currently registered for use in sheep or goats in Australia.

Giardia

Giardia duodenalis comprises a species complex consisting of eight genetic assemblages. Assemblages A and E have been widely reported in Australian sheep,[8,9,11,16] and assemblage B has been sporadically reported in sheep overseas. *Giardia duodenalis* assemblages A and B are widely considered zoonotic and of public health significance. There is emerging evidence that assemblage E may be also be zoonotic.

Giardia shedding in faeces is common and widespread in Australian sheep with shedding detection ranging 11-44% for studies conducted in asymptomatic sheep flocks sampled on a single occasion.[6-9,16] There is less data on shedding by Australian goats, but a recent study identified shedding in 12% rangeland goats after capture.[12]

Clinical disease

Giardia infections have been associated with diarrhoea in lambs and kids. In Australia, naturally-acquired infections have been associated with looser faecal consistency in lambs up to 10 months old.[6,14] As with other protozoan parasites, giardiasis may occur concurrently with other infections. Asymptomatic infections are common, and the role of *Giardia* as a primary cause of diarrhoea in livestock remains poorly understood.[17]

Giardia is generally considered as a self-limiting infection in sheep. However, experimental and natural infections have been associated with reduced growth rate and reduced carcase weight weeks or months after detection of infection.[13,14,18]

Diagnosis

Diagnosis of giardiasis is not straightforward. Cysts can be detected in faeces using microscopy techniques or immunofluorescence assay. However, sporadic cyst-shedding, presence of low numbers of cysts and steatorrhea can interfere with *Giardia* detection using microscopy. The sensitivity and specificity of immunoassays is highly variable. As with *Cryptosporidium*, molecular techniques have improved sensitivity and capacity to distinguish genotypes,[16] but are not widely available in animal health diagnostic laboratories. Serological tests are not considered to be a reliable indicator of disease.

Treatment

There is limited evidence for efficacy of treatments for giardiasis in sheep and goats. A European study found that a three-day course of fenbendazole reduced cyst shedding in 12 week old lambs, but had no impact on faecal consistency or weight gain.[19] Fenbendazole is not registered for treatment of giardiasis in sheep in Australia.

Conclusion

Protozoan infections are common in Australian sheep and goats. Improved diagnostic methods are improving our understanding of the epidemiology and impacts of infections on sheep and goats. Protozoan infections should be considered as a potential contributor to scouring as a component of mixed infections with nematodes and pathogenic bacteria. Recent work in Australian sheep and goats has challenged previous perceptions about impacts of protozoan infections on health and production in sheep and goats older than 3 months of age, with impacts on growth and carcass weight noted even in animals without evidence of scouring post-weaning. Some protozoan infections in sheep and goats have public health significance.

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Reproductive wastage in young ewes

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Summary

The reproductive performance of maiden ewes in Australia is poorly understood. Here we provide preliminary data from a project exploring the timing and causes of reproductive wastage in maiden ewes. This data suggests foetal losses in the period between pregnancy scanning and birth contribute overall reproductive wastage. The magnitude *of in utero* wastage varies between farms. Opportunities to contribute to ongoing R&D projects exploring opportunities to improve reproductive performance will be highlighted.

Background

It is widely accepted that the reproductive performance of maiden ewes is lower when compared to mature, multiparous ewes. However, the extent of this gap in reproductive performance between maiden and adult ewes is not well quantified. Furthermore, the causes of increased wastage in younger ewes are not fully understood.

Reproductive wastage can be assessed by comparing the number of foetuses present at scanning with the number of lambs present at marking.[1,2] However, wastage between scanning and marking does not distinguish losses occurring during pregnancy from those in the perinatal period. Studies largely conducted with adult ewes show that most lamb deaths occur in the perinatal period.[3-5]

Accurate assessment of the timing of reproductive wastage requires monitoring ewes during lambing to record the number of lambs born. This is labour intensive and is generally not feasible for extensively managed commercial sheep enterprises. Those enterprises that do record the number of lambs born often report discrepancies between the number of lambs scanned and number of lambs born. Anecdotally, discrepancies between the number of lambs at scanning and birth are more commonly observed and are of larger magnitude for maiden ewes, and especially for maiden ewe lambs joined at 8-10 months of age. Discrepancies between the number of lambs identified at scanning and lambing are reported in flocks with no visual evidence of an abortion storm.

It is not clear if these discrepancies represent errors at pregnancy scanning, wastage during pregnancy (abortion), errors recording the number of lambs born or a combination of these factors. Overseas studies suggest that foetal losses during mid- to late-pregnancy may be an important source of wastage in young ewes.[6] If this is the case for Australian sheep, *in utero* foetal losses (abortion) could explain discrepancies between the number of lambs scanned and number of lambs born as reported by farmers.

In 2018, a project to determine the magnitude and causes of foetal losses in maiden ewes in Australian sheep enterprises was initiated by Meat and Livestock Australia. Here we present preliminary data from the first year of a fieldwork study. The aim of this study is to quantify the extent and timing of reproductive wastage in maiden ewes joined either as lambs (7-10 months old) or hoggets (18-20 months old).

Methods

This project will involve 30 on-farm research sites between 2018 and 2020. Research was conducted on four farms in south west Western Australia in 2018. Two of the farms joined maiden Merino hoggets and two farms joined maternal composites as ewe lambs (Table 2).

Approximately 200 ewes at each site were monitored from joining until lamb marking (Table 1). The joining period ranged from 35 to 46 days. Weight, condition score and blood samples were collected from ewes at five time points. Condition score was measured using the scale of 1-5.[7]

Pregnancy scanning was conducted twice using transabdominal ultrasonography to determine foetal number and viability. The first pregnancy scan was conducted at 76-88 days from the start of joining (41-45 days after removal of rams). The second scan was conducted at 116-119 days from the start of joining. Foetal viability was determined by the presence of cotyledons and a heartbeat and/or foetal movement. Foetal number at day 116 pregnancy is subject to error, so foetal number recorded for scan 2 was determined using combination of number of foetuses observed, viability and scan 1 record. Scanning was performed by the same operators at all farms for both scans.

Ewes and lambs were monitored daily over the lambing period. Lambs were tagged, recorded as live or dead and their dam was identified. Lamb survival per ewe was assessed to marking. Ewe udders were assessed to determine lactation status (wet or dry) at marking. Ewes that failed to lamb (no lambs born) were identified using lambing records (no lamb/s allocated to ewe) and ewe lactation status at marking (dry). Lambs that were dead at lambing rounds (died between birth and tagging) were categorised as 'born', therefore wastage included in birth-marking period (i.e. born but did not survive to marking).

Table 1: Timing of measurements used to assess extent and timing of wastage

	Pre-joining	Scan 1 (d76)	Scan 2 (d116)	Pre-lambing (d140)	Lambing (birth)	Marking
Ewe condition score	✓	✓	✓	✓		✓
Ewe weight	✓	✓	✓	✓		
Blood sample	✓	✓	✓	✓		✓
Foetus count		✓	*			
Foetus viability		✓	✓			
Lamb identification					✓	✓
Lamb status (dead/alive)					✓	✓
Ewe lactation (wet/dry)						✓

* determined using combination of number of foetuses observed, viability and scan 1 record

Farmers were asked to collect aborted foetuses or foetal membranes, plus lambs that died within two days of birth. In most cases, dead lambs collected by farmers were frozen for storage prior to conducting a post mortem. This impacted accurate interpretation of necropsy findings, however attempts were made to categorise the cause of death using methods previously described.[8] Stillborn lambs (no evidence of breathing or walking) that had evidence of cerebral or CNS oedema or haemorrhage were classified as 'dystocia'. Predation and decomposition of some carcases made post mortem examination observations challenging to interpret, therefore these cases were classified as 'undiagnosed'.

Proportions (%) for magnitude of reproductive wastage (foetal or lamb loss from start to end of period) between farms, timing of reproductive wastage within farms, and cause of death (post mortem exam death category) within farms were compared using two-tailed z-test. Statistical significance was accepted where $p<0.05$.

Results

Ewe characteristics

Ewe joining weights and condition score profiles across all four farms are shown in Table 2. Ewes were managed to body weight and condition score targets at all four farms.

Table 2: Characteristics for ewe flocks with mean weight and condition score (scale 1-5)

	Farm 1	Farm 2	Farm 3	Farm 4
Ewe type	Merino	Merino	Maternal	Maternal
Age at joining (months)	18-20	18-20	7-9	7-9
Start of lambing	July	July	July	August
Ewes joined (*n*)	212	188	198	199
Live weight - joining (kg)	50.0	48.0	51.9	44.9
Condition score - joining	3.3	3.2	3.4	3.3
Condition score – scan 1 (d76)	3.0	2.8	3.4	3.2
Condition score –scan 2 (d116)	2.8	2.8	3.1	2.9
Condition score - pre-lambing (d140)	2.7	2.7	3.1	3.1
Condition score - marking	2.8	2.8	3.1	3.1

Magnitude and timing of wastage

Higher wastage occurred from birth to marking compared with pregnancy (scan 1 – birth) for three of the four farms (Table 3).

Greater wastage during pregnancy (foetal loss between scanning and birth) was observed for maternal ewe lambs compared to Merino hoggets (Table 3; $p<0.001$). For Farm 3, 36/74 total loss between scanning-marking occurred during pregnancy (between scan 1 and birth). For Farm 4, 20/49 foetal loss occurred during pregnancy. Notably, this occurred without visual evidence of an abortion storm under routine monitoring of ewes by farm staff, with only one aborted foetus and one ewe with retained foetal membranes noted at Farm 3.

In utero foetal loss between scan 1 (day 76) and scan 2 (day 116) was evident in both maternal ewe lamb flocks (Table 3). Specifically, 12/148 (Farm 3) and 2/129 (Farms 4) ewes had evidence of foetal loss between the two scans. Foetal loss between the two scans was not evident at either farm with Merino hoggets (Farm 1 and Farm 2). Foetal loss between scan 1 and scan 2 was higher at Farm 3 than other farms (Table 3).

Table 3: Magnitude and timing of reproductive wastage for four flocks of maiden ewes

	Farm 1	Farm 2	Farm 3	Farm 4
Ewes empty (dry) at scanning (%)	13	5	25	37
Scanning rate (day 76) (%)[1]	117	116	98	106
Marking rate (%)[2]	94	81	61	81
Magnitude of wastage				
Overall wastage scanning-marking (% foetuses)[3]	19^{A}	30^{BC}	38^{B}	23^{AC}
Wastage day 76 – day 116 (% foetuses)[3]	0^{A}	0^{A}	7^{B}	1^{A}
Wastage day 116 – birth (% foetuses)[3]	0^{A}	4^{B}	12^{C}	8^{BC}
Wastage birth – marking (% lambs)[3]	19^{AB}	27^{B}	24^{B}	15^{A}
Timing of wastage				
Pregnancy: day 76 – birth (% wastage)	0	14	49	41
Birth – marking (% wastage)	100	86	51	59
P-value[4]	<0.001	<0.001	0.630	<0.005

[1] Number foetuses scanned / number of ewes joined
[2] Number of lambs marked / number of ewes joined
[3] % foetuses (or lambs) lost from start to end of respective period. Lambs dead at birth (full term) are included in birth – marking wastage
[abc] Wastage values (% foetuses) in rows with different superscripts are significantly different (two-tailed $p<0.05$)
[4] two-tailed z-test comparing proportion reproductive wastage in pregnancy (scan 1 – birth) vs birth to marking

Perinatal wastage

A total of 135 post-mortem examinations were performed, representing 135/237 (57%) foetal or lamb mortalities between scan 2 and marking. Cause of death category was established for 70% cases (Table 4).

The main cause of death varied between farms but overall most lambs died from stillbirth, dystocia and starvation-mismothering-exposure complex (Table 4).

Table 4: Cause of death category for foetus/lambs submitted for gross post-mortem examination

	Farm 1	Farm 2	Farm 3	Farm 4	Overall
Post-mortem examinations (n)	33	39	48	15	**135**
Abortion (%)	0^{a}	0^{a}	4^{a}	0^{a}	**1**
Stillbirth (%)	31^{b}	10^{b}	23^{b}	7^{ab}	**19**
Dystocia (%)	24^{b}	10^{b}	25^{b}	46^{c}	**23**
Starvation-mismothering-exposure (%)	21^{b}	38^{c}	21^{b}	27^{bc}	**27**
Primary predation (%)	0^{a}	0^{a}	0^{a}	0^{a}	**0**
Primary exposure (%)	0^{a}	0^{a}	0^{a}	0^{a}	**0**
Misadventure (%)	0^{a}	0^{a}	0^{a}	0^{a}	**0**
Undiagnosed (%)	24^{b}	42^{c}	27^{b}	20^{bc}	**30**

[abc] Cause of death proportions within farms (columns) with different superscripts are significantly different (two-tailed $p<0.05$)

Discussion

The key finding from this pilot study was that foetal losses between scanning and birth may be sporadically evident in maiden ewes, even in the absence of signs of an abortion storm that would normally trigger veterinary investigation. Foetal losses between scanning and birth may be an important contributor to overall reproductive wastage in ewe lambs.

Foetal wastage during pregnancy was higher for ewe lambs with 41-49% wastage occurring between scan 1 (approximately day 76) and birth. Wastage during pregnancy is challenging to detect in typical Australian sheep production systems. Signs of abortion were not evident to producers, despite monitoring that would be considered typical or greater than standard management for commercial production. Aborted material (foetus, membranes) were recovered for only two ewes at Farm 3. In both instances, aborted tissues were recovered by project staff as a consequence of the additional observation of ewes as part of the project.

Differences between the number of lambs scanned at the second scan and the total number of lambs identified at birth could represent either abortion in late gestation, or birth of stillborn or weak lambs that died soon after birth and were not identified during lambing rounds. Less than 9% of foetuses were lost between scan 2 and birth at Farms 1, 2 and 4. Whilst errors in assessing foetal number at scanning could contribute to apparent wastage, operator error is unlikely to explain all losses in these cases. The number of foetuses at scan 2 was within 2% of the first scan, and viability as well as number was assessed as part of the experimental protocol. It is likely that a proportion of apparent wastage between scanning and birth was largely failure to find lambs that were born and died soon after birth. This highlights the challenges with identifying timing of reproductive wastage on extensively managed sheep properties, even when lambing rounds are regularly performed by experienced managers. It is possible that poorer maternal instincts of maiden ewes may contribute to difficulties in matching weak or dead lambs to their dams or to recover dead lambs, as inexperienced ewes may be less likely to show interest in the lamb.[9]

It is likely that true foetal loss (abortion), rather than simply failure to find dead lambs during the lambing period, represented a significant component of reported wastage between scan 2 and birth for Farm 3 based on a number of observations. Firstly, foetal loss was also observed between scan 1 and scan 2 at Farm 3, supporting the conclusion that *in utero* losses were occurring in that flock. Secondly, a ewe from Farm 3 that had a viable foetus at scan 2 (day 116) was observed at pre-lambing (day 140) with retained foetal membranes, and subsequently identified as dry at marking. This suggests foetal loss (abortion) had occurred during late pregnancy.

Lamb losses between birth and marking represented the largest source of reproductive wastage. This is consistent with previous observations for mature ewes.[3-5] The causes of perinatal deaths based on gross post-mortem examination were consistent with other Australian studies that showed dystocia and the starvation-mismothering-exposure complex were the most common causes of perinatal death.[3-5] Stillbirths were an important source of wastage for Farm 3, and work is ongoing to identify possible infectious aetiological agents.

Variable wastage between scanning and marking was observed, reinforcing the notion that reproductive performance of maiden ewes is variable. This study is being expanded to total of 30 sites across a wider geographic area (WA, SA and Vic) in 2019-2020. This will be supported with a survey of reported wastage scanning-marking based on farmer records. Data from expanded fieldwork and survey will establish variability for reproductive wastage across a range of production systems and environments.

Association between wastage and infectious disease
Aborted tissues and samples collected from stillborn lambs are being screened for infectious diseases associated with abortion including *Brucella* spp., *Campylobacter* spp., *Salmonella* spp., *Chlamydophila*, *Leptospira* spp., pan-pestivirus, *Toxoplasma gondii* and *Coxiella burnetii*.

Serology (*Toxoplasma gondii, Neospora caninum, Coxiella burnetti, Campylobacter fetus* fetus, *Campylobacter jejuni*) is being conducted for ewes with evidence of abortion and ewes that failed to rear a lamb. These will be case-matched to control ewes that successfully reared lambs to marking. Analyses will be performed to determine association between disease exposure (seroconversion) and the pregnancy outcome. Data will be reported on completion of the project.

Limitations and weaknesses for this project
Most farms involved in the fieldwork include stud flocks as part of their sheep enterprise. This was due to requirement for conducting lambing rounds that is typically only performed by studs to tag the lambs, record their dam and measure lamb birthweight. All four farms involved in this study during 2018 ran studs, although the mob used for Farm 3 was part of the commercial ewe flock. It is not clear whether risk factors for reproductive wastage are influenced by differences in management practices between farms that include studs compared with those that run commercial flocks only.

Take-home messages

- *In utero* losses are likely to be an important component of reproductive wastage in some mobs of ewe lambs in Australian production systems. The causes of this are not well understood, and can occur without obvious signs of an abortion storm
- Practitioners should encourage clients to submit stillborn lambs to regional animal health laboratories for testing to identify infectious disease that may be contributing to wastage and to support exotic disease exclusion programmes
- Results from this ongoing project will be reported in 2020-2021, including the impact of infectious disease and other risk factors on reproductive wastage in young ewes.
- Producers that are joining at least 200 maiden ewes, can perform lambing rounds and are interested in identifying opportunities to reduce wastage can contact the project team for more information on participating in the project in 2020.
- Veterinarians and advisors should encourage clients to participate in the national maiden reproductive wastage survey. This will provide data to help benchmark performance of ewe lambs and maiden hoggets across a range of production environments.

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Wildlife Conservation Programs in Pastoral Landscapes in Northern Australia

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Introduction

Pastoralism is a dominant land-use in Northern Australia, making an important contribution to local employment and regional economies, and providing beef to markets. At the same time, Northern Australia is of international significance for biodiversity conservation, supporting extensive areas of relatively intact savannas and a mosaic of other vegetation types. These ecosystems in turn support a rich fauna including many endemic and threatened species[1,2].

Conservation in Northern Australia

A moderate proportion of northern Australia is formally reserved for conservation (10-20%, varying between regions), primary in national parks (e.g., Kakadu National Park, 2 Million ha) and, more recently, in Indigenous Protected Areas. A smaller, but still significant, area of land is managed by private nature conservation organisations. Of these, Australian Wildlife Conservancy (AWC) is a leading proponent. AWC owns or is contracted to manage nearly 5 Million ha of land in northern Australia, in addition to properties in central and southern Australia. AWC's property portfolio covers a range of tenures, including substantial areas of pastoral lease[3].

Formal reservation alone is insufficient to protect the natural systems and biodiversity of northern Australia. Large declines have been reported in small-medium sized mammals and ground-active birds in relatively intact landscapes in Northern Australia, including populations inhabiting Kakadu National Park and other protected areas[4,5]. Research has demonstrated that predation by feral cats is a major factor in causing declines in these species, exacerbated by loss of ground-cover as a result of frequent, extensive wildfire and grazing[6,7,8]. Active conservation is required to protect the biodiversity of Northern Australia, regardless of tenure[9].

Wildlife conservation in pastoral landscapes in Northern Australia

AWC has conducted a suite of research programs investigating the impacts of pastoralism of biodiversity[6,10,11]. Based on this evidence, AWC is working both on its own properties and with a number of cattle producers to achieve biodiversity conservation on pastoral leases. Primarily, on these properties, AWC manages fire, excludes stock and controls feral herbivores on high-value conservation land to promote the persistence of native wildlife, while cattle production is focused on the better quality pastoral lands. Landscape-scale management of fire across these management zones has benefits for biodiversity conservation and pastoral production[12].

Given that many pastoral properties include a balance of land suited for pastoral production and (often rougher) country better suited to biodiversity conservation, this model has the potential to be more widely adopted across Northern Australia. Its wider adoption would not only benefit conservation of wildlife but can permit diversification of income for pastoralists through tourism and carbon projects.

In this presentation, I discuss the evidence base that underpins the model, and illustrate its adoption.

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How supporting others creates wins for everyone: a case study of 'Pets In The Park'

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Background

In an ideal world, when a companion animal pet gets sick, a pet owner would take them to a veterinarian who would assess the animal's condition and prescribe a remedy. However, for people who cannot afford pet care, and especially for pet owners experiencing homelessness who may have little to no disposable income, no transport, and few people they can call on for help, a sick pet presents a major issue. Even the task of providing routine preventative care for a pet can be a significant challenge for people who are homeless. There is no direct government assistance available to help people to look after their pets, or to access pet-friendly housing, for those who are socially-disadvantaged or those with mental health issues. This is despite a growing evidence base of the value that pets can bring in helping improve people's mental and emotional health, and their physical and sociological health.

The importance of pets, for people of all walks of life

Pets are now being recognised as not only providing a source of satisfaction for people who want to have an animal in their home, but as also playing a variety of important roles that benefit human health. Pet owners report that their pets help them to feel calm, happy and able to handle stress.[1] Pets themselves have been shown to help reduce blood pressure in children and adults,[2–4] and the presence of a pet was better able to keep people's blood pressure down, during a stressful activity, compared to a best friend[5] or a spouse.[3] The presence of spouses and friends actually resulted in increased blood pressure, possibly due to feelings of 'judgement' by the person performing the task.

In studies involving older people, having a pet was associated with less deterioration in ability to perform activities of daily living, compared to not having a pet.[6] Pet owners (in particular dog owners) were more likely to use parks, perform leisure-time physical activity, walk (and walk for longer), compared to non-pet owners.[7] Owning a pet was also found to improve cardiovascular ability, and to relate to better recovery after a cardiac event.[7] Geriatric patients with schizophrenia who interacted with animals were found to have significant improvements in social functioning, impulse control, and daily activities.[4] Improved socialisation, weight-maintenance, reduced fear and anxiety and less loneliness have all been reported associated with pet ownership.[7]

In a study of people with mental health problems, they reported their pets to be in their most valued circle of support. Pets provided a source of secure and intimate relationships that otherwise were not available and were reported to be a valuable source of "illness work" that helped people to manage their feelings through distraction from symptoms and upsetting experiences, and provided a form of encouragement for activity.[8] Despite all of the above benefits of pet-ownership, pets are generally not considered part of mental-health plans,[8] and financially-disadvantaged people are offered no direct government assistance to care for their pets. The opposite is often more the case, that pet-friendly government-assisted accommodation or affordable-housing is generally unavailable and as a result many people choose to live on the street, rather than give up their pets.

A growing global trend to help vulnerable members of society and their pets

There is a growing global trend in Australia, the USA, Canada, Great Britain and other countries, for members of the public and professionals who are more financially and socially advantaged to help those who are disadvantaged. This trend carries through to the realm of pet-care and veterinary care.

The birth and growth of Pets In The Park

Pets In The Park (PITP) was started in 2012 in Sydney Australia, when a group of veterinarians and veterinary nurses realised there was an urgent need for a service dedicated to caring for the pets of those experiencing homelessness, or at risk of becoming homeless. The original model included two 'pop-up' veterinary clinics, one in Darlinghurst, near Kings Cross on the outskirts of the CBD, and the second in Parramatta, approximately 20km west of the CBD. In 2014, a program was launched in Frankston, Victoria (50km south of Melbourne) and in 2015 / 2016 a Melbourne CBD program started. In 2016, a Brisbane CBD program was started and in 2017 a Canberra program began. In 2018, a Hobart program started and in 2019, PITP launched in Perth (WA) and Caboolture (Qld). PITP provides free veterinary clinics for people experiencing homelessness who have pets. At the clinics, qualified veterinarians perform free health checks, vaccinations, administer flea and worm treatment and dispense basic medication for pets. Desexing programs are also provided throughout the year. Veterinary clinics can donate services and surgeries to the program (including desexings and other surgical or diagnostic procedures) which allows people experiencing homelessness to have these procedures performed when they otherwise could not afford to do so. All programs are totally volunteer-run and the organisation currently has a single paid staff member (CEO) who provides an invaluable service to ensure the smooth running and growth of the organisation. Corporate supporters have also provided generous donations, particularly in-kind donations of vaccinations and medicines that allow the programs to run. Sincere gratitude goes to the organisation's current national supporters – Virbac Animal Health, Applaws Natural Pet Food, Microchips Australia and BRC Lift and Interior Fixtures. Donations from the public are much-needed for the many costs of running the organisation and also allow for new programs to be launched, such as the facilitation of an Outreach Program in Sydney which allows PITP greater reach across the Sydney metropolitan region via a dedicated van and volunteer staff.

Why services like PITP are necessary

People experiencing homelessness are among the most marginalised groups in the world, and pet ownership amongst homeless people in Australia may be as high as 23-25%.[9–11] With Australia's homeless numbers estimated by the Australian Bureau of Statistics at 116,000 in 2016*, this could mean in excess of 29,000 pets belonging to people without homes.

Homeless people may feel pressured to give up their pets due to a lack of financial ability, or an inability to find pet-friendly accommodation. Yet pets perform vitally important human-health roles as discussed above, and pets have also been suggested to help people

* http://www.abs.gov.au/ausstats/abs@.nsf/lookup/2049.0Media%20Release12016

marginalised from society to construct a 'positive personal identity',[12] which is of critical importance for the mental health of these individuals. Caring for pets has been demonstrated to result in improved self-care behaviours and reduced self-destructed behaviours, including numerous documented anecdotes where individuals cite the need to care for their pets as being their reason to give up addictions, stop self-harm, and avoid behaviours that might see them return to prison.[12] Pets give people a 'reason' to live, which carries through even after a pet may pass away from old age (usually replaced by another pet).

However, without financial capability to provide for themselves and their pets, homeless people will often neglect themselves to ensure their pets are fed and looked after. They will go without food and give up other necessities in order to provide for their pets, and through this process, they can redefine their self-image, from one of hopelessness to that of 'carer'.[12] Being able to care for their pets can allow homeless people a path to improved self-actualisation and self-belief. When significant medical issues for a pet occur, this can be a challenging time for a homeless pet-owner. However, with organisations like PITP, and generous veterinary clinic supporters, available to help, this can also be a time when an individual who has become isolated can begin to re-connect with society and begin to understand that people do care for them and the wellbeing of themselves and their pets.

The benefits are not only for clients but also for 'givers'

Interestingly, the benefits of helping others extend beyond those actually being helped. Examining PITP as a case study, there are many direct and indirect benefits felt by those who participate in helping running the clinics or helping run the organisation. These kinds of benefits extend to many other charities and charitable activities also.

Some of the more obvious benefits to volunteering include a sense of achievement, advancement of professional growth, and the positive recognition and feedback[13] that volunteers get for their activities. Many volunteers are quoted to say that volunteering is actually a 'selfish' rather than an altruistic activity, because they get back more than they give[14]. It has been shown that volunteering can be mood-enhancing, social-integrating, health-promoting, or even death-delaying.[14] Volunteering is also shown to result in greater longevity for participants, better functional ability, lower rates of depression in later life, and less incidence of heart disease.[13]

Volunteers learn to interact in different environments, embrace cultural differences, and grow in their understanding and empathy towards others. On a professional level, they are exposed to different roles that might not be available in their current career, including leadership, governance, communications, risk-assessment, or even events-management. All of these roles are available to volunteers at PITP, depending on their interest. For younger or less experienced people especially, volunteering also looks great on a CV.

Other benefits of volunteering include socialisation and community engagement, and making friends outside of one's current circles. Volunteering has even been known to lead to successful (romantic) relationships (in the author's experience).

For veterinarians who have clinical experience but who are currently working outside of clinical practice (for example those working in industry, government, academia, or a non-veterinary role), PITP can also present an opportunity to continue to work to 'keep their skills up' for a few hours a month in a low-stress environment, and to perform simple consultations, under the supervision of experienced vets and nurses.

For non-volunteering supporters, including corporates, supporting veterinary clinics, and other donors, a relationship with PITP can provide an opportunity for ‘corporate good citizenship’, and can help further engage staff in a collegial-giving opportunity that can help foster a better working relationship at an organisational level. Staff of a supporting organisation may subsequently also wish to have active participation in the charity, or conduct fundraising in aid of the charity, creating further wins for the charity and further, ongoing engagement at multiple levels. (Staff might then also start to see some of the indirect benefits of volunteering as discussed earlier, and this can have further ongoing benefits for their organisation as well.)

The future

With all the benefits associated with both the giving and receiving from the programs run by PITP and other charities, hopefully there is a long future ahead for all such organisations. Anyone interested in joining PITP is encouraged to contact their local PITP in their area, or if there isn’t one, and someone is interested in starting a region of their own, please contact mail@petsinthepark.org.au or feel free to contact the author.

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Disclosures

Dr Mark Kelman is a director of Pets In The Park Inc. The views shared in this paper are his own, and do not necessarily reflect those of the organisation. Dr Kelman receives no remuneration, as a director of this charity.

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Non-neoplastic and neoplastic disorders linked to desexing in dogs

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Introduction

Throughout most of the developed world, surgical sterilization has become a common tool for combatting the overpopulation of unwanted dogs and eliminating the risk of reproductive diseases in pet dogs (e.g. mammary gland cancer and prostate hyperplasia /infection).[1-6] In the United States, 64% of dogs have been surgically sterilized.[7] For this review, ovariectomy and ovariohysterectomy (spay) or castration (neuter) will be collectively referred to as gonadectomy, since each of these methods for surgical sterilization includes gonad removal (ovaries or testes).

In the normal adult mammal, the hypothalamus secretes gonadotropin-releasing hormone (GnRH), which stimulates the anterior pituitary gland to release of luteinizing hormone.[8] Luteinizing hormone (LH) stimulates the secretion of gonadal steroid hormones (testosterone in males and estrogen/progesterone in females). These gonadal steroid hormones then negatively feedback to the hypothalamus and anterior pituitary to decrease the secretion of GnRH and LH, respectively. However, in the gonadectomized (desexed) mammal, there is no negative feedback, which results in supraphysiologic circulating concentrations of LH. In desexed dogs, LH concentrations are more than thirty times the concentrations found in normal adult dogs.[9] Although the main role of LH is for reproductive functions (e.g. ovulation, corpus luteum formation), there are LH receptors present throughout the body, not just limited to the reproductive tract. The purpose of LH receptors in non-reproductive tissues is not known but may induce cell division and stimulate nitric oxide release.[10] With constant activation following gonad removal, these receptors are upregulated, further magnifying the effects of the supraphysiologic LH concentrations in non-reproductive tissues. In this review, I have summarized several non-reproductive long-term health complications resulting from canine desexing and discussed the possibility of how these effects are mediated by LH receptor activation in these non-reproductive target tissues.

Non-neoplastic disorders

Obesity is serious medical problem defined as an excessive accumulation of fat beyond the physical and skeletal limits.[11] Gonad removal is the single largest risk factor for the development of obesity in dogs.[12] Up to 68% of desexed dogs are obese.[13-18] Gonadectomy induces obesity through two main mechanisms: increased appetite and decreased metabolic rate. Gonad removal stimulates food intake[19] and increases indiscriminate appetite.[20] In unaltered dogs, food intake suppresses the secretion of gastrointestinal hormones (cholecystokinin and glucagon) resulting in satiety (alleviation of hunger).[21] However, within 1 week following de-sexing, food intake increases by 20% and then persists.[22-23] It is possible that stimulation of LH receptors (present in the gastrointestinal tract following desexing) increase cholecystokinin and/or glucagon release. It is also possible that LH receptors in the hypothalamus are involved in the increase in appetite[24] as lesions within the ventromedial hypothalamus result in hyperphagia.[25]

Urinary incontinence is an involuntary leakage of urine resulting from either a weakened or complete loss of urinary sphincter control. The association between urinary incontinence and gonad removal in female dogs was first described by Jo (1965).[26] Urinary incontinence is a common long-term health complication of desexing female dogs, with a reported incidence ranging from 5-30%.[27-30] Early age desexing (under 5 months of age) may further increase the risk of occurrence of urinary incontinence.[30-31] LH receptors are expressed in all regions of the canine lower urinary tract, from the body and neck of the bladder to the proximal and distal urethra.[32-33] Desexed female dogs with urinary incontinence have a significantly higher number of LH receptors in the lower urinary tract compared with unaltered females.[34] Urinary continence can be restored in desexed females by reducing circulating LH concentrations using estrogens,[29,35-38] GnRH agonists[39-40] or GnRH immunization.[41-42]

Urinary calculi are solid particles (concretions) in the urinary system, usually composed of mineral salts that can form in any part of the urinary tract.[43] After evaluating records from more than two million dogs, Banfield Pet Hospital found that all urinary calculi (urine crystals, kidney stones, and bladder stones), occurred at a rate three times higher in desexed dogs compared with unaltered dogs.[44] Under normal circumstances, there is a balance of urinary calculi promoters and inhibitors. However, this balance appears to be disrupted from the influence of an abundant LH environment.

Diabetes mellitus results from the impaired secretion of insulin with variable degrees of peripheral insulin resistance leading to hyperglycemia. In dogs, the incidence of diabetes mellitus is 0.4-1.2%[45] and has been increasing over the past 30 years.[46-47] Gonad removal doubles the risk for developing diabetes mellitus in dogs.[46] Although desexing increases the risk for obesity, the increased prevalence for diabetes mellitus in desexed dogs is unrelated to obesity[48-49] and may be a direct effect of LH on the pancreas.

Hypothyroidism is a common endocrine disorder in which the thyroid gland does not produce sufficient quantities of thyroid hormone.[50-51] Gonad removal has a profound effect on thyroid function[52] and is the most significant cause for the development of hypothyroidism in dogs.[53] Thirty percent more desexed dogs develop hypothyroidism compared with unaltered dogs.[54] The concentrations of thyroxine in desexed dogs were significantly lower in both genders when compared with intact dogs.[55] Women who have undergone gonadectomy are also at an increased risk for developing hypothyroidism.[56] LH receptors are expressed in normal and adenomatous human thyroid glands.[57] Our laboratory has reported on the presence of LH receptors in the canine thyroid gland co-localized with thyroid stimulating hormone (TSH) receptors.[58] It is possible that continuous LH receptor activation is interfering with the mechanism of action of TSH in the thyroid, resulting in hypothyroidism.

Canine hip dysplasia is associated with the abnormal joint structure and laxity of the muscles, connective tissue, and ligaments that would normally support the hip.[59-61] As hip joint laxity increases, the articular surfaces between the acetabulum and the head of the femur lose contact with each other, resulting in subluxation. Over time, subluxation results in a significant change in the size and shape of both articular surfaces and varying severity of osteoarthritis. It is important to note that most dogs with hip dysplasia are born with normal hips but then develop hip dysplasia secondary to intrinsic and/or extrinsic factors. The incidence of hip dysplasia can be as high as 40-83% in giant and large breed dogs.[59,62-63] Independent of the occurrence of obesity, desexing significantly increases the incidence of canine hip dysplasia.[64] Compared to unaltered dogs, desexing increases this by 1.5 times[59] to 2 times[65] the occurrence in unaltered dogs. The mechanism for the increased incidence is not known but our laboratory has demonstrated the expression of LH receptors within the ligament of the head of the femur, the hyaline cartilage, and subchondral bone of the femur head.[66] It is possible that an increase in LH receptor activation in the structural support tissues within the hip joint results in increased laxity, which is responsible for the higher occurrence of hip dysplasia in de-sexed dogs.

The cranial cruciate ligament serves to prevent cranial displacement of the tibia relative to the femur, to limit internal rotation of the tibia relative to the femur, and to prevent stifle hyperextension.[67-68] Cranial cruciate ligament rupture is another musculoskeletal disorder that initially involves the degeneration of the cranial cruciate ligament, which leads to a partial rupture and then progresses to a complete rupture following an unspectacular traumatic event.[69-70] Similar to hip dysplasia, most dogs with cranial cruciate ligament ruptures are born with normal stifle joints but then develop the tendency for cranial cruciate ligament rupture secondary to intrinsic and/or extrinsic factors. Gonad removal significantly increases the prevalence of cranial cruciate ligament rupture,[71] doubling the occurrence reported for unaltered dogs[72] with an incidence as high as 5.1% and 7.7% in males and females, respectively.[65] Prepubertal desexing delays tibial growth plate closure,[1] which extends the length of tibia and the steepness of the tibial plateau.[73-74] Increased steepness of the tibial plateau can increase the cranial tibial thrust, which is a risk for cranial cruciate ligament rupture.[75-76] Despite the skeletal deformations that occur with pre-pubertal desexing, even dogs post-pubertally desexed have an increased risk for cranial cruciate ligament rupture.[65] There is some evidence that hormones (estrogen and relaxin) may play a role in altering cranial cruciate ligament laxity and modify risk factors in humans.[77-78] Our laboratory has demonstrated the expression of LH receptors within the cranial cruciate ligament.[66] It is possible that an increase in LH receptor activation in the cranial cruciate ligament results in increased laxity, which is responsible for the higher occurrence of ligament ruptures in desexed dogs.

The role of gonad removal on behavior is complex. Evidence for benefits as well as detriments following desexing has been reported. Reproductive-related behaviors (such as urine marking in house, mounting, and roaming) are all reduced or eliminated following gonadectomy.[79-81] However, fear and aggression tend to be exacerbated.[82] Fear of storms, fear of gunfire, fear of noises, fear biting, timidity, separation anxiety, and submissive urination all increase significantly following desexing. Desexed females are also more reactive to the presence of unfamiliar humans and dogs.[83] Although some dogs may become less aggressive following gonadectomy,[80] dominance aggression[84] and owner-directed aggression[20,85] occur with a significantly higher frequency in desexed dogs compared with unaltered dogs. The hippocampus and hypothalamus both play important roles in controlling behaviors, especially those pertaining to fear and aggression. Luteinizing hormone receptors are abundant in the hippocampus and hypothalamus.[86-88] In addition, administration of supraphysiologic concentrations of LH to gonadectomized animals can induce aggression and other behavioral changes.[89-91]

Cognitive dysfunction syndrome is a neurodegenerative disorder of senior dogs, which is characterized by both cognitive changes and neurophysiological pathologies.[92-93] Memory impairment, poor problem solving skills, social disconnect, confusion, and day-night reversal may occur as the condition progresses. Gonad removal significantly increases the development and progression of cognitive dysfunction syndrome in dogs.[94] Increases in luteinizing hormone are associated with declines in cognitive performance.[95] In addition, elevated LH concentrations increase beta amyloid plaque formation and are implicated in the development of Alzheimer's syndrome in humans.[96-97] Therefore, it is possible that LH and its receptor are important in the development of cognitive dysfunction syndrome in desexed dogs.

Neoplastic disorders

Unlike the condition in men, the aggressive nature of the canine prostate adenocarcinoma and the lack of a screening test make the identification of early-stage prostate cancer in dogs extremely problematic.[98] In dogs, desexing is the largest risk factor for the development of prostate adenocarcinoma.[99-100] Luteinizing hormone receptors are abundant in the prostate gland and increase in expression following gonadectomy.[101-103] Prostate carcinomas in dogs are associated with a high rate of metastasis at presentation and poor prognosis even with aggressive local therapies.[98] Prostatectomy is associated with significant postoperative morbidity, in particular urinary incontinence, without significantly extending survival times.[104-105]

Transitional cell carcinomas can arise from the bladder or urethra, including the prostatic urethra.[106-110] Even with surgical removal, radiation treatment and chemotherapy, the prognosis for dogs with transitional cell carcinomas is poor with only 16% of treated dogs surviving for over one year.[111] Desexed dogs have a significantly higher risk of developing a transitional cell carcinoma compared with unaltered dogs.[111] Luteinizing hormone receptors are widely distributed throughout the bladder and urethra and increase in expression following gonadectomy.[32-33,112-113] Our laboratory has also demonstrated the abundant expression of LH receptors in transitional cell carcinoma tissue.

Osteosarcoma is a highly metastatic cancer of bone tissue. Despite many advances over the past 20 years, survival times for dogs diagnosed with osteosarcoma have not changed, with the principal cause of mortality being the development of pulmonary metastases.[114] Osteosarcoma occurs with significantly higher frequency in desexed dogs.[115] The incidence of osteosarcoma in desexed dogs is 1.3-2.0 times higher than in unaltered dogs.[116] It is not known if LH receptors exist in the bone or if LH could be using an indirect mechanism to mediate the increased incidence of osteosarcoma.

Hemangiosarcoma is a rapidly growing, highly invasive cancer arising from the lining of blood vessels and occurring almost exclusively in dogs. Primary tumors can arise in any vascular tissue but the spleen and heart are the most common locations for hemangiosarcoma to develop. Even with surgical removal, the mean life expectancy is 86 days (range, 10–202 days) without adjunctive chemotherapy and 189 days (range, 118–241 days) with adjuvant chemotherapy.[117] Many studies have confirmed the presence of LH receptors in vascular endothelial and smooth muscle cells.[118-119] Desexed female dogs have two times the risk for developing splenic hemangiosarcoma and five times the risk for developing cardiac hemangiosarcoma compared with unaltered females.[82,120] Our laboratory has also demonstrated the expression of LH receptors in hemangiosarcomas, which may explain why this cancer is more common in desexed females.[121]

Mastocytoma is the most common skin tumor in dogs.[122] Luteinizing hormone receptors are abundant in the skin.[32,123] Several studies have documented an increased risk for developing mastocytoma following desexing in dogs.[65,82,124] Our laboratory has shown that not only do mastocytomas express LH receptors, but that these tumors express three distinct patterns of LH receptor immunoexpression.[125] Moreover, mastocytomas from desexed dogs had significantly higher more LHR-positive mast cells (84.2±8.7%) overall. In addition, LHR-positive mast cells exhibiting the type 2 pattern (66.6±15.3%) compared with mastocytomas from intact dogs (64.3±4.2% and 49.2±8.4%, respectively).[125] The higher expression of LHR provides a mechanism that could be exploited in intervention strategies (e.g. using GnRH agonists) for mastocytoma recurrence in desexed dogs leading to prolonged survival time.

Lymphosarcoma is a cancer of lymphocytes and/or lymphoid tissues. Lymphosarcoma is the most common cancer diagnosed in dogs accounting for up to 24% of all canine cancers.[126] LH receptors are present in lymphocytes and lymphoid tissue (medulla of thymus).[127-128] Our laboratory has demonstrated that the mean percentage of circulating LH receptor-positive T lymphocytes is significantly higher in desexed dogs (16.6%) than in sexually intact dogs (10.5%); whereas the percentages of circulating LH receptor-positive B lymphocytes did not significantly differ by reproductive status.[128] Gonadectomy increases the incidence of lymposarcoma.[82] Desexed males are three times more likely to develop lymphoma than unaltered males and about 1 in 10 neutered males will develop lymphoma.[65] Our laboratory has demonstrated that 12.4% of cells in canine neoplastic lymph nodes expressed of LH receptors.[128] In addition, we showed that in vitro activation of LH receptors on T-lymphoma cells stimulates cell proliferation.[129]

Conclusion

Unrelated to any particular disease or major cause of death, years of gonad retention prolong longevity.[130] Based upon the review of the literature, it becomes clear that canine gonads are not merely reproductive organs but critical to endocrine, musculoskeletal, behavior, and anti-neoplastic health. Among the non-reproductive functions of gonads, suppression of LH secretion and resulting LH receptor overexpression appear necessary in maintaining homeostasis. Therefore, a surgical sterilization method that enables the dog to keep gonads intact while still preventing reproduction is likely to prolong their health.

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What do Australian customers say about vets? Findings from a series of research groups

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Onswitch has a long history of undertaking consumer research with pet owners across Australia - each year we carry out both formal and informal data gathering with pet owners and clients of veterinary practices, in order to identify what drives their behaviours and choices. In May 2018, three focus groups were held with cat and dog owners in Brisbane. Attendees were recruited to represent a wide range of pet experiences, species and age, and all had visited their practice within the last six months. Almost without exception, these owners referred to their pets as family members, speaking of the money, time and love lavished on them. All spoke of the importance of their chosen veterinary professionals 'getting' the human-animal bond and many had switched practices where they had experienced vets and nurses lacking warmth towards their pet. Australian clients mirror those in the UK in wanting their practices to 'feel' right, with vets and nurses involving owners in decision-making, acknowledging that they know their animals best, and recommending treatment options that put the pet's needs first, not profit or ease.

It's not all about price

Vets often believe that owners choose a practice primarily based on cost, yet Onswitch research both in Australia and the UK finds that price is the primary factor in less than 10% of initial practice choices (and then it is weighed up against what you get for the price). Of far greater importance are convenience (location, proximity and opening hours) and rapport with the team. Our ongoing research consistently shows that decisions on practice choice for Australian pet owners are rooted firmly in emotional factors, with several in the latest qualitative groups reporting that they drive further than necessary because the vet is lovely enough to justify it.

We asked our Brisbane groups to talk about the regular healthcare routines they follow with their pets and how they feel about their practice. The results showed:

- Owners are accepting of level of vets fees, "they are all the same"
- Pragmatic decisions are made about their animal's care - unexpected bills go on the credit card
- Owners like to see the same vet each time, many had switched from practices where locum numbers are high
- Bad experiences lead to switching practice
- Recommendation from family and friends is key
- Reviews on Google and Facebook also help greatly in choosing a new practice
- Lots of cost-benefit analysis is made about vet and pet care - when it comes to healthcare plans, owners want to get back what they pay in (just as they do with their own plans)
- Importance of trust - vets need to demonstrate love and make a connection with the pet and the owner
- There is plenty of choice of practices, many owners use a practice 1-2kms away and make a conscious choice to travel because they like the people and / or service there

The ideal practice

Crucially, pet owners predominantly describe the service they would like to receive, rather than the building it takes place in. The three groups were consistent in their feedback that the 'ideal' practice would:

- Feature a personal touch from everyone in the team
- Have authenticity - a genuine love of animals
- Be small enough to be friendly and familiar
- Allow enough time to be heard and not feel rushed
- Offer good parking
- Have charges stated clearly up front
- Feature more innovative waiting area designs and layouts in order to reduce stress for pets (cats in particular)

The average Australian practice

In May 2018, Onswitch carried out a swathe of practice visits in Melbourne, Sydney, Brisbane and on the Gold Coast - visiting as a potential customer we found that the 'average' Australian urban practice is not quite yet at the level of the focus group's 'ideal' practice:

- Very few leaflets and educational materials were given out
- No appointments were offered, even when prompted
- Pricing was vague when discussed and price charts were rarely visible
- Wellness plans and offers were not routinely promoted to potential clients
- Pet's name and history were rarely enquired after
- Name badges are rarely worn
- Compared with human healthcare premises, practices often look shabby and are poorly laid out

The crucial customer experience

The qualitative groups offer a unique insight into Australian customers' hearts and minds. It's vital that any service business understands the needs of its clients if it is to deliver a superior customer experience that can't easily be matched by competitors. For veterinary practices, providing an excellent customer experience drives new client registrations and boosts turnover - it's not hard to do, but it's important that it's done, and crucial that it's done right.

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Onswitch qualitative research, May 2018, Brisbane

Onswitch practice visits, May 2018

Mobile Abattoirs: One more day on the farm.

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Declaration of Conflict of Interest

I am a founder, shareholder and paid director of FarmGate MSU Pty Ltd, which operates a mobile slaughter unit under the brand Provenir. Funding to establish this business has come from SproutX (the Victorian Agtech Start-up accelerator program), MLA and private investors.

Introduction

Mobile abattoirs offer a real alternative to traditional static abattoirs for producers of livestock for food production. Since the 1980's, there has been a 75% global reduction in the number of static abattoirs, meaning a large number of producers must transport there livestock further for processing. In Australia, this reduction coupled with the size of our land mass, has meant in some cases transport distances of 2-3 days between farm and abattoir. Coupled with increasing commoditisation, less than perfect traceability, reduced access for small consignments of livestock, and recurrent charges of failing to maintain animal welfare standards, large abattoirs are meeting the needs of a smaller group of larger producers, while reducing access to an increasingly diverse producer demographic, as well as increasingly conscientious consumers.

Mobile abattoirs, with their small-scale, flexible model, slow work rate, and ability to go where the livestock is, offer many advantages, with greater respect for the animal at the end of its life, and improved access to processing for farmers being two of the major ones.

Background and Design

Mobile abattoirs first appeared in Scandinavia in the 1960s for use during seasonal reindeer hunts.

They have appeared for processing farmed livestock in the last 15 years across many countries, in many forms, with operations continuing today in the USA, Canada, Sweden, Chile and several other countries. Units operated in the UK but did not prove viable for many reasons (1).

The design has been built around a semi-trailer-sized unit, with or without on-board refrigeration, and commonly reliant on slaughter occurring outside the unit followed by winching the body into the unit. Later models have modified the units to accommodate an internal knocking box, conceivably enhancing hygiene. Business models have included service or contract processing only, or livestock purchase, or a combination of both; management has varied across producer co-operative-owned, or government-run, NGO-run, and privately operated.

In Australia, mobile abattoirs have periodically operated in recent times for camel and buffalo in the Northern Territory, and for emus in Pyramid Hill in Victoria (1997)(3). In 2010, Annabelle Coppin, a Pilbara beef producer, argued in her ISS Institute report that the northern Australian beef industry was ready for MSUs, given the vast distances that producers often needed to transport their cattle to processors (2).

Provenir, at the time of publication, has been operating a 40 foot custom-designed MSU pulled by a prime mover in the Riverina, NSW, for a little over a month. It is the first MSU in Australia for processing cattle and sheep.

Operation and Applications

Provenir enters into partnership with livestock producers based on production management factors, the quality of the livestock, and the accessibility and suitability of the infrastructure on the farm. Many of the conditions of the agreement are designed to maximise animal welfare; the dividend of these measures (2 months on farm in the same group; calm handling techniques, pre-exposure to yards with a non-stressful experience)(4) is enhanced biosecurity and food safety, and better meat quality. The day before the arranged date for processing, the farmer yards the livestock to be presented and leaves them yarded overnight.

The MSU arrives at the farm, and the livestock presented are given an ante mortem inspection by the Provenir meat safety inspector. Livestock that pass inspection are purchased and moved into a holding pen prior to one-by-one entering the MSU for slaughter and dressing. The entire process, from stunning to dressed carcase, occurs inside the MSU and is out of view and hearing of the remaining livestock. Refrigerated trucks then transport the carcases and edible offal to a further processing centre within 1-2 hours drive.

The potential for different applications of the MSU in the Australian setting is great, from remote Indigenous social enterprise, to increasing the efficiency and food safety of wild/feral animal harvests.

Benefits

The benefits of bringing the abattoir to the farm are numerous.

Animal Welfare studies over the last 30 years have pointed towards correlations between longer livestock transport journeys and increased stress and injury (6,7). Animals yarded in close proximity to, or with, unfamiliar animals also register increased signs of stress (4,5). Mobile abattoirs eliminate both these problems, with familiar handlers adding to the welfare dividend. Finally, the livestock are unaware of what is about to occur until the last minutes.

MSU Worker benefits are largely associated with the type of work and the work environment: less repetition and more varied tasks; slower chain speed; different location of the MSU every day or two; less exposure to refrigeration and freezer temperatures, so potentially less cold injury. There is no significant restriction in workspace compared with a fixed abattoir in our design. Better worker welfare potentially has a positive effect on pre-slaughter animal welfare.

Producers stand to benefit in many areas. Access to processing is the key benefit, with an improvement being realised in flexibility of livestock production as a result. This will improve cash flow. There will be no livestock transport fees to the abattoir, no agent's fees, no yield loss realised on live transport so more kilograms of animal to sell to the mobile abattoir. Premium price at the retail outlet will flow back to the farmer, improving farm income.

Community benefits stem from the locations of the mobile abattoirs and the processing hubs in regional Australia. The viability of smaller farming operations may be secured, and workers will be able to settle in smaller towns, due to the presence of mobile processing.

Local economies will be boosted, and regional development will follow. Agri-food tourism centres will be able to add a true, local, paddock-to-plate offering to their menu.

Meat quality will be improved; with greater glycogen stores in the muscles of the animal under minimal stress at time of slaughter, more lactic acid can be produced after death, which drops pH, and prolongs shelf-life.

The traceability of the product from the individual animal to the retail display cabinet will be greatly enhanced, through barcoding attached to every part of the body along the supply chain till packing and labelling. Live weights and carcase weights of each animal are available to the farmer on the day. Provenir is looking to utilise biomarker technology soon; this will reduce food substitution and fraud to almost nothing, and will be the best traceability tool available.

Five Years On?

Factors that will affect the future progress of mobile abattoirs in Australia include many that affect the wider Australian meat industry, including seasonal breeding times, climate change-induced extreme weather events and prolonged droughts, domestic and export market demand and regulatory changes.

Consumer preferences for meat that has been produced with highest welfare may increase, favouring the mobile abattoir-derived product. The consumer's ability to access the story behind the meat they have chosen, or buy/eat meat grown and processed locally, may also influence purchasing choices. Producer realisation of higher income, and greater flexibility in processing times should strengthen supply of livestock to a mobile abattoir.

Beyond these cautious predictions for slow development of this niche sector of the meat industry, it is difficult to divine where the model will position itself.

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Mitigating eye disease in sheep and cattle destined for live export

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This article contains excerpts of Dr Fraser Murdoch's PhD thesis and modified for publication here by Dr Laurence. This research was funded by the Live Export Programme (Meat and Livestock Australia).

Introduction

Sheep

Western Australia started exporting live sheep in 1845[1]. Today Australia remains overall the world's largest exporter of livestock and the industry's welfare standards are a global benchmark[2]. Sheep are a major part of this export trade.

The live export trade remains a heavily scrutinised and controversial industry. All exported animals are subject to the Australian Standards for the Export of Livestock (ASEL) legislation[2]. These 23 standards are in place to ensure animal health and welfare remain a priority throughout the export chain and that livestock sourced for export are fit and healthy to travel. Any animals failing to meet these standards should be removed from the export chain.

Anecdotal figures from exporters indicate that approximately 0.5-1% of sheep are rejected annually because of the presence of Infectious Ovine Keratoconjunctivitis (IOK). These sheep are deemed to have no commercial value and are typically sold to the pet meat trade. Although sheep will typically recover from IOK with appropriate treatment, there is a cost associated with this treatment in medication, labour hours and additional feeding. In addition to the financial costs associated with the disease, IOK has been recognised as having a detrimental effect on animal welfare. Conjunctivitis is an irritating condition in humans[5] and corneal ulceration is considered a very painful condition in humans[6]. Assessment of pain and discomfort in a prey animal such as a sheep is notoriously difficult[7]. Considering the findings of Leibowitz (200) and Wirbelauer (2006) it is reasonable to believe that sheep suffering from these conditions are also feeling pain, even though they show little or no signs of discomfort. However, it is essential to act on the assumption that the sheep are in pain, so that animals suffering from IOK receive effective treatment to minimise any possible impact on their welfare and long-term health. This is not only in the obvious interest of the animals but in the interest of the live export industry, whose animal welfare standards must bear rigorous scrutiny.

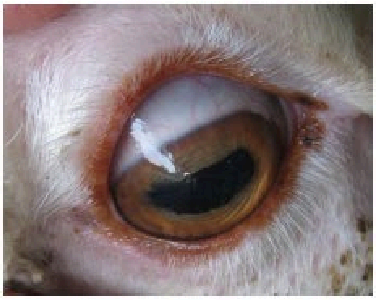
General aims

Among others, some of the general aims of this extensive body of research were:

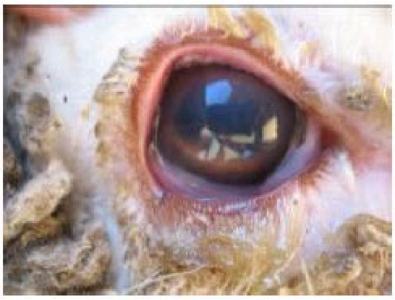
- considering that much research has been published on microbiological causative agents of IOK, to determine if these agents are present in feedlot conditions and associated with clinical disease
- to test the efficacy and impact on the animals of various common treatments for these organisms
- to identify practical and effective treatments for IOK in pre-export feedlots that house up to 80,000 sheep
- given that IOK is a progressive disease, to determine if a severity of disease treatment cut-off can be established so that a decision can be made on which treatment, if any, would be most appropriate at different stages of the disease

Grading system

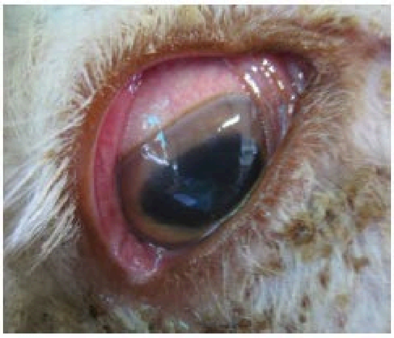
The characterisation of IOK in the eyes of sheep was done using a one to five scale. Here is the chart that we used:



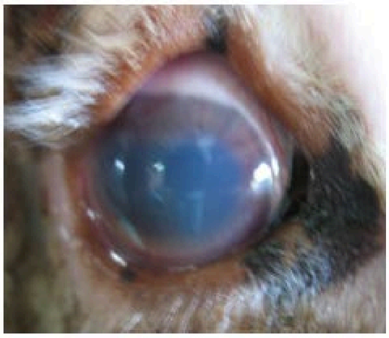
Grade 0 – normal eye



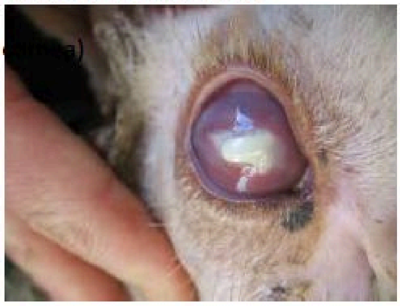
Grade 1 – epiphora (weeping eye)



Grade 2 – conjunctivitis

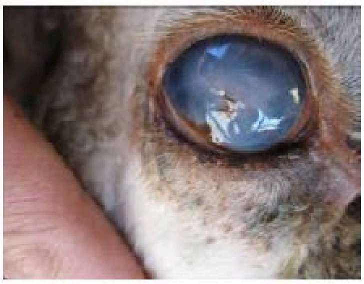


Grade 3 – corneal oedema (clouding of the cornea)



Grade 4 – corneal ulceration

Grade 5 - corneal neovascularisation



Grade 6 – chronic eye damage

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Figure 1: Grades of severity of Infectious Ovine Keratoconjunctivitis.

Experiment 1 – Identification of the microbiological causes and possible treatment of infectious ovine keratoconjunctivitis in a pre-export quarantine feedlot.

The aims of this experiment were to:

- describe normal ocular flora in sheep in a pre-export feedlot
- assess the efficacy of several commonly used treatments at reducing the clinical appearance of IOK in naturally infected sheep
- assess the impact of those treatments on the bacterial population in the ocular environment.

Methods

Fifty-one merino and merino cross sheep with no clinical signs of ocular disease were selected from the pre-export feedlot. Both eyes were swabbed using a cotton-tipped, wooden-shafted swab which was inserted into the fornix, between the conjunctiva and the globe. Eighty merino and merino cross sheep with naturally occurring IOK infections were also selected from those sheep held back from a shipment owing to the presence of clinical eye disease. Sheep were selected based on the clinical grade of infection. The grading system was used. Only sheep with grade 2 (conjunctivitis) or grade 3 (corneal oedema) in both eyes were selected for this experiment.
We tested several common treatments:

Group	Given
1	Oxytetracycline injectable (Alamycin LA 300, Norbrook Lab. Aust. Pty Limited) 20mg/kg given as a single intramuscular injection into neck muscle. Dose calculated based on accurate sheep weights.
2	Cloxacillin 500mg/3g (Orbenin Eye Ointment, Pfizer Animal Health) administered at a dose of 125mg per eye (one quarter of a tube applied as a streak to each eye).
3	No treatment, control group
4	Oxytetracycline powder 200mg/g administered orally in the drinking water. 2g of powder(400mg drug)/head/day based on each sheep drinking 4L water/day.
5	Oxytetracycline 20mg/g, (Terramycin Pink Eye Powder, Pfizer Animal Health), applied topically twice daily to eyes for 5 days.
6	Oxytetracycline 20mg/g, (Terramycin Pink Eye Powder, Pfizer Animal Health). Applied topically to eyes twice daily for 5 days PLUS Oxytetracycline injectable 20mg/kg (Alamycin LA 300, Norbrook Lab. Aust. Pty Limited) given as single intramuscular injection into the neck muscle. Dose calculated based on accurate sheep weights.
7	Oxytetracycline 2.0mg/g, (Terramycin Pink Eye Aerosol, Pfizer Animal Health). Applied topically to the eyes twice daily for 5 days.
8	Oxytetracycline (Alamycin 300 LA Norbrook Lab. Aust. Pty) at a dose of 20mg/kg on day 0 and day 4 by intramuscular injection into the neck muscle. Dose calculated based on accurate sheep weights.

Table 1: IOK treatments tested

Results

A total of 20 different organisms were cultured from the 102 control eyes. Of these organisms, *Bacillus* species were the most commonly isolated with 87.3% positive. *Moraxella ovis* was the second most commonly isolated organism with 38.2% followed by *Staphylococcus chromogens* with 33.3%. Only 4.9% of control animals were positive for *Mycoplasma* species, only one of which was positively identified as *Mycoplasma conjunctivae.*

It was found that two injections of long acting Oxytetracycline (OTC) treatment (Group 8) and Oxytetracycline single injection combined with topical oxytetracycline powder (Group 6) were the most effective treatments.

Discussion

Up to 20 different species/genera of organisms were isolated from the eyes of healthy sheep including *Moraxella ovis* and *Mycoplasma conjunctivae*. This result highlights the potential carrier status of healthy sheep that can act as a source of infection to naïve animals.

This experiment has highlighted the limited value of using topical treatments for IOK in sheep. The use of topical OTC products has been found to worsen the clinical signs of IOK and giving no treatment is more effective that using either of these products. Injectable OTC was shown to be the most effective treatment, and this result is like studies carried out under more extensive situations in other countries. Although not the most effective treatment tested, in-water OTC was more effective than giving no treatment. Given the relative ease of administration of medication in water to large numbers of animals, this treatment warrants further investigation. This investigation was done and while in-water OTC was clinically efficacious, the use of OTC-medicated pellets yielded better results. In addition, in-water OTC depressed appetite significantly and caused changes to the rumen microbiome that were detrimental to animal health. Establishing that injectable OTC is the most effective enables this treatment to be used as a useful standard with which to compare any treatments used in subsequent experiments.

Experiment 2 – The treatment of infectious ovine keratoconjunctivitis with in-feed medication in a pre-embarkation feedlot.

Introduction

It was important to test whether the positive responses seen clinically in sheep treated with OTC-medicated feed in controlled animal house environments were replicated in an actual pre-embarkation feedlot. Up until this point all experiments had focused on small numbers of animals with frequent and close individual monitoring. The experiment reported here mimicked the feedlot environment. This experiment was designed to validate the previous results in an industry context so that the efficacy of in-feed treatment of IOK could be demonstrated as a practical, affordable and realistic option for exporters. Both productivity and animal welfare were hypothesised to improve should the results concur with previous research.

A range of severity of infection was considered in this research, based on selection of sheep with eye grades between 1 and 6. The experiment aimed to assess the efficacy against varying grades of IOK of in-feed OTC medication compared with two injections of OTC, which has been consistently found to be the optimum treatment.

Hypotheses

- Oxytetracycline-medicated feed will be effective in treating mild, up to and including grade 3, IOK
- Two injections of OTC will be effective against all grades of IOK

Methods

Two hundred and seven Merino cross, mixed age sheep with naturally occurring clinical IOK were selected from those rejected at a pre-export feedlot. No sheep had received treatment prior to selection.

Sheep eyes were graded using the grading system previously described. All sheep had clinical IOK in both eyes, not necessarily of equal severity. Following grading, sheep were drafted into two groups, one group with clinical eye grades 2-4 and the other with clinical eye grades 5-6. From these groups, sheep were randomly drafted into 3 treatment groups:

Group 1 - control group receiving no treatment (n=69 with 40 grade 2-3 and 29 grade 4-5)
Group 2 - intra-muscular injection OTC (Alamycin 300 LA, Nobrook Laboratories Australia PTY Ltd, Tullamarine, VIC, Australia) (2 doses of 20mg/kg 4 days apart) (n=70 with 45 grade 2-3 and 25 grade 4-5)
Group 3 – in-feed OTC (Terramycin 200, Phibro Animal Health, Girraween, NSW, Australia) (n= 68 with 36 grade 2-3 and 32 grade 4-5).

Sheep were housed in three separate raised pens in a standard feedlot. Free access to water was given to all sheep. Those in groups 1 and 2 had *ad lib* access to pelleted feed as would be typical in a feedlot. Sheep in group 3 received medicated feed for 5 days. Accurate measurements of feed intake were not taken, but subjective assessment of residual feed was made; residual feed was removed daily. Accurate measurement of residuals was not possible owing to contamination of feed and crumbling of pellet diet. *Ad lib* access to non-medicated pellets was given following cessation of medicated pellets.

Eye grades were recorded every second day from day 0 up to day 10 using the same grading system as before.

Results

Eye score on Day 0 was significantly associated with eye scores post treatment ($P <0.001$). When treatment means were corrected for score on Day 0 there was a significant effect of Treatment ($P<0.001$) on daily eye score but this effect interacted with day-post-treatment such that the effect of treatment on eye score was different on different days' post treatment ($P<0.001$).

At the end of the experiment animals treated with injections had lower eye scores than those treated with in-feed medication. Both treatments had lower mean eye scores than the control animals.

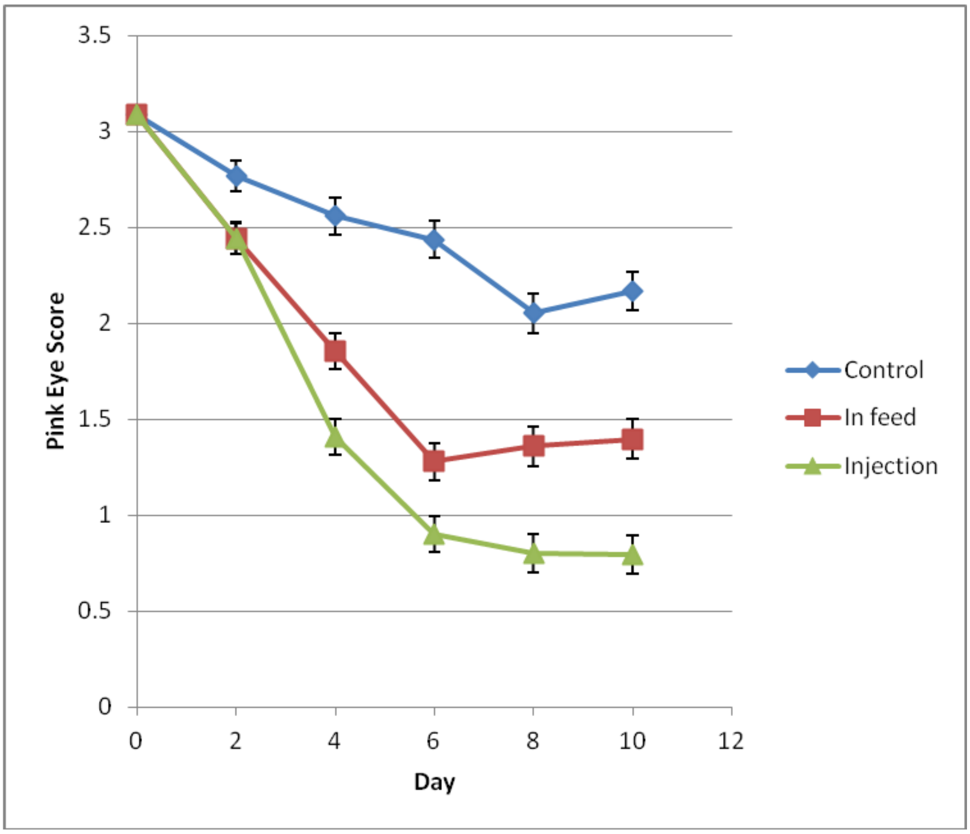

Figure 2: Adjusted pink eye mean scores for each treatment and day.

In-feed OTC for 5 days is an effective treatment for sheep with IOK up to and including grade 3, whereas injectable OTC is effective in treating IOK up to and and including grade 5 (Table 13).

Score	In-feed	Injection
1	Treat	Treat
2	Treat	Treat
3	Treat	Treat
4	Not effective	Treat
5	Not effective	Treat

Table 3: Treatment recommendations (Treat vs Not effective) of different routes of administration (In-feed vs Injection) at different degrees of severity of disease (Score) on day 8 of experiment.

Discussion

The results of the feedlot experiment are consistent with results seen in the previous experiments. Although, subjectively, feed intake in the medicated feed group did decrease, the amounts of residual feed remaining were small. In-feed OTC is considered effective in treating sheep with eye grades up to and including 3. The hypothesis was that in-feed OTC would be effective up to grade 4; however, the experiment demonstrated that those sheep with grades 4 and 5 need to be treated with intra-muscular OTC to have the best chance of recovery to the point where they could be shipped.

The use of in-feed medication is significantly less labour intensive than injecting individual animals which greatly reduces the cost of the treatment. Within a cohort of sheep at the pre-export feedlot there is likely to be variation in severity of clinical signs of IOK. Survey results from inspectors outlined in Chapter 4 indicated that up to 50% of a shipment could have mild IOK during the high-risk summer period. Given the numbers of animals involved and the labour required to inject those with mild disease it is encouraging that in-feed medication is an effective treatment. Given that IOK is considered a painful condition[10,11] having an effective treatment available that requires minimal labour could contribute to improved welfare of animals in pre-export feedlots.

General conclusions

Infectious ovine keratoconjunctivitis is a disease that continues to challenge not only the live export industry but also the farming community worldwide. The multitude of factors known to contribute to the occurrence and severity of an outbreak continue to frustrate veterinary practitioners and producers alike. Given the presence of several key risk factors within the pre-export feedlots, IOK outbreaks would appear to be unavoidable. It is for this reason that effective and realistic treatment strategies must be in place to ensure treatment of the disease, optimization of animal welfare, reduction in economic losses and an assurance to the public that the industry is focused on the health and welfare of the animals it exports.

Cattle

This research was commissioned after anecdotal reports from veterinarians and exporters working in the cattle live exports supply chain of sporadic, severe outbreaks of eye disease in cohorts of *Bos taurus* cattle destined for long-haul shipping to Russia or China. Out breaks occurred in pre-export quarantine, on board the vessels or during post-arrival quarantine. Eyes were often so severely affected that it resulted in the loss of the animal. The costs associated with treatment and loss of animals suitable for sale was significant.

Because the reports from industry were inconsistent with respect to time of year, severity, clinical signs and impact, a workshop as held to better define the problem. veterinarian working in the live export supply chain, export company representatives, makers of immunotherapeutics used to reduce the incidence of eye disease and epidemiologists contributed to the planning of this project. It was concluded that eye disease in these cattle was a multifactorial disease with different possible causative agents and several risk factors. One risk factor over which exporters had some control was the degree to which cattle were immune to disease before receival in pre-export quarantine. It was agreed that an experiment would be conducted to test the hypothesis that providing adequate immunotherapy to cattle in time for full immunity to develop before receival in pre-export quarantine, would greatly reduce the incidence of eye disease in quarantine, on vessels and during post-arrival quarantine. The trial was to be conducted over three voyages.

Sourcing suitable cattle for the experiments proved to be difficult, least of all because gaining access to cattle on-farm, five weeks before transport to pre-export quarantine seemed to be almost impossible. Most exporters didn't buy cattle until much closer to the shipping date.
A cohort of Angus cattle destined for China on agistment on a farm in Victoria was deemed suitable for the experiment, although the animals were a bit older than normal. A pathogen survey was conducted before animals were drafted in a Control and Treatment group where the Treatment group received additional vaccination for bovine viral diarrhoea virus, *Moraxella bovis, Manheimia haemolytica* infection and bovine herpes virus 1.2 infection.

Many of the animals had antibodies to BVDV, PI3 and were antigen positive to *Moraxella bovis* and *Moraxella bovoculi* on-farm. In pre-export quarantine and on board vessels there was a zero incidence of eye disease. It was not possible to directly confirm the hypothesis although the degree to which the animals were already immune to causative agents is postulated to be the reason for the low incidence of disease in quarantine.

One problem with this type of research is the difficulty in gaining positive association with treatment when disease outbreaks are rare. To show the effect of treatment (in this case appropriate use of vaccines) there needs to be significant disease in the Control group. This is fundamentally very difficult to predict in field-based research such as this. That and the difficulty in gaining access to cattle on-farm in a timely manner such that full vaccine courses could be delivered meant that the research was revised such that the results of the one experiment, a review of the scientific principles, an understanding of eye disease causation and epidemiology and well-established treatment protocols were used to produce best practice guidelines for the live export industry. the challenges associated with implementing these guidelines are well recognised.

It is suggested that wherever possible and practical, exporters aim to access cattle destined for export at least four weeks before collection at quarantine such that full courses of appropriate vaccines can be given and eye disease outbreaks minimised.

Best practice

The following are recommendations that will reduce the incidence of eye disease in pre-export quarantine and on vessels at sea:

1. Choose cattle that have been yard-weaned to minimise stress
Producers are directed to the MLA-produced resources on minimising the stress of weaning: https://www.mla.com.au/research-and-development/animal-health-welfare-and-biosecurity/husbandry/weaning/ and it is recommended that exporters source cattle from farms where cattle have been yard-weaned
2. Gain access to cattle while still on the farms of origin to provide appropriate immunotherapeutics in enough time (usually five weeks before receival in quarantine) that seroconversion has provided for protective antibody titres at the time of induction to pre-export quarantine. Some vaccines need two doses of vaccines to be protective against the disease and these can be up to one month apart. One dose of vaccine is not protective and cattle inducted to pre-export quarantine after one dose of a vaccine that requires two doses will not be protected and be susceptible to disease.
It is suggested that appropriate vaccines include those that provide protection against:
- bovine viral diarrhoea virus – (Pestiguard – Zoetis)
- *Moraxella bovis* (Piliguard – Coopers Animal Health)
- *Mannheimia haemolytica* (Bovilis MH)
- Bovine herpes virus – a few products are available. It is acknowledged that the presence of antibodies to BHV1.2 may be contrary to the requirements stipulated in export protocols. The use of live vaccines could be considered once blood testing is complete
3. Source local cattle if possible to minimise truck transport times
4. Adapt cattle to mixed rations slowly in pasture holding paddock to allow for acclimation to concentrated feed
5. Minimise over refining of feed to reduce dustiness
6. Use insecticidal ear tags or fly traps to minimise fly populations in feedlots
7. Employ dust mitigation strategies in feedlots such as careful choice of bedding and laneway sprinklers to reduce the impact of dust on eyes
8. Try to maintain cohorts of animals in their lines and minimise mixing of cattle groups which facilitates spread of disease
9. Remove and treat affected animals as soon as they are identified because pinkeye is very contagious.
Treatment includes; immediate removal from population and housing in hospital pens for affected animals; checking for foreign bodies like grass seeds in affected eyes; the use of topical cloxacillin antibiotic ointment in mild cases; long-acting intramuscular antibiotic (oxytetracycline) treatment and anti-inflammatory (meloxicam) treatment for more severe cases; the use of eye patches for severe cases.

Conclusions

While this research initially aimed to prospectively demonstrate the benefits to furnishing young cattle with suitable immunity such that their risk of developing eye disease in pre-export quarantine was diminished, it became apparent that this was not going to be possible. In the four years allocated to experimental work, on three occasions planned experiments were cancelled just days before field work was to take place. In addition, sourcing suitable cohorts of cattle five weeks before transport to quarantine proved an insurmountable travel. In consultation with the LEP programme manager, representatives from the Live Export Research Development Advisory Committee and an experienced epidemiologist, it was agreed that resources and money would be best redirected, given the chance of proving the hypothesis were low.

Practical implications for industry

This report includes a best practice guide. Sadly, it is recognised that it is unlikely that industry will adopt these suggests. This is not a reflection on the willingness or commitment of exporters to improve animal welfare, rather a comment on the nature of the supply chain itself. Decisions ae made quickly in a changing environment governed by the whims of byers and changing market forces. It is highly unlikely that access to cattle on-farm, more than five weeks before receival in quarantine, will be a readily-adopted recommendation, despite this being the best was to provide immune-protection for the animals. Nevertheless, every attempt should be made to reduce the risk factors associated with eye disease, including the strategic use of vaccines.

Future work

It is here argued that the basic principles of controlling eye disease are well understood, as outlined in the literature review. Further experimental work will likely not yield valuable findings. Research should be conducted in the cost of eye disease to the industry. Results of a desktop survey exercise like this that carefully characterise the number and extent of outbreaks of eye disease and the cost to exporters would be useful. At the start of this project it was anecdotal reports of serious disease events that prompted the development of the project and the true significance of this syndrome is still not well understood. Measuring its actual impact would be a useful exercise.

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Oral Anatomy and Dental Charting for Practitioners

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Introduction

The study of oral anatomy helps the veterinarian to accomplish any kind of surgical procedure more quickly and with less damage to tissues, especially in cases of major oral surgery.[1]

This presentation highlights the basic dental/oral anatomical knowledge essential for performing veterinary dentistry in a safe and timely manner. Special attention will be given to landmarks and highly significant anatomy.

The examination of the oral cavity should be performed in the conscious patient followed by a more detailed examination in the anaesthetised patient. A definitive examination can only be performed in the anaesthetised patient and may require intra-oral radiography to completely assess subgingival pathology.

The findings of the examination, any extra diagnostic tests and therapy, should be recorded in the patient's record and ideally on a chart. A dental chart is an easy to read pictorial summary that assists clinicians to rapidly record any dental pathology and treatment. It is also useful for explaining the findings to the client upon discharge of the patient and as a permanent record for future reference and comparison. The dental chart also represents a medico-legal document.

Anatomy of the tooth

Crown. The crown of the tooth is covered by a thin layer of hard protective enamel. It is demarcated from the root at the cemento-enamel junction which is located at the neck of the tooth. It is important to note that the hard enamel layer on the outside of the crown is produced by ameloblasts but only during development in the tooth bud and once eruption occurs no more enamel can be produced. Enamel is 96% inorganic, primarily hydroxyapatite crystals, with the remaining 4% being water and fibrous organic material.

Root. Roots may be single, or multiple. Furcation is the term used to describe where roots diverge. Exposure of the furcation of a tooth due to periodontitis is a poor prognostic indicator. The root is covered by the thin cementum layer.

Dentine. Dentine is produced by odontoblasts lining the pulp cavity. Dentine development continues throughout life making the tooth stronger as it ages. The thickening of the dentine results in the progressive narrowing of the pulp cavity as the animal ages. It is important to remember that the dentine consists of dentinal tubules, these are microscopic tubes (approximately 50 000/mm^2) traversing the dentine from the pulp cavity to the dentino-enamel junction. Sitting within the dentinal tubules are processes extending from the cell body of the odontoblast. Exposure of dentinal tubules to the oral cavity, for example in tooth fracture, can result in death of the tubule's individual odontoblast and subsequent ingress of bacteria into the pulp cavity even though the pulp is not exposed. There are three main types of dentine:

- Primary dentine: i.e. dentine present at tooth eruption
- Secondary dentine: Dentine that develops as the animals ages
- Tertiary or reparative dentine. Produced after trauma to odontoblasts. It is denser, has less tubules and may stain a darker colour.

Dentine is 70% inorganic material, mainly hydroxyapatite, and 30% organic fibrous material and water.

Alveolar bone. The root is embedded into the alveolar bone of the jaw. Radiographically the inner surface of the tooth alveolus appears as a bright white line, the lamina dura.

Anatomy of the periodontium

The periodontium consists of the gingiva, cementum, alveolar bone and the periodontal ligament and represents the tissues that surround and support the tooth. Periodontitis, inflammation and subsequent destruction of these tissues, is the most common oral disease in mature animals.

The gingiva is the keratinized mucosa that covers the alveolar processes of the mandible and maxilla surrounding the neck/cervical aspect of the tooth. It consists of:

- Free gingiva
- Attached gingiva
- Sulcular gingiva
- Junctional epithelium

The attached gingiva is tightly adherent to the underlying connective tissue and alveolar bone. Whereas the free gingiva is the collar of gingiva around the neck of the tooth. The inner aspect of the collar is called the sulcular epithelium and this space between the tooth and the sulcular gingiva is the gingival sulcus. The gingival sulcus should measure less than 3mm depth in the dog and less than 1mm in the cat. At the bottom of the gingival sulcus is the gingival attachment represented by the specialised junctional epithelium and underlying gingival connective tissue. The gingiva is distinguished from the loosely attached, non-keratinised oral mucosa at the muco-gingival line. Determination of gingival inflammation and anatomical integrity is an important diagnostic tool in assessment of periodontal disease.

Directional anatomy

Specific oral directional anatomical terms are used to help describe the position of structures in the mouth.

Mesial – directed towards the midline of the body

Distal – directed away from the midline of the body

Lingual – directed towards the tongue

Labial – directed toward the lips

Buccal – directed toward the cheek

Dental formulae

Dogs and cats are diphyodont, i.e. they have two successive sets of teeth, the deciduous teeth and the permanent teeth

Dog primary teeth	2x (i 3/3 : c 1/1 : p 3/3) = 28
Dog permanent teeth	2x (I 3/3 : C 1/1 : P 4/4 : M2/3) = 42
Cat primary teeth	2x (i 3/3 : c1/1 : p3/2) = 26
Cat permanent teeth	2x (I 3/3 : C 1/1 : P 3/2 : M 1/1) = 30

Eruption times (in weeks)

Teeth	Primary		Permanent	
	Puppy	Kitten	Dog	Cat
Incisors	4-6	3-4	12-16	11-16
Canines	3-5	3-4	12-16	12-20
Premolars	5-6	5-6	16-20	16-20
Molars	-	-	16-24	20-24

Occlusion (from AVDC)

Occlusion is the relationship of the maxillary teeth with the mandibular teeth. Key aspects of normal occlusion include:

- Maxillary incisor teeth are positioned rostral to the corresponding mandibular incisor teeth with the cusps of the mandibular incisors sitting on the cingulum of the maxillary incisor.
- The mandibular canine tooth is situated between the maxillary canine and the opposing 3rd maxillary incisor. The mandibular canine tooth is inclined labially.
- The maxillary premolar teeth do not contact the mandibular premolar teeth.
- The crown cusps of the mandibular premolar teeth are positioned lingually to the maxillary premolar teeth.
- The crown cusps of the mandibular premolar teeth bisect the interdental spaces rostral to the corresponding maxillary premolar teeth.
- The mesial crown cusp of the maxillary fourth premolar tooth is positioned lateral to the space between the mandibular fourth premolar tooth and the mandibular first molar tooth.

Breed related differences in occlusion are common.

Anatomical structures to avoid

There are numerous important structures in the oral cavity or adjacent to the oral cavity that must be avoided when performing oral surgery. These include:

- Incisive papilla
- Sublingual caruncles
- Nasal cavity
- Infraorbital canal and foramen with its neurovascular bundle
- Middle mental foramen
- Molar lingual salivary gland in the cat (within the membranous molar pad)
- Mandibular canal and associated neurovascular tissue
- Lingual nerve
- Eye
- Branch of the major palatine artery between maxillary canine tooth and maxillary incisor 3, lateral nasal artery
- Salivary papillae (parotid, zygomatic) adjacent to maxillary molar 1

Dental charting

Dental charting is part of complete, concise and accurate record keeping. Dental charting means less writing thereby saving time. There are numerous veterinary dental charts available. The Australian Veterinary Dental Society provides basic charts that can be used. Electronic charts are also available.
The most commonly used tooth identification method used is the modified Triadan system whereby each tooth is assigned a three-digit numeral. This provides a quicker way of referring

to each tooth. It also is useful on computer-based records. A useful way to remember the numbers is the rule of 4 and 9 where canines are assigned the number 4 and molar 1 is assigned the number 9.

Information that may be recorded on the dental chart may include:

- Skull type – brachycephalic, mesocephalic, dolichocephalic
- Occlusion
- Tooth abnormalities – persistent deciduous teeth, absent teeth, supernumerary, tooth resorption, caries, damage
- Periodontal health –
    - gingivitis index (severity of gingivitis)
    - plaque index
    - calculus index
    - gingival sulcus/periodontal pockets depth
    - gingival contour – recession, hyperplasia
    - tooth mobility
    - furcation exposure
- Treatments performed

There are standard abbreviations that may be used on the dental charts. The AVDC (American Veterinary Dental College) abbreviations are a useful reference for abbreviations.

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Setting up a Dental Service

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Introduction

Establishing or improving our veterinary dental service offers several advantages, namely better patient care, more satisfied clients, enhanced clinician experience, and an increased practice profitability.

This discussion will focus on dental equipment useful in small animal general practice. Instruments and equipment will be listed, and their uses will be briefly outlined.

Why set up or improve our service?

According to Owen McCafferty, CPA, most general veterinary practices average 2.5-3.5% of gross sales related to dental care, including anaesthesia, IV fluids, extractions, suture, medications, etc. used during dental procedures. He feels that the potential is to have 15-20% of your gross sales be dental-related. Obviously, this makes a huge difference to practice profitability.

What is required?

Dental operatory. Ideally a separate dedicated room should be available, but this is often not feasible. To reduce bacterial contamination from aerosolization the area should be located away from sterile procedures.

Table. A dental table with a grill and drainage is optimal as water coolant from dentistry can me messy. These tables can also be used for other 'dirty' procedures such as enemas or abscess drainage.

Ergonomics and OHS safety. Some dental tables are height adjustable and the clinician might be able to stand to perform the procedure, however with varying heights of staff this may not be possible and sitting may be better. Sitting in a fixed position performing dentistry can be hard on the back, neck and wrists therefore it is important to have an ergonomic seat such as a saddle chair or gas-lift chair with lumbar support.
Eye protection, face masks and gloves are essential. Instruments and drills present operator risk and great care should be taken using them.

Oral examination. Diagnosis and assessment of the oral cavity can be aided by adequate lighting and magnification. A good quality dental examination light either wall mounted or mobile in addition to head mounted light source to help visualise in the oral cavity. LED lights are ideal as they produce little heat, are long lasting, are efficient and can be dimmed without colour shift. Magnification to enable proper assessment of pathology and aid in procedures is often useful, especially in cats. Choosing loupes with the correct magnification (usually 3x) and working distance (usually 38-46 cm) allows for appropriate ergonomics and reduces eye strain and neck/back tension. Choosing the lowest comfortable magnification level is useful as it provides the widest field of view. Typically for general dentistry magnification is 2.5x, 3x, 3.5x. Working distance is the distance from the operator's eyes and the animal's mouth. It is related to the height of the practitioner. i.e. the taller you are the longer the working distance.

Dental machine. This is a vital piece of equipment. The dental machine or cart is usually powered by compressed air and drives high and low speed handpieces, it may also have a suction unit. The dental machine also provides water and/or chlorhexidine to the high-speed handpiece and the scaler. A 3-way or triplex syringe is usually part of the machine. This delivers water (or chlorhexidine solution), air or a combination to produce mist and is useful to irrigate the mouth with chlorhexidine prior to commencing a procedure and to help visualisation by flushing airway blood and debris. There are many different brands of dental machines on the market, but good back-up, advice and service may be worth paying a little more.

High speed handpiece. These handpieces operate at 350 000-500 000 rpm when not under stress. The high-speed handpiece is typically used with burs that are used for cutting teeth, e.g. taper fissure burs, or removing alveolar bone to aid in extractions, e.g. round burs. Most commonly used burs are carbide steel, but diamond burs are available. We dispose of the bur after a single use and bill it as a consumable to the client, thereby ensuring that the bur is always sharp. It is important to use a light touch when using the high-speed handpiece or it will stall due to its low torque.

Low-speed handpiece. This is typically used for polishing teeth with a polishing cup on a low speed contra-angle handpiece, or as a disposable snap-on oscillating prophy angle. Polishing is performed using prophy paste at a speed of 5000 rpm or less.

Scaler. Scalers are used to remove plaque and calculus from the tooth surface. Scalers may be either sonic or ultrasonic. Ultrasonic types are more commonly used in veterinary practice. There are two main types of ultrasonic scalers, 1. Piezoelectric, 2. Magnetostrictive, either with a ferrite rod or a ferromagnetic stack.

They have various advantages and disadvantages. Piezoelectric scalers work by expansion and contraction of crystals in the handle when powered by electricity resulting in vibration of the scaler tip. They tend to produce less heat than the magnetostrictive type. The ferrite rod type scaler is active vibrating over the entire 12mm tip and may be more efficient. Cooling the tip with water spray is essential. Subgingival tips, which are finer to assist in access into narrower spaces, are available for some scalers. A scaler that can be used sub gingivally is useful, as the mainstay of periodontal disease treatment is removal of plaque and calculus from the gingival sulcus.

Intra-oral radiography

Dental radiography allows detection of potentially painful pathology that is not evident on a visual inspection. If you are undertaking small animal dentistry on a regular basis then this is a critical diagnostic tool.

Dental X-ray machines. A specific dental X-ray machine is preferred over a general veterinary X-ray machine as positioning is easier and they are well collimated. Dental X-ray machines have a fixed mA and kV, but have a variable time setting. Many machines have anatomical settings, i.e. the tooth of interest is selected on the controls and settings are automatically calculated.

Dental X-ray machines may be wall mounted on articulated arms, on a wheelable stand or hand-held in a camera type set-up. Government regulations for set-up varies from state to state but suppliers are often very helpful in assisting your set up.

The image capture may be via:

- Conventional X-ray film
- Indirect digital system (CR) – phosphor plate

- Direct digital system (DR)

Most practices will find that digital radiography is advantageous over film for the following reasons:

- Less radiation
- Quicker processing
- Enhancement and magnification of image
- Ease of image storage
- Cheaper to run in long term
- No chemicals
- Greater number of grey shades

Suppliers of the CR systems may have a variety of plate sizes, e.g. size 2, size 4, size 5
Most DR systems are size 2.
CR systems require a scanner to convert the latent image on the phosphor plate into an image on the computer screen. This processing takes a few seconds once the plate is loaded into the scanner. DR systems connect directly to the computer and the image is almost instantly attainable.

Instruments
Most of the dental work in general practice revolves around treating and preventing periodontal disease and extractions. The list of instruments below is a useful starting point for your dental practice.
Instruments used for examination
Dental mirror – this is an under-utilised instrument, it is particularly useful in examining palatal/lingual surfaces of teeth in the caudal mouth. It can also be used to gently push tissue out of the way when performing procedures.
Penlight – used to transilluminate teeth to assess vitality, useful in assessing presence of plaque.
Periodontal probe/Dental explorer – this two-ended instrument has an explorer at one end (e.g. Shepherd's hook) and a periodontal probe at the other. The periodontal probe is probably the single most important instrument for veterinary dentistry. It is used to assess attachment loss to the tooth by measuring the depth of the gingival sulcus or periodontal pocket and gingival recession that may be present. This is useful in assessing the severity of periodontal disease. The dental explorer is used to gently probe sub-gingivally to assess the presence of calculus or lesions such as tooth resorptions.
Instruments used for treating periodontal disease
Scalers. Hand instrument used for removal of supra-gingival calculus and plaque. There are various types including sickle scalers, e.g. Jacquette scaler, H6 scaler.
Curettes. These are scaling instruments for use sub-gingivally or supra-gingivally. They have a round toe so are less likely to damage the gingiva than scalers. There are two major types of curettes - universal and area specific. The Gracey curette is an example of an area specific curette. There are many different sizes as they are designed for parts of different teeth in human dentistry where access may be difficult. Curettes include: Mini 5 Gracey curette 5/6 or 7/8, Columbia curette 4R/4L, Barnhart curette 5/6, Gracey curette 13/14

Instruments used during tooth extraction and oral surgery

- Scalpel handle (size 3) for scalpel blade size 15
- Periosteal elevator for raising mucoperiosteal flap, Molt number 2 (for cats and small dogs), Molt number 4, and Freer periosteal elevator
- Selection of alveolar elevators. Stretches and fatigues the periodontal ligament. May be plain or winged type depending on preference. The winged shape conforms to the root. Sizes from 2mm – 5mm commonly used. Various handle sizes are available to suit the hand size of the operator.
- Coupland chisel. A different type of elevator preferred by some operators.
- Luxators. These are used as a wedge and cut the periodontal ligament. Recommended for more advanced operators. Cislak Ic 1 and 2mm useful for cat teeth.
- Root pick. Useful for retrieval or fractured root apices. Also useful as an elevator in cat's teeth.
- Root tip forceps. Useful to grasping fractured root tips or very small teeth
- Extraction forceps. Large and small sizes required.
- Scissors. Iris scissors or La Grange for cutting gingiva.
- Burs: Taper fissure cross-cut: size 699L, 701L, 701LS, 702. Round: size #2, #4, #6, #8. For cats a size #0.5 and size #1 round bur may also be useful.
- Minnesota retractor. To move vulnerable soft tissue out of harm's way
- Needle drivers. Small sized Olsen-Hegar or Mayo-Hegar are useful.
- Adson-Brown thumb forceps
- Suture material. Monocryl or monosyn 4/0 or 5/0 on a P3 reverse cutting needle is useful.
- Mouth gag. Spring loaded mouth gags are not recommended in cats as they may cause blindness due to compression of the maxillary artery. Cut down syringes may be more appropriate, and their use restricted to short periods. Perm rollers (for hair) may also be used.
- Small bone curette. For debriding alveolus after extraction.
- Arkansas stone sharpener with oil

Other products

Plaque disclosing agent. Used post professional cleaning to assess the effectiveness of plaque removal.

SANOS dental sealant. Applied to dry teeth at the end of cleaning. It acts as seal to the subgingival area preventing formation of plaque.

Orastrip. A test strip used in the consult room to detect the presence of thiols (volatile sulphur products) produced by pathogenic bacteria. Not currently available in Australia

Tricalcium phosphate/hydroxyapatite (Synergy). Biosynthetic bone graft used in large extraction sites to support bone growth.

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AFAST and TFAST Imaging in the emergency setting

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Summary

TFAST: thoracic focussed assessment with sonography for trauma
AFAST: abdominal focussed assessment with sonography for trauma

These tests have increased in popularity in recent years after a number of sentient publications and increasing access to portable ultrasound. In veterinary medicine, the tests are applied commonly to any animal presenting in an emergency situation (including, but not limited to dyspnoea, tachypnoea, pyrexia, abdominal distension, collapse, trauma) and therefore the use of “trauma” in the acronym FAST may not be relevant. In human imaging the tests have evolved in the emergency room and in post-trauma assessment.

TFAST has become an important modality in the early assessment of dyspnoeic patients. It useful in diagnosing pleural effusion and pneumothorax. Its usefulness for interpreting lung pathology is more problematic. It does require some degree of operator skill and experience, but this can be learned. It should not replace thoracic radiography as an imaging modality and is complementary to radiography.

AFAST is a useful tool for the detection of peritoneal effusion in the emergency setting and requires relatively little training. Its use beyond peritoneal fluid assessment is difficult to quantify. Complete sonographic assessment of the peritoneal cavity and organs is challenging and well beyond the scope of AFAST; practitioners are cautioned not to overinterpret other imaging findings. Abdominal radiography provides a globoid assessment of the abdomen (with limitations) but can be shared for interpretation. Similarly, quality complete abdominal sonographic assessment (e.g. by a specialist radiologist or competent practitioner) should be recommended in the diagnostic assessment of abdominal disease.

This presentation will review the methods of performing AFAST and TFAST, including indications, patient positioning, image optimisation and probe selection and provide clinical examples of the benefits and pitfalls of both modalities.

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Diagnosing the dyspnoeic patient

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Summary
Be calm and orderly in your approach to the dyspnoeic patient.
First, do no harm! Supplemental oxygen, sedation, IV access: handle gently and minimally.
Imaging: XR vs US: merits of each will be discussed with clinical examples provided.

Notes
Causes of dyspnoea in dogs and cats include

- Airway diseases: upper airway collapse including brachycephalic syndromes and laryngeal paralysis, dynamic airway disease, bronchitis/allergic airway diseases and foreign bodies. Brachycephalic syndromes and laryngeal paralysis are common and often breed related, the others are less common causes of dyspnoea.
- Airspace diseases: anything that displaces air from the alveolar air sacs. The main categories are
    - Fluid:
        - Modified transudate (congestive heart failure, non cardiogenic pulmonary oedema)
        - Exudate (aspiration or bronchopneumonia)
        - Haemorrhage
    - Cells: neoplasia
- Diseases causing loss of lung volume: including pleural effusion and pneumothorax.

Once your patient is stable consider refining your DDx list based on signalment, breed, history and make a selection of XR or US based on what is available.

Radiography:
A go-to modality, it will provide a global assessment of the pleural space, heart and pulmonary structures. Patients will typically cope well with mild sedation (even with dyspnoea) but flow-by oxygen will be required. Aim for minimising stress with a DV projection to help rule in/out pleural disease, asses the caudal lung lobes and start the assessment of pulmonary lobar vessel size (for evidence of left congestive heart failure). The lateral projections will be required for cardiomegaly assessment. Simple strategies will be presented to help you refine the DDx above based on radiographic appearance.

Radiography shares the benefit of being easily sharable (digitally) for a second opinion. It does not require any specific technical expertise to acquire and should be available in all small animal practices. Interpretation of the study may be difficult with complex cases, and patients can struggle in lateral recumbency.

If even if TFAST gets you the answer, it would be rare that thoracic radiography would not be involved in the work up (e.g. for a more global assessment or to get a better baseline of disease, or to assess response to therapy).

Sonography:
TFAST has become the first imaging modality of choice in emergency practice based on its ability to provide assessment in the treatment room (or the oxygen cage). Place the patient in sternal recumbency on oxygen and apply isopropyl alcohol or gel to the thoracic wall: start ventrally and look for evidence of pleural effusion (anechoic), moving cranially and caudally. Then slide up the thoracic wall to evaluate for air-filled lungs and try to determine whether there is pulmonary pathology present (comet tails, B lines). Finally, TFAST can provide some clues that a pneumothorax is present.

The major benefit is that pleural pathology can be sampled immediately. The major problem is that assessment of the lungs with any detail is not possible, cardiac assessment requires a significant degree of skill and the sample is limited to a small field of view (the transducer width) so global assessment is not possible.

The presentation will provide tips for approaching imaging in both of these scenarios.

Canine Aggression: When dogs don't like anyone (1 and 2)

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There are many topics that can divide people, what to do about dogs that are aggressive is one of these. As veterinarians our job is manage the health of animals and to advocate for animal welfare. To do this well for dogs with aggression problems we need to be aware of our biases about aggression so it does not cloud our assessment and treatment plans for these dogs. Then the steps of gathering a history, observing the patient, investigating any findings and developing a diagnosis and treatment plan occur. Lastly a discussion of the prognosis and risks related to the diagnosis and management is undertaken.

Aggression and Culture

To understand why aggression in dogs is treated so harshly, we have to first understand culturally what aggression means for us before we can consider what the purpose of aggression is for the dogs who use it. Then we can start to consider if the aggressive behaviour expressed by a dog is normal and appropriate, or due to malfunctioning emotion systems. We can then discuss treatment plans opening and without bias so clients can make the best decision for their dog and themselves.

In no other area of veterinary medicine do people's opinions affect whether a patient is even offered the chance of treatment than in behavioural medicine. Would a diabetic dog ever be euthanised without an attempt to discuss causes of the disease and treatment options? Why does this happen to dog that show aggression?

Culturally, we love aggression and we hate it. In some areas aggression by individuals is desirable and lauded and those who show it are rewarded. But at the same time, aggression in other areas of life are considered unforgivable.

There are certain rules that can, consciously and unconsciously, bias our thinking about aggressive behaviour in ways that are not always beneficial for our patients. In our culture, it is not acceptable for an adult to beat up child, for a younger individual to beat up an elderly individual and for bigger to bet up smaller, regardless of the child's, elderly individual or the smaller individual's behaviour.

So how does that fit with our consideration of aggression between dogs or aggression by dogs towards people. Dogs are allowed to be aggressive- in specific situations described by humans. Dogs are allowed to defend the defenseless against bigger or more dangerous enemies. So, for example, a dog is allowed to defend a child against an adult. Dogs are allowed to be aggressive in their role as heroic police dogs or military working dogs. Dog are also allowed to be a little bit aggressive when in great pain- but only to veterinarians and their staff.

So how does it feel when a dog bites a child or bites their owner? Or bites and kills a small white dog belonging to a 72 year old widow? What should happen to that dog?

When we give advice it is important that we are aware of our personal biases so that the advice is scientific and lawful. To understand how to treat these dogs we must first understand how and why dogs use aggressive behaviour.

Normal aggression

Aggression is part of the normal behaviour of dogs. Just like getting angry and expressing frustration with shouting and waving our hands around is within normal limits of behaviour for people, growling and barking and even lunging and biting are completely normal for dogs to do- sometimes. Aggressive behaviour may be motivated by fear, anxiety, frustration and anger. It is generally associated with negative emotional states. When an animal is in a negative emotional state, they may be more inclined to react aggressively.

The way we decide if aggressive behaviour is a normal response or an abnormal response is to look at three factors

1. The *Context* in which the aggression was used. If the answer to our question "was aggressive behaviour an appropriate response in this situation?" is yes then we might just have normal behaviour.
2. The *Intensity* of the aggressive behaviour is also considered. If the dog used a growl to stop unwanted attention, this could be considered normal. If the dog lunged up and bit someone for stopping and talking in a friendly manner, this intensity is not appropriate.
3. The *Frequency* of aggressive behaviour is also considered. If the dog is aggressive nearly all the time in a situation that does not warrant it then it is probably not a mentally healthy animal.

Predatory behaviour- not an aggression

Some texts include predatory behaviour as an aggressive behaviour. It is not part of aggressive behaviour because the aim is to cause death to acquire food- not to move perceived threats away. Predatory behaviour is very different from aggressive behaviour. Predatory behaviour is silent with a stalking component. The attack is swift without any warning and the aim is to be lethal.

In contrast aggressive behaviour often has a relatively orderly progression through threats to violence. Threat behaviour is performed so the other party can see it and the aim is to use threats to avoid having to use violence.

Dogs may use Aggressive Behaviour Normally

Dogs may be aggressive in many situations as a normal and appropriate response to their environment and social interactions.

Dogs evolved as scavenging, hunting species. They eat a wide range of foods from fresh prey, eggs, carrion, vegetables and plant matter. This habit of eating carrion and other found objects means several dogs would gather around a carcass or other source of food. Wherever there are resources in restricted supply, a system has to be worked out to minimise injury to parties wanting the resource. As a species related to the Great Apes, we tend to feel comfortable with a hierarchy but not always (think about the post-Christmas Sales- first come, first served scrum). Dogs, in contrast, to what is written about them by humans, do not work on a hierarchy. Dogs manage access to resources on the idea of "if I can hold onto it, it is mine" or Resource Holding Potential.

Resource Holding Potential describes weighing up of different factors that decide if a dog will fight to hold onto a resource. The factors involved include physical characteristics such as weight, age, size and motivators such as how hungry/thirsty/tired the dog is, previous experience in general and with the challenger, and personality characteristics such as their tenacity. Other strategies that may be used to obtain access to a resource include feigning indifference and sneaking in to steal it or to standing close to and barking at other dog until they get frustrated and leave (or frustrated owners intervene).

It is within normal limits of dog behaviour for a dog to growl when approached while eating food, bones or other treats. It is also normal for a dog to react aggressively to someone playing with the dog's food as it attempts to eat. It is within normal limits of behaviour for a dog to growl when approached on a bed, couch or other resting place. Some dogs will show growling, teeth flashes, muzzle punches and air snaps towards other dogs when playing with toys. Many dogs will show some sort of protective behaviour around their owners or other valued people when there are several dogs or people around. This may take the form of positioning themselves close to the owner, between the owner and others, staring at approaching dogs or people or even growling.

What becomes frustrating is that these normal behaviours are seen as inappropriate and the dogs needs to be "gotten out" of performing them. But they are normal. The result is that we are pushing against instinct. If the dog is resilient enough, it learns to adapt. But many dogs with high normal anxiety or anxiety disorders have endured extra stress due to well-meaning dog owners doing what they are told they must to prevent problems.

Advice for Owners about Dogs and Aggressive Behaviour

- Growling is a request by the dog for more space. It is not a sign that the dog is turning bad or is vicious or a threat to the children.

Food and Feeding

- Let the dog eat. Cut out the tricks, cut out handling the food, cut out putting treats in the bowl. Just let the dog eat.
- If you don't want to eat it, don't take it from the dog. If it something the dog should not eat, offer something better so it will leave the other item alone.
- If there is a problem with people or children being near the food bowl, move the bowl and dog so they eat in a quiet, out of the way place for feeding.
- Teach children to respect the dog's food and treats.
- If food is very valuable to the dog and they tend to want to defend it, feed more frequently (and possibly a lower calorie food). Feeding once a day makes food incredibly valuable and may heighten stress for the dog.
- If there is more than one dog in the household, feed them in separate areas if there are issues around the food bowls and feeding time.

Resting place

- Everyone, including the dog, needs a place where they can rest without being bothered. Encourage all dog owners to provide this space.
- If the dog is in its resting area, let it rest. Don't pat, cuddle, or otherwise bother the dog.
- If the dog growls when moved off furniture, there are two choices. The dog is not allowed on the problem furniture ever. A place for it to rest needs to be provided. Or the dog can be trained to move off when asked. This is done with treats or other activities that the dog likes to encourage it to get up.

Play

- Normal Dog play is accompanied with growling and biting. The difference is that the dogs inhibit their bites and body slams to minimise hurting their play partner(s).

Fear

- Just like humans, some dogs show aggressive behaviour when frightened. They may growl, snarl or lunge and bite if feeling threatened. This is an appropriate response in some situations. If the dog growls, we all need to stop, pull back a bit and reassess the situation. This goes for at home, in public and in the veterinary clinic.
- It may not be the human's intention to be frightening, but if the dog perceives the behaviour of the human as threatening, then it is right and the human needs to pull back.

Abnormal Aggressive Behaviour

As we have seen aggressive behaviour is used to ask for or demand space. It can be motivated by several emotions including fear and anxiety. These last two emotions are very important. Aggressive behaviour may be a strategy that an animal experiencing abnormally high levels of anxiety and fear uses to try and improve its emotional state.

The most common mental health disorders seen in VBM practice are anxiety and fears. One of the signs of an anxiety disorder may be aggressive behaviour. Increased irritability may also be reported by owners. Many dogs with anxiety disorders are aggressive and many will use aggressive behaviour deliberately and confidently to control their environment. They do this to help relieve the discomfort of feeling anxious and fearful.

Anxiety and fear are uncomfortable emotions. If the systems in the brain that produce the changes in the body and brain that cause these uncomfortable sensations are overactive or under regulated then the affected individual feels physically uncomfortable and emotionally distressed in *NORMAL* situations. Aggressive behaviour may be directed to unfamiliar individuals (dogs and/or humans) to create a safe space around the individual. Other dogs manage their anxiety about being handled or change in their environment by using aggressive behaviour to control other pets or their owners' behaviour.

Aggression may also be used when an animal feels frustrated. Sometimes these patients have impulse control problems or problems regulating their emotional state and therefore controlling their behaviour. Aggressive behaviour may also be a sequela to other mental health disorders such as separation anxiety or Obsessive Compulsive Disorder (OCD).

Diagnosis of the type of aggression occurs through collecting a detailed history of recent and historical incidents. These are examined for 'triggers'- events or individuals that reliable cause aggressive behaviour. Investigating the outcomes of attempts to manage the situation can also help with identifying the types of aggression.

Treatment

Broadly the treatment is the same, regardless of the condition diagnosed but the devil is in the detail. The detail will vary with the individual dog, owner, family situation and routines.

Environmental Management-

Safety for everyone, support and reassurance of the patient is needed. Luckily, providing a safe, supportive environment for the patient tends to keep everyone safe too.

Avoid triggers- the dog has no appropriate coping strategies so continual exposure is not helpful. Separation (from other dogs, from children) at home may be needed.

Let the dog dictate. This is a big change in thinking but very important if a dog is aggressive within a family. Many times aggressive incidents occur because the human does not respect the dog's requests or need.

Modify Emotional State-

This is the behavioural management but really the behaviour is secondary to the emotional state. If the animal feels safe and calm then it is less likely to be aggressive. So exercises emphasising calm behaviour, that practice moving into and out of arousal levels and maintain control are all helpful.

And we do not test the dog.

Training for happy obedience is encouraged, as are trick training, scent work especially if a dog cannot be exercised in public.

Medication

Typically an anxiolytic medication such as Fluoxetine or Clomipramine is used with or without a situational medication such as Clonidine, Trazodone, Mirtazapine, or Gabapentin is used to help stabilise and rebalance the neurochemistry. Zylkene is often very useful. Adaptil collars or plug ins may also help.

Euthanasia

Euthanasia should be approached the same as in any veterinary case involving serious ill health. It is a valid treatment if the animal's welfare is at risk. The decision to euthanise for reasons other than the animal's welfare (safety concerns) is a decision for owners to make- for themselves and for their family and for their pet. It is not a decision that veterinarians should make for owners (check your biases at the door!).

Aggressive dogs and children can live together safely. Children learn that dogs have feelings and they need respect. Not a bad thing for them to learn. Safety and management need special attention and owners must be made aware of the need for runs, barriers, child proof barriers, gates and door and locks. If these things are used then aggressive dogs can stay in families safely. This is a decision for the owners to make- not for veterinarians to make for them (again- check your biases at the door).

Aggressive behaviour by dogs may be normal or it may be a sign of a mental health disorder. Treatment and management is possible and many affected dogs are great companions once treated. It is imperative in anxious dog cases that we are aware of our biases about aggressive behaviour in general, and aggressive behaviour by dogs in particular, so that these do not impinge on the diagnosis of the cause and subsequent treatment and management plans.

Trauma And its Effect on the Brain

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Many veterinary patients experience some level of trauma throughout their lives. They may be the victim of accidents such as road traffic incidents, falls or attacks by other animals and some may experience traumas as abuse from people. The perception of events as traumatic is individual and a failure to process traumatic events can have far reaching events for the individual. How we manage our patients after accidents and other traumas is important for that patient's full recovery.

Traumatic events are defined as extraordinary events that overwhelm an individual's normal adaptions to life. Events may be experienced directly as violence to self or to important others such as caregivers. In humans Secondary Traumatic Distress is recognised when an individual suffers emotional difficulties after hearing or seeing violence done to others. It is possible that this may be an issue for animals, but it has not been researched to the authors knowledge.

In contrast, Post Traumatic Distress Syndrome (PTSD) has been diagnosed in animals. PTSD is a debilitating condition that some people develop after experiencing traumatic events. The symptoms and signs of PTSD in humans can include re-experiencing traumatic events, avoiding reminders of the trauma and hypervigilance. It may also be co-morbid with depression.
PTSD has been diagnosed in chimpanzees captured in the wild, laboratory chimpanzees, wild caught parrots and elephants after culling events [1-3] and anecdotally in military dogs working in war zones and other animals in war zones. These animals showed withdrawal, tension, hypervigilance, problems socially, reduction of play, reluctance to explore and self-soothing and comfort seeking behaviour.

Trauma can affect the brain at any time along the life of an individual. Importantly, the perception of what is traumatic is very individual and is affected by things such as personality and pre-existing mental health. The brain processes a traumatic event by working back through and comparing it with past and new experiences in the same situation. Through this, the memory of the trauma is reduced and linked to a time and a place. But some brains do not seem able to do this reorganising of the memories. At present we do not have one test that can categorise an individual as being at greater risk of PTSD. However, differences have been identified in the brains of individual humans with PTSD compared to humans who do not have the diagnosis.

Humans with PTSD tend to have:

- Less grey matter in their brain - fewer cell bodies, dendrites, astrocytes, capillaries
- Smaller brain volumes - fewer cells and fewer connections, less complexity. Higher
- Smaller Hippocampal volumes - Organisation of memory, with the medial frontal cortex manages the Amygdala
- Smaller Corpus Callosum - Integration of both hemispheres of the brain
- Poorly regulated Amygdala - hyperactive responses to trauma related stimuli[4]

It has also been found that higher levels of intelligence appear to be protective against PTSD. [5] What is not known is if these differences are due to the Trauma itself or due to the genetics or early life experiences. Four factors have been found to be important to the healthy development of the brain. These are genetics, environment, health of the dam and caregiving experienced by the young brain.

Genetics
The genes are the building plans for the brain. Mentally healthy parents have a better chance of producing mentally healthy offspring.

Maternal Health
The physical and emotional health of the mother while pregnant will affect the emotional development of the foetus she carries. This has been shown in human and animal studies and the effects may run through to the grandchild generation

Caregiving
The caregiving an individual receives has effects on their ability to resilience. Resilience and Sense of Coherence are important protections against PTSD. A sense of Coherence is defined as “the extent to which one has a pervasive, enduring, though dynamic, feeling of confidence that one's environment is predictable and that things will work out as well as can reasonably be expected.” It is a mix of optimism and a sense of control or predictability of the world.[6]

Resilience is governed in part by personal characteristics of the individual but the caregiving the individual experiences can be protective against the effects of trauma. Reliable, good caregiving helps the individual develop a sense of coherence and resilience.

Environment
The environment of the young individual experiences is also very important for developing a healthy brain that has some resistance to trauma. Complex, enriched environments result in individuals with brains that are dense and complex with lots of connections.

As Veterinarians there are many things we can do to help our patients, especially those coming to us from less than ideal situations. The aim is to try and trauma proof young animals as much as possible. With the growing trend to buy puppies over the internet without seeing where they were raised or meeting their parents or relatives, it is possible that we will see a rise in puppy farmed dogs that are more susceptible to the effects of trauma. There are many things that we can be doing, even in just everyday practice to help our patients.

Managing Pregnant mothers and managing neonates

- Help her take good care of her babies. This may mean the obvious such as good food, it may mean minimal handling to minimise her stress.
- Species specific type of care for orphans maybe more important than we think. Native animal carers have learned to provide this, but we may need to provide this for kittens and puppies. For example, avoid leaving puppies or kittens alone for long periods if they are not at that level of development.
- Use pain relief in any and all procedures that may be painful. Use anti-inflammatory/pain relief if inflammation is present. There are times in brain development where the brain is very sensitive to things such as pain- especially in the neonatal period [7]

For older puppies and kittens

- Encourage owners to provide an inviting environment that is supportive of their pet’s development. Puppy class and Kitten classes, if done well, help show owners how to support their puppies and kittens as they explore. The caregiver is a source of support, encouragement and reassurance rather than being a punitive figure. Adaptil collars can also be used effectively.
- Veterinary Visits for puppies and kittens must avoid being traumatic. This can be done utilising Fear free handling and low stress handling methods to avoid upsetting the animal. Positive reinforcement for exploration and just providing treats after something painful can help keep them focussed on the good things.

- If a painful or frightening procedure needs to be done, then consider sedation or anxiolytic medication given before the consultation. Pain relief is needed- especially as part of caring for them after accident or illness.

A special note about Puppy farm pups
These puppies need very good and very calm care. They may have been born to a stressed mother in a poor environment. The result is that the real world is overwhelming for them- their brains have not developed the complexity needed to process all the information and stimulation. Some of these pups may be resilient enough to adapt but many will not. Their owners need to understand this and the animals probably need to be under the care of a veterinary psychiatrist as they may require medication from a young age.

Managing Adult Animals with PTSD
If you are suspicious that a patient has PTSD then they need referral to a veterinary psychiatrist. PTSD is a psychiatric condition and needs proper diagnosis and treatment
The Treatment Plan can be summarised under the headings of:

Environmental Management
Medication
Behaviour Modification

Environmental Management: the aim of environmental management is to provide a rest for the patient and to calm down sensitised pathways. We can also use this to help build trust between the pet and the owner. Reassurance and support from the owner can be important to helping these patients calm.

Medications are needed and may include SSRIs, TCAs, Alpha 2 agonists, SARIs and Benzodiazepines. We can also use Pheromones and nutraceuticals such as Zylkene.

Behaviour Modification is also part of the treatment plan. It can be started at any point in the treatment but cannot advance until the brain is calm and settled. In people, trauma related exposure (TRE), is used very successfully. Through controlled re-experiencing of the events (listening to a bland recording of the event for example) there is physiological habituation. New memories and assessment of old memories is also helpful but requires trust and self-awareness.

While animals do not appear to have the level of self-awareness to let them consider their previous responses, we can help them through very careful desensitisation and counterconditioning utilising a relax on cue exercise around reminders of the trauma. Just like in people, this requires a lengthy period of preparatory work. It cannot progress until the patient is ready.

However, for some patients, this will not be in their best interests and we should always discuss with the owner if getting this animal out into the 'real' world is in their best interests or if their life is good without these exposures.

To Summarise
Trauma has long lasting effects on the brain and subsequent behaviour of affected individuals. Animals affected by trauma need more than treatment of physical wounds, psychological damage needs to be addressed especially as it is not possible to identify if a patient will suffer long term, debilitating effects from the trauma. Animals of different ages may need different strategies. For example, young animals can be encouraged to develop resilience while older patients may more structured treatments.

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You Know You Want It-Medication!

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“I don’t know what to use.” “What can I use?” “What works fast as Fluoxetine takes so long to work!” “Fluoxetine didn’t work, what can I use?” “Nothing has worked immediately, what else should I use?” Psychotropic medications are a fascinating group of medications that can be the key to helping a pet with mental illness back to normalcy. But not many veterinarians are comfortable with the actions, side effects, indications and contraindications for these medications.

My aim is to take you through the medications that you have on your shelves. Nothing fancy but the ones that most clinics are going to have some. Let’s have a close look at the classes, some of the members, how they work and the indications for their use.

Classifying Psychotropic Medications

Medications to treat psychological problems may be grouped by their structure (TCA, Gabapentin) or more commonly by their point or mode of action. You may see acronyms such as SSRI, SARI and NaSSA. These are a short hand way of describing the action of a medication. The table below shows some of the acronyms.

TABLE OF ACRONYMS

Acronym	Translation	Examples
NaSSA	Noradrenergic specific Serotonergic Antidepressant	Mirtazapine
SSRI	Selective Serotonin Reuptake Inhibitor	Fluoxetine
SARI	Serotonin Agonist and Reuptake Inhibitor	Trazodone
TCA	Tricyclic Antidepressant	Clomipramine
Other useful acronyms to know		
NA/NE	Noradrenaline	Neurotransmitter.
5-HT	Serotonin	Neurotransmitter. The subscript numbers and letters refer to subclasses of receptor.
NAT	Noradrenaline Transporter Protein	The protein that collects Noradrenaline from the synapse and takes it back into the neurons for reuse.
SERT	Serotonin Transporter Protein	The protein that collects Serotonin from the synapse and takes it back into the neurons for reuse.
CYP	Cytochrome P450 Enzymes	Hepatic Enzymes responsible for much of the medication metabolism in the liver.

We can also describe medication by how we plan to use them for our clients. A common combination used is a baseline anxiolytic medication and a second medication used as needed to help a patient manage an unavoidable and stressful situation (situational anxiolytic). We can also use medications to add to the effect of another medication- the added one is an adjunct medication. One use for psychotropic medication that is gaining popularity is to give a fast acting, anxiolytic medication before veterinary visits to animals that find the veterinary visit very stressful. We also have nutraceuticals and pheromone products that can used in conjunction with the more traditional medications.

A word about Cats

Cats, in general, have lower doses of medication compared with dogs and with humans. It is well understood that they have differences in their type and number of enzymes for the metabolism of many medications.[1] A lack of glucuronidation enzymes is the most described in the literature and most known.[1] Many psychotropic medications are metabolised slowly in cats. For example Clomipramine is bio transformed into its metabolites more slowly by cats compared with than dogs and rats.[2] Therefore, doses may need to be much lower and spaced further apart in cats compared with dogs.

The Medications

Nutraceuticals

Alpha Casozepine

This is a psychoactive peptide made by hydrolysing the Casein protein of cow's milk. It binds to the GABA receptor where Valium, alcohol, and barbiturates all bind. Alpha-casozepine's binding affinity is much weaker which is why we get a gentle calming rather than heavy sedation, anaesthesia or disinhibition. Alpha Casozepine has no identified side effects and is highly palatable. It is available in Australia formulated for dogs and cats as Zylkene.

It can be used daily as an adjunct or it can be used situationally. Some animals respond just using it on the day others have a better response if it is started a few days before the stressful event. Some animals may need a higher dose so can have 1.5 or even 2 times the dose listed.

Zylkene is not a registered veterinary product so can be bought over the counter without a prescription.

Uses

- Reducing anxiety in dogs and cats (and horses)
- Adjunct to conventional medications
- Situational Anxiolytic

Pheromones

Pheromones are small chemicals released from one individual that are detected by others of the same species and that cause changes in the receiver's behaviour and physiology. There are two types commercially available for small animals in Australia. Dog Appeasing Pheromone (DAP) is available for dogs as Adaptil. It is a synthetic copy of the pheromone released from the mammary gland area of the bitch soon after she has given birth. It is thought that this pheromone causes calming and relaxation in the neonate and helps them bond to the mother. Some adult dogs also show a calming response when exposed to DAP.

It is available as a spray, room diffuser and collar. There is some anecdotal evidence that the collar may have better efficacy than the room diffuser. Owners need to be told that the Adaptil collar must be fitted firmly (1 finger under the collar) to allow the body heat to diffuse the pheromone. Also, it cannot be covered by other collars. I suggest that people put the collar on so it sits high on the dog's neck.

When using the diffuser, pay attention to the volume that the diffuser covers. Large areas may need more than one, rooms that open to the outdoors are not suitable and areas with unique architecture such as voids may also not be suitable for the diffuser. As it needs to be down low, I am cautious about using them in rooms where babies and toddlers roam. Lastly, it MUST be left on. It takes up to 24 hours for it to diffuse through a room. So, turning it on and off in areas where the dog is spending time regularly is false economy.

Specific Conditions with Labelling

- Fear of noises, storms, visitors, being home alone

Uses

- Used alone to help calm mild anxiety cases
- Used as an extra treatment with anxiety cases.
- Help puppies settle into their new homes and keep them calm while navigating the world.[3,4]

Feliway is a different type of pheromone. It is a synthetic analogue of the Feline Facial Pheromone (FFP). The message it seems to give cats is "this is my territory" and it helps them relax. A word of warning though, some highly territorial cats may react to the Feliway diffuser as if another cat has entered and marked their territory and spray on the diffuser.

Specific conditions with Labelling

- Spraying and Urine marking
- Urine marking
- Scratching
- Calming for anxious cats

Uses

- Sole products in very mild anxiety cases
- In combination with conventional medications.

Tricyclic Antidepressants (TCA)

These are older medications grouped by their chemical structure. Clomipramine is one such medication and is widely available in veterinary medicine, with 2 products labelled for use in companion animals. Clomicalm is the original product and many of you may be using Clomav.

Clomipramine is a SNRI, that is a Serotonin and Noradrenaline reuptake inhibitor. Its main effects are through its action on the SERT protein resulting in an increase in serotonin in the synaptic cleft. It also increases noradrenaline concentrations in the synaptic cleft by blocking the action of the NAT which also seems to help in reducing anxiety.

Clomipramine is the most serotonergic of the TCAs. This means that it has the effect of increasing the amount of serotonin available in the synapse. Clomipramine also has anti-adrenergic, anti-histamine, anti-serotonergic, anti-dopaminergic and anti-cholinergic activities through its actions with the different receptors. This is the cause of some of the unwanted effects seen with Clomipramine such as dry mouth, constipation and sleepiness. In humans, it is not used as frequently because of the side effects. This should not be taken that it is not an effective medication and it does have some indications where it is the medication of choice.

Clomipramine in cats can prevent voluntary urination by preventing the detrusor muscle from contracting. This is due to the anticholinergic effects of clomipramine. [5] Many cats adapt but it is something that owners need to be told to watch for when they start their cat on medication. Stopping the medication and supporting the cat until the bladder functions voluntarily is needed if a cat has this effect of the medication.

It takes time to be certain that Clomipramine is not working for a patient. For many it starts to work within a few days but for some it may take as long as a month or more. Therefore, give it 6-8 weeks to be certain it has had every chance to start showing an effect. Clomipramine is metabolised by the liver and kidneys so ideally these should be screened with a blood test before the medication is started. However, if a patient struggles with visits to the vets and is otherwise well, it may be best to post-pone blood tests until the animal is calmer.

Specific conditions labelled

- Separation Anxiety
- Urine Spraying and Urine Marking in cats

Uses-

- Anxiety disorders especially Generalised Anxiety Disorder, Panic disorder, Separation Anxiety, Phobias, aggression related to anxiety
- Obsessive-Compulsive Disorder- medication of choice.

Selective Serotonin Reuptake Inhibitors

This family of medications is used more frequently for treating anxiety as it is much more targeted to the SERT. SSRI's also have fewer side effects so are generally easier to take. Fluoxetine, under the brand name Prozac, is one of the most widely known SSRIs. Reconcile was a veterinary product for dogs but is now off the market.

Fluoxetine stays in the body a long time. It has a $t_{1/2}$ of 6.4 hours in dogs but two of its metabolites, norfluoxetine and desmethylfluoxetine act as serotonin re-uptake inhibitors, so increase the duration of action of the medication. Norfluoxetine has a $t_{1/2}$ life of 42 hours in dogs.[6] The practical side of this is that it takes a long time for Fluoxetine to completely leave the body of a patient. When changing from Fluoxetine to a new medication, wean the patient off the fluoxetine slowly once they have been on it for more than 2 months.

Side effects on starting Fluoxetine are uncommon. The two most frequently seen are Calm/Lethargy/Depression and Loss of appetite (and subsequent weight loss).[6] All adverse side effects resolved when the fluoxetine was stopped. [6] Side effect studies for cats have not been carried out in detail although personal experience has been that Fluoxetine can cause an inability to voluntarily urinate in cats. This is probably due to a centrally acting effect of serotonin. Owners need to be told to monitor urination in their cats when starting fluoxetine treatment.

Uses;

- Anxiety disorders- generalised anxiety disorders, aggressive presentations, separation anxiety, phobias
- OCD

Alpha 2 agonists

The alpha 2 agonist medications were originally marketed as treatments for hypertension due to their effect on alpha 2 receptors in the brain stem. Clonidine is one that is frequently used in veterinary medicine. Clonidine reduces blood pressure by binding to the autoreceptors on the presynaptic neuron and reducing Noradrenaline release. Its use in people has spread beyond hypertension and it is used in human medicine for treating a wide range of conditions from Tourette's syndrome and ADHD and hot flushes and cluster headaches.

In veterinary medicine Clonidine has found use as a situational anxiolytic. It is quick acting so can be given before an expected or planned event known to be stressful for the animal. Owners will need to trial the medication in their pet to find out how long before the event to give the medication. A good rule of thumb is 30-90minutes, but it may be more. Also, the dose range is quite wide so testing the dose before the event is a good idea.

Side effects most commonly reported by human users include hypotension, dizziness, stiffness, constipation and dry mouth. In veterinary behavioural medicine, side effects are few but probably include dry mouth, constipation and stiffness. Be aware that Clonidine can potentially cause problems with urinary retention through activation of the internal sphincter of the urethra.[5] Alone it may not be a problem but if combining medications, it is something of which to be cognisant.

A final point for Clonidine's use as a pre-visit anxiolytic. If sedation or anaesthesia with Medetomidine is planned, then Clonidine should NOT be used as a pre-visit anxiolytic due to both medications having an adverse effect on blood pressure. Suitable substitutes for a pre-visit anxiolytic may be gabapentin, trazodone, midazolam or mirtazapine.

Uses

- Situational anxiolysis
- Adjunctive medication for Generalised Anxiety Disorder
- Reactivity

Gabapentin

Gabapentin is an interesting medication used for several neurologic and psychological conditions. Its exact mode of action is not fully understood. However it has been shown to enhance GABA synthesis, slow down GABA catabolism, inhibit Glutamate synthesis and inhibit monoaminergic transmission.[7] To translate; GABA is a major inhibitory neurotransmitter of the brain, Glutamate is a major excitatory amino acid neurotransmitter. Monoamines are Dopamine, Serotonin and Noradrenaline. Thus, the result of giving Gabapentin is that the brain is calmer and harder to excite.

Gabapentin is used to manage epilepsy, neuropathic pain, movement disorders, migraines, anxiety disorders, bipolar disorders and substance abuse and withdrawal in humans and has few drug interactions or side effects.[7] The use in animals is similar.

Side effects include sleepiness, sedation, ataxia and dry mouth. Overall is it well tolerated.

Uses

- Neuropathic pain (may present as severe self-mutilation)
- Adjunct medication for anxiety disorders
- Pre-visit anxiolytic (especially good for cats)[8]

Mirtazapine

Mirtazapine is used mainly as an appetite stimulant in veterinary medicine, but it has broader potential effects for helping manage anxiety.

Mirtazapine is classed as a Noradrenergic and Specific Serotonergic Antidepressant (NaSSA) and an atypical antidepressant.[9] It is a strong antagonist of serotonin receptors 2 and 3 (5HT2, 5-HT3) and central alpha 2 adrenergic receptors. The Serotonin 1A presynaptic autoreceptors increase their activity due to the blockade of 5-HT2 and 3 receptors so give Mirtazapine even better selectivity for serotonin transmission than the SSRI medications. Through blocking the alpha 2 adrenergic receptors, there is greater release of serotonin and noradrenaline at the axon terminals. Mirtazapine also acts on the Histamine 1 receptors. It does not have any effect on Dopamine receptors or dopamine or serotonin reuptake.

It has a great profile for helping relieve anxiety, depression, sleep disorders, chronic pain conditions, and appetite stimulation in oncology cases in people.[9] However, its big drawback, in humans, is that it increases appetite leading to weight gain as well as causing somnolence and sleepiness. In veterinary patients, it is helpful for appetite stimulation in hospital and could be considered if applicable for oncology cases. Its main use is as a second or third tier anxiolytic medication. There is also a use for it as a situational anxiolytic and a pre-veterinary visit anxiolytic medication.

It is metabolised by in the liver and mostly excreted in the urine. Cats have a tiny dose or are dosed every 72hours. Care is also needed with very small cats (and dogs) and elderly cats. Renal or hepatic disease may increase the clearance by 50% in humans. This is likely to be applicable to dogs. As the tablets are small, it can be hard to section them to suitable doses with accuracy.

Trazodone

Trazodone is a tricky medication because of its status in Australia. We do not have a commercially available product. All trazodone must be compounded by compounding pharmacies on the receipt of a prescription for a specific animal for whom the veterinarian has a bona fide client-patient-veterinarian relationship. This makes having it on the shelf to give a few doses to trial and use for pre-visit anxiolytics illegal.

Putting Trazodone's status in Australia aside, it is a useful medication for helping relieve anxiety. It can be used as a situational anxiolytic and as an adjunct to Fluoxetine or Clomipramine. Trazodone is a SARI- a serotonin antagonist and reuptake inhibitor. It has a unique structure and complex pharmacological profile.[10]

Choosing what medication or combination of medication to use can be difficult- especially as there are few hard and fast rules. In this field, we start with our best choice considering the patient's signalment, their diagnosis and their individual presentation and history, the owner's situation, routine and financial constraints. Then we wait to see the effect.

A psychotropic medication should be life enhancing for the patient. Side effects are not tolerated so it may take a little while to find the right combination of a patient. However, the desired effect may take much longer to come about so we don't change just because it is not working quickly enough. Also, we don't necessarily need something to work immediately. For example, it is not always necessary to use something like trazodone when starting fluoxetine "because" the fluoxetine takes a long time to work. The trazodone should be used if its effects are needed to help the patient manage. Sometimes the patients just need some patience, quiet, empathy and reassurance.

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Acupuncture and Rehabilitation for Acute Polyradiculoneuritis (Coonhound Paralysis)
Useful tips and tricks

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Introduction

Acute polyradiculoneuritis (APN) is an immune-mediated lower motor neuron disorder[1] that results in flaccid tetraparesis or tetraplegia and, in some cases will also affect cranial nerves and respiratory function.

APN is a condition that has increased in incidence in Australia in recent years. Previously, it was more commonly referred to as Coonhound paralysis[1]. In the past it was thought that raccoons in North America were responsible for transmitting the disease to dogs[1]. There is no specific, recognized treatment to date for APN. Conventionally treatment for APN is restricted to supportive care and physical therapy[2]. The recovery period has been quoted as several weeks to months[3].

Useful acupuncture points and Traditional Chinese medicine patterns will be discussed in the treatment of APN. A lack of research in the area does not preclude their effectiveness. Related research looking at other lower motor neuron disease and acupuncture, moxibustion and herbal supplements have been conducted[4]. The recovery period is greatly enhanced when biomechanical imbalances are also corrected. Various rehabilitation techniques appropriate to the severity of the paralysis/ paresis, weakness will be discussed.

Chinese medicine pattern and acupuncture points selection

Since 2006, canine patients have been referred for suspected Acute Polyradiculoneuritis. All dogs were originally diagnosed by their initial or specialist veterinarian with lower motor neuron disease and peripheral neuropathies. Although most of their clinical signs were variations of flaccid tetraparalysis or paresis, the Chinese medicine patterns were varied and this enabled a more accurate acupuncture point selection for each patient at each consultation session.

Patterns found were based on these parameters:

1. Is there weakness/ deficiency
2. Is there excess/ reactivity
3. Is there stagnation/ blockage
4. Is there imbalance between upper(chest), middle (abdomen) and lower (pelvic) jiao/ regions (cranio-caudal)
5. Is there imbalance left and right
6. Is there imbalance top to bottom (dorso-ventral)
7. Which layer of the Shang Han Lun or Wen Re Lun is the immune response trapped in?

Acupuncture

1. Qi deficiency
   a. moxibustion, electrostimulator (+ variation 'jump starting'), piezo stimulator, laser
   b. Local points
   c. Command points – especially deficiency in extremities
   d. Governor vessel (channel) deficient points

2. Imbalance
   a. piezo stimulator, needles, laser
   b. Bladder channel reactive points
   c. proximal and distal points
   d. balance technique
   e. Eight Extraordinary Channels

Eight Extraordinary Channels

Extraordinary channels coordinate and regulate the Qi and Blood within the 12 regular channels they link. The opening and closing points (Confluent Points) of these extraordinary channels are located around and distal to the carpus and tarsus and used to treat disorders of the corresponding extraordinary channel.[3]

Channel	*Location*	*Confluent points*	*Moves Qi*
Dumai Governor Vessel (GV)	Dorsal midline	SI3, BL62	Back to front
Renmai Conception Vessel (CV)	Ventral midline	LU7, KID6	Front to back
Yangweimai Yang Linking Vessel	Lateral stifle, shoulder	TH5, GB41	Up to down
Yinweimai Yin Linking Vessel	Medial hind limb, neck	HC6, SP4	Down to up
Chongmai* Penetrating or thoroughfare vessel	Parallel to Kidney channel	SP4, HC6	Internal to external
Daimai* Girdle or Belt Vessel	Encircling the Hypochondriac region Shaoyang syndrome	GB41, TH5	External to internal

Channel	*Location*	*Confluent points*	*Moves Qi*
Yangqiaomai Yang Hand Vessel	Lateral hind limb extremities, shoulder, head	BL62, SI3	Left side to right
Yinqiaomai Yin Heel Vessel	Medial hind limb extremities, eye	KI6, LU7	Right side to left

*Acupuncture points along the Dai Mai have been found to insert into the internal and external obliques.[5] Tension in these muscles would promote a kyphotic posture. Another Extraordinary meridian the Chong Mai which links the internal to the external has recently been found to have parallels with the vascular system, especially the vena cava. [6]

Biomechanical Dysfunction

- Compensatory fascial tightness - Corrected with osteopathic releases to improve lymphatic drainage, vascular circulation and neurological function
- Joint restrictions – Chiropractic adjustments as necessary to maintain joint health and correct compensatory biomechanical imbalances.

Rehabilitation

Rehabilitation is an extremely important component of the healing process.
Appendix 1 briefly outlines the restrictions and exercises at different stages of the rehabilitation process.

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Appendix 1: Rehabilitation techniques for lower motor neuron disease

	Paralysis	Paresis	Can't lateral to sternal	Can't sternal to sit	Can't sit to stand	Can't walk, can stand supported	Can't stand to walk, can stand unsupported	Weak, can get up, Can walk, knuckling	Some ataxia, weak, wobbly, can walk, not knuckling	Constipation
Mashed pumpkin										x
Tap and tickle back, legs, feet	x	X	X	X	x	x	X	X	X	
Massage back, skin rolls if compliant, portable massager	X	X	X	X	x	X	X	X	X	
PNF – sit to stand					sequencing					
Balance/ peanut ball support- cr/cd, side to side					sequencing	X				
Proprioception- balance disc, trampoline						X	X	X		
Passive range of motion limbs- shoulder rolls, cycling, joint pumps lymphatics,	x	X	X	X	x	x	X			

	Paralysis	Paresis	Can't lateral to sternal	Can't sternal to sit	Can't sit to stand	Can't walk, can stand supported	Can't stand to walk, can stand unsupported	Weak, can get up, Can walk, knuckling	Some ataxia, weak, wobbly, can walk, not knuckling	Constipation
circulation, flexibility, neurological input										
Withdrawal reflex	X	X	X	X	X	x	X			
Hydrotherapy – standing in water						X	X			
Hydrotherapy – underwater treadmill							X	X	X	
Hydrotherapy-paddling with lifejacket						x	X	X	X	
Wading						X	X	X	X	
Supportive sling,towel, harness- help em up					x	X	x	x		
Tail standing →walking						X	X	X		
Assisted walking in cart/ frame on treadmill							x	x		
Walking on								X	X	

	Paralysis	Paresis	Can't lateral to sternal	Can't sternal to sit	Can't sit to stand	Can't walk, can stand supported	Can't stand to walk, can stand unsupported	Weak, can get up, Can walk, knuckling	Some ataxia, weak, wobbly, can walk, not knuckling	Constipation
stimulatory surfaces										
Dog boot+/- toe up sling								x		
Short walks often								X	X	

How to build the equine dental stream in your equine or mixed practice

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Introduction

Since the 1990's, around the world equine dentistry is growing in demand from owners to veterinarians, to levels approaching those before the invention of motor vehicles. Many mixed and equine practices have embraced it, and it has become an important part of their business models. But with estimates of up to 80 % of domestic horses still not receiving an annual or bi-annual dental check up, there is still abundant opportunity for even more equine and mixed practices to build the equine dental streams within their practices.

Why equine dentistry could become a valuable part of a veterinary practice.

Unlike fads such as heart scores, bleeding racehorses, cutting superior check ligaments for bowed tendons and pin firing for shin soreness, equine dentistry is not a fad and there has been a large increase in the amount of scientific research, papers, and inclusion in mainstream veterinary conferences and undergraduate courses worldwide over the past two decades.

Dental care is an integral part of optimal welfare and care of domesticated horses. Nature did not intend for horses to be bitted or restricted in paddocks with a narrow spectrum of feed types. Domesticated breeding programs do not have the powers of natural selection in breeding good conformation mouths as seen in brumbies such as the Guy Fawkes Brumbies in far north NSW which are familiar to the author.

Owners usually can relate to dental pain and the importance of a healthy mouth, unlike many other conditions that vets need to educate them on? Most owners have not suffered from gastric ulceration, liver disease, gut parasites, seedy toe, shin soreness, laminitis, Cushing's disease etc. So it quite easy to convert them to believe the need for higher quality dental care for their beloved horses.

Most A and B class clients are already willing to invest in good quality dentistry, but they need to be assured that it is a competent and confident operator performing it, and that it is value for money. Note that value for money perception is not all about being the cheapest!

Business models are often seeking to make the business more like a conveyor belt than a roller coaster, and equine dentistry can help to achieve that in mixed and equine practice. It is not seasonal, but can be used to make quieter times of the year busier for the practice should they desire that.

So long as there are standard operating procedures and minimal levels of competence and confidence amongst the veterinarians in the practice, many owners will be willing to accept the new vet or recent graduate to do the dental checks and maintenance on their horses. It can be the ideal way for a new vet to get to know the owners, develop rapport, impress them with their knowledge, skills, bedside manner, horse handling skills etc. Meeting owners in a low stress, elective situation is a superior way for the new vet in the practice, compared to a high stress, after-hours emergency situation where the costs and outcomes may be far less optimal for the owner – no matter who the vet is.

With the increasing emergence of online pharmacies competing directly with veterinary clinics for sales of S4 medications, there is the realisation that veterinary clinics will need to charge better for their professional skills. The reliance on pharmaceutical sales to supplement the professional fee income to the business will likely diminish over the next few years. Unlike pharmaceuticals, physical professional skills cannot be sold online.

It is unanimous amongst equine veterinary dental experts around the world that an oral exam cannot be done properly without sedation, so sedation is an integral part of a quality dental service on a horse. The charge for sedation is best being charged within the overall dental fee, rather than charged separately. The reason to include it is that it reduces the request to do the dental without sedation, plus allows proper charging for the sedatives. Owners will soon realise it is better value for the one professional to do both the sedation and the dental, rather than a veterinarian coming to sedate the horse for a lay person to do the dental. Unfortunately though, there are still reports of widespread illegal use of sedation by lay tooth floaters in Australia, with some veterinarians thinking it is acceptable to supply these drugs.

Equine dental services are a good way to get you onto horse properties at least once a year, and this will give access to more work for the business. It is not uncommon for an owner to seek an opinion on another problem whilst you are there doing the dentals. Addressing this request is value adding and can easily lead to more work which can be a “win win” for the client and veterinary clinic.

Wellness packages can be appealing to A class clients, and dental services, parasite control and vaccinations are the foundation of these packages. They can help bond clients to your practice, especially if loyalty programs and discounted packages are used to incentivise clients to commit and invest in the package.

How to integrate equine dentistry into a mixed or equine practice

The author has assisted many veterinary practices to integrate equine dentistry into their practices, and has seen some spectacular successes, failures and mediocre results too.

Like any successful venture, there needs to be careful planning, commitment and effort to achieve something worthwhile. It is far easier to successfully implement dentistry into a practice if that practice is already held in high regard within the local equine community. For anyone else, there is a lot higher chance of failure, but it is still definitely possible to succeed.

HOW? Step 1: Set Goals.
Firstly it is necessary to set goals. Begin with the end in mind. What do you want to build? Do you want to be doing 1-5 dentals per week, 5-10, 10-20 or more than 20? Who do you want to be doing the dentals – some or all of the equine vets? If some are too busy to take on more work than they currently are, as will likely be the case with the senior vets, it is still important for them to be able to do a proper and thorough oral exam, and then be able to refer the case to the dental focussed vet/s in the practice.

HOW? Step 2 Develop strategies.

Strategic planning is important in any new venture, and this is best done involving the whole team. Some steps in developing strategies to implement equine dentistry into a veterinary practice may include:

a) Generate enthusiasm and desire for the venture to succeed. You may need to review the “whys” of developing this new line of work? Remember that past negative views of doing dentals by traditional equine vets are exactly that – the past! Most full time equine dental vets now are by and large very fulfilled and passionate professionals who have used this field of equine medicine and surgery to reinvigorate their careers , even from the point of them leaving the profession altogether. They truly enjoy the certainty that they are making a positive effect on almost every patient, yet have the enormous variety and challenges to allow them to grow in their careers, contributing to large scale welfare improvements, and enjoying becoming important members of the health care teams of some leading training stables etc.
b) Education – of the veterinarians, the other staff of the practice and finally the clients. Failure to educate ALL of these will lead to failure of the venture. There will be no point relying on your receptionists to sell your services if they don’t understand the value of good dentistry and have faith that your vets are the best professionals for the clients.
c) Equipment – like most manual skills, the task soon becomes unpopular if the right tools and education on how to use and maintain them properly is not undertaken. You will fail if you don’t have the right tools for the job, and vets will soon lose the passion for the work.
d) Systems and standard operating procedures – to become a major line of work it is important to standardise the processes of oral examination, sedation, odontoplasty, nerve blocks, taking radiographs etc. The practice’s reputation will suffer if one older vet insists on not sedating for dentals, not doing a thorough oral exam etc. And the new vets may not be embraced or trusted if they do dentals a lot different to the already established and popular senior vets.

   It will be important for the vets to have been educated, and have practiced and refined their systems of setting up equipment, using dental equip, sedating and examining the horse, floating the mouth, charting the mouth and invoicing etc. Failure and lack of confidence at any of these points may incite doubt in the client’s mind and so contribute to them seeking alternative dental service providers next time. Practice makes perfect, so insist on good levels of practice and revision prior to trying to convince owners and seek loyalty.
e) Marketing and client education – Despite all the possible options for marketing and education, a few stand out when it comes to equine dentistry in veterinary practices. With the advent of social media, the tasks of marketing, client education and branding have never been so easy or important. The use of videos, photos and information or links to good articles become powerful marketing tools on social media, and will help to brand your clinic as experts who are serious about helping owners to help their horses. Other cost effective and valuable marketing and client education media include fridge magnets, use of dental charts, a good up to date website, client education seminars, and of course the age old “word of mouth” – especially from key opinion leaders within the equine industry.

   Ultimately the Key to successful marketing and education is changing the focus from how good you are, to what’s in it for the client and what you can do to serve and help them.

HOW? Step 3 – The use of role models.
Using role models helps prevent wasting time and money trying to reinvent the wheel.

HOW? Step 4 – Review your results.
In any new venture, you need to track your results and notice what is working and what is not. Then determine what to change in the attempt to improve results. Reviewing different results each month, 3 months, 6 months and annually would be needed. Things to track can include:

- Tracking where your new clients are coming from
- Number of clicks on your social media posts
- Number of enquiries about dental work and number of conversions to work
- Responses to different marketing methods
- Set a marketing theme to run for 3 months, and check the responses to each theme e.g. broodmare dentals being set to be done in Winter, so that you are avoiding the high risk periods for losing pregnancies such as early and late pregnancies.
- Number of new dental clients
- Percentage of clients who keep getting your clinic back for dental work
- Number of dentals being done
- Response to dental reminders and number of reminders needed to achieve a booking.

If your results are not good enough, then try surveying your clients, using tools such as Survey Monkey, to ask those questions on their perceptions on your dental services.
Successful modern businesses do this routinely to ensure they are keeping up with serving their clients in the best way they can. They know it takes 7 times more effort and cost to find a new client than retaining an existing client.

HOW? Step 5 – Constant and never ending improvement.
Wowing your clients will help keep you perceived by them as the expert who they can entrust as their preferred equine dental service provider – who is not only the best, but also the best value for money. They like to be able to brag to their friends about how good you are with their horses etc.

HOW? Step 6 – Maintenance and inventory of equipment
It is well worthwhile to develop a culture that encourages vets and nurses to look after equipment as if it is their own. Tools to help grow this culture include having good systems in areas such as:

- Where the equipment is to be kept and put back each time,
- Lists and videos on how to maintain the equipment, that all staff and shown and sign off that they understand it.
- A budget for equipment repairs that if kept below a certain level, will give the staff a bonus at Xmas etc.
- Monitoring and audits on how well equipment is being maintained, and demerit points for non-compliance by individuals.

Step 7 – Keep focusing and encouraging the basics!
One of the hardest things to do within a multi vet practice is to ensure that ALL of the veterinarians continue to do the basics well every single day. Omitting the basics of an oral exam will lead to missed pathology, omitting the basics of odontoplasty may lead to iatrogenic damage to teeth and omitting the basics of equipment maintenance will contribute to increased equipment failure and break downs. Misfortunes such as missed pathology,

iatrogenic tooth damage and equipment failure ultimately result in clients losing faith, less repeat work and ultimately less revenue for the business.

Some DO's and DON'TS for successful implementation of equine dentistry into a veterinary practice.

Through surveying some successful mixed and equine multi-vet practices, who have successfully implemented equine dentistry and seen continual growth over the past 5-10 years, we will now discuss some key DO's and DON'TS,

DO:

1) Do set goals as a team for what you want to achieve and how you intend to get there. Review them often, and change tactics if not working.
2) Do IN house training so that ALL of your vets, nurses and receptionists all believe in it, and offer incentives for the lay staff to use your vets to do their horses' teeth rather than other providers.
3) Do look at all the possible benefits of equine dentistry to your clinic, discuss them with all team members to find increased levels of enthusiasm, and keep feeding that enthusiasm.
4) Do start to look at every horse on your books as a potential dental patient, showing interest in its mouth even if you are there to treat it for other issues. Showing interest in the dental health of the horse is sowing the seed to the client that you can potentially do the dental work for them in the future.
5) Do offer incentives for clients to try your clinic to meet their equine dental needs.
6) Do continue to improve your systems, education and skills in equine dentistry to ensure there is consistency between vets.
7) Do set minimum skill expectations for all vets – especially the basic fundamentals of being able to proficiently perform a thorough oral exam.
8) Do use loyalty programs – especially digital tools such as flok.
9) Do use social media and have themes that run for 3 months at a time.
10) Do invest in good equipment that makes the work easier, faster, and more profitable.
11) Do seek feedback from clients on what they are looking for
12) Do Offer incentives for group bookings, as they can easily be your most profitable periods of work.
13) Do look to lever off your veterinary privileges and offer packages where you can do more than just a dental and deliver great value e.g. free sheath cleans, vaccinate whilst there, faecal egg counts, lateral x-rays of front feet to help their farrier with break over angles, preventing lamenesses etc.
14) Do develop and train your receptionists and nurses in how to script to enquiries the value of the dental service you provide and why it is worth it etc.
15) Do spend time explaining to owners how many pathologies will need revisits and cannot be fixed forever in just one treatment.
16) Do seek opportunities to educate the Key Opinion Leaders in your area, and look after them, as their recommendations are valuable.

DON'T:

1) Don't bother with ads in papers, radio, TV, yellow pages.

2) Don't bad mouth other dental service providers to your clients as it is unprofessional and will get you nowhere.

3) Don't get discouraged or disheartened if you experience a lag period after the first wave of dental business. Just keep chipping away at doing a good job and wait for those horses to need a dental again and hope that word of mouth and good marketing brings new clients in.

4) Don't try to convert owners to use you if they have little faith in your equine vet abilities in other areas

5) Don't continue to reschedule dental appointments because of emergencies. Your best clients are often the owners of a few horses, and are usually busy people, who have to take time off work to have their horse's teeth done etc. If you keep rescheduling them because of emergency call outs etc, it may become inconvenient for them to use you and force them to look elsewhere for a dental service provider who does not do emergencies. When you set an appointment for dental work with a good client, do whatever it takes to keep it and not reschedule it.

6) Don't trust the lay tooth floater who you used to sedate for etc. Once they know you have become direct competition to them, they may start to subtly ridicule your techniques etc. Common phrases such as, "your horse is not naughty or bad and so doesn't need sedation to do its teeth", or "I don't need a light and mirrors to see inside your horse's mouth as I can see with my fingers", or "the vets don't get taught dentistry at vet school but do a weekend course".

7) Don't isolate yourself on cases. Set up and liaise with a network of colleagues and experts, and don't be afraid to refer the cases that you worry will not go well in your hands.

8) Don't think that the routine dental work is not important to retain as part of your services, in the belief that you can focus on the advanced work. The routine work is often more profitable that advanced cases, especially if they don't respond as well as expected, demanding a lot more time and resources than quoted for. The routine work is where your veterinarians develop and improve the skills and experience needed to attempt the advanced cases. The routine cases are where the advance case pathology will be detected and shown to the owners, and able to explain to the owners the need to investigate the problem further before being able to treat it as discussed.

In summing up, equine dentistry is rapidly becoming a very significant line of income production and sustainability in successful equine and mixed veterinary practices. But the success of the venture largely depends on how it is implemented.

Livestock Export – The Role of Veterinarians

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Introduction

Veterinarians play a vital role in every aspect of the livestock export trade from negotiating market access to regulation, certification, and many aspects of the trade's day to day operations. Without the involvement of veterinary professionals, the livestock export trade would not exist. Veterinarians also play a vital role in trade improvements through research into animal welfare, disease control and improving productivity and performance during transport and after arrival in overseas markets.

Similar to other intensive agriculture industries, private veterinarians working in livestock export deliver professional services and often form an integral part of a business model to manage animal health and welfare risks. As veterinarians, we can prioritise animal health and welfare outcomes while taking economic and other constraints into account. Our professional training and experience give us an in-depth perspective and understanding of animal welfare. Veterinarians are well placed to measure and assess welfare outcomes and to advocate for optimal welfare standards within a production system.

Veterinary involvement can be broken down into specific areas related to their activity and their part in the livestock export process. Australian Government Accredited Veterinarians (AAVs) are responsible for pre-export preparation and shipboard work while non-AAV roles vary from regulatory positions to consultants delivering advice on animal health and welfare. Only veterinarians accredited by the federal government are permitted to oversee the preparation of livestock for export or accompany livestock on voyages. There are around 120 AAVs who are registered, under federal legislation, to undertake export related work[1]. AAVs are either accredited for pre-export preparation work, for shipboard work or in many cases both.

This document aims to give an overview of the Australian livestock export supply chain and to outline the roles that veterinarians play in the industry. There have been significant improvements to industry regulation and legislation over the last 12 months, the details of these changes are beyond the scope of this paper. However, veterinarians have played a vital role in putting forward and implementing these improvements.

The Livestock Export Supply Chain

Securing Market Access

Australia only exports livestock to countries that have trade and biosecurity agreements in place, the industry's social license to operate also requires us to sell to markets that agree to uphold our Export Supply Chain Assurance System (ESCAS).

Trade and Market Access & Animal Biosecurity

Australian trade discussions require technical expertise to negotiate with importing countries. These roles are often filled by Australian veterinarians who provide advice on Australia's disease and biosecurity status. These discussions ensure Australia has fair-trade access in line with risks identified in disease control and biosecurity protocols.

Importing Market Facilities

Australian livestock exported as feeder or slaughter animals are managed under ESCAS when they reach their destination market. This means that an export company can only trade with receivers who have been pre-approved as having appropriate facilities for the handling, housing and slaughter of Australian animals. The standards that we require from our trading partners are similar to, if not higher than those required in Australia. The implementation of ESCAS has involved significant investment from industry and extensive consultation with Australian veterinarians, ultimately improving animal welfare outcomes for both Australian animals and local animals in importing countries.

Pre-export Preparation

The Australian livestock export industry is legislated using a whole-of-chain risk-based approach to minimise the chance of adverse animal health and welfare outcomes at any stage of the export process. Each consignment undergoes thorough planning and risk assessment. Risk mitigation strategies are put in place from Australian farms through to the point of slaughter in the foreign destination markets. Veterinarians work with export companies to navigate the legislative framework and plan consignments to meet exporting country requirements, importing country requirements, commercial expectations and acceptable animal welfare standards and outcomes.

The livestock export trade is regulated by the Federal Department of Agriculture and Water Resources working under the Australian Meat and Live-stock Industry Act 1997, Export Control Act 1982 and Export Control (Animals) Order 2004. Enforced standards and operational guidelines for the livestock export industry are outlined in the Australian Standards for the Export of Livestock (ASEL). Licenced exporters must have an Approved Arrangement in place to describe how the company operates, records and manages its operations. The exporters Approved Arrangement is associated with specific Standard Export Plans (SEPs) stipulating all requirements for meeting the ASEL and importing country requirements for each particular market and mode of transport (sea or air), and species they export. In conjunction with Approved Arrangements and SEPs, exporters must have an Approved Export Program (AEP) which outlines the specific instructions of activities and reporting that AAVs must perform to meet ASEL and importing country requirements for each specific SEP.

Export Company Veterinarians – Spanning the Whole Supply Chain

Veterinarians can be employed directly by export companies in various roles or provide services on a contractual basis. They use their problem-solving skills and knowledge of animal health in many areas throughout the supply chain to ensure that animal welfare is a key part of the export business model. Veterinarians working for export companies typically engage with all other veterinary professionals involved in the industry. Responsibilities and services provided by an export company veterinarian will vary greatly and include, but are not limited to, export legislation compliance and documentation, consignment planning and risk assessments, overseeing livestock health and welfare throughout the supply chain, engagement with other industry veterinarians in Australia and importing countries, as well as communication with industry bodies.

Export companies are required to document and record all operational and governance systems and the processes used to meet legislative requirements. A key role of a company veterinarian is their involvement in the development of Approved Arrangements and Approved Export Program documentation. Understanding the legislative framework to meet importing country requirements requires some technical understanding of the disease and biosecurity status of different regions in Australia. In addition to providing technical advice to meet legislative requirements, a company veterinarian can also ensure there is an operational understanding of compliance frameworks, appropriately trained personnel and systems in place for reviewing consignment outcomes.

The involvement of an animal welfare professional in an export company's consignment planning and risk assessments can optimise welfare outcomes. Having a detailed understanding of the export company's business model, the importing country demands, and the relevant regulation and legislation puts an export company veterinarian in an informed position to provide useful advice, formulate risk assessments and guide management decisions. Risk assessments cover the sourcing of livestock, their management in registered premises, nutrition, health and management during transport. Company veterinarians or contracted veterinarians are also involved in the identification and implementation of risk mitigation measures at all stages of the supply chain.

Livestock selection and preparation greatly influences the health and welfare of exported livestock further down the supply chain. This part of the export process is one of the most significant areas of influence for veterinarians. Livestock preparation begins on farm and veterinarians can provide advice and services to ensure selected animals meet all requirements and are suitable for transport by sea or air to the specific market. Preparation may include logistics to coordinate the services of other veterinarians and AAVs. Consignments can vary from 10,000 head of cattle to be exported by sea to 50 goats exported by air. The preparation of consignments is likely to involve on-farm testing and extensive data and document verification. The transport process requires careful planning and benefits from the oversight of an experienced veterinarian. Management of animal health and welfare also extends beyond the discharge port with ESCAS and breeder programs to be implemented in importing markets. The identification of animal health or welfare issues to be addressed in importing markets, especially in developing or new markets, is an integral part of the role.

Communication with importing country veterinarians often takes place during the preparation of a consignment and particularly during pre-export quarantine periods. International trade partners will often send their government or private veterinarians to check that export processes are in line with the import permit, commercial arrangements and compliant with agreed health protocols between countries. Company veterinarians are best placed to coordinate and communicate with these stakeholders as they have an understanding of both the commercial and legislative requirements.

Export company veterinarians frequently engage with industry bodies such as the Australian Livestock Export Council (ALEC) and LiveCorp on industry projects and initiatives. The involvement of veterinary professionals in industry working groups is significant and has been promoted and welcomed by exporters and industry to ensure animal welfare is balanced with commercially driven considerations on all issues relating to livestock export.

The unique understanding of both commercial and operational aspects of the export trade means that a veterinarian's position in an export company provides valuable services and advice to ensure that good animal welfare outcomes are a priority.

Federal Government - Live Animal Exports Division

The federal government regulator is responsible for applications and audits of a licenced exporter's Approved Arrangement, Approved Export Program (AEP) and Approved Supply Chain under ESCAS. In addition, they are responsible for the accreditation and auditing of AAVs. DAWR Live Animal Exports operational personnel will review applications for export permits and determine certification of importing country requirements before granting approvals to export. Veterinary guidance and professional skills are required in this area.

Federal Government - Regional Veterinary Officers

DAWR have regional veterinary officers in each state which carry out final inspections of livestock before export, review the compliance with Approved Arrangements and Approved Export Programs, and issue health certification and export permits. This role is key to final certification that livestock have been prepared in accordance with all ASEL and importing country requirements and requires unique skills and understanding of the industry.

State and Territory Government Veterinarians

Property of origin clearances required for federal government certification are issued by state and territory government veterinarians to verify that on-farm disease status complies with importing country requirements. State and Territory veterinarians are also involved in animal welfare inspections in line with state-legislated animal welfare acts.

Private Veterinarians Registered in State or Territory

Private veterinarians provide services to the livestock export sector through activities such as pre-export pregnancy testing, ensuring livestock meet the customer's selection criteria not related to ASEL, and by carrying out on farm livestock inspections. Private practitioners work with producers and buyers to ensure that livestock are eligible for specific export orders based on the health status of the property.

Pre-Export Australian Government Accredited Veterinarians

AAVs involved in pre-export preparations perform clinical inspections on livestock and provide declarations to the exporter that animals meet the ASEL and importing country requirements before export. International trade negotiations and agreements require AVVs to conduct pre-export testing, administer treatments and make declarations to the exporter that animals meet the importing country requirements set out in the Approved Export Program (AEP) or the import permit. AAVs identify animal welfare risks and review compliance with both the ASEL and importing country requirements.

Transport by Sea

Shipboard Australian Government Accredited Veterinarians

The health and welfare of the animals during all long-haul voyages by sea and some short-haul voyages is overseen by a shipboard AAV. Shipboard veterinarians also provide Daily Reports and an End of Voyage to DAWR to meet the reporting requirements outlined in ASEL.

A shipboard veterinarian's role requires a good working relationship with an exporting company so that their experience and expertise is also sought during the voyage planning and preparation phase. The active involvement of an experienced shipboard vet in the preparation of appropriate livestock, provision of suitable resources such as bedding and feed, formulating the stowage plan, and considerations for the livestock vessel's mechanical systems and infrastructure, will have a significant impact on the voyage outcomes.

Veterinarians may be present during the loading of livestock on to the vessel to ensure that safe animal handling practices are being followed and that animals are loaded according to the stowage plan. When the ship departs, the veterinarian will work closely with the LiveCorp accredited stockpersons and the ship's crew to manage the daily care of the livestock. Daily care includes the provision of feed and water, management of bedding and the manure pad, while also monitoring the performance of the ship's systems to ensure a stable microclimate for the animals.

The livestock are closely inspected 2-3 times daily by the AAV and accredited stockpersons, animals that are sick or injured, or those not coping with the pen environment, are drafted into hospital pen areas where they can receive individual care and attention. The veterinarian will provide clinical care, or humanely euthanase and post mortem animals where required. A shipboard AAV spends the majority of their time at sea working with the livestock and providing direct care to animals.

The unloading process occurs under the supervision of the AAV or accredited stockpersons. Animal handling and care are critical at this time as livestock are relocated from the ship to the receival facility. The shipboard AAV can communicate with the receiver about the history of the consignment, recommendations regarding follow-on care, and the medications and treatments administered during the voyage.

Details regarding the health and welfare of animals, their access to resources, and the environmental conditions onboard long-haul voyages are recorded and reported daily by veterinarians. As well as fulfilling the reporting requirements of the regulator, the AAV will liaise with the exporting company, the shipping company, and the foreign receiver to encourage ongoing improvements in the performances of voyages and animal welfare outcomes. Not all voyages have and AAV onboard, industry accredited stockpersons will perform this role on most short haul voyages, although veterinarians are integral in the provision of the training and skills required for industry accreditation.

Shipboard Independent Observers

In April 2018, the federal Minister for Agriculture and Water Resource announced that an Independent Observer (IO) would accompany livestock voyages in an effort to improve DAWR's regulatory capabilities and to ensure compliance with animal welfare outcomes as stated in ASEL. The IO role onboard long-haul voyages have mostly been filled by government veterinarians who review and audit the AAV's compliance with the AEP instructions.

Destination Markets

Veterinarians have been engaged around the world to deliver post-delivery animal health and welfare programs and consultation in areas such as training, nutrition, infrastructure design and abattoir management. Australian veterinarians and stockpersons work alongside local veterinarians and stock handlers to design and install facilities that are suitable for Australian livestock. Training, infrastructure and slaughter techniques must be adapted to suit the type of animals being handled. ESCAS and the influence of the

Australian livestock export industry have improved standards for local animals in foreign markets as well as animals sourced from other countries by improving facilities and providing training to local workers.

The licensed exporter is held accountable for ensuring that the maintenance and ongoing inspections of the ESCAS approved facilities are carried out. Exporters are also responsible for the traceability of all feeder and slaughter livestock exported from Australia. The traceability of every animal (982, 236 cattle and 1, 105, 740 sheep in 2018[2]) must be documented to ensure that Australian animals are not sold outside the supply chain to farms or slaughterhouses that do not meet ESCAS standards. Veterinarians often work with export companies to facilitate ESCAS compliance, training, traceability and reporting requirements.

Ancillary Roles in Livestock Exports

External consultants

External consultants provide exporters with advice on departmental requirements, logistics, data collection, health issues, disease mitigation and MLA/LiveCorp funded research. External veterinary consultants often have areas of specific expertise such as nutrition, epidemiology and production animal economics in addition to their veterinary skills base. The involvement of veterinary consultants in a production animal business can significantly improve the performance and profitability of a production system or a trading and transport business model.

Research and Development

Veterinarians are involved in delivering industry research and development projects. The live export sector has an active R&D program and receives funding from industry levies, the federal government and donations from industry partners to implement research that has been identified by industry to ensure ongoing improvement. Technology is actively being sought to optimise environmental conditions onboard export vessels and in supply chain feedlots and slaughterhouses. Improved technologies for monitoring and recording livestock health and welfare and environmental conditions are also being sought and adopted. Veterinary science is an evidence-based profession; veterinarians are trained to assess evidence and understand the importance of properly conducted research and are therefore an essential resource for carrying out epidemiological, clinical and field research as well as implementing research findings within industry. Continued industry improvement is vital to ensure that community expectations are being met and the industry operates competitively on an international market.

Conclusion

It is rewarding to work as a veterinarian in an industry that is prioritising progress and seeking to adopt new technologies and best practices. Veterinarians and especially AAVs can be advocates for animal welfare while working towards fulfilling the importing country's consumer demands, the Australian community's expectations, the implications of a socially and commercially viable trade, and the extensive regulatory requirements that govern the industry.

The AAV role is unique and diverse, it provides an opportunity to work directly with livestock and alongside multiple stakeholders with animal welfare as a shared goal. The AAV role differs from the traditional transactional structure of veterinarians working in private practice, but it is not a purely regulatory position either.

Veterinarians are well placed to provide balanced and informative feedback to industry, the regulator, lobby groups and the Australian public. They have the skills and experience to use evidence and reason to guide industry. Ultimately all veterinary roles help to ensure optimal health and welfare outcomes for livestock exported from Australia.

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Reporting anti-microbial usage at the point of administration

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Introduction

The development of anti-microbial resistance (AMR) is becoming an increasing concern worldwide. A national animal health reporting system was able to capture anti-microbial usage (AMU) at the point of administration, and we present some examples and discuss the implications

Background

A number of diseases that should by now be of limited significance are causing increasing problems as existing therapies have sub-optimal performance. The worst case scenario is a return to a world where antibiotics are no longer a reliable part of the practitioner's tool kit.

Management of the overuse of these drugs is typically focussed on campaigns with positive messages for reduction in anti-microbial usage (AMU). Such messages include reasons such as loss of efficacy and side effects, and of course the potential for residues in food-producing animals. Although these efforts are making some progress, it is difficult to measure the change in prescribing habits in veterinarians, as there may be no central system for monitoring the use of such drugs. Points for AMU surveillance can include importers, manufacturers, wholesale distributors or retail sale. A common alternative is end use monitoring (such as residue testing in meat). By their nature, these methods tend to be expensive and offer little certainty about the risks associated with any given animal. In many countries such systems do not exist at all.

The national animal recording system we assessed (iSIKHNAS[1]) collects reports by instant or text message from veterinary staff. For disease investigation cases, staff can report a final diagnosis and the treatment(s) that were administered at the time. We looked at data collected and compared recorded diagnosis with the corresponding pharmaceutical treatment using visualisation and exploratory data techniques.

Results

The system receives around 40 000 "diagnosis" responses per month, and administration of antimicrobials occurs in association with about 25% of these cases. In most of the cases the decision to administer antimicrobials appears to be reasonable, however in some cases uncertainty may exist over the need to use such treatments (for example, when a viral disease was recorded).

Discussion

The system produces reports which can identify these inappropriate AMU, and individual instances can be investigated by contacting the prescribing veterinarian. This closed loop is relatively inexpensive, as it can be shown that only a small number of veterinarians routinely administer antimicrobials where it is not indicated. In contrast to the cost of untargeted national campaigns, this approach provides a cheap and effective mechanism to monitor and manage antimicrobial usage.

Notes

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Application of virtual herding technology on sheep

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Introduction

Virtual fencing is an emerging technology that is currently applicable to cattle with an eShepherd™ system (Agersens Pty Ltd). Through the use of an algorithm that combines GPS with animal behaviour, the device uses audio cues and an electrical stimulus to train the animal to stay within a virtual boundary. Virtual fencing has the ability to contain and control the movements[1] of animals and offers potential benefits for land and animal management. There is interest in applying virtual fencing to sheep, this paper will discuss the research that has been conducted to determine if virtually fencing sheep is feasible and to determine the potential impact of virtual fencing on sheep welfare.

Methods

For all experiments virtual fencing was implemented using manually controlled dog training collars (Garmin TT15, Garmin Ltd., USA) capable of administering an audio (70–80 dB, 2.7 kHz) and an electrical stimulus (320 V, 20 us,16 pulses per s). The electrical stimulus level was determined by an initial study[2] using 30, 4 y.o. Merino cross ewes. Sheep were taught the virtual fence through an associative learning training protocol, in which sheep would receive an audio cue when approaching the designated fence line and would receive an electrical stimulus immediately after the audio cue ceased. As the audio cue was applied, if the sheep stopped walking or turned away from the fence they would not receive an electrical stimulus. Sheep were trained in an individual and group setting using methods from Marini et al. [2, 3].

Individual training

Twenty, 2 y.o. Merino ewes were trained to the virtual fence in two small familiar paddocks (65 x 65m). In the centre of the paddock was a pen containing 10 sheep which were used to attract the sheep to approach the virtual fence. Training was conducted over 8 days, with one training session a day, each training session being 3 minutes long. Behaviour in response to the electrical stimulus was scored from 1, no response; 2, the sheep pulled its head back, but continued walking; 3, the sheep lifted its front legs off the ground and ran. Scores were analysed using a Fishers exact test.

Group training

Eighteen 4 y.o. Merino ewes were acclimated to a paddock (40 x 60m) for 2 weeks without the virtual fence. They were then trained to the virtual fence as two flocks of nine sheep, for one week each. The sheep were given access to the paddock for 6 h during the day and were moved out into a holding pens overnight.

Number of audios and number of electrical stimuli applied were recorded. Virtual fence interactions were analysed using a logistical regression, or paired t-test and binomial proportions test where a regression wasn't feasible.

Results

Individual training

There was variation between sheep and their ability to respond to the audio cue. Out of the 20 sheep, 2 did not respond to the audio cue alone following paired interactions of the audio cue followed by the electrical stimulus. The sheep had a moderate proportion of electrical stimulus to audio cue (57%). Sheep in this study responded to the audio cue without prior encounters, due to this there was no clear point of learning as with previous studies[2, 3]. Sheep had a tendency to lessen their reaction from moderate to mild in response to the electrical stimulus as training progressed ($\chi 2$ (4) = 8, P = 0.06). The proportion of animals that scored a 1, 2 or 3 were 4.34%, 36.9% and 58.7% respectively at the beginning of training and 6.67%, 73.33% and 20.0% respectively at the end.

Group training
In a group the proportion of animals likely to receive an electrical stimulus before and after learning the virtual fence was low (Table 1). Sheep were able to learn the virtual fence through associative learning within an average of 3 interactions. Interactions with the fence was affected by surrounding sheep, with sheep in the front of the flock receiving an audio or electrical stimulus turning other sheep around and away from the fence.

Table 1: The proportion of animals that receive an electrical stimulus before and after learning the virtual fence when trained individually or in a group. All sheep were naive to the virtual fence when training began in these studies.

		Before learning	After learning
Individual Training	This paper	57%	N/A
	Marini, D., et al[2]	99%	48%
Group Training	This paper	24%	10%
	Marini, D., et al[3]	33%	19%

Discussion

The virtual fence was effective at preventing sheep from entering an exclusion zone in individual and group settings. Training sheep individually to a virtual fence highlights the variation between sheep and their ability to learn[2]. It is important to determine what leads to this individual variation in learning, as animals that are unable to learn to avoid the electrical stimulus are at risk of having poor welfare.
When trained in a group the ability for sheep to learn the virtual fence and avoid the electrical stimulus improves. Many fence interactions occurred as the flock approached the fence line together, leading to peer-induced behaviours when interacting with the virtual fence. This is of particular interest as it has implications for sheep management and how individuals learn within the flock.
Previous work looking at behavioural and physiological measures have indicated that the stimuli used in virtual fencing are no more adverse than stimuli encountered by sheep during common husbandry[4]. However more work needs to be conducted looking at the effect of the virtual fence on welfare in long term group studies.

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Ten Ways to Improve your Veterinary Mentation

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1. **Exercise**

There is mounting evidence that exercise plays a preventative role in helping us to avoid depression[1]. The medical profession regularly puts exercise at number one for good mental and physical health.
Busy veterinary professionals and in particular those with young families often find it difficult to exercise regularly. Compliance improves if you exercise with a friend, join a club or pay for a gym membership. The more vigorous the exercise the greater the likely benefit you will get.

2. **Stress Relief**

Each veterinarian has different stressors. One person may experience difficulty with clients, another surgery and perhaps another with euthanasia. It is important to identify your stressors and reframe them as your interpretation may not be correct. Cognitive Behaviour Therapy may be required.
In general terms, exercise, humour, meditation, mindfulness and yoga are excellent for stressed veterinarians.
Hobbies that are distractive and demand total focus are recommended.

3. **Connection**

Our genes are those of the tribe. Relationships are vital for us as social beings. The maximum number of close friends we can have is 5 according to Prof. Robin Dunbar from the University of Oxford. This is because of the social investment time required. Family and close friend relationships need to be fostered by veterinarians so they have support structures in place should the corrugated road of life present problems.
It requires a considerable effort to keep relationships in good shape. Communication is the lifeblood of relationships.

4.**Diet**

Barely a day goes by without new information on the human gut microbiome and its role in health and mentation[2]. Large quantities of neurotransmitters are produced in the gut. The vagal nerve is a conduit from the gut to the brain which circumvents the blood brian barrier. Raw foods and a diet rich in fruit and vegetables, omega 3 and 6 are very beneficial. Soft drinks rich in sugar have been linked to depression in teenagers. We need to move away from processed foods and sugar rich drinks.

5. **Gratitude and Compassion**

Buddhists determined through antiquity that Gratitude and Compassion are central to human happiness. In a world hooked on the dopamine reward system we need to get back to the oxytocin system.
Clinical Psychologists have long advocated writing down 3 good things that happen each day and reading back through the entries regularly. It is easy to focus on the things that go wrong in practice but we must revisit what went well.

6. **Hobby**

The benefit of a hobby is that it can be beneficially distractive and if absorbing can be akin to meditation. It is also a great way to make new friends.

7. **Voluntary Work**
When we help others we help ourselves. Voluntary Work improves your perspective on your own life and when we work where the need is greatest it often puts our own issues into a new perspective. Overseas voluntary veterinary work can be a great way to see a new country and meet local people.

8. **A Job you Enjoy**
If your work makes you miserable you have to change that situation. A veterinary degree can take you in so many different directions. It is worth the effort to find your niche as then it will not feel like work. Some veterinarians move away from practice to administrative jobs or different careers completely. It is about you finding fulfilment.

9. **Sleep**
Most of us need 8 hours or there will be serious health consequences later. Stress, anxiety and depression all play with sleep patterns. Debt and difficult relationship issues do not help either. At night our brain still works on the problems of the day.
One of the greatest enemies of sound sleep in veterinarians is caffeine. The half -life of caffeine is usually 6 hours and approx.15 hours in pregnancy. It can be prolonged in some individuals. The removal of all sources of caffeine from the diet for a two week test period can be a good way to determine if it is the basis for insomnia.
If you have sleep problems it is best to avoid caffeine, alcohol and chocolate late in the day. Exercise can be very helpful to encourage sleep but not before going to bed.
Screens at night time are also a hazard.

10. **Positivity**
Professor Martin Seligman informs us that to be happy and fulfilled we need PERMA[3]. This stands for Positive Emotions, Engagement, Relationships that are positive, Meaning in Life and Accomplishment.

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doi:10.1001/jamapsychiatry.2018.4175
[2] Evidence Mounts that gut bacteria can influence mood, prevent depression/Science/AAAS
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Books
[3]"Authentic Happiness" Prof. Martin Seligman
"Flourish" Prof. Martin Seligman
"The Depression Cure" Prof. Stephen Ilardi

Medical Management of Colic in Practice

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Introduction

Primary large colon impactions are a frequent cause of colic in horses. Impactions usually develop at sites of reduced luminal diameter such as the pelvic flexure or right dorsal colon, although they can form anywhere along the intestinal tract. Typically, the impacted material will be ingesta; however, obstructions due to the ingestion of geosediment (sand, soil or gravel) can also occur. Common predisposing risk factors for the development of a large colon impaction include dehydration and hypovolaemia, inappropriate mastication of feed, parasite migration, sudden reduction in exercise, increased stabling, recent travel and the administration of gastrointestinal motility-modifying drugs. Factors that play a role in increased sand ingestion include geographical location, soil type, frequent access to pasture, reduced access to long-stem roughage, boredom and indiscriminant eating behaviour.

Clinical Signs

Large colon impactions tend to develop gradually and are therefore associated with mild to moderate signs of abdominal pain. Colic signs are usually insidious in onset and often intermittent or spasmodic, rather than acute severe. Signs observed include frequent stretching or posturing to urinate, flehman, flank watching, pawing or lying down. As the large colon becomes more distended (with ingesta, sand or gas) and the colonic mucosa becomes increasingly inflamed, the clinical signs observed may become more consistent and severe. In cases of severe large colon impactions, diarrhoea may also be a presenting sign and is associated with the presence of a significant colonic obstruction that allows only water to pass through. Similarly, cases of intestinal geosedimentation may also result in diarrhoea secondary to mechanical irritation of the colonic mucosa, and weight loss.

Diagnostic Investigation

History: When investigating cases of colic, it is important to obtain as much information as possible pertaining to their dietary management, water intake, pasture turnout, stabling, exercise, deworming, concurrent medication and dental treatment particularly. Horses presenting with large colon impactions will frequently have a history of reduced faecal output; and when questioned further, owners will often describe the manure as being dry, "bally", or of smaller volume than usual. Appetite may or may not be reduced. In cases of sand enteropathy, owners might also report some weight loss, recurrent colic, chronic or intermittent diarrhoea or the passage of faecal liquor following defaecation.

Physical Examination: Most horses presenting with large colon impactions or sand enteropathy are still reasonably bright, alert and responsive. They are usually normovolaemic, with pink mucous membranes, a capillary refill time < 2.5 seconds and good pulse quality; however some cases may be hypovolaemic or dehydrated. Mild tachycardia (up to 48 beats per minute) and tachypnoea (up to 20 breaths per minute) are frequently observed, and affected horses are usually normothermic. Reduced gastrointestinal borborygmi over one or more abdominal quadrants is a common finding, with or without the presence of abdominal distension. In cases of sand accumulation, a classic "waves crashing on the beach" sound may be auscultated, as the sand moves with the peristaltic colonic motility. It is important to remember that false negatives can occur, particularly if the sand accumulation is large or gastrointestinal motility is reduced.

Assessment of Hydration: Determining the hydration status of colic patients is imperative as it will have a significant impact on their treatment. As previously stated, most cases presenting with large colon impactions or sand enteropathy will be normovolaemic, however more severe cases or those that have been going on for an extended period of time may present as either hypovolaemic or dehydrated. Hypovolaemia refers to patients that have a loss of circulating volume, and will frequently be tachycardic with an increased capillary refill time (> 2.5s), increased urine specific gravity (> 1.040), increased blood lactate concentration (> 2.0mmol/L) and evidence of haemoconcentration (increased packed cell volume and total solids). Dehydration refers to a loss of total body water and results in tacky mucous membranes, increased skin tent (beware of age-related changes in skin tent due to loss of elasticity – use the skin of the eyelid as the more reliable indicator), sunken eyes and a decrease in bodyweight.

Rectal Palpation: Rectal palpation is an incredibly useful diagnostic tool when investigating colic, and should be performed whenever possible. Safety for the veterinarian, handler and patient is paramount when performing this procedure, and ideally would be undertaken with the horse appropriately restrained in stocks. Depending on the temperament of the horse, sedation with an alpha-2 agonist may also be required. To improve the quality of rectal examination and limit rectal straining, infusion of local anaesthetic (50ml lignocaine 2% or mepivacaine 2%) following evacuation of the rectum should be performed. Hyoscine-N-butylbromide is a smooth muscle relaxant that is very effective in reducing rectal straining. In Australia however, this pharmacological agent is currently only available in combination with dipyrone, a weak nonsteroidal anti-inflammatory (Buscopan® Compositum [Boehringer Ingelheim] or Spasmogesic [Troy Laboratories]), thus may not be routinely used.

Typical rectal examination findings in a horse with a large colon impaction include a smooth distended viscous, which may or may not be indentable depending on how dehydrated the impacted ingesta is. In cases of a pelvic flexure impaction, the viscous will be palpable running laterally and ventral to the pelvis. As sand typically accumulates in a cranioventral and ventral location, affected horses will often have a normal rectal examination. In horses with larger burdens, or accumulations that result in an obstruction, gas-distended viscous may be identified at any location.

Nasogastric Intubation: Passage of a nasogastric tube should always be performed as part of the diagnostic work up for any horse presenting with signs of abdominal pain. In some cases of colonic obstruction, net reflux may be elicited. In the authors' experience, the amount is usually less than 8 litres, typically as a once-off yield. The nasogastric tube may then be left in place to facilitate administration of enteral fluid therapy.

Abdominocentesis: Analysis of peritoneal fluid can be a useful diagnostic tool in investigating causes of colic, particularly when it comes to differentiating between a strangulating and non-strangulating intestinal lesion. Care should be taken when performing abdominocentesis in patients with abdominal distension due to the increased risk of intestinal perforation. Peritoneal fluid findings in cases of large colon impactions are usually fairly unremarkable; the fluid is typically clear yellow, with a normal to mildly increased protein concentration. Peritoneal lactate concentration tends to match that of the peripheral blood. Inadvertent enterocentesis is not uncommon in horses with sand accumulation as the increased weight of the colon causes it to sit much closer to the ventral body wall. The presence of sand in an enterocentesis sample is consistent with sand enteropathy.

Faecal Sand Sedimentation Test: This test is simple to perform and can easily be done by owners at home. Whilst a positive faecal sand sedimentation test is consistent with sand excretion, this test has a poor correlation with the presence of colonic sand (high false negative rate) and provides no quantitative information on the intestinal sand burden.

Diagnostic Imaging: Transcutaneous abdominal ultrasonography can be helpful in increasing a clinician's index of suspicion of a large colon impaction, however in the authors opinion it is not possible to definitively diagnose an impaction via ultrasound. Common ultrasonographic findings include reduced colonic motility, distended large colon walls and flattened haustra of the ventral colon. Thickness of the colonic walls tends to be within normal limits.

The use of ultrasonography to diagnose sand enteropathy in horses has been described in the literature, although is considered of limited value in regards to quantification of the sand burden. Reported ultrasonographic findings include: acoustic shadowing caused by the accumulation; a hyperechoic outline of the ventral haustra; immobility of the intestine; visualisation of the ventral colon lying directly on the body wall or compressing ventral intra-abdominal fat; and stretching or loss of ventral haustra.[1]

Abdominal radiography is considered the gold standard diagnostic test for identifying sand accumulation in horses, as it is easy to perform, non-invasive and can be used quantitatively. For an average-sized horse (up to 500kg) in our hospital, standing lateral projections of the ventral abdomen are typically obtained using a grid and the following exposure settings: 100kVp/40mAs (cranial view) and 110kVp/64mAs (caudal view). Accumulation of geosediment can be clearly identified within the colon, appearing as a single or multiple opaque mass(es) within the cranioventral abdomen (Figure 1). Frequently the accumulation(s) will conform to the shape of the colon, and often the margins of the colonic haustra will be obviously outlined. If the sand has settled out from the ingesta the dorsal margin of the accumulation tends to be horizontal.[2] Parameters such as the number of accumulations, location, length and height of the accumulation(s), opacity, and homogeneity of the accumulation(s) should be evaluated and can be helpful in determining the clinical significance of the sand burden.[2,3]

Figure 1: Left lateral radiograph of the cranioventral abdomen. Note the large sand accumulation within the ventral colon with a flattened dorsal margin and clearly outlined haustra.

Abdominal radiography is not only utilised for the definitive diagnosis of sand enteropathy, it is also very useful in monitoring efficacy of treatment (Figures 2a and 2b). Serial radiographs can be obtained during the course of therapy to determine the duration of treatment or whether more aggressive intervention is warranted. For those patients that are known "accumulators" or have a history of sand enteropathy, routine radiography every 3 - 6 months is recommended as part of their preventative health program.

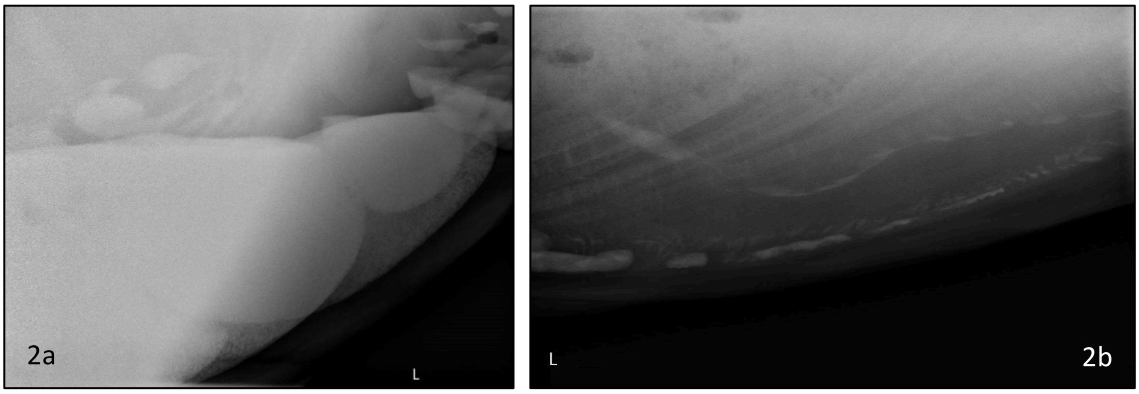

Figure 2a and 2b: Left lateral radiographs of the caudoventral abdomen. Note the large sand accumulation within the ventral colon (left image) and its subsequent resolution following treatment (right image).

Treatment of Large Colon Impactions

Medical management of large colon impactions incorporates four main goals: 1) Appropriate analgesia; 2) Correction of fluid deficits; 3) Clearance of the impaction; and 4) Prevent recurrence.[4] First and foremost however, **feed must be with-held** until the impaction has resolved.

Analgesia: When it comes to providing analgesia for colic patients, practitioners will typically opt for a non-steroidal anti-inflammatory drug, alpha-2 agonist, opioid, or a combination thereof. Flunixin meglumine (1.1mg/kg intravenously q12-24h) is commonly used in cases of equine colic as it is cost-effective and provides excellent visceral analgesia. Other NSAIDs available for use in horses include phenylbutazone, meloxicam, ketoprofen and carprofen, however in the author's opinion these drugs provide an inferior level of pain relief compared to flunixin.

In equine practice alpha-2 agonists (namely xylazine, detomidine, and romifidine) are frequently used as sedation, however they also have profound analgesic effects. Intravenous administration of xylazine (0.2 – 1.0mg/kg)[5] can be used to facilitate examination and diagnostic workup of colic cases, and is the author's preferred choice over detomidine and romifidine due to its analgesic potency and shorter negative effects on gastrointestinal motility. The use of opioid drugs in horses with colic may be considered controversial, due to the possibility of gastrointestinal stasis. In a practice setting, the administration of butorphanol (0.01-0.1mg/kg IV or IM)[6] can be useful, particularly if additional pain relief is required whilst allowing time for your other medical therapies to take effect. Similarly, butorphanol can be used in combination with an alpha-2 agonist to facilitate transport to a surgical facility if indicated. Anti-spasmodic drugs such as N-butylscopolammonium bromide (Buscopan™) have no direct analgesic effects and result in significantly decreased gastrointestinal motility. Intravenous administration of Buscopan™ (0.3mg/kg) will frequently provide appropriate relief in horses affected by spasmodic colic, however it's use is seldom indicated in large colon impactions or cases of sand enteropathy.

Fluid therapy: Generally, horses presenting with large colon impactions or sand enteropathy will be normovolaemic, particularly if veterinary attention is sought in a timely manner. However, if these patients are hypovolaemic or dehydrated, intravenous fluid therapy (IVFT) is warranted to restore their circulating volume and improve perfusion. The volume of fluid required will be dependent on the patient's fluid deficit, which can be calculated using an estimate of percentage dehydration and bodyweight. For example, a 500kg horse who is 5% dehydrated will require 20L of fluid to correct the deficit. Isotonic polyionic crystalloids (Hartmanns and Lactated Ringers Solution) are an appropriate choice for replacement fluids, due to the similar electrolyte composition to plasma; plus, they are available as 5L bags.

When implementing IVFT in the field, it is important to consider the facilities available, the time required to administer the required volume of fluid, and the financial implications. Administration of intravenous fluids as a bolus tends to be more practical compared to a constant rate infusion; and typically a rate of 20-40ml/kg is used. Use of a 12-14 gauge catheter in combination with a large-bore giving set will result in a faster flow rate, which can be further encouraged by hanging the fluid bags as high as possible. Re-evaluation of the patient's clinical parameters (ie. demeanour, mucous membranes, capillary refill time, heart rate and pulse quality), should be performed following the first 10L bolus to determine if additional IVFT is necessary. Urine output and urine specific gravity are excellent indicators of hydration; however, care must be taken in using these parameters in horses that have been sedated with alpha-2 agonists, due to the resultant dose-related increase in urine flow. An improvement in gastrointestinal borborygmi is frequently detected following fluid administration, attributable to improved perfusion to the gastrointestinal tract and subsequent improvement in motility.

It is now widely accepted that the correction of large colon impactions is more effective when using enteral versus intravenous fluid therapy, and several studies have demonstrated faster resolution, lower financial costs and shorter hospital stays when enteral fluid therapy is used.[4]

<u>Enteral fluid therapy:</u> The content of the large intestine is predominantly fibrous ingesta suspended in water. As the large colon plays a major role in water absorption, the amount of water containing the ingesta is dependent on the horse's fluid intake and their fluid requirement to maintain appropriate systemic hydration. In times of need (ie. when water intake is reduced or the horse has an increased water requirement), the large intestine acts as a reservoir and the vasculature will draw water from the colon to support the systemic circulatory system, thereby reducing the amount of fluid the ingesta is suspended in.[7] Consequently, enteral fluid therapy is very effective, as it increases the volume of water the ingesta is suspended in, prevents further loss of colonic water and acts to rehydrate and soften the impaction. It is important to remember that enteral fluid therapy will only be effective if the patient's gastrointestinal tract is functional. Therefore, if more than 4ml/kg of gastric reflux is obtained following nasogastric intubation or there is evidence of small intestinal distension or ileus, oral fluid therapy is contraindicated.

It is well reported that the administration of high volumes of isotonic enteral fluids is an effective means of promoting hydration of colonic content and motility through the intestinal tract. It is proposed the gastric distension that occurs following the administration of enteral fluids stimulates the gastrocolic reflex, thereby encouraging peristalsis and the passage of ingesta through the intestinal tract. An isotonic balanced electrolyte solution can easily be made by adding 5g table salt (NaCl) and 5g Lo-Salt (KCl) per litre of water (30g NaCl + 30g KCl in 6L water).[4] Alternatively, 9g NaCl can be added to each litre of water, however most horses presenting with large colon impactions will be hypokalaemic thus the addition of potassium chloride is recommended.

Enteral fluids can be provided from a bucket in the stable (relying on voluntary water intake by the horse) or via nasogastric intubation either intermittently or as a constant rate infusion. In the author's experience, the majority of horses with large colon impactions are unlikely to voluntarily drink sufficient water to maintain their hydration, let alone result in colonic overhydration, even if electrolytes or molasses are added. Administration via a nasogastric tube is therefore the preferred method, as it gives the practitioner precise control over both the volume and timing of fluid administration.[4] To calculate the volume of fluid to be administered, a dose of 10-15ml/kg bodyweight is used, with a dosing frequency of 30-60 minutes.[4,7] Some horses may demonstrate signs of colic following administration of high volumes of fluid, therefore walking them horse immediately after can be beneficial in alleviating their discomfort. When the described volume of fluid and frequency of administration is used, the majority of large colon impactions will resolve within 24 hours, following an average of 5 treatments.[4] However, the number of treatments required to clear an impaction is case-dependant, and the decision to discontinue treatment is made based on evidence of resolution on rectal palpation and the development of cow-pat faeces.

If administering enteral fluids frequently (ie. every 30-60 minutes), the nasogastric tube can be left in place. The tube can be secured to the halter and a muzzle placed to prevent the horse from rubbing it out, particularly if it is unsupervised for the period in between dosing. It is also important to check for gastric reflux prior to administering each dose. If reflux is elicited, abdominal ultrasonography should be performed if possible, to evaluate stomach size, and to ensure there is appropriate small intestinal motility and no small intestinal distension. If small intestinal ileus has developed, enteral fluid therapy should be discontinued. If no small intestinal distension is evident and there is appropriate intestinal motility, increasing the interval between fluid doses is recommended to allow sufficient time for gastric emptying. Reducing the volume of fluid administered should also be considered.

Enteral fluid therapy can also be administered as a constant rate infusion (CRI) using a narrow indwelling nasogastric tube or equine feeding tube; and is useful in cases which do not tolerate high volumes of fluid. Enteral fluid therapy kits are commercially available, however due to the small size of the tube, endoscopy to ensure correct placement of the tube within the oesophagus is recommended. Fluid rates up to 2L/100kg/hr can be used, however most clinicians will use a CRI rate of 1L/100kg/hr.[7] It can be argued however, that administering fluids as a CRI results in loss of the gastrocolic reflex, which is thought to play an important role in gastrointestinal motility and impaction clearance. Constant rate enteral fluid therapy should ideally be administered in-hospital so that the patient can be closely monitored, hence is not routinely performed in a field setting.

A number of different types of fluids have been suggested for the treatment of large colon impactions, including mineral oil, magnesium sulphate, dioctyl sodium sulphosuccinate and sodium sulphate. With reference to large colon impactions, the only use for mineral oil is as a marker of gastrointestinal transit time. Despite being frequently used in practice, mineral oil has no effect on softening or breaking up an impaction,[7] and may even prevent water permeating the impacted ingesta. Magnesium sulphate is commonly used in the treatment of large colon impactions, due to its cathartic properties. Although not as effective as balanced electrolyte solution in improving colonic water content, it has been shown to improve the water content of faeces in the small colon.[7] A maximum dose of 1g/kg $MgSO_4$ is recommended, once daily for up to 3 days. Increased doses can result in magnesium toxicity; the clinical signs of which include tachycardia, tachypnoea, sweating, increased excitability, muscle fasciculations, flaccid paralysis and recumbency. Importantly, magnesium sulphate should only be used once the patient's hydration status has been corrected. Compared to $MgSO_4$, sodium sulphate is a more potent cathartic, however consistently causes significant electrolyte disturbances, namely hypernatraemia and hypocalcaemia.[7] As electrolyte monitoring is not easily performed in a field setting, the use of sodium sulphate is not recommended. There is no evidence to suggest the use of dioctyl sodium sulphosuccinate is more effective than water alone, and given the potentially fatal effects if overdosed, should not be administered.

Treatment of Sand Enteropathy

The treatment of colic secondary to sand enteropathy encompasses the same principles as large colon impactions, and it is important to stabilise the patient in regards to pain control and correction of fluid deficits, acid-base and electrolyte disturbances. Feed should be with-held until appropriate faecal output is observed and colic signs have resolved. Ideally, specific therapy targeted at sand clearance will only be initiated once the signs of abdominal discomfort have been abolished and the patient has been started back on feed.

Psyllium mucilloid either in-feed or administered via nasogastric intubation remains the mainstay of treatment of sand enteropathy and over recent times, a number of treatment protocols have been evaluated for their effectiveness in sand clearance. The recommended dose of psyllium for sand evacuation is 0.5 - 1g/kg, however getting a horse to eat that amount of psyllium in-feed daily for 7 consecutive days is often difficult. Also, it has been shown that repeated daily administration of psyllium (1g/kg bwt) via nasogastric intubation for up to 7 days is more effective at clearing sand accumulations than feeding psyllium at home.[8]

Psyllium, mineral oil and magnesium sulphate (and their combinations) are all commonly used in clinical cases of sand enteropathy. In 2014 Niinistö *et al* reported the administration of psyllium (1g/kg) plus magnesium sulphate (1g/kg), mixed with 15ml/kg water and administered via nasogastric tube daily for 4 days, was more effective in clearing large sand accumulations than either psyllium (in 15ml/kg water) or magnesium sulphate (in 15ml/kg water) alone.[9] In the author's experience, a combination of psyllium (1g/kg) and mineral oil (up to 8ml/kg) administered via nasogastric tube daily for up to 7 days, is also an effective means of clearing large sand burdens; plus, it is much easier to administer the psyllium via nasogastric tube when it is combined with mineral oil versus water. There have been a small number of anecdotal reports of gastric rupture following the administration of psyllium in water (personal communication), therefore it is suggested to allow sufficient time for gastric emptying following feeding, prior to administering the treatment dose.

Treatment of sand enteropathy using any of the above strategies is associated with a risk of colic, thus it is recommended these patients are hospitalised for the duration of their treatment. Not only does this allow them to be monitored closely throughout the treatment, their access to sand can potentially be better controlled. Management strategies aimed at limiting exposure to sand should not only be implemented following discharge, but also throughout the treatment phase. Horses undergoing treatment should ideally be stabled and bedded on shavings, with minimal or no access to pasture. Long-stem roughage should also be offered *ad libitum*.

Prognostication

With reference to both large colon impactions and sand enteropathy, surgical intervention is warranted if a poor response to appropriate treatment is elicited, clinical parameters continue to deteriorate despite medical intervention, or the signs of abdominal pain become refractory to analgesia. Overall, with appropriate medical treatment, the prognosis for short-term survival in horses with uncomplicated large colon impactions and sand enteropathy is good.

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Strategic Deworming: The Current Thinking

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Introduction

One of the most frequently asked questions to equine practitioners is "How often do I need to deworm my horse?" As a result of the development of anthelmintic resistance over the past few decades, the answer to this question has changed considerably. No longer is it acceptable to advise frequent deworming at regular intervals, as was the traditional approach, instead a more selective and individualised approach should be considered. Currently, cyathastomins (small strongyles), ascarids (*Parascaris equorum*) and tapeworm (*Anoplocephala perfoliata*) are considered the most important parasites causing clinical disease in horses. Unfortunately, there remains limited information regarding the efficacy of available dewormers in Australia; nonetheless, anthelmintic resistance is of increasing concern to equine practitioners, and strategies to minimise developing resistance whilst continuing to prevent disease are paramount.

Anthelmintic Resistance

Simply defined, *"Resistance is the ability of worms in a population to survive treatments that are generally effective against the same species and stage of infection."*[1] For resistance to develop in the first instance, resistance genes within the parasite population must be pre-existing, even before a particular drug is used for the first time. When an anthelmintic drug is appropriately administered, only the resistant parasites should survive; and for a short time (until the horse is re-infected with drug-susceptible parasites) they will be the only parasites producing eggs, conferring resistance to the next generation and increasing the resistant gene pool.[2] Consequently, having a population of susceptible parasites consistently present on the pasture that are able to dilute the frequency of resistance genes (refugia), becomes an incredibly important component in delaying the onset of anthelmintic resistance. The rate of resistance development is determined by a number of factors such as frequency of treatment, inappropriate dosing of anthelmintics, mass treatment and single drug regimens; and it is now widely accepted that the traditional approach to deworming, whereby frequent blanket treatment was administered to all horses at regular intervals, has significantly contributed to the development of anthelmintic resistance.[3]

Importantly, the presence or occurrence of resistance is highly variable between geographical locations, thus appropriate testing should be undertaken on individual farms to conclude whether resistance is truly present. Tools used for monitoring parasite infection and detecting anthelmintic resistance include faecal egg count analysis (FEC), faecal egg count reduction test (FECRT) and egg reappearance period (ERP).

<u>Faecal Egg Counts:</u>

Faecal egg counts can be easily performed in practice, and are predominantly used to detect the presence of strongyle and *P.equorum* eggs in the faeces of individual horses. It is important to note that it is not possible to distinguish between large and small strongyles microscopically (larval culture or PCR are required); however, in managed horses it is widely accepted that the majority (>99%) of strongyle eggs identified come from the cyathostomins.[4] When collecting faeces for testing, it is essential to choose the freshest sample (<12 hours). Due to the non-uniform distribution of nematode eggs within faeces, it is recommended multiple samples are obtained from different parts of the manure pile, and that those different samples are thoroughly mixed prior to performing the FEC.[5] If the FEC is not going to be processed immediately, the sample should be anaerobically stored (ie. in an airtight container) and refrigerated.

There are a number of accepted methods for enumerating strongyle eggs in equine faecal samples, however the McMaster (and it's modified versions) and Whitlock methodologies are commonly used.[6] To perform the standard modified McMaster test, a 2, 3 or 4g sub-sample of the collected fresh faeces is dissolved in 28ml, 42ml or 56ml flotation solution respectively. The faecal suspension is then mixed well and filtered through a tea strainer to eliminate large particulate matter. The filtrate is mixed again to try and ensure randomised distribution of eggs, before being dispensed into the 'McMaster' counting slide. The slide is then allowed to stand for 5 minutes, giving the eggs time to float to the chamber surface, before being examined under the microscope (x10 magnification) and the number of parasite ova are counted. To calculate an 'egg per gram' value, a multiplication factor is applied, dependant on how many grids or chambers are counted. By counting all eggs in both chambers (1.0ml faecal suspension), a detection limit of 15 is achieved, resulting in a more accurate count.[6]

Results of faecal egg counts do not accurately reflect the total adult *Strongyle* or *Parascaris* burden of the horse, however they do allow for the categorisation of individual horses on the basis of their egg shedding and subsequent environmental contamination potential. Although horses that graze together are exposed to the same parasite population, individual horses will have markedly different levels of egg shedding. This concept is known as over-dispersion, and it can be assumed that in a group of adult horses, 15-30% of that population will contribute to 80% of the pasture egg contamination.[4,6] Based on the results of their faecal egg counts, individuals will be classified into three groups: low shedders (< 200 epg), medium shedders (200 – 500 epg) and high shedders (> 500 epg).

<u>Faecal Egg Reduction Tests:</u>
The faecal egg reduction test evaluates anthelmintic efficacy, based on its' ability to reduce the faecal egg output following treatment.[7] When conducting a FECRT it is recommended to use a minimum of 6 horses per yard/property that have not received any anthelmintic treatment for at least 8 weeks; and ideally those with the highest pre-treatment egg count will be included.[4] To perform the test, a FEC is performed prior to anthelmintic administration (FEC_{pre}) and again 14 days following treatment (FEC_{post}). The percentage egg reduction (% FECR) is then calculated using the following formula:[7]

$$\textbf{\% FECR} = ([FEC_{pre} - FEC_{post}] / FEC_{pre}) \times 100$$

The American Association of Equine Practitioners (AAEP) created the following table, with suggested cut-off values for the interpretation of strongyle FECRT.[4] In the absence of any published specific guidelines, the values below are recommended for evaluation of test results.

	Expected efficacy if no resistance	Observed Results of the FECRT		
		Susceptible	Suspected Resistance	Resistance
Fenbendazole/Oxibendazole	99%	> 95%	90 – 95%	< 90%
Pyrantel	94 – 99%	> 90%	85 – 90%	< 85%
Ivermectin/Moxidectin	99.9%	>98%	95 – 98%	< 95%

Egg Reappearance Period:

The egg reappearance period can be defined as the time interval between the last effective anthelmintic treatment and the recommencement of significant strongyle egg shedding (AAEP); where significant egg shedding is defined as an epg value >10% that of the pre-treatment value.[8] A shortened strongyle ERP is indicative of reduced drug efficacy and the early development of anthelmintic resistance, and although no Australian-specific information is currently available (to the author's knowledge), numerous UK studies have demonstrated a shortening of strongyle ERP following treatment with both ivermectin and moxidectin.[8]

Goals of Strategic Deworming

The main goals of any deworming strategy include: 1) the prevention of clinical disease caused by parasitic infection; 2) control of parasite egg shedding; and 3) the minimisation or reduction in the rate of anthelmintic resistance development. Ideally, each horse property would have a tailored deworming program, based on their individual FECRT and ERP results. For the majority of yards however, this is considered unfeasible, therefore recommendations for deworming should be based on the individual's contamination categorisation and level of risk for parasitic infection. Individual and management factors considered useful for determining a horse's level of infection risk include: results of repeated faecal egg counts, their age, the number of times manure is collected from the pasture, pasture management, stocking density, presence/absence of youngstock, horse movement, yard quarantine policy, previous history of parasitic disease and previous history of colic.[9]

Regardless of their FEC categorisation, it is recommended that all adult horses receive 1-2 anthelmintic treatments per year (spring and autumn), targeted against large strongyles, tapeworm, bots and *Habronema/Draschia* spp. Ideally, tapeworm antibody testing would be performed to assess whether treatment against *Anoplocephala* is in fact required, however at present this diagnostic is not widely available in Australia. Horses categorised as "medium shedders" may require an additional treatment (2-3 treatments per year in total) and "high shedders" may be dewormed 4 times per year, with the target parasite being cyathastomins. Close attention should be paid to the timing of anthelmintic administration, and ideally treatments will be performed at times of peak parasite transmission (typically spring and autumn), when levels of pasture refugia are high.[4] To preserve the long term larvicidal efficacy of moxidectin, routine use is not recommended; rather it should be reserved for use in horses with clinical disease.

Selective anthelmintic therapy is not recommended for young horses, due to their reduced immunity and increased contamination potential. During their first year of life, it is recommended foals be dewormed at least four times, approximately 3 months apart. The first deworming is advised at 2-3 months of age, using a benzimidazole effective against *Parascaris spp*. The second treatment should be administered prior to weaning (at 4-6 months or age), where the anthelmintic used is based on whether strongyles or ascarids make up the majority of the parasite burden, as determined by a FEC. The third treatment is typically undertaken at 9 months, where strongyles and tapeworm are targeted; and a fourth treatment, targeted at strongyle parasites, is administered at 12 months of age. Following this, yearlings and 2-year-olds should continue to be treated as "high shedders."[4]

Environmental control is an important part of a successful deworming strategy, and improving pasture hygiene is one of the most effective means of minimising egg contamination. In order for horses to become infected with strongyles, they must ingest the infective larvae from the pasture. As the lifecycle of a strongyle parasite begins as an egg in a manure pile, removing the faeces at least twice a week will significantly limit the number of larvae that subsequently develop, especially during the times of year when the weather is mild (10 - 33°C). Frequent and routine removal of manure from the pasture may also help to prevent the development of "roughs", which are known to act as a reservoir for parasites. Although it is desirable to minimise pasture contamination with parasite eggs, the goal is by no means to eliminate parasites completely. By adopting a selective approach to anthelmintic therapy, preservation of refugia is possible, ensuring continued exposure to a population of drug-susceptible parasites.

Consideration should also be given to maintaining appropriate stocking densities and the consistent grouping of horses. Greater stocking densities are likely to result in greater parasite burdens, particularly when young horses (<2 years of age) are paddocked together. Younger horses (foals, weanlings and yearlings) typically excrete higher numbers of parasite eggs, due to their reduced immunity against parasites, thus parasites are likely to accumulate more rapidly. Consequently, frequent manure removal from these pastures should be prioritised.

Over the past few years there has been some concern regarding the re-emergence of *Strongylus vulgaris*, associated with the implementation of selective anthelmintic therapy.[10] Interestingly, it has recently been reported that *S. vulgaris* is endemic in Australian wild horse populations.[11] As the opportunity for transmission to domestic horses is certainly possible, equine practitioners should be aware of the potential of clinical disease associated with infection, however this should not discourage a continued effort to target anthelmintic therapy. Additionally, by adopting a selective approach to deworming, it is anticipated that equine veterinarians will regain some influence and control over parasite management, which is desperately needed to slow the rate of development of anthelmintic resistance in horses.

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Decision making practises in practice

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Introduction

As veterinarians we make decisions all day every day, some routine and others critical. Decisions are made at a variety of speeds, based on intuition or made heuristically, from evidence based practise and analytical assessment. We are considered to be inherently good decision makers and trained to refine these skills from our high school years and then throughout our veterinary careers. However, do we take time to reflect on how we are making these decisions, what it is we do not see or consider, or if key elements or considerations are possibly even hidden by our own bias, thinking preferences and efficiencies. Are we at risk as a profession of becoming overly efficient in diagnostic decision making and problem solving, such that we are taking an increasingly linear, predetermined path towards our diagnosis? Do we recognise the power of "solution seduction" or the in-practice-implications of time constraints, inconsistent access to appropriate resources and a community culture of peer and experiential or anecdotal support. If so, do we have the tools to identify when this is happening? This paper seeks to explore some key aspects of decision making, how we identify when our decision-making approaches are at risk and questions what resources would better support a more holistic approach to not only making but reflecting on how we make diagnostic decisions as veterinary professionals.

The "art" of diagnosis

"Pathology is a scientific "art" requiring continuous learning and professional development, particularly as diagnostic challenges are continually emerging. " [1]

What is commonly described as a stepwise process, a linear progression towards a satisfactory (satisfying) end result, is actually far from linear. It requires us to capture, distil, query and conclude against a range of unknown outcomes, accommodate a wide range of variables and concurrently apply introspection and extrospection to a situation. Many models of thinking and problem solving exist, with those such as the Markov Model focussed on a medical or clinical setting and others, such as "design thinking" or "human centred design" more broadly applied for a diverse range of approaches, and currently in vogue.[2] However, rather than seeking a predetermined frame, considering some of the key elements that underpin how we make decisions and embracing our personal strengths is more likely to generate a sustainable, flexible, consistent and robust, personalised approach.

Bias

"We are increasingly challenged with novel syndromes…." [1]

As veterinary scientists we are familiar with the concept and potential impact of bias, although mostly in the research or experimental frame. Reflection on how we productively or presumptuously apply bias in a diagnostic sense may provide some insight into how we might better capture the broader context of a situation and possibly generate a different approach or outcome. Bias has been explored in social research for decision making in medical and veterinary practitioners and has been documented in how we approach exploring a case (farm, herd or individual animal level), apply treatment recommendations and explore diagnostic pathways.[3] The research outcomes determined that most commonly, at least for experienced veterinarians the way decisions were made was based on positive similar experiences or a history of encountering and dealing with similar presentations. Medical practitioners have also been documented to preferentially follow a particular treatment pathway, despite it being identified as a suboptimal treatment choice, or apply particular diagnostic tools, guided by their past experience rather than the evidence apparent in real time. When participants were formally requested to apply an evidence-based approach, some of these outcomes were adjusted favourably. [3]

Extending conclusions towards a differential diagnosis, or "most likely scenario" without bias, is almost an inhuman feat. Our thinking preferences and the way we assimilate data creates bias. This is an essential process that means we do not become overwhelmed with data and experiences and are able to prioritise and filter, such that we can make decisions and move through each day. As a cohort, veterinarians' thinking preferences tend to gravitate towards data, information, process and rigor and this serves us well as a practitioner. That is not to say imagination and creativity are wholly absent, however, we refine these inherent data filtering skills and thinking preferences from a young age, through our focus in school and then as we are successfully selected into the profession. All these successes are built on our ability to perform particular types of tasks and solve problems to expert level. This is increasingly refined and celebrated as we progress through our undergraduate training and further reinforced in our professional years, compounded by financial and time metrics. The more able we are to recognise how we bias or filter data, what our thinking preferences are and how this shifts under emotional, situational or physical stress, can facilitate us executing a more effective and holistic decision making process in all situations, professional and personal.

Experiential learning, presumption and efficiency

"Older veterinary scientists mostly find it remarkable that so many new disorders have emerged in their careers of veterinary public health." [1]

Our decision making can be improved by being consciously aware, amongst the time poor and high pressure environment in which we often work, that the questions we choose to ask, and how we ask them will determine the answers we receive. As we take the first steps along a diagnostic pathway, are we attempting to prove our intuitive diagnosis correct or explore or even disprove this and demonstrate there are things we don't yet understand? Finding the balance in these approaches such that we are able to move forwards, not encumbered by doubt or facilitated by overconfidence, but consciously considering each step we take is a process that takes practice and continuous reflection and refinement. I would postulate that we are a community ill at ease with the concept of leaving unknowns unknown, that is, the pressure to make a diagnosis can often be overwhelming in driving clinical decision making, narrowing our perspective to what we already know to be true and creating more efficiencies or introducing bias. Are we able to comfortably approach a case with an acceptance that a definitive diagnosis may in some cases, in fact not be possible? It is reasonable to accept that there will always be times when relying on our intuition is appropriate. However, although we are commonly lauded by our clients and peers for this ability, teasing apart assumption based on experience (intuition) and applying this skill in a more rigorous, evidence based framework offers the opportunity to better celebrate our strengths.

Developing evidence based practises requires practice

It has been proposed, investigated and demonstrated that veterinarians commonly adopt an approach that has been deemed imperfect in the context of applying evidence based medicine (EBM). Research identified that consultation with colleagues, specialists, laboratories, and the Internet rather than a scientific databases or peer-reviewed literature is common practice - again driven most often by time available and the practicalities of access to these resources. It was concluded that more time spent in this frame in undergraduate training, would create efficiencies such that this time spent could be optimised and therefore more likely utilised in a clinical setting. Ensuring mentors and teachers openly and overtly apply these approaches, offering tips and tools to how to do so efficiently in a clinical setting, may also assist in establishing and reflecting on how we make decisions as graduate veterinarians. This is supported by an identified and documented gap or lack of understanding of perspective and the lived reality of practice between academics providing training in these approaches and those compelled to apply them in a practical setting. A need therefore exists in veterinary training to facilitate closing this gap.[4,5]

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"From over the Fence?"
What is the future role of vets in extensive livestock farm businesses?

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Overview

The opportunities and challenges facing the veterinary profession in service provision for animal health to livestock owners has been well documented and described, including exploration of key ways to leverage change.[1-5] Despite this there has been no considerable difference in approach, engagement nor repositioning of the profession, even in the face of an overtly expressed desire for change. A number of hypotheses have been presented and explored in previous workshop and survey work as to why this status quo remains. These include challenges of a fiscally sustainable business model for service provision by veterinarians in general practice, the lack of training, mentoring and employment opportunities for young vets or those with a desire to offer these services, competition with other service provision and in particular veterinary services or animal health advice perceived free of charge (through resellers, government vets, industry bodies and the pharmaceutical industry), and the deep seated culture and values of our profession, most notably how we identify and are perceived as veterinarians by farmers and farm communities. This paper seeks to provide an overview of discussion, findings, collective learnings and opinion explored in a workshop conducted at the Australian Sheep, Camelid and Goat Vets conference in Melbourne in late 2018, augmented by debate conducted at the Australian Cattle Vets conference in 2019 and survey results conducted in other projects (2017-2018), indirectly related to the key issue but highly relevant to veterinary service provision in this context.

The majority of information provided comes from comprehensive discussion at the Melbourne workshop (SCGV 2018). Here workshop participants were asked to consider the current situation and challenges for veterinary service provision to large scale, grazing livestock farm businesses in the following frames;

- What is the current situation (where are we now)?
- What would we like the situation to be (where do we want to be)?
- What is required to make that change (how will we get there)?
- What are the most important aspects to act upon now (best/most important next steps)?

The workshop offered to delegates of this conference (AVA Perth 2019) seeks to utilise the information in this paper to further progress this discussion, working towards developing and formally proposing alternative approaches or models for veterinary service provision and identifying who is best suited to lead and facilitate this change.

What is the current situation (Where are we now)?

The Melbourne workshop concluded that considerable opportunity exists "the time is right to champion this change" that vets are inherently trusted in society personally and professionally, are identified as best positioned to provide comprehensive animal health and biosecurity advice and oversee surveillance activities and that other sectors perceived as competition in providing these services are in decline (government vets). This is further augmented by buoyant livestock markets and high commodity prices, a new generation of farm owners and land managers who have not experienced the old (or existing) paradigm of service provision, and a consumer sentiment of increasing expectation and transparency around animal health and welfare, and for food security and safety. However, participants felt that vets have been historically poor at self-promotion, or worse, have enabled the perpetuation of a perception that we provide acute response services in a high cost, high input model of business and are not able to engage in a more holistic sense with farm businesses - reflected directly as a lack of confidence from farmers in the appropriateness of vets to provide the services their business needs.

It was also identified that there are a lack of post graduate training and mentoring programs to support vets who want to upskill or transition into these roles, and that where training exists, it is outdated, overly academic or not adequately contextualised to current and future industry needs. A reflection on undergraduate training concluded that undergraduate curricular are already overwhelmed by content required to meet day-one-competencies and overseas accreditation requirements. Further discussion unpacked a sentiment that there is additional risk for graduating veterinarians to become quickly disheartened and in fact feel anxious or underprepared in offering services to livestock owners. As current veterinary business models don't support these services, this leads to a loss in confidence and competence in key skills that were developed in their undergraduate training (eg diagnostic investigations of livestock, post mortem examination, understanding feed requirements and pasture management for livestock health). Participants also felt that not only was there a lack of vets with adequate skills, there were no overt plans for succession, with a large proportion of vets currently servicing farm businesses in a particular generational bracket and creeping closer to retirement.

A valid question was asked – are vets in fact the best people to provide these services, are the challenges with time constraints, remuneration, motivations too great and does the next generation of veterinarians in Australia want to pursue this – if so, is there a model of service provision and professional interaction with farm businesses that has to date not been explored?

What would we like the situation to be (where do we want to be)?

It was agreed there is a need for greater unity in approach to addressing the underlying challenges and for working toward the development of a trusted, competent, confident, sustainable, relevant community of professionals providing routine and expert advice to farm businesses. The concept of working in partnership with other service providers such that networks of advisors offer greater value to farm businesses was raised. This was in contention to the "us and them" culture of many traditional approaches to service provision where the perception is that advisors work in competition rather than to augment and support one-another's skill base, and are mostly working in isolation either as individuals or within businesses.

The development of a new culture supported by and invested in continuous learning was explored – where networks are built and then revisited and consolidated or strengthened over time, recognised broadly by producers and industry for the expertise this would offer. The need to build and maintain optimism was also discussed, to increase the likelihood that people are drawn to the concept which in turn is framed in a positive, achievable light rather than what was reflected as a fairly negative, unsuccessful and challenging one currently. The need for work to change the external frame within which vets are perceived was also raised. The workshop offered that currently vets are perceived in a particular way which limits engagement by farmers. Changing this perception, even possibly changing terminology used to differentiate vets who are providing consultancy services may assist in creating a change in perception for external parties (farmers, farm communities, other service providers) This concept was also raised in the ACV debate, where the room fell divided, some vocally loyal and unwavering in the veterinary practitioner frame, insisting the change was required from outside of the profession not within.

What is required to make that change (how will we get there)?

A process of consultation was proposed, to better understand what landowners and farm mangers want, rather than crafting change based on the assumption that this is already known. It was equally felt, that opportunity exists to show producers what they do not know or understand about veterinary service provision, as what is currently in view fits a more traditional frame of the in-practice-vet, or one who has little knowledge of farm businesses. Working to establish and offer a more holistic advisory service was identified as not only a way forward but necessary to be able to be perceived as of value to farmers; offering a grass-roots-based service, spending time on-farm building trust and rapport. This concept was explored at the ACV debate, where it was recognised that this is not traditionally something vets have done well, if at all, where we are commonly time poor and limited by the concept of "chargeable hours" such that a paddock walk or kitchen table discussion is almost unachievable in most practitioners' day.

Leveraging existing reasons for veterinary contact was explored, for example through prescribing or regulatory activities (ram sedation/antimicrobials), consumer expectations of veterinary input into animal welfare, biosecurity, traceability and transparency in food production, and learning from existing successful businesses to better understand how to transition to this type of service provision. The development of mentoring networks supported by industry or professional organisations was proposed as a pathway to utilise and build on existing social and professional infrastructure.

Both the workshop and debate acknowledged the need for a more proactive approach to marketing and to create unity and alignment across veterinary sectors (government, private, industry). A more creative approach to the development and use of on-line resources such as YouTube, Twitter, web based platforms etc was proposed and to provide user friendly, pithy, seasonally relevant advice to engage the new generation of tech-savvy producers.
The workshop discussion started to explore some of the changes required to veterinary businesses to facilitate providing the services identified. The need to create teams of professionals working together was again offered, including identifying that changes in mindset may be required to do so. It was proposed that the profession is more likely to manifest change if we work on what is within our influence and control, and recognise our limitations, but not be limited by our perception of what is possible based on past experience.

What are the most important aspects to act upon now (best/most important next steps)

To distil the discussion, participants were asked to prioritise what they saw as the key changes required and the next best steps to take. The following list provides a brief summary of what were considered the most important areas to prioritise in either action or building understanding;

- Be prepared to take advantage of the changes in consumers expectations and their engagement with food production - for many there is probably an assumption that vets are already involved in farms
- Do this collectively as a profession not in competition - collaborate with other experts
- Have an aligned voice and approach
- Consult with producers
- Upskill in basic skills that demonstrate competence
- Leverage contact with farmers eg through biosecurity plans, prescribing, grass roots activities etc
- Better understand if vets (especially younger/next generation) view this as a good business opportunity? – is this what the profession wants?
- Change the way we promote/talk about ourselves as a profession

- Explore alternative business models for provision of these services more collectively – learn from successful existing businesses.
- Create mentoring opportunities
- Work towards creating the mindset/attitude change required within our profession
- Advocate for support from industry, government, pharmaceutical industry to give credence to these proposed services

Intentions for AVA Conference Perth 2019
The Perth workshop will aim to build on these learnings to discuss and develop priority strategies for actualising the changes identified as key. If appropriate, these will form the basis of a formal proposal to industry, professional organisations and government to further develop and resource our profession in this direction.

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Handling horse emergencies at an event

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Handling horse emergencies at an event can be a challenge for even the most experienced veterinary practitioner. Emotions can run high, control of both the animal and the human element is required, and often a veterinarians clinical skills are the least useful part of the equation.

Types of emergencies

Incidents at horse events come in all shapes and sizes. Some involve single animals and sometimes there are multiple animals and possibly riders involved as well. Incidents may also involve structures such as jumps, mobile starting barriers, fences and floats. Water hazards may also be involved.

Plan ahead with the event organisers

Where practical meet with the event organisers to ascertain what physical provisions they have in place to assist you to deal with horse emergencies. This can include discussion around the roles of various event personnel, horse handling staff, provision of horse ambulances/floats (winches), disposal of deceased animals, privacy screens and crowd control measures. Having adequate crowd control measures in place is important for the safety of the animal, the vet, the public and the attendant staff. Have a good idea of the layout of the grounds and where the various resources are. Don't assume plans are in place when they may not be.

Medications and equipment

At a minimum the veterinarian should have a lightweight easily carried bag with the following in it; euthanasia solution, suxamethonium, needles/syringes of various and appropriate sizes, anti-inflammatory injection, sedation, butorphanol, ketamine, dexamethasone injection, flunixin, anti-spasmodic injection, eye ointment, penlight, bandages, cotton wool roll, Kimsey splint, rubber tourniquet bandage, wound spray,

Have an emergency drug dosage chart i.e. sedatives/anaesthetic doses, laminated in your bag – under pressure it is helpful to have these dosages written clearly and accessible.

Assessing the incident

Ensure you have transport made available to get to the location of the injured animal. Assess the surroundings before proceeding – are there loose horses in the vicinity, has the progression of other horses on the course been halted? The immediate safety of both yourself and other people is a responsibility.

Assess the location of the injured horse – are there any hazards nearby i.e. water, ditches, electricity?

TLAER courses

Technical Large Animal Emergency Rescue courses are available in most states via the EVA or state horse council bodies. These courses generally make use of life size and weight horse mannequins and are very useful for the equine practitioner who officiates at horse events. The courses generally teach techniques from simple drags for moving horses trapped under fences etc. to more complicated procedures for moving horses from ditches and water hazards. Glides or slides are very useful equipment to have at an event as it may be required to move an anaesthetised or deceased animal onto an ambulance/float.

Media inquiries

Often following a horse emergency or serious injury at a high-profile horse event there may be media inquiries. These inquiries may at times be directed straight to the practitioner that was present. It is important to have a good knowledge of the expectations of the event organisers in respect to their media policy. For experienced horse event organisers they will have an established protocol whereby media inquiries will be directed towards and handled by a communications team. In these cases it is essential that any inquiries are re-directed to the event organiser's communication steam for comment. Veterinarians should not make individual comment on these occasions. However in many cases at lower level events the event organisers will not have a clear media policy or communications plan so it is essential that the vet has a good understanding of what is expected of them in this regard. It would be broadly advisable that in any situation veterinarians refrain from making comments to media unless they are directed to by the event organiser. There may be incidents where there is associated human injury or even fatality and "off the cuff" comments may later be scrutinised as part of any investigation into the incident. It may not be determined on the day of the event what the causative factors are so unsupported comments are not advisable.

Post mortem examinations

The practitioner should always be prepared to have to write a report to an insurance company where a horse has died or been euthanased. If the owner of the horse is not present at the time of the injury the insurance status of the horse may be unknown until later.

Taking photos/video of the injury and any relevant peripheral areas, hazards etc. is always advisable as they may be useful later in a report to the insurance company. Also consider developing a process to send the horse for post mortem examination where required and practicable – this is especially useful in the case of unexpected death, cases where the injury is not externally appreciable, or situations where there is severe injury to a person or human fatality as these matters often end up being examined by a higher authority i.e. Work Safe or Coroners court.

Floats

Where animals are transported to events in floats or trucks it is a possibility that the practitioner may have to deal with animals that are entrapped within float structures. This is an example where TLAER courses can be invaluable. Access by the vet to sedate the animal can also be compromised. Pole syringes for sedation can be invaluable tools for sedating animals in restricted space. Consideration may have to be given to anaesthetise an animal for removal from structures such as floats or barriers and glides are useful here.

Biosecurity

Consider biosecurity issues – this is especially relevant in areas where Hendra virus is endemic. It is highly likely that the institution receiving any deceased horse for a post mortem examination will require some certification or declaration from the attending veterinarian regarding the health status of the horse or in endemic areas policy may dictate that Hendra vaccination is required before post mortem examination can occur.

Performing a postrace veterinary examination and common findings

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When a horse race meeting is in progress it is a requirement that at least one veterinarian is present on course to carry out both emergency and regulatory roles. It is part of the veterinarian's race day responsibilities to perform postrace examinations of horses.

Why perform a postrace examination

There are several indications why a postrace examination might be required:

- The on course veterinarian is required to monitor horses returning to the mounting yard after the race and identify any obvious issues such as lameness, distress, epistaxis or lacerations. If these conditions are noted in the mounting yard then the veterinarian should perform a follow up examination in the stall area and report to Stewards accordingly.
- Stewards request – usually a runner that under performs according to expectations or the betting market.
- Interference in running – look for abrasions, lacerations to limbs, heels, lost shoes etc. that may indicate interference in running.

When to examine

Where a horse finishes a long way behind the field it is good practice to auscultate the heart immediately upon the horse returning to the mounting yard. This is a good opportunity to detect arrhythmias as some will convert quickly and may not be present when the horse reaches the stall area. The mounting yard also provides an opportunity to observe a poorly performed runner for early signs of distress or heat stress, as they can deteriorate quickly and may be prone to ataxia or collapse on the way back to the stall area. These horses can be identified and closely followed back to the stall area.

For routine postrace examinations as requested by the Stewards it is ideal to let the horse return to the stall area and be hosed down, scraped and allowed to drink prior to being assessed. Ideally this primary examination should be performed at approximately 15-20 minutes postrace.

Areas to examine

Clinical assessment in suggested order:

- Visually assess the horse's general demeanour – this is valuable for ruling out postrace heat stress/distress.
- Heart rate and rhythm – average heart rate is around 100 bpm approx. 15-20 minutes after racing. Heart rates above 100bpm but below 120bpm may be reported to Stewards as "slightly elevated heart rate". Heart rates above 120bpm may be reported as "elevated heart rate". Heart rates greater than 130bpm can often be associated with cardiac arrhythmias.
- Assess respiratory rate and character.
- Epaxial muscle enlargement – variable findings.
- View nostrils for evidence of epistaxis – head and eyes for any injuries.
- Palpate lower forelimbs – tendons, joints and shins in young horses especially important – look for any lacerations especially to hind limbs. Observe feet for lost shoes.
- Upper body for abrasions/haematomas – contact with starting gates.

- Observe horse both at the walk and trot for lameness – trotting in a triangle can be helpful. Generally accepted practice to report the lameness to Stewards as localised to a certain limb or limbs without further clinical diagnosis required.
- Discussion with the trainer can be helpful – recent history/impressions of the way the race was run

Common findings

Some common findings during a postrace examination include:

- Exertional heat illness – otherwise known as "heat stress" – may occur in some horses despite relatively mild conditions but often seen on days of high temperature, high humidity and low wind. Clinical signs can include agitation, kicking out, ataxia, collapse, rearing backwards or lunging forwards.
- Synchronous diaphragmatic flutter – "Thumps" – may be an incidental finding and association with poor performance is unclear.
- Exhaustion/exercise intolerance – increased respiratory rate and depth/nostril flare
- Epistaxis – may be from one nostril or two – racing rules describe what is defined as a "bleeding episode".
- Lameness – a common finding and may be mild to severe in nature. It is not a requirement to diagnose the exact location of lameness, unless clinically obvious, as the Stewards only require a general report on which limb/s are involved - the horse can be referred off course for the trainer to seek further advice from their usual veterinarian.
- Eye injuries – may be the result of being flicked by another riders whip or from a clod of dirt hitting the eye.
- Colic – spasmodic – uncommon.
- Lacerations – location - might signal interference in running or be self-inflicted.
- Lost shoes – toe clip dislodgement can be result of stumbling.
- Cardiac arrhythmias.

Cardiac arrhythmias

It is not uncommon for a racecourse veterinary clinician with access only to a stethoscope for cardiac auscultation in the post exercise period to hear an arrhythmia, but from that limited test be unable to assess whether that arrhythmia is/was likely to be clinically significant and possibly the reason for a poor performance during a race.

In this respect the Alivecor™ monitor can be very useful to the racecourse veterinary clinician in order to collect data in the crucial immediate post exercise phase and then be able to retrospectively analyse this data to determine if the arrhythmia detected was clinically significant or normal. Further investigation of the heart during exercise with a digital ambulatory ECG recorder (Holter monitor) may be warranted.

Correlating the ECG findings with the horse's performance during the exercise is also crucial to consider as normal post-race arrhythmias may be considered incidental findings, and if poor performance was noted this could be due to other factors such as lameness etc.

Using the Alivecor™ Veterinary Heart Monitor during a postrace examination and interpreting the ECG results

The Alivecor™ Veterinary Heart Monitor is a mobile veterinary electrocardiograph (ECG) recorder for use on dogs, cats and horses. The device obtains a single-lead ECG trace and transmits that data to the iPhone™ where it can be viewed in real time via an "app". The data is then also wirelessly transmitted to a server or "cloud" which is used to store, view and print ECG traces from the data collection.

To use the recorder - wet the intended area (left side chest wall over the heart) and place the monitor at a 45 degree angle parallel to and above the long axis of the heart. Orient the monitor so that the iPhone's™ round button is closer to the animals head for proper polarity - otherwise the ECG trace will be displayed upside down.

When the heart rate is extremely rapid after exercise the recording speed on the device is best set on 50mm/s, rather than 25mm/s, in order to best evaluate both the complexes and complex intervals.

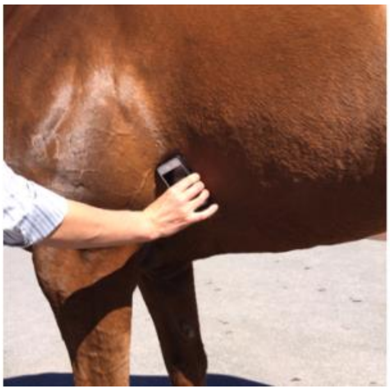
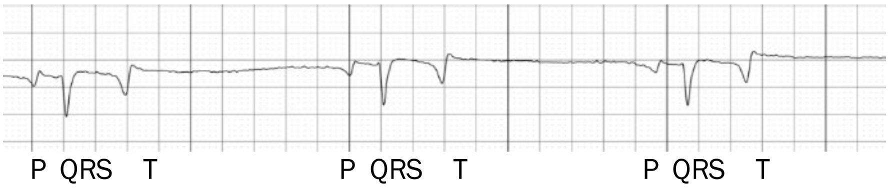


Image 1. Correct orientation and placement of the device on the horse.

A normal base-apex ECG appears as such;



Some examples of ECG recordings from Thoroughbred racehorses recorded immediately after racing (all flat races) using the Alivecor™ monitor are discussed below.

<u>Horse 1</u>

Signalment - 7-year-old gelding which finished 13/13 (1300m race distance) beaten 10.25 lengths.

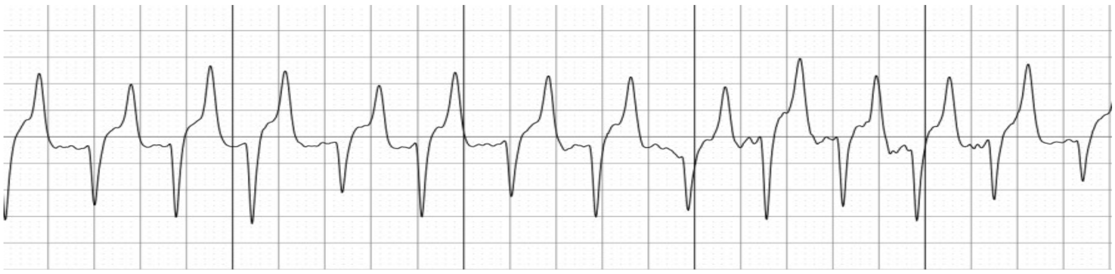


Figure 1. ECG recorded 20 minutes post racing.

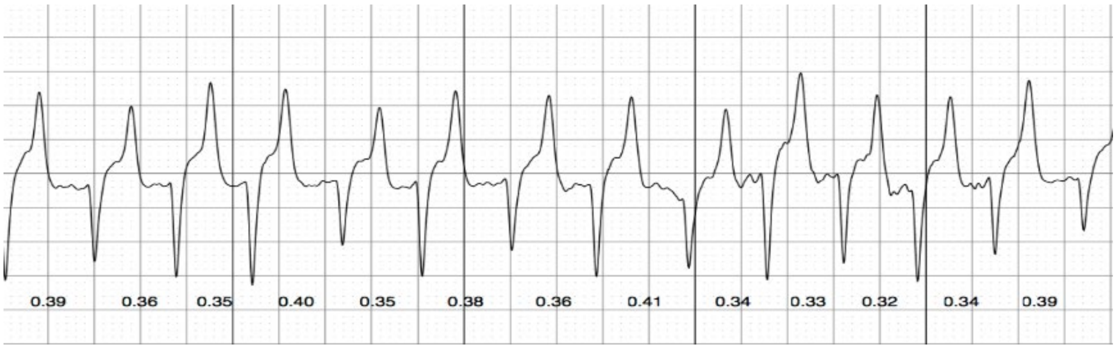


Figure 1a. ECG with QRS intervals measured manually

After the ECG was recorded the QRS complex intervals were measured. This ECG trace revealed an irregular rhythm with irregularly and inconsistently spaced QRS complex intervals. Further there is an absence of the appearance of clearly defined P waves. Due to the rapid heart rate it can be difficult to appreciate the F or fibrillation waves in this ECG recorded immediately post exercise. This is a pattern typically associated with atrial fibrillation.

<u>Horse 2</u>
Signalment - 5-year-old gelding finished 8/9 (1200m race distance) beaten 7 lengths. A post race veterinary examination requested by Stewards revealed a cardiac arrhythmia on auscultation.

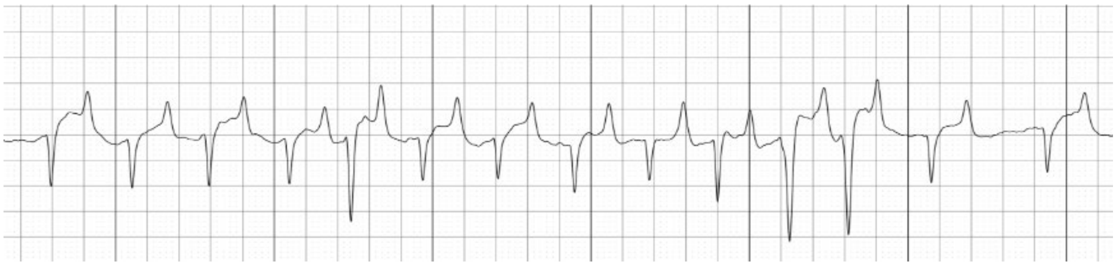
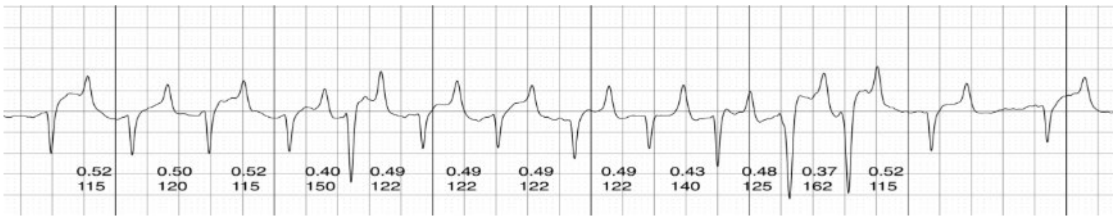


Figure 2. ECG recorded 20 minutes post racing.



SVPD SVPD SVPD

Figure 2a. Same ECG in Figure 2 with QRS intervals manually measured. Heart rate at each QRS complex has also been manually calculated using QRS intervals and ECG speed (25mm/s) as inputs.

Clearly there are three supraventricular premature depolarisations (SVPD) that occurred during this section of the ECG as indicated. These premature beats would explain the arrhythmia that was auscultated. Note the amplitude variation of the SVPD's. A supraventricular premature depolarization is characterised by a premature beat (QRS complex) followed by a normal P wave to P wave interval (re-set). P waves are identifiable within the ECG. Runs of SVPD's are termed atrial tachycardia.

<u>Horse 3</u>
Signalment – 2-year-old filly finished 9/10 beaten 69.2 lengths.

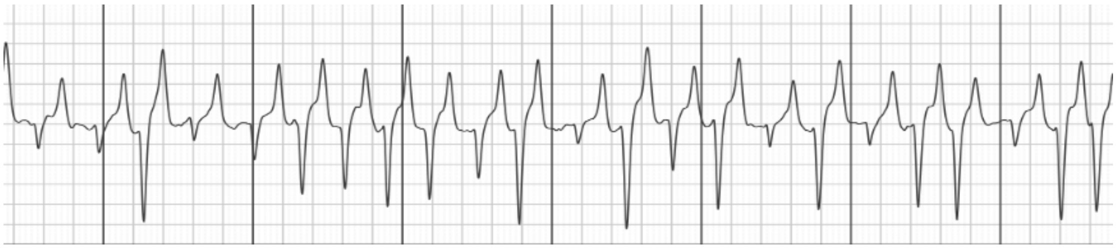


Figure 3. ECG recorded 23 minutes post racing.

From the ECG in Figure 3. the heart rate (ventricular contraction rate) is measured at an average of 25 beats per 8 seconds (187 beats per min) when using the QRS complexes to calculate this figure.

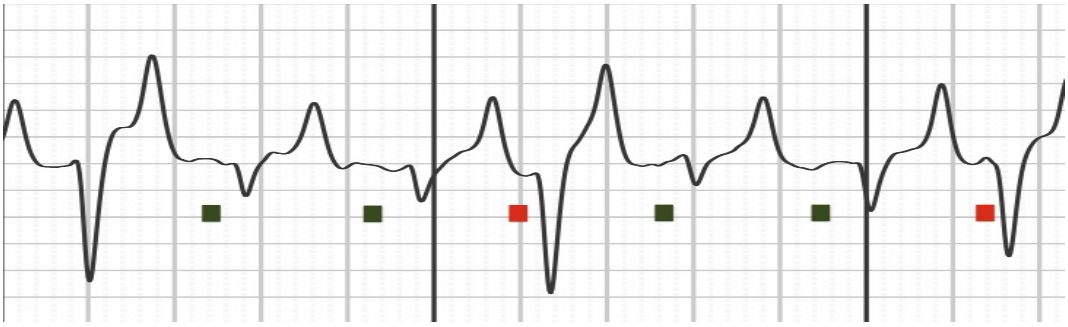


Figure 3a. The P waves (atrial contractions) are marked on this section of the Figure 3. ECG in green. The red marks indicate an absence of a P wave immediately prior to a QRS complex.

From this section the atrial heart rate is calculated as 22 beats per 8 seconds (165 beats per min). This rate is lower than the calculated ventricular rate (QRS) of 187 beats per minute due to the intermittent absence of P waves indicated in Figure 3a.

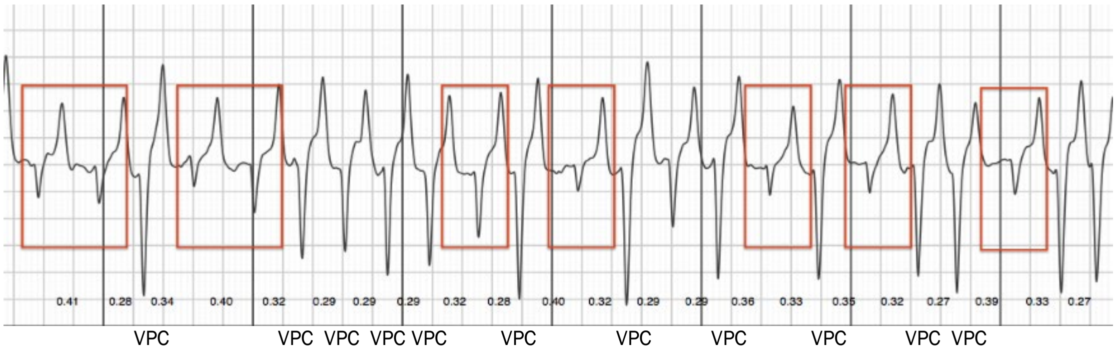


Figure 3b. Same ECG recorded in Figure 3. with QRS intervals manually calculated and the ventricular premature contractions (VPC) marked.

This filly is having both isolated, and runs of, ventricular premature contractions (VPC), which are characterised by an early QRS complex that is not preceded by an atrial contraction or P wave. Whilst isolated VPC's in the post exercise period are not considered abnormal current guidelines suggest that VPC's, otherwise known as premature depolarisations (PD), are clinically important if >2 isolated PD are detected during peak exercise; or if multiple (>5) pairs or paroxysms of PD are detected immediately after exercise.[1]

Horse 4

Signalment – 5-year-old mare finished 11/13 beaten 39.7 lengths (2000m race distance). A post race veterinary examination revealed a cardiac arrhythmia on auscultation.

Figure 4. ECG recorded 20 minutes post racing.

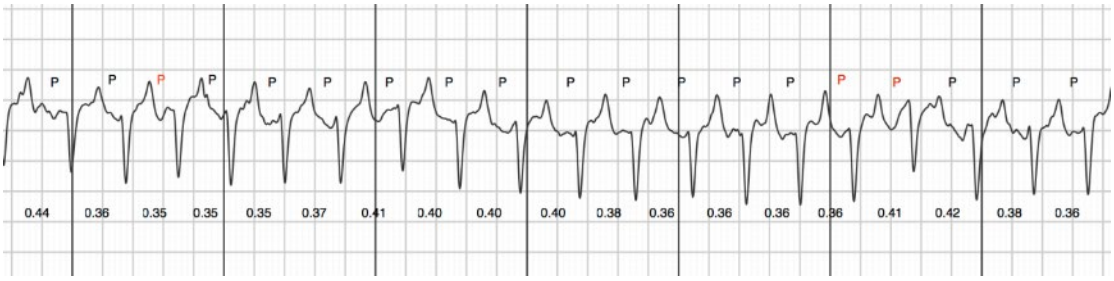


Figure 4a. Same ECG in Figure 5. with QRS intervals calculated and P waves marked.

In this case the heart rate is speeding up and then slowing down. P waves are present. This is an example of sinus arrhythmia at a high heart rate. The mare was retired from racing and so lost to follow up.

Aetiology of common post exercise arrhythmias

Hypoxia and electrolyte imbalances have been shown to result in ventricular arrhythmias during and after exercise.[2,3] Large fluctuations in autonomic tone are said to account for sinus arrhythmia, second degree AV block and isolated premature depolarisations in the immediate post exercise period.[4]

TABLE 1: Prevalence of cardiac arrhythmias during exercise in Thoroughbreds in training with no known performance limitations

	Warm-up (n = 105)		Exercise (n = 101)		Recovery 1 (n = 99)		Recovery 2 (n = 98)		Post exercise (n = 99)		At least 1 phase (n = 98)	
	N	P (CI)	N	P (CI)	N	P (CI)	N	P (CI)	N	P (CI)	N	P (CI)
Sinus arrhythmia	55	52.4% (43.1–61.8%)	0		3	3.0% (0–6.3%)	22	22.4% (14.3–30.5%)	28	28.3% (19.66–37.0%)	64	65.3% (55.9–74.4%)
Second degree atrioventricular block	3	2.9 % (0–6.0 %)	0		2	2.0 % (0–4.7%)	15	15.3% (8.3–22.3%)	2	2.0% (0–4.7%)	16	16.3% (9–23.6%)
Ventricular premature depolarisations	13	12.4% (6.2–18.6%)	3	3.0% (0–6.3%)	8	8.1% (2.8–13.4%)	7	7.1% (2.1–12.1%)	6	6.1% (1.5–10.7%)	28	28.6% (19.6–37.5%)
Supraventricular premature depolarisations	11	10.5% (4.8–16.2%)	0		7	7.1% (2.1–12.1%)	8	8.2% (2.9–13.5%)	4	4.0% (0.2–7.9)	22	22.4% (14.2–30.7%)

n = No. of horses in which ECG was successfully recorded; N = no. of horses in which arrhythmia was found; P = prevalence; CI = confidence interval.

Table 1. Prevalence of cardiac arrhythmias during exercise in Thoroughbreds in training with no known performance limitations.[5]

Table 1 demonstrates that a high proportion of horses in this study were shown to have premature depolarisations in the warm up period. As hypoxia and electrolyte imbalances are unlikely to be present and cause arrhythmias in the warm up phase, psychological factors and fluctuations in autonomic tone were more likely to be the cause of premature depolarisations in the warm up phase.[5]

In the post exercise phase second degree atrioventricular block and sinus arrhythmias were more commonly detected as during this phase the sympathetic input is decreasing rapidly and vagal tone is becoming more prominent. Second degree atrioventricular block and sinus arrhythmias tend to disappear as the heart slows further.

Discussion

Sinus arrhythmia, second degree atrioventricular block and isolated premature depolarisations are known to occur in normal horses at rest and following exercise.[6,7,8] Horses frequently develop dysrhythmias in the post-exercise period[5] and many of these dysrhythmias are considered to be normal and are most likely caused by a rapidly changing autonomic nervous system control. However, it is thought that any form of arrhythmia at peak exercise is abnormal.[2,3,4] Current guidelines suggest that premature depolarisations (PD) are clinically important if >2 isolated PD are detected during peak exercise; or if multiple (>5) pairs or paroxysms of PD are detected immediately after exercise.[1]

A high prevalence of isolated premature depolarisations detected in the post exercise period may signal a concern, and warrant further investigation though, as supraventricular premature depolarisations can be the inciting cause of paroxysmal atrial fibrillation, while isolated ventricular premature depolarisations can trigger ventricular tachycardia or even fibrillation.[5] Paroxysmal atrial fibrillation (PAF) is an important cause of poor performance, as distinct to many other normal post exercise dysrhythmias, however PAF can be difficult to document because horses often convert to a sinus rhythm before it is possible to obtain an electrocardiographic examination.[9] Frequent runs of SVPD's in a 24 hour period after exercise might support a tentative diagnosis that PAF had occurred during that exercise. Likewise, frequent runs of VPD's in a 24-hour period after exercise may indicate that a ventricular dysrhythmia was present during that exercise.[9]

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Take my breath away… A highway to the danger zone

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Introduction

There has been a massive rise in the popularity of many of our brachycephalic breeds in the past few years, especially Pugs, Boston terriers and French Bulldogs. Advertising campaigns and movies have been partially responsible for the increase in numbers of these dogs as pets, as well as a perceived increase in the number developing obstructive airway syndrome as breeders and owners select for some of the "cuter" facial characteristics. In recent years, new obstructive conditions and treatments have been described adding to our knowledge on brachycephalic airway syndrome. The aim of this presentation is to review in a systematic fashion current understanding of this condition.

Pathophysiology

Brachycephalic conformation of a greatly foreshortened facial skull is the result of premature fusion of the basicranial centres, particularly between the basioccipital, basisphenoid and presphenoid bones, which then limits growth and shortens the longitudinal skull axis. This culminates such that all the normal oral and nasal tissues must exist in a smaller compact area. These tissues are compressed leading to regions of narrowing and increased resistance to normal air flow. Regions of narrowing include stenotic nares, relative hypertrophy of turbinates with aberrant turbinate formation and mucosal contact in the nasal passage, potential nasopharyngeal narrowing due to macroglossia and elongated soft palates causing dorsal occlusion of the larynx. Brachycephalic dogs must therefore produce an increased respiratory effort to overcome these regions of airflow resistance in order to allow a sufficient volume of air to enter the chest during inspiration to meet respiratory requirements. As air is increasingly "suctioned" in relative to physiological needs and the severity of the fixed obstructions, the velocity of the air flow must increase. According to the Bernoulli principle, an increase in the speed of air flow occurs simultaneously with a decrease in pressure. It is this principle that explains standard wing engineering to allow aeroplanes to fly. Unfortunately, in brachycephalic dogs, the decrease in luminal pressure resulting from increased air flow speed leads to constant concentric drag on the lumen walls resulting in inflammation, oedema and eventually stretching or eversion of tissue and lumen collapse. As this occurs, airflow resistance is further increased and the ability to adequately respirate worsens creating a disastrous cycle.

Additionally, brachycephalic dogs have been shown to have increased goblet cells (mucus-producing) in their upper airway, as well as a thickened submucosa layer, leading to increased mucoid phlegm and exacerbating stertor. They are prone to regurgitation and hiatal herniation, and it has been shown that addressing GI issues peri-operatively will reduce post-operative complications such as aspiration pneumonia.

Stenotic Nares

Due to thickened keratinized covering and oversized dorsolateral nasal cartilages stenotic nares can be easily diagnosed through direct visualisation. Amputation of the dorsal lateral cartilages was first described by Trader in 1949, but a wedge resection rhinoplasty to widen the nostrils is likely the most widely described technique for correction in modern surgical texts. Despite its popularity, little has been published on its effectiveness to increase air flow.

One concern that has been recently raised is the lack of removal of alar fold (an extension of the ventral nasal concha) to the level of the nasal vestibule with standard rhinoplasty, which provides a cosmetic improvement but potentially not a functional improvement to every patient. In 2004, Ellison published a technique referred to as alarpexy in an attempt to provide constant “nostril flaring”. More recently Oechtering has described the vestibuloplasty technique which is a more radical version of the rhinoplasty procedure removing dorsolateral cartilage to the level of the vestibule.

Intranasal Obstruction

In 2016, Oechtering et al. reported on 132 brachycephalic patients following rhinoscopy and nasal imaging. In their study they found 91.7% of patients to have conchal mucosal contact secondary to relative conchal hypertrophy and that more than two thirds of assessed patients had either rostral or caudal aberrant turbinate development further obstructing air flow through the ventral and middle meatuses. As a result of these findings, laser assisted turbinectomy (LATE) was employed as a potential treatment for the disorder. Some regrowth is noted with this technique and often a revisional procedure is required. Computed tomography is another modality that can be used to help document aberrant turbinates and may play a role in aiding clinicians in providing a more accurate prognosis where LATE is unavailable.

Nasopharyngeal Obstruction

Tonsillectomy has often been discussed as a potential aid in helping patients with airway obstruction. The author has often struggled with the concept of oral surgery to improve air flow when most dogs are preferred nasal breathers. A more compelling argument has recently been mounted for tonsillectomy and is referred to the “meat in the box” theory. The argument is that many of these breeds have macroglossia and thickened soft palates. Pressure placed on the ventral aspect of the mandible appears to cause nasopharyngeal and retrolingual airway obstruction as these structures compress on the soft palate and obstruct the nasopharynx. Enlarged tonsils that outside of their crypts add further “meat to the box” resulting in more obstruction to the nasopharynx when the mouth is closed. Using CT, airflow can be modelled through these airways using computational fluid dynamics. This will provide accurate evaluation of which upper airway components are causing the major disruption to laminar flow.

Soft Palate

A long soft palate has been implicated in airway obstruction as the tip of the palate hangs or is drawn into the rima glottidis during inspiration. In 1942 Farquharson first published staphylectomy to address the long palate. In more recent years, studies have looked at the use of vessel sealing devices to perform staphylectomy. The author does not encourage use of these devices and prefers the traditional blade and suture technique having seen several patients have increased postoperative morbidity with their use. Another technique to address the soft palate is Findji and Dupré’s Folded Flap Palatoplasty. This technique allows both shortening as well as “debulking” of the palate in patients with palatine thickening.

Everted Saccules

Described in many texts as stage 1 laryngeal collapse, the eversion of the lining of the laryngeal ventricles is seen in many patients, with some as young as 8-9 months of age. These are essentially the equivalent to having your jeans pockets turned inside out. Treatment via sharp sacculectomy is still the treatment of choice. It is important not to nick the vocal folds when undertaking sacculectomy.

Conclusion

Prophylactic airway surgery to decrease airflow resistance in brachycephalic breeds is often indicated to prevent eventual laryngeal collapse, end stage obstructive disease. Numerous points of static obstruction may exist and a thorough examination of the patient including imaging should be considered to assess potential treatment options. The addition of LATE, vestibuloplasty and folded flap palatoplasty to the list of treatments available for patients with brachycephalic airway obstructive syndrome will hopefully provide some patients improved outcomes in the future. As always, close monitoring in the immediate post-operative period is imperative for all patients.

Homecare advice – what should we be telling clients?

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Introduction

Successful treatment and prevention of periodontal disease requires a combination of professional treatment in the clinic and a personalised dental homecare regime. Owner commitment to perform dental homecare will ultimately effect the decisions we make in the clinic regarding periodontally compromised teeth. For this reason, dental homecare needs to be integrated and delivered in a manner that is realistic, achievable and affordable for both pet owners and practitioners.

Our homecare mission is to reduce the accumulation of dental plaque as a means to prevent gingivitis and/or progression of periodontal disease and maintain good oral hygiene. Dental homecare refers to activities clients can perform for their pets at home on a daily basis to achieve this. It is important to recognise that homecare is not effective for treating pre-existing disease and it is not a substitute for regular professional examinations and treatment. A personalised dental homecare regime should only be implemented after appropriate professional treatment has established a healthy, pain free mouth.

Control and reduction of the accumulation of dental plaque can be achieved by active or passive techniques. Active techniques require the participation of the client and includes tooth brushing, oral rinsing and application of barrier sealants. Whereas passive techniques use ingredients to help reduce bacterial numbers associated with plaque formation, or to impede the formation of calculus by interfering with the binding of salivary calcium to plaque. Passive techniques tend to be more attractive to clients and include the use of dental diets, treats, chews, water additives and dental sealants. Active techniques are the most effective in achieving our homecare mission. We can incorporate passive techniques into the homecare regime but they are not a substitute for active techniques.

A range of products are available and important to assist clients with providing at home dental care in between professional teeth cleaning. Homecare products can be classified into mechanical, chemical, dietary and dental sealants. The Veterinary Oral Health Council (VOHC) was established in 1997 and consists of independent board-certified veterinary dentists who award the VOHC seal of approval to those products that were shown in controlled studies to reduce the development of plaque and calculus. The VOHC does not conduct the controlled studies but reviews the results of tests performed in accordance with approved VOHC protocols (at least 20 percent average plaque and calculus reduction compared with controls on two blinded studies). I strongly encourage you to look at the VOHC website (www.vohc.org) when reviewing dental homecare products to help you find any evidence to support the products claim.

Mechanical plaque control

Tooth brushing is considered the gold standard for reducing the accumulation of dental plaque[1]. Performed on a daily basis it is one of the best methods for homecare plaque control by mechanically disrupting plaque accumulation. Any less frequent than once every two days is ineffective. Despite being the most effective method, client compliance is generally low with a study reporting six months after a professional treatment only 53% of clients educated on oral hygiene continued to brush their pet's teeth several times a week[2]. Low client compliance can be due to manual difficulties, a non-cooperative pet or lack of time.

In order to increase client compliance it is important to demonstrate the tooth brushing technique to your clients and then ideally observe the client performing the brushing technique on their pet. The technique recommended is known as the Modified Bass method. Children's or a pet's toothbrush are ideal, ensuring they have a soft filament brush. The Modified Bass method is a circular sweeping motion concentrating on the tooth/gingival margin with the brush angled 45 degrees to the tooth surface. Pet flavoured toothpaste can be applied to help increase acceptance of brushing. One study demonstrated the benefit of using a product during tooth brushing whose efficacy against plaque formation had been proven, to be more effective than tooth brushing alone[3]. The only toothpaste currently available in Australia given the VOHC seal of approval for plaque in dogs and cats is HealthyMouth™ Gel. Human toothpaste should not be used as it can cause gastric irritation if swallowed. It is important that clients work with their pets and take time introducing tooth brushing. Clients should start with the pet tolerating gentle manipulation of the face and lips, then the placing of a finger in the mouth gently rubbing the teeth to eventually using a toothbrush. Taking the time to give lots of praise, affection and rewards will result in an enjoyable and beneficial human-animal bond.

Chemical plaque control

Chemical plaque control focusses on reducing bacterial numbers associated with plaque formation, or interfering with the binding of salivary calcium to plaque in order to inhibit the formation of calculus. Chemical control can be achieved by adding the ingredients into food, mouth rinses, gels, sprays, water additives and dental wipes.

Chlorhexidine gluconate

Chlorhexidine is a broad spectrum antimicrobial with excellent efficacy in the oral cavity[4]. It is a very effective agent for controlling plaque accumulation and gingivitis. It causes precipitation of the cytoplasmic contents of the bacterium, which makes bacterial resistance unlikely to develop. Chlorhexidine binds to hard and soft tissues within the oral cavity well, leading to prolonged contact time and sustained antimicrobial activity. Prolonged use can lead to staining of the teeth, which is temporary and can be removed by professional polishing. It can alter taste perception and is bitter which can be difficult to mask. Hexarinse™ and Mavlab™ Dental spray gel are products available which contain 0.12% Chlorhexidine gluconate. It is recommended to use at home and in clinic as a pre-operative oral rinse and during the healing period following professional treatment.

Polyphosphates

Polyphosphates like sodium hexametaphosphate and sodium tripolyphosphate bind salivary calcium, making it unavailable for incorporation into the plaque biofilm to form calculus. Polyphosphates have no direct effect on oral bacteria and plaque formation.

Soluble Zinc Salts

Zinc compounds like zinc ascorbate and zinc gluconate have been used for their antimicrobial activity and ability to bind to sulphur which results in insoluble compounds that emit little odour therefore reducing halitosis. Maxi/guard™ Oral Cleansing Gel contains zinc ascorbate and aims to reduce bacterial growth, plaque formation and gingivitis in cats[5]. It is relatively tasteless and alcohol free.

Oral gel, sprays and drinking water additives

A variety of oral gels, sprays and drinking water additives are available on the market. We will focus on the only product to have the VOHC seal of approval for plaque control. Healthymouth™ is a veterinary only product and marketed as an additional strategy not an alternative to brushing and professional veterinary dental care. It is available in a variety of forms including an oral gel, dental spray or water additive for dogs and cats. It does not contain synthetic chemicals, pharmaceuticals or alcohol. It contains a range of natural ingredients with its effectiveness claimed to be from the combined effect of at least two active ingredients; papain (a natural enzyme to reduce the build-up of proteins in the mouth) and zinc gluconate (antibacterial activity). Studies revealed plaque reduction scores between 60-80% with daily use. The company recommends incorporating the product into a dental homecare plan; ideally for puppies and kittens prior to the development of periodontal disease and in older patients with existing periodontal disease following a professional dental examination and clean.

Dietary plaque control

Complete and balanced diets

Dental diets are a component of good oral health. Dietary plaque control can be achieved by mechanical disruption of plaque via dietary texture and calculus control by a chemical coating (polyphosphate). Chemical coating helps reduce calculus formation but their effect on preventing plaque accumulation is less compared to the textural diets. Polyphosphates reduce calculus formation via chelating calcium found in saliva. Textured diets are formulated to be used as a normal maintenance diet, the benefit of the diet is diluted proportionately if not used as the entire diet.

Commercial dry kibble diets are not any better at controlling plaque than moist diets except in the case of specifically formulated dental dry diets. To provide effective mechanical cleansing, the kibble must promote chewing and maintain contact with the tooth surface. Most commercial dry kibble diets are hard but brittle so that the kibble shatters without much resistance and there is little or no abrasive effect from chewing. Hills t/d has a special fibre matrix technology which allows the tooth to penetrate the whole way through the kibble before the kibble shatters which provides mechanical disruption of plaque.

Hill's Pet Nutrition

Hill's currently has five products for the control of plaque, calculus and gingivitis in dogs and cats in Australia that have the VOHC seal of acceptance for both plaque and calculus.

- Prescription Diet™ Canine t/d™, Original and small bites
- Science Diet™ Oral Care for dogs
- Prescription Diet Feline t/d™
- Science Diet™ Oral Care for cats

The products are formulated for use in dogs and cats over one year of age. Oral Care™ is a denser kibble than t/d™. Oral Care™ is intended for use in those animals without established periodontal disease, whereas t/d™ is for use in animals with established disease. There are no abrasives or active chemicals in Hill's t/d™ kibble. Prescription Diet™ t/d™ reduces plaque by 31% and calculus by 50% in dogs. In cats it reduces plaque by 58% and calculus by 64%.

Royal Canin

Royal Canin Veterinary Diet™ dental hygiene™ is a complete and balanced diet that is coated in sodium tripolyphosphate and the kibble is of a larger size than their standard kibble. Royal Canin claim the dental hygiene diet reduces plaque by 16% and calculus by 55% in dogs and only reduces plaque by 20% in cats. The diets have undergone studies but currently the only diet to have the VOHC seal of approval is the Royal Canin feline dental diet which is for plaque only.

Eukanuba
Eukanuba do not have a specific dental diet but incorporate a 'Dental Defense System' into their adult range of canine complete and balanced diets. The 'Dental Defense System' is a coating of sodium hexametaphosphate which chelates calcium aiming to reduce calculus. The Eukanuba adult maintenance diet for dogs with the 'Dental Defense System' has the VOHC seal of approval for calculus only.

Purina
Purina Pro Plan Veterinary Diets™ DH Dental Health Canine and Small Bites Canine Formula has the VOHC seal of approval for calculus only. Whilst the Purina Pro Plan Veterinary Diets™ Feline Formula has the VOHC seal of approval for plaque and calculus.

Advance
Advance Dog dental and Cat dental have larger, more textured kibble than their other diets and have added sodium tripolphosphate to help reduce calculus formation. Advance dental diets do not have the VOHC seal of approval.

Dental treats/chews
There are a significant number of treats and chews available, however the majority have little research to back their claims. Treats and chews are only a part of the homecare plaque control program and should be used as well as, not instead of tooth brushing. There are currently no products on the market with the VOHC seal of approval for both plaque and calculus for our feline patients.

Greenies
Feline greenies have the VOHC seal of approval for calculus only whereas the Canine greenies range of edible treats have the VOHC seal of approval for plaque and calculus. The size of the greenie chew must match the weight of the dog for maximum effectiveness as they work by mechanical abrading the tooth surface.

Product with the VOHC seal of approval for calculus only include:

- Purina DentaLife™ Daily Oral Care Dog treats use a unique shape featuring eight distinct ridges to help provide mechanical abrasion.
- Virbac CET™ VeggieDent™ Chews for dogs utilise a similar concept and feature a unique Z-shape.
- Purina DentaLife™ Daily Oral Care Cat treats have a porous texture to help the tooth penetrate the treat.
- Merial OraVet Dental Hygiene chews for dogs focusses on an active ingredient known as Delmopinol hydrochloride (a surfactant). This active ingredient aims to form a preventative barrier to reduce the cohesion of plaque bacteria to the tooth by reducing the viscosity of glucans which are a part of the salivary pellicle[6].

Pedigree™ Dentastix™
Dentastix™ for dogs have a special texture and are long straight chews with an X-shape. Trials have been conducted using the VOHC guidelines but they do not have the VOHC seal of approval currently. The trials showed an average reduction of 40% in reducing calculus build up when used daily[7].

Rawhide chews/dried animal tissues (pig ears)

These items can be classified as chew aids as they promote chewing and are not considered nutritionally significant. These treats/chews require supervision as some animals attempt to swallow leading to a possible GIT obstruction and choking hazard. These products especially dried animal tissues are also generally sold from bulk bins and there is no assurance of quality control which may lead to bacterial contamination. Rawhide chews and items like pig ears stimulate chewing and saliva flow with some studies performed indicating some benefit to calculus reduction in the short term. There are no long term studies that I am aware of that provide evidence of their long term effectiveness on plaque control, acceptance by dogs and their use in cats. To avoid damage to teeth and the oral cavity clients should be cautioned about feeding any product that does not easily bend, compress or dissolve when placed in a fluid environment.

Bones

Currently there are no studies that prove bones prevent or help control periodontal disease. At this point in time, the risks of feeding bones outweigh the unproven dental benefits. Risks of feeding bones include fractured teeth, GIT upsets, obstructions, perforations and constipation as well as exposing our patients and clients to bacterial or parasitic pathogens.

Dental sealants

Dental sealants aim to prevent plaque accumulation and are applied following a professional teeth cleaning procedure. They are tasteless and odourless, placed along the gum line around all the teeth.

Merial OraVet Dental Sealant™ is a waxy polymer that adheres to the tooth by electrostatic attraction which creates a hydrophobic barrier. This barrier inhibits bacterial adhesion to the tooth, helping decrease plaque and calculus formation[8]. OraVet is applied after the professional teeth cleaning and comes with a take home kit for owners to apply once weekly. Currently OraVet does not have the VOHC seal of approval for plaque and calculus.

SANOS Dental Sealant is a hydrophilic polymer that forms a film in the gingival sulcus, hindering plaque attachment to the gingival margin and sulcus. It is also placed following a professional teeth cleaning but does not need any take home applications and is clinically proven to last up to 6 months. This product does have the VOHC seal of approval for plaque and calculus.

Conclusion

A personalised dental homecare regime can benefit the patient, client and your clinic. The patient benefits from being able to be pain-free and eat without discomfort and maintain better oral health. A stronger human-animal bond can form and clients become more aware of dental disease leading to an increase in requests for more professional dental treatments, ultimately benefiting your clinic. Clients will be more likely to detect small problems early when they are easily treatable, instead of you discovering extensive dental disease during routine check-ups.

Professional examination and treatment should be performed on a regular basis to prevent and/or control periodontal disease. The frequency of professional intervention depends on the effectiveness of the dental homecare regime and the level of dental disease present. Tooth brushing remains the gold standard for controlling dental plaque, with passive techniques incorporated as no single product or technique is 100% effective. When selecting products to be used in a dental homecare regime look at the efficacy for controlling plaque and for peer reviewed studies or the VOHC seal of approval. To ensure maximum benefits are achieved when using dental homecare products ensure the client understands how to use the product and its limitations. Take time to educate your staff and clients on the value and need for professional and properly-timed dental care.

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Understanding tooth resorption and how to manage cases

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Introduction

Tooth resorption is a painful disease of the teeth and periodontium, most commonly affecting cats and now also recognised in dogs.[1] Various studies report prevalence rates of tooth resorption in cats to range from about 30 to 70%, depending on the composition of the population examined and on the diagnostic tools used to establish the diagnosis of tooth resorption.[2] Prevalence increases with age, with a significantly higher number of cats >10 years old having tooth resorption.

Tooth resorption is a progressive disease process caused by multinucleated cells known as Odontoclasts. Odontoclasts are mainly involved in resorption of dental hard tissues. The process begins as pathological resorption of root surface cementum that extends into the underlying dentin. As the disease progresses the lesion may eventually be exposed to the oral cavity leaving the tooth painful and susceptible to infection. Proposed causes that have been investigated include viral and bacterial disease, nutrition and hereditary and environmental influences. However, to date there is no proven aetiology for this disease process.

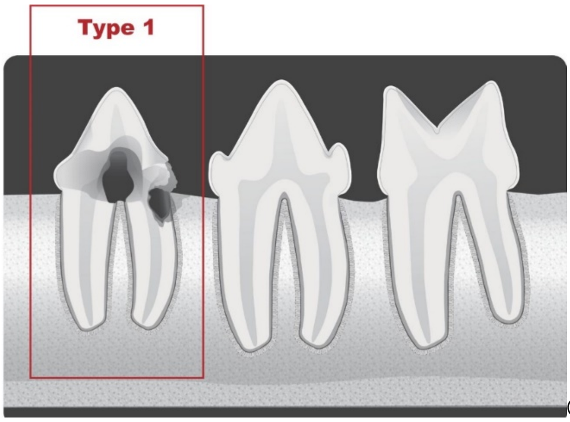
Clinical Signs and Diagnosis

As our patients do not necessarily show overt clinical signs of dental disease clients will not always pick up on disease processes at home. In fact, most patients will actually continue to eat normally despite discomfort. Clinical signs which may be shown at home include salivation, pawing at the mouth and rubbing of the face, decreased appetite ('fussy' eaters) and halitosis. Lesions are generally discovered incidentally on oral clinical exam during routine/non-related check-ups. A thorough oral exam is part of a complete physical examination and should be performed on all patients, particularly those aged >10 years.

On oral examination tooth resorption is normally detected as a hard tissue defect at the cementoenamel junction covered by hyperplastic gingiva, granulation tissue or calculus. The combination of an oral examination with a dental explorer and intraoral dental radiography is important in diagnosing tooth resorption. A dental explorer can be placed just below the gingival margin at a right angle to the long axis of the tooth and run mesiodistal across the tooth surface to feel for rough areas. The mandibular third premolar is the most commonly affected tooth, other teeth frequently affected include the mandibular first molar, the maxillary canine and the maxillary fourth premolar.[2, 3] Detection of a single lesion can indicate that multiple teeth are affected, as the distribution pattern in most patients is often symmetrical.

Dental radiographs are imperative in the diagnosis and treatment process. There are three distinct radiographic types of tooth resorption.[4]

Type 1 – the tooth is characterised by focal or multifocal radiolucency of the tooth, with an otherwise normal radiopacity and normal periodontal ligament space.



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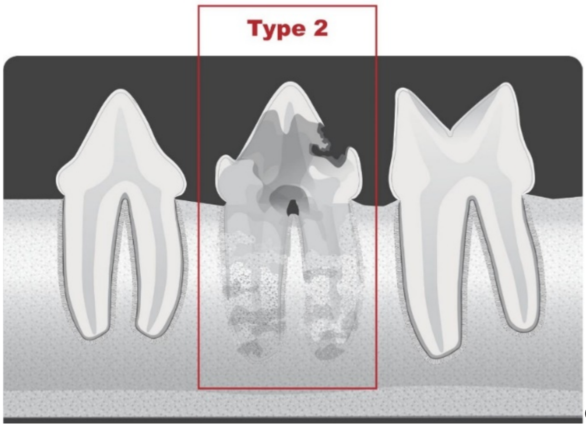
Type 2 – there is narrowing or disappearance of the periodontal ligament space and generalised decrease in radiopacity of the root that is consistent with replacement of root structure by alveolar bone. Root structure and alveolar bone take on a similar radiographic appearance and cannot be easily distinguished from one another.



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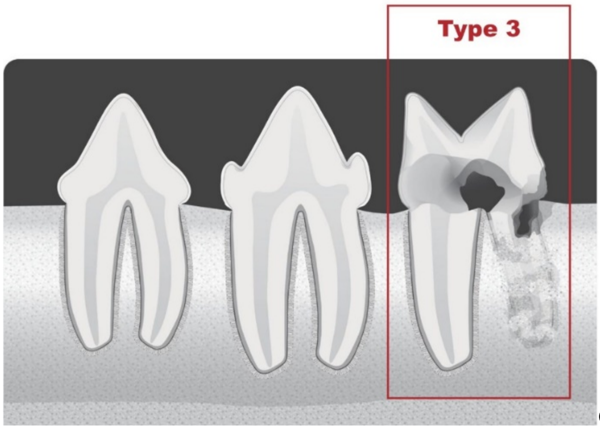
Type 3 – has both type 1 and type 2 features that are present in the same tooth, such as normal and narrow or lost periodontal ligament space, focal or multifocal radiolucency, and areas of decreased radiopacity.



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The American Veterinary Dental College has developed a staging system (stage 1-5) to quantify the extent and location of the disease process. However in a general practice setting it is important for the veterinarian to be able to recognise the three radiographic types as this will help you determine the most appropriate surgical option for your patient.

Treatment

In treating this disease process our main goal is to minimise patient discomfort. As no aetiology has been confirmed there is no known treatment that prevents development and/or progression of tooth resorption. Currently attempts to salvage affected teeth by applying dental restorative materials typically fail and the most effective treatment option for this disease requires surgical intervention.

Extraction of teeth with tooth resorption can be difficult due to tooth weakening and dentoalveolar ankylosis. It is advised to perform the extractions surgically with full thickness mucogingival flaps to decrease the incidence of fractured roots and allow for complete removal of tooth material, alveoloplasty and closure of the extraction site. Teeth exhibiting type 1 tooth resorption on dental radiographs require complete removal of tooth material (inclusive of all root fragments).

Crown amputation with intentional root retention is an acceptable alternative to extraction in teeth exhibiting type 2 tooth resorption on dental radiographs. Crown amputation is only acceptable for teeth where there is advanced root replacement resorption, ankylosis and no periodontal ligament or pulpal tissue evident radiographically. There must be no radiographic evidence of endodontic disease or periodontitis otherwise crown amputation will result in painful retained roots that may never resolve.

When performing crown amputations it is important to at least remove all dental tissue above the alveolar crest and 1-2mm into the socket before performing alveoloplasty and suturing the wound closed with a fine absorbable monofilament (I use 4/0 monocryl in dogs and 5/0 monocryl in cats). For type 3 teeth, roots with type 1 classification should be extracted and roots with type 2 classification can be treated by crown amputation with intentional root retention.

As radiographic classification of tooth resorption is subjective, extraction is most often the preferred and recommended treatment. Veterinarians without access to dental radiographs should not perform crown amputations. The affected teeth should either be extracted without intentional root retention or the patient referred to a facility with dental radiology. Following extractions post-op radiographs should be performed to document removal of all dental structures.

Conclusion

Tooth resorption is now recognised in both cats and dogs. It is a painful, progressive disease process with no known aetiology. A thorough oral examination on all patients when possible is important for early detection. To achieve an accurate diagnosis dental radiographs are imperative and once detected surgical management is required.

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Interactions between land use change, flying-fox ecology and Hendra virus dynamics

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Typically, flying foxes are nomadic nectar feeders and pollinators of native forests. Natural cyclical transitions from El Niño to La Niña periods impact Eucalypt phenology, causing intermittent acute food shortages for flying foxes and temporary fissioning of flying fox roosts. More recently, these 'fission' roosts are persisting outside of acute food shortages, manifesting as an exponential increase in flying fox roosts in urban areas. Immediately pre-dating these recent changes in flying fox ecology, the mid-1990's saw a peak in destruction of their key habitats and the emergence of four novel zoonotic viruses from flying foxes. One of these, Hendra virus, stands out as an excellent model system for understanding bat virus transmission and spillover globally. Hendra virus spillover to horses tends to be associated with seasonal 'pulses' of viral excretion within bat populations, but the interactions between proposed broad-scale and roost-level drivers of Hendra virus transmission are complex and have not been fully elucidated. Our results indicate that landscape-scale processes driving flying fox roost fissioning are linked to processes driving Hendra virus excretion and spillover to horses. By gaining insights into the interactions between environmental change, bat ecology, viral dynamics and spillover, we hope to identify the root causes of viral spillover from wildlife hosts and develop new ecological interventions to prevent bat virus spillover in Australia and globally

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Automated mastitis diagnostics during milking – technology, performance and challenges

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Introduction

A clear trend, in most dairying countries, is increasing herd size and a concurrent increasing number of cows per labour unit. Anecdotal evidence from Australia and New Zealand also points towards larger herringbone and rotary milking parlours being preferred for new constructions – the days of building a 10 aside parlour are likely behind us. With these trends comes increasing time pressure on milking staff and the desire to build automation, or technology enabled methods, into routine tasks. Detection of clinical mastitis (CM), and milk unfit for processor supply, is one such routine task.

Alongside the increase in herd size is an explosion in the uptake of Automatic Milking Systems (AMS) also known as milking robots. This is particularly evident in Western Europe and Canada although pasture based dairying countries have also experienced moderate expansion of this technology. It is estimated that there are now 38,000 AMS units worldwide [1]. Automatic milking systems rely on sensor based systems for mastitis detection and diversion of milk not suitable for supply.

This paper will explore sensor systems for mastitis detection in both conventional milking systems, which are the majority in Australia, in addition to AMS.

Overview of sensors and mastitis detection systems

Dairy farmers are under regulatory direction (and pressure) to supply milk for human consumption that is free of visual abnormalities including those associated with CM such as flecks, clots and watery milk. Where an AMS is installed, detection of abnormal milk which should be diverted from supply is largely governed by sensor systems built into the milking unit. The sensors, and their accompanying information systems, are an intended first line of detection prior to further farm staff assessment.

An entry point for understanding how these systems operate is a review by Rutten *et al.*[2]. Sensors in dairy production are attached or non-attached. In AMS and conventional milking, sensor systems are non-attached. Non-attached sensors are subsequently categorized into in-line or on-line. In-line sensors take measures from a continuous flow of biological material from the cow (such as milk) while on-line sensor systems take a sample for analysis. Any sensor system used in conventional or AMS milking can be conceptually thought of as operating at 4 levels:

1) The sensor employs a technology that measures something about the cow
2) Interpretation that summarize changes in sensor measurements resulting in information about the health status of the cow
3) Integration of sensor information with other, sensor derived or non-sensor, information to produce advice
4) Creation of an autonomous decision or farmer led decision

The bulk of literature describing sensors systems used in either conventional or AMS milking deals with levels 1 and 2. The relationship between the sensor, the gold standard (GS) for sensor data interpretation, plus any ancillary data from other sources, is described by an algorithm [2].

Sensor systems involved in mastitis detection vary in technology and underlying approach to detection. From approximately 30 peer reviewed publications, sensor systems have incorporated:

- electrical conductivity (EC)
- color
- ICSCC or a proxy measurement for cell count such as an automated Californian Mastitis Test (CMT) or Rapid Mastitis Test
- temperature
- milk yield
- Hydrolytic enzymes (eg: NAGase) or proteolytic enzymes released as a result of inflammation

Regardless of the milking system being used, cows that are flagged for human assessment following a milking "mastitis" alert will be checked within a milking session or between milkings. In pasture based AMS installations, assessing animals post-alert is more complex involving segregation of the animal at next AMS presentation. It is common practice for cows undergoing CM treatment in AMS to be milked using conventional equipment for the period of treatment and milk withhold.

Irrespective of sensor system detection performance, farmers and their advisers need to be cognisant of factors concerning sensor system installation costs, running costs, maintenance and calibration requirements, plus hardware robustness [3]. Accurate and timely detection of CM in and milking system is important for milk quality, animal welfare and antimicrobial stewardship.

Detection system performance

Mastitis detection sensor systems can have performance described as for any other biological test using test sensitivity (SE) and specificity (SP) - these are inherent qualities of the sensor system being described. Sensitivity describes test performance in truly diseased animals while SP describes test performance in truly non-diseased animals. With mastitis sensor performance, a diseased or non-diseased animal should be thought of as a cow that has mastitis (generally CM) at an individual milking.

With any detection system, care should be exercised in what is being described. A cow that is defined as "truly having mastitis" does not provide sufficient clarity when assessing sensor performance. For example, this description may define a cow infected with a mastitis pathogen (based on milk culture) but classified as a subclinical infection, or a cow where the true infection status is unknown but remains classified as a subclinical infection through individual cow cell count (ICCC) level, or a cow classified as a CM case based on a defined description of the visual signs (and other tests) necessary for this diagnosis. An assessment of material describing sensor system performance must be specific in describing what is actually being described if "mastitis case" is used as the gold standard. The overriding constraint in automated mastitis detection is the reality that there is no continual monitoring of the mastitis infection status of a cow – only discrete observations at set time points normally associated with milking [4].

The International Standards Organization describes in ISO/FDIS 20966, Annex C (Automatic milking installations – requirements and testing) methods for assessing abnormal milk which should be withheld from bulk tank supply [5]. This document further describes a minimum standard of 80% SE and 99% SP for detection of abnormal milk.

Any diagnostic test can further be described according to positive predictive value (PPV) being the proportion of positive test results that are truly diseased. Positive predictive value is not an inherent quality of a sensing system as PPV is influenced by disease prevalence. Both Sherlock *et al.* [4] and Hogeveen *et al.* [6] have proposed that PPV should be described as "success rate" (SR) in mastitis sensor systems as it is a more useful descriptor for the milking team.

Similarly, these groups of authors [4,6] have also suggested that sensor system SP is a poor term to use with dairy farmers, in part, because of the opportunity for "numerical perception" to be skewed. For example, a SP of 95% may appear numerically to be high, but a mastitis sensing system with this performance would lead to a frustratingly large number of sensor alerts to be checked which would prove to be non-diseased. As an alternative, False Alert Rate (FAR) is proposed as a more intuitive measure of sensor system performance. False Alert Rate is defined as the number of false sensor alerts per 1000 cow milkings and is calculated by:

- FAR = 10*(100% - SP%) per 100 cow milkings [4,6]

The importance of these concepts concerning performance of sensing systems is highlighted in Tables 1 to 3 which have use an example adapted from Sherlock *at al.* [4]. Here we consider an example 100 cow herd milked, with an average 12 hr milking interval, over 5 days. Further, for simplicity of the example, we assume that there are 25 cases of CM that all occur on the 5th day of milking. Here the disease event we wish to define is a CM case per cow milking. For each example the prevalence of CM does not alter (25 CM cases per 1000 cow milkings), nor does the sensor system SE (set at 80%).

In Table 1, the sensing system SP is 96.9% resulting in a FAR of 30/1000 cow milkings and a SR of 40%. In Table 2, the change in sensing system performance is a decrease in SP (from 96.9% down to 94.9%) resulting in a FAR of 50/1000 cow milkings and a SR of 29%. Finally, Table 3 describes and increase in sensor system SP to 98.5% (just below the 99% recommended in ISO 20966). In this scenario the FAR becomes 15/1000 cow milkings and a SR of 57%. At initial appraisal, a sensing system SP of 96.9% may appear "numerically" satisfactory but the result is 30 cows being flagged for CM assessment by milking staff over 5 days in a herd only milking 100 cows. Even an increase in SP to 98.5% still results in a potentially unacceptable number of cows to be checked. There are no farmer sourced guidelines on acceptable levels of false alerts [7]. However, a survey conducted with 139 Dutch farmers indicated that a low level of false alerts was one of 3 specific considerations when assessing sensor system performance (the other 2 being alerts delivered in a timely way and an emphasis on severe CM cases) [8].

Table 1: 100 cow herd, 1000 cow milkings (5 days at average 12 hr MI), total CM based on gold standard = 25

	CM + cow milking	**CM – cow milking**	
Test +	20	30	**Total 50 test +**
Test -	5	945	**Total 950 test -**
	Total 25	**Total 975**	1000 cow milkings

Sensitivity = 20/25 = 0.80 (80%)

Specificity = 945/975 = 0.969 (96.9%)

Positive predictive value (Success Rate) = 20/50 = 0.40 (40%)

False alert rate = 10*(100%-96.9%) = 30 per 1000 cow milkings

Table 2: Same scenario as described in Table 1 but increased False Alert Rate

	CM + cow milking	CM – cow milking	
Test +	20	50	**Total 70 test +**
Test -	5	925	**Total 930 test -**
	Total 25	**Total 975**	1000 cow milkings

Sensitivity = 20/25 = 0.80 (80%)

Specificity = 925/975 = 0.949 (94.9%)

Positive predictive value (Success Rate) = 20/70 = 0.29 (29%)

False alert rate = 10*(100%-94.9%) = 50 per 1000 cow milkings

Table 3: Same scenario as described in Table 1 but decreased False Alert Rate

	CM + cow milking	CM – cow milking	
Test +	20	15	**Total 35 test +**
Test -	5	960	**Total 965 test -**
	Total 25	**Total 975**	1000 cow milkings

Sensitivity = 20/25 = 0.80 (80%)

Specificity = 960/975 = 0.985 (98.5%)

Positive predictive value (Success Rate) = 20/35 = 0.57 (57%)

False alert rate = 10*(100%-98.5%) = 15 per 1000 cow milkings

In both the review paper by Hogeveen *et al.* [6] and a later review by Rutten *et al.* [2], the authors reported that none of the peer reviewed papers describing AMS sensing system performance claimed results that were equal to, or exceeded, the ISO standard target. Mein and Rasmussen [9] have suggested that the SP target described in Annex C [5] is a minimum acceptable level for AMS as it implies 10 false alerts/1000 cow milkings.

In an AMS installation, farmers generally do not check all alerts due to time constraints [10] so a Dutch study assessed the approach of farm staff incorporating non-AMS information on individual cows to help decide which cows to check for CM [11]. However, the authors found that the effect of using cow data, by the 12% of farmers who undertook this approach, resulted in only minor improvements in determining true CM cases from false alerts.

Gold Standard Definition

The choice and description of any GS used to assess sensor system performance is vital [2]. Annex C in ISO 20966 attempts to deal with methods of detecting abnormal milk and interpretation of test results and the guideline implies that a true CM case can be defined based on the first few squirts of foremilk [5]. Mein and Rasmussen [9] purport that a more robust approach of defining a CM (or "clinical episode") would be clots >2mm in average diameter persisting for more than 3 squirts of foremilk for at least 2 of 3 consecutive milkings.

Mein and Rasmussen's proposal is supported by re-analysis of NZ data from a 650 cow herd monitored for 21 days (26,974 milkings) as part of a study assessing the performance of a novel mastitis sensor system. Clinical mastitis was used as the GS but, depending on the 4 possible interpretations of a "true" clinical case, the SE of the sensor system ranged from 68% to 88% and the FAR from 3.9 to 7 alerts per 1000 cow milkings. Currently, there is no agreement on a suitable GS for CM in milk harvesting research. This situation is compounded by the reality that CM is a relatively infrequent event leading to a weak statistical inference [9]. The requirement for careful assessment of the GS description in any study, combined with the range of sensor types and associated algorithms, makes comparison of sensor system performance even more complex for the farmer or adviser [6].

System alert period

Any sensor system description should include the sensor alert period (alert window). This may also be described as the time window of detection [6] or the time resolution of measurement by a sensor to alert [2]. This describes the time period required for the sensor system (sensor data and associated algorithm) to register a test negative or test positive result – that is, the cow milking is defined by the system as no alert or an alert. In some systems, the initial mastitis alert may be distinguished from a repeated alert at following milkings.

Results from the previously discussed survey of 139 Dutch farmers supported an ideal alert period of 24 hrs only [8]. Hogeveen *et al.* proposed that the alert period should ideally be no longer than 48 hrs [6]. With an alert period of 24 to 48 hrs one study found there were no GS episodes of CM with more than a single alert [4]. While Khatun *et al.* found that altering the alert period with an electrical conductivity (EC) sensor system resulted in only small changes in SP [12], consensus in the literature indicates that wider alert periods results in increased SR (or PPV) and decreased FAR [4,7]. On balance, alert periods of between 24 to 48 hrs, while reducing sensor system performance, strikes an acceptable compromise between the workload created by mastitis alert checks by farm staff and the desire to reduce false positive alerts.

Conceptually, alert period is further explained in Figures 1 to 3. In all cases in this example, the observed CM episode, or GS detection, is defined as 24 hrs. In Figure 1 the alert period is 48 hrs with both alerts here being classified as false alerts (false positive alerts) as they do not overlap with the CM period defined by farmer observation. In Figure 2 the alert period is also 48 hrs but, in this scenario, there is overlap between the alert period and the CM period resulting in a true positive alert (represented in the numerator of the SR calculation). Figure 3 describes an alert period of 96 hrs (4 days) to illustrate that alerts being received by the milking staff at each milking up until the confirmed observation period for CM would likely result in up to 6 periods where the cow would be checked without identifying her as a CM for treatment.

Figure 1: Illustration of relationships between gold standard detection and sensor system alert detection

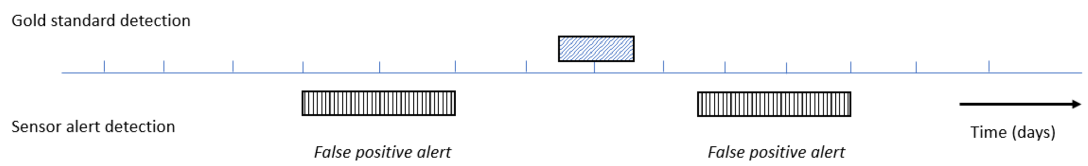


Figure 2: Illustration of relationships between gold standard detection and sensor system alert detection

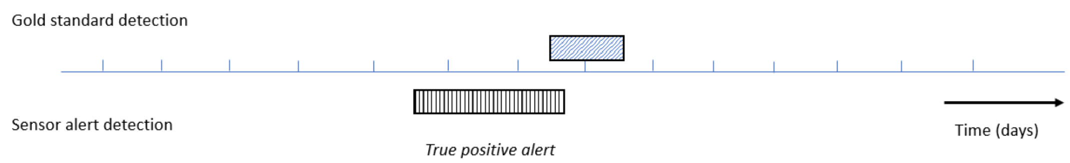
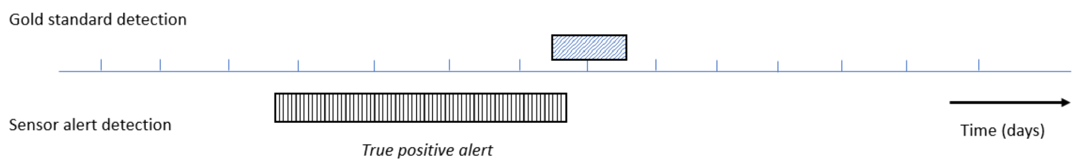


Figure 3: Illustration of relationships between gold standard detection and sensor system alert detection



Electrical conductivity

Milk electrical conductivity (EC) is principally determined by the amount of Na^+ and Cl^- dissolved in milk [13]. This is normally at a lower concentration compared to blood serum. During inflammation (ie: subclinical mastitis or CM) tight junctions of the mammary epithelial cells loose integrity resulting in an increase in Na^+ and Cl^- ions in secreted milk [14]. Milk EC can be affected by factors other than inflammation such as milk transport, air turbulence, sensor configuration, fat content and temperature [15, 16].

In the literature there are reports of threshold values for EC indicting the presence of mastitis. Hogeveen and Ouweltjes [17] described a threshold for mastitis at 4.6 mS/cm while Hamann and Zecconi [18] described healthy foremilk samples with an EC of 4.9-6.4 mS/cm, an EC increase of 0.4 to 1.2 mS/cm in quarters infected with a minor pathogen and a larger increase to 8 mS/cm in quarters infected with a major pathogen. A constraint around the use of EC as a singular indictor of mastitis is the reality that the measurement alters during milk letdown [19]. Cisternal milk (milk which has drained by gravity into the udder cistern prior to milk letdown) has an inherently higher EC when compared to alveolar milk. Once milk letdown occurs, and alveolar milk starts to mix with cisternal milk, EC will drop. This process takes around 40-60 s after initial teat stimulation. As a result, in AMS with highly standardized teat preparation procedures, there is little chance that any milk being tested by a quarter in-line EC sensor will be only cisternal in origin. As a consequence of milk letdown, the SE of EC as a mastitis indicator decreases [20]. No guidelines are reported on the EC threshold to use in conventional milking or AMS for the detection of CM [12].

Comparing expected changes in ICCC as a result of a subclinical, or clinical infection, the rise in EC is linear while the increase in ICCC is logarithmic [15]. In any milking system, the SE of EC as the primary indicator of mastitis, could be improved by comparing quarters within cow (only possible in AMS), or by comparing repeated measures within cow and between milkings [10, 15]. The advantage with quarter measurement is that this comparison can occur within a single milking [6]. With detection of CM using EC, the range of SE is reported as 47% to 86% while SP ranges from 56% to 99% [2, 12]. Various approaches using index creation with EC measurements alone, resulting in suboptimal sensor system performance relative to ISO 20966, has led Khatun *et al.* to conclude that options for using EC for satisfactory CM detection, in isolation from ancillary data in AMS milking have been exhausted [12]. Using multiple sensor system information is likely to result in better detection of CM but many milking systems do not employ secondary sensor systems for mastitis detection such as ICCC or a proxy measure [21]. Electrical conductivity probes employing 4 probe heads instead of 2 may be more accurate as there is less chance of sensor drift [10].

Somatic cell count or an equivalent, proxy test

Sensors which measure (via direct measurement or proxy) ICCC, or individual quarter SCC, are the other common type of detection system for mastitis in AMS and have reasonable uptake in some conventional milking systems. Sensor systems which use automated counting of stained cell nuclei, based on fluorescence images, have good correlation with laboratory based measures of SCC – an R^2 of 0.86 [22] or up to 0.99 [3]. This type of sensor is generally on-line (uses a defined milk sample). A fixed sampling frequency of 24 to 36 hrs had the lowest sampling rate in a study reported by Sorensen *et al.* but also the lowest test performance [23]. Sampling frequency is important because of running costs which have to be taken into account – note that EC sensor running costs are negligible. In the Sorensen *et al.* study, dynamic sampling with a frequency of every 24 hrs delivered better test performance and was recommended. The SE was 89% when all data was used. However, in practice, IMI alerts may not be very practical because of multiple alerts recorded over time.

On-line sensors which perform as automated CMT devices provide a proxy measurement for ICCC. The correlation of this sensor system output with laboratory based ICCC is around 0.76 and less when compared with stained cell nuclei methods. Kamphuis *et al.,* when assessing this sensor system for detection of CM, found a 2 to 3-fold increase in SR and the same degree of decrease in FAR when a fuzzy logic information system was used in the algorithm creation compared to the use of SCC or EC methods alone [7].

Colour change

A study assessing the degree of blood concentration in milk able to be assessed visually by human test subjects found that 0.1% blood in milk was scored as pink (to be withheld from the bulk tank) where there was reference milk containing no blood for comparison [24]. Reflecting this work, it was recommended that AMS should be able to detect and divert milk with 100 mg/l blood (0.1%) as at this level, milk will have a red tinge. The combination of EC with sensor information such as colour, increases mastitis detection system SE and SP where algorithm creation uses fuzzy logic or neural networks [3].

Incorporation of other sensor or cow information

Milking interval change has been postulated as potentially useful information to incorporate into mastitis detection algorithms but the expected increase in interval (decrease in milking frequency) with mastitis has not been reported and so this parameter may be not suitable and is only potentially applicable in AMS [20].

Milk yield losses due to CM will vary according to the month of lactation and can vary from 8% (month 1) through to 1% (month 9) with an average of 5% [21]. Combining this information into a mastitis detection algorithm makes theoretical sense but milk yield is difficult to predict in a milking system with variable, within cow, MI such as in AMS [22, 25] and pasture based conventional milking systems. Milk production rate may be a more suitable parameter to incorporate into an algorithm for the detection of an IMI [26].

Conclusion

Diligent and careful consideration of research is required in assessing any given mastitis detection system incorporated into the milk harvesting process. While (infrequently published) material on the performance of inflammation enzyme tests has not been addressed in this paper, the core questions for an assessment remain the same:

- can the sensor system be described in the 4 stages from sensor measurement to final advice?
- is performance reported using robust methods, clear parameters such as SR and FAR, and relative to a well described GS?

Through this type of information, farmers and their advisers should be in a better position to make valid decisions concerning conventional or AMS milking system mastitis detection systems on farm and how they should be applied.

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The future of milking management in Australasia

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Introduction

Milking management can be defined as activities during milking time which affect both milk harvesting and milk quality control. These activities, or processes, are all in proximity of the milking parlour and can include aspects of milking an individual cow that occur prior to cups-on, during cluster attachment or immediately after cups-off. This paper will examine recent research in milking management and its implications for milking in Australia and New Zealand. The framework for this will discussion will be via the structure provided for milking management examination using the Countdown Technotes and Mastitis Investigation Pack.

Veterinarians and other farm advisers undertaking a mastitis and milking management investigation are encouraged to use CountdownTechnote (TN) 13 [1]. One aim of this paper is to re-focus attention on making the correct connections between these milking time observations and their likely influence on new mastitis infections.

The logic of TN 13 is summarised in the Flowchart of the general approach for investigating a mastitis problem (TN 13, Figure 1). The key steps are:

1) Describe the presenting problem
2) Define the problem more specifically using milk culture results
3) Activate the advisory team
4) Collate and assess findings with the advisory team
5) Develop a farm plan with the farm team
6) Review progress

Step 3 and 4 are the steps where a single investigator, or team, conduct milking time observations and interpret the results. These milking time observations are described in more detail in both TN13 and the accompanying Mastitis Investigation Kit (Countdown and Smart SAMM [2]).

The milking time tests covered in TN13 are:

Section E: Milking machine dry test
F: Performance test of milking machines
G: Milking routines and cup slips
H: Clinical cases
I: Teat condition
J: Cow behaviour and milking time per cow
K: Completeness of milking and cluster alignment
L: Teat disinfection
M: The environment

Milking machine dry test (TN 13 Section E)

The dry test report is the record of a milking machine test with only air flowing through the plant. The dry test report has 3 primary functions in a mastitis investigation. These are:

- Measurement of the system vacuum (generally measured at both the pump inlet and receival vessel – the latter being the more important measurement)
- Measurement of the pulsation phases in ms
- Measurement of air admission through various plant components and vacuum reserve (l/min)

The system vacuum will give advisers a guide to what the likely claw vacuum will be under peak milk flow depending on if the plant is a high-line milk line (herringbone) or low-line (rotary). There is a larger difference between system vacuum and claw vacuum in a herringbone under peak milk flow as lifting milk 1-1.5 m into the milk-line (high-line) "consumes" vacuum.

The important pulsation characteristics to note are the duration of the d-phase in ms (target is greater than 150 ms) and b-phase (not greater than 500 ms). At a d-phase of less than 150 ms teat-canal congestion will be seen [3] and a b-phase of greater than 500 ms may result in within pulsation cycle teat congestion [4]. Just describing pulsation phases as a % of the total pulsation cycle time is of little use unless the conversation to ms time is made.

Vacuum consumption of plant components, in addition to vacuum reserve, will determine if the plant is high or low risk for teat cup slips which will have a deleterious effect on milk harvesting efficiency but not necessarily new infection risk.

Performance test of milking machines (F)

The primary reason for a milking plant performance test is to obtain the claw vacuum during the peak milk flow period. Average claw vacuum, measured during this time, should be within the range 32 to 42 kPa [5]. This test should be applied to at least 5 cows, and preferably 10, during a single milking with the vacuum recorder measuring point as close as possible to the bowl.

Where possible, mouthpiece chamber vacuum should also be obtained during the same milking. Despite mouthpiece chamber vacuum being one of the physical forces preventing cup slip during milking, to prevent teat-canal congestion the vacuum in this part of the liner should be as low as possible [6]. Liners used in both the NZ and Australian markets tend to be wider bore and so mouthpiece chamber vacuum in excess of 10-15 kPa during the peak milk flow period can be expected. Square and triangular liners may have higher mouthpiece chamber vacuum compared to round liners during the peak milk flow period and the no flow period. It is usual for this vacuum measurement to increase after the cessation of milk flow at the end of milking which will, in turn, increase the risk of teat barrel and teat end congestion.

Were a handheld vacuum gauge is used to obtain claw or mouthpiece chamber vacuum levels, the device should use an averaging algorithm which allows for average vacuum to be measured over a prescribed time period (eg 10-30 secs). Handheld devices with a constant readout are not fit for purpose with this test. The VaDia device (Biocontrol, Norway) is able to record vacuum in both the short milk tube and mouthpiece chamber simultaneously on a single time tracing and are a preferred device for measuring vacuum at locations around the cluster during an entire milking on a single unit. An excellent review on the practical application of this diagnostic technology under Australian conditions is described by Malmo and Mein (2015) [7].

Vacuum measurements taken during a plant performance test should always be assessed in relationship to the teat condition assessment (I) and milking time per cow (J) – taken as a group of observations, they should create a sensible story relative to each other.

Milking routines and cup slip (G)

Assessment of milking routines involve three main observations: a) cups-on procedure b) cups-off procedure and c) consistency of routine. In the main, (a) and (b) are about limiting air admission into the claw during cups-on and cups-off which aids in vacuum stability in the milkline. It is questionable whether this has an influence on new infections through changes in claw vacuum (and the formation of “impacts”). Consistency of routine, particularly in long herringbone parlours, is linked in with milking efficiency (cows per hr) and the risk of overmilking (teat cups attached during the low milk flow rate phase). Overmilking is to be avoided as far as possible (see further section in this paper).

Measurement of cup slip was deemed to be important as an indicator of new mastitis risk, but it is now no longer viewed as critical. This is because work describing the “impact” mechanism of new infection [8,9] used small volume bowls and liners with small diameter short milk tubes. It is unlikely that modern milking claws with bowls greater than 150 ml capacity and short milk tubes in excess of 9mm diameter can generate the airflows needed to form an “impact” against the teat end (reverse flow of milk due to a pressure gradient) [10].

Clinical cases (H)

Assessment of clinical case management during the milking routine has a focus on systems to detect clinical cases as a function of normal milking practices along with segregation of clinical cases (either newly detected or under treatment). Where possible, detection of clinical cases (via BMCC monitoring, filter sock assessment, cow surveillance during milking, routine stripping or automated sensor) should be organised in such a way that there is minimal time between the onset of a clinical case and detection.

Where possible, clinical cases should always be milked last to reduce the risk associated with increased bacterial load on the liner between cows. This is despite the likelihood that the risks associated with cross contamination, with the liner as the vector of bacterial transmission, are less compared with previous best practice opinion based on milk harvesting research from the 1960’s to the 1990’s.

Teat condition (I)

The teats can be viewed, in concept, as 4 individual milking machine testing units for each cow. How sample results should be interpreted is summarised in TN 9 [1]. In general, increasing the sample size will improve the robustness of results and it is usually practical to assess at least 100 cows during an individual milking.

Where teat hyperkeratosis or teat congestion are found to be approaching, or above trigger levels described in TN 9 (and TN13) [1], further investigation of vacuum, pulsation and liner characteristics are recommended [11]. However, based on research into the new infection risk posed by hyperkeratosis [12] and teat congestion [13,14], the influence of any findings in a herd should not be overstated. An increased prevalence of teat end hyperkeratosis, or teat congestion, is important in an individual herd but it should also be kept in context compared to factors such as environmental exposure of pathogens to the teat-end. If teat-end hyperkeratosis is above trigger levels, yet teat-end vacuum during the peak milk flow period is acceptable along with the degree of overmilking, liner selection may need to be re-considered. This is because increasing liner compression is a risk factor for hyperkeratosis [11]. Frustratingly, neither liner compression, nor its biologically relevant indicator, overpressure [15] is not reported for commercial liners by manufacturers.

Cow behaviour and milking time per cow (J)

Cow behaviour assessment has been referred to as "cow comfort" during milking in some forums. This should be differentiated from milking gentleness which more specifically describes how gentle the action of the liner is on teat tissue. The two concepts are linked but different. Where the assessment of kicks and steps (KiSt) is above the trigger level for investigation (TN 13) [1], the teat assessment score should be closely examined for evidence of teat congestion (red, blue teats, ringing at the teat base) along with claw vacuum results. If there does not appear to be an association between cow comfort measurements and teat assessment it may be that observations of increased kicks and steps is due to factors other than poor milking gentleness.

Milking time per cow is primarily concerned with overmilking. Any degree of overmilking, as judged on very low or nil milk flow through the bowl, will increase vacuum at the teat end (particularly in a herringbone, high-line shed) and increase vacuum in the mouthpiece chamber. An increase in vacuum in both of these parts of the liner will increase the risk of teat-end and teat barrel congestion [6]. Because of the differences in quarter milk flow rate and production, when nil-flow in the bowl changes from flow to low flow, it is likely that at least one of the quarters has been in the nil-flow period for a minute or more with deleterious effects on that teat. The degree of overmilking, combined with teat assessment, is an important milking time measurement.

Measurement of incomplete letdown is an indicator of poor milk harvesting efficiency and, potentially, a risk factor for teat congestion. This is a difficult area to assess in Australian and New Zealand cows as all the research into oxytocin response indicates that tactile stimulation (eg: pre-milking preparation) is required for milk letdown prior to teat cup attachment [16]. There is no published research to indicate that cows are able to have an oxytocin release based on the "sights and sounds" of routine milking without tactile stimulation of the teat and udder tissue. In both Australia and New Zealand we assume this to be the trigger for oxytocin release, but it has never been demonstrated in a controlled study.

Completeness of milking and cluster alignment (K)

It has been postulated that undermilking would increase the risk of mastitis, but research into automatic teatcup removal milk flow threshold levels indicates that residual milk in the udder can be increased without an associated increase in clinical case rate [17,18]. It is likely that measurements examining completeness of milking are of minimal importance to new mastitis infection rate and observations of cluster alignment are also of secondary importance with regards mastitis risk. Cluster alignment may be a useful indicator of milk harvesting efficiency, however.

Teat disinfection (L)

It is not the purpose of this paper to review the published science of teat disinfection as a mastitis control measure. Suffice to say, however, that provision of adequate teat disinfection, at each milking, is a major mitigator of new infection risk. Both TN 7 and TN 13 [1] outline the importance of teat disinfection chemical concentration, coverage (all the teat surface in contact with the liner) and application volume. Of all the milking time observations, this single area likely carries the largest impact on mastitis risk.

Environment (M)

Pre-cups on, cleanliness of the udder and teats can be scored according to the assessment process described by Schreiner and Ruegg [19] (2003) using a 1 to 4 scale. If 20% of cows score 3 or 4 there is an association with increased mastitis risk and attempts should be made to isolate and minimise exposure to dirt, mud and faeces. The cleanliness score should always be reviewed in light of the available culture results as part of the TN13 step #2: Define the problem more specifically using milk culture results.

Pre-milking preparation

In many dairying countries, a pre-milking preparation (PMP) routine is mandated as a requirement for the right to supply milk. A good example of a regulatory framework is the Pasteurized Milk Ordinance (PMO) [20] in the US which directs dairy farmers to undertake a wipe/disinfection-follow up wipe process prior to cluster attachment. Here the disinfectant contact time is around 30-50 seconds with an average total time for this PMP process of approximately 103 seconds according to one recent study in the US Midwest [21].

The application of PMP, such as that described in the US PMO, would typically require an additional labour unit at each milking regardless of the dairy parlour type. A core question for Australian and NZ farmers, and their advisers, is does the environmental teat contamination usually observed in pasture based systems result in a new infection rate that warrants PMP? Few studies have examined this question in pasture based systems – however, a recent 5 herd case-control study in Australia described no statistically significant difference in clinical mastitis incidence, nor new infection rate, between cows treated with PMP for 60 days during the calving period compared to control cows [22]. Despite no statistically significant differences overall, the authors did report a markedly lower clinical mastitis incidence in one herd. The conclusion on this study stated that a decrease in clinical mastitis incidence of around 50% would be required to justify the additional labour costs associated with PMP and that this process may be justified on pasture based farms in times of high environmental challenge. It is reasonable to suggest that the udder cleanliness scoring system described by Schreiner and Ruegg [19] is suitable for assessing the degree of environmental challenge to teats.

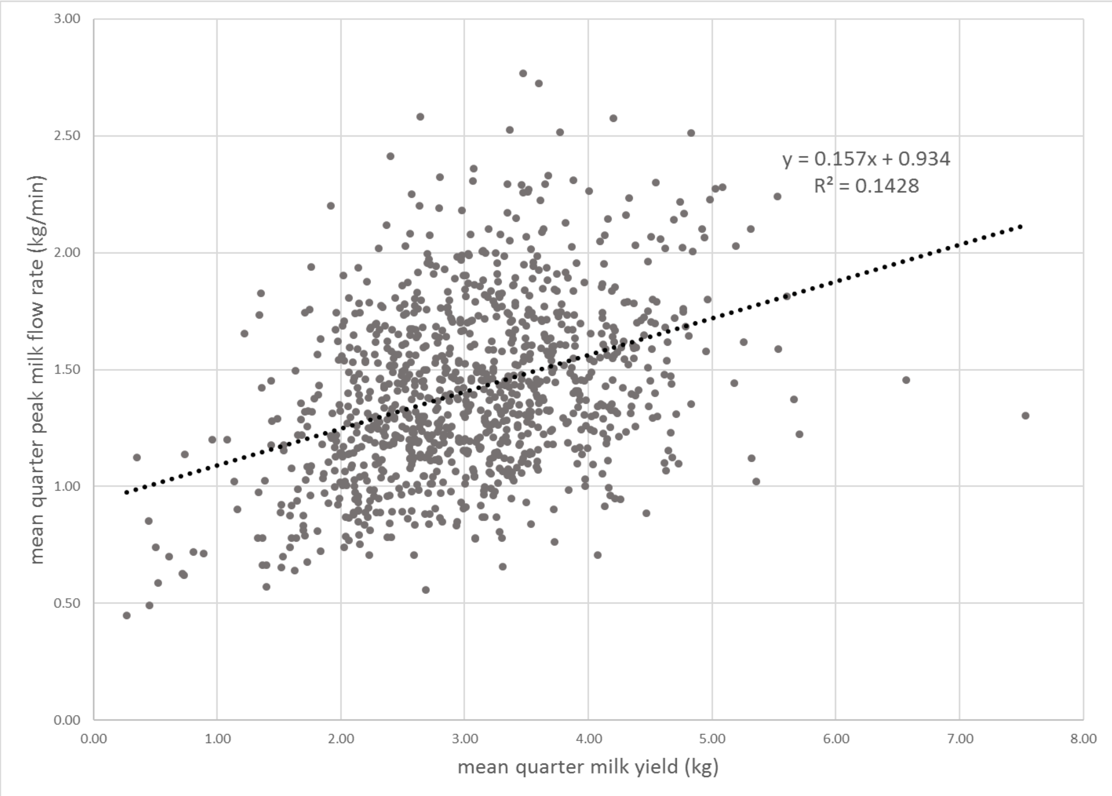
Regardless of how PMP influences new mastitis infection rate, it may be justified as a means of increasing the proportion of the herd experiencing oxytocin release and letdown prior to cluster attachment. Anecdotal evidence gathered from multiple milking time visits in New Zealand indicate that the majority of cows do not have a letdown until after cups-on based on VaDia vacuum device recordings. Poor letdown prior to cluster attachment, and the associated bi-phasic milk flow rate at the start of milking leads to an overall decrease in average milk flow rate and a "dribble finish" to milk flow near the end of an individual cow milking. It decreases milk harvesting efficiency. Interestingly, the study reported by Moore Foster et al. [21], described a mean of 25% of cows with delayed letdown in 64 Michigan herds with what appeared, on initial assessment, adequate PMP practices. The dominance of 3X milking herds in this study may have influenced the results due to the increased time for milk transit from the alveolar spaces to the gland cistern in 3X milking compared to 2X.

Milking speed

Milking speed is a term commonly used in describing milking management, but it needs to be carefully defined. It can mean either average milk flow rate or peak milk flow rate (both in kg/min). In this instance, manipulating teat-end vacuum, pulsation settings or liner compression will influence peak milk flow rate and hence average milk flow rate [23]. For example, increasing system vacuum or b-phase duration will usually increase peak milk flow rate (but not in all cases) while increasing liner compression tends to increase the range of potential peak milk flow rate. However, milking speed can also mean cows per hour through the parlour. In general, cows per hr is the primary metric to use to assess milk harvesting efficiency. Increasing peak milk flow rate, with a potential decrease in milking gentleness, is unlikely to have a large impact on cows per hr and hence should be considered carefully before application [24].

As an additional note on peak milk flow rate and average milk flow rate, a commonly held mis-conception is that increasing quarter or udder milk yield is associated with increasing milk flow rate – more plainly, large volume cows milk quickly. However, there is essentially no association between quarter or udder milk yield (per milking) and peak milk flow rate [25]. Knowing one parameter does not assist in predicting the other. This is illustrated in Figure 1 which describes milking data from a 1200 cow herd milked using an automatic milking system – note the wide scatter of datapoints around the regression line which has a weak coefficient of determination (R^2).

Figure 1: Plot of per cow, left front quarter milk yield in kg vs peak milk flow rate (QPMF) in kg/min for d in milk (DIM) window 50 to 60. All lactation numbers included.



Conclusion

There are 8 milking time test sections described in TN 13. Of these 8 areas, the most important influences on new infection risk are: teat disinfection (section L), environment (M), teat condition (I) and clinical case management (H). The performance test of machines (F) and milking time per cow (J) should always have their results compared to observed teat condition to ensure that a cohesive story around true risk factors for new infection can be made by the investigating team. Where an increased risk of environmental teat contamination is observed, the application of PMP for a short, or protracted period, may result in a decrease in new mastitis infection rate.

Any change in milking management which results in more reliable oxytocin release prior to cluster attachment will have the benefit of increased average milk flow rate. While peak milk flow rate and average milk flow rate are important metrics of milking speed, it is likely that the more important measure of milking speed is cows milked per hour. Milking gentleness should never be traded off for increased peak milk flow rate.

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Keeping Vets Happy at Work
UK Evidence of gender discrimination in our profession – result of the British Veterinary Association project

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Does Happiness in the workplace matter?

The United Nations World Happiness Report 2017 found that, worldwide only 20% of people are actively engaged in their work. This means that a large percentage of the worlds' workforce are potentially in a state of psychological exit – they are present but not engaged. In a veterinary practice there is nothing to say that this means they are less committed to animal health and welfare, but they are probably less committed to the advancement of the practice and disengagement can have a negative effect on a person's mental wellbeing. Unsurprisingly studies have shown that happiness and work are interlinked, and we know that happiness affects our mental wellbeing but there is also evidence that happiness can also be of economic benefit. A happy workforce can increase productivity and company performance. A paper by Warwick University showed that happy workers can be 12% more productive and unhappiness was linked with lower productivity.

Is the UK veterinary profession happy at work?

That's all great but how does this fit in with the veterinary profession? The UK veterinary profession has been going through a crisis of recruitment and retention with practices finding it more and more difficult to recruit vets, with 47% of practices taking over 3 months to recruit and 31% of practices failing to recruit at all. There are also plenty of anecdotal reports of a leaky bucket in terms of retention. Surveys by the British Veterinary Association have shown that when asked "Would you choose to be a vet again?" 15% of respondents said no and 58% were unsure. In another survey half of graduates, who were eight years graduated said their career had not met expectations.

Compounded by the UKs exit from the European Union the UK veterinary profession has found itself in a crisis. This led to a greater need to understand and evidence some of the problems that may be causing there to be such a problem. While there was some evidence of the problems and we could articulate long lists of these such as career progression, long hours, out of hours, pay transparency and more what we were missing was the linkages between the problems that would highlight the core reasons that recruitment and retention was failing.

Findings of a study on motivation, satisfaction and retention in the UK veterinary profession

This is where the British Veterinary Association collaborated with Dr Chris Begeny and Professor Michelle Ryan of Exeter University who had a wealth of experience in this area to try and find any linkages between the many factor raised regarding recruitment and retention problems. In short, our initial work set out to probe why there was a reduction in motivation early in the career, vets' lack of confidence, work-life balance and the gendered nature of the profession. Our results for this phase of the study showed some fascinating things around job satisfaction, motivation and retention:

- Motivation:
    - Vets' motivation is affected by their day-to-day experiences in the profession
    - While confidence levels were generally very high, career potential and ambition were only moderate
    - The most important predictors of these motivational feelings are feeling valued and admired, a sense of fitting in, and having role models
- Satisfaction:
    - Wellbeing and satisfaction are also linked with day-to-day experiences
    - Job satisfaction was only moderate and linked to having role models, feeling valued at work and endorsing a culture of long work hours
    - Levels of stress and burnout were not insignificant and were lowered by having role models and (worryingly) by endorsing the long hours culture.
- On retention:
    - We found evidence of psychological exit – those feeling less ambitious or a lack of engagement with their careers.
    - 37% reported actively thinking about leaving the profession.
    - The strongest predictor of retention was the availability of role models, followed by having a sense of fitting with those who have been successful before them.

Evidence of gender discrimination

So how did gender play into these results? The results above were similar for men and women but interestingly there were some findings that identified the gendered nature of the profession.

- Female vets report lower career ambition, lower confidence, as having less career potential, and higher burnout
- And female vets reported experiencing significantly more overt gender discrimination than male vets and less frequently being treated in a positive manner by their colleagues.

This led on to the second phase of the study to look in more detail at gender in the veterinary profession. This study showed how two experimentally identical vets were treated differently by their employer in terms of how they were perceived, treated and paid. And the only difference between the vets was that one was called "Mark" and the other "Elizabeth." One finding was that employers that believed that women no longer face discrimination in the profession awarded Mark between £1,100 and £3,300 more in salary and believed that "Mark" was more competent than "Elizabeth". On the basis of gender alone "Mark" was given a higher salary and deemed to be more competent than "Elizabeth."

The collaborative research between the British Veterinary Association and the University of Exeter has moved us away from anecdote and shown us evidence. The challenge to the profession now is to act on this and show true equality. A vet is not a female vet or a male vet, they are a vet, and should be given the same opportunities no matter what their gender. As a profession we must reach the point where we are not thinking: "Am I Mark or Elizabeth?". We need to confront any unconscious bias and install open and transparent approaches to employment, pay and progression.

National Wildlife Biosecurity Guidelines and Australia's wildlife health system

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Introduction

Wildlife Health Australia (WHA; https://www.wildlifehealthaustralia.com.au/) is the peak body for wildlife health in Australia. WHA's vision is "**Healthy wildlife, healthy Australia**" and our mission is to develop strong partnerships to better manage the adverse effects of wildlife diseases. WHA's roles include surveillance, reporting and communication and coordination of information to assist response to wildlife disease.

Biosecurity, in the national context, is defined as "the management of risks to the economy, the environment, and the community, of pests and diseases entering, emerging, establishing or spreading". Biosecurity can also be explained as the set of precautions taken to minimise the risk of introducing a pest or infectious disease into an animal or human population.

Biosecurity is important for everyone who works with wildlife and everyone who works with, or interacts with wildlife, has a role to play in wildlife biosecurity.

For our purposes "wildlife biosecurity" means managing risks, primarily associated with infectious diseases, transmitted from wildlife to humans (and vice versa), from wildlife to domestic animals (and vice versa) and between groups of wildlife; it refers to both individuals and populations. Good biosecurity focuses on risk assessment and management, appropriate work practices, hygiene, isolation and housing, reduction of stress, appropriate support from veterinarians and other professionals including diagnosis and treatment of sick or infected animals and people.

Australia's National Wildlife Biosecurity Guidelines

The National Wildlife Biosecurity Guidelines (www.wildlifehealthaustralia.com.au/AboutUs/News/NationalWildlifeBiosecurityGuidelinesreleased.aspx) (Wildlife Health Australia 2018) describe nationally agreed protocols for good biosecurity practices for the handling of wildlife, to reduce the risk of disease transmission between wildlife populations, as well as reducing disease risk for people and domestic animals. They contain simple, applicable and consistent protocols for good biosecurity practices for the handling of wildlife that can be used by all who work with wildlife, to reduce the risk of disease transmission.

Infectious diseases can have serious impacts on wildlife, humans and domestic animals. They can affect the individual, the population or, in some cases, the broader ecosystem itself. The direct impacts of pests and infectious disease on individuals (animal or human) can include ill health, increased mortality, reduced fertility or congenital disease. There may be more subtle effects which predispose individuals to death e.g. the individual is compromised by disease to such an extent that death occurs by other means (such as starvation, trauma or predation). Some diseases with wildlife as part of their ecology can negatively affect Australia's animal and human health, trade and market access, biodiversity and tourism.

Many people, including wildlife researchers, managers, wildlife carers and veterinarians work with wildlife in Australia. Diseases emerging from wildlife are well recognised as a significant threat to human and domestic animal health. Wildlife are vulnerable to diseases originating in human and domestic animal populations and disease can be spread between wildlife populations by human activities. Some examples of important biosecurity concerns in the wildlife sphere (in Australia or globally) include:

- Hendra virus (a fatal zoonotic disease), transmitted from flying-foxes to horses, and then to humans; an example of a disease that does not affect the wildlife host but can be transmitted to a domestic animal and then to humans
- sarcoptic mange in wombats (and other native mammals); the protozoal disease toxoplasmosis and koala chlamydia disease; are all examples of infectious diseases that have been transferred from feral or domestic animals to wildlife
- Australian bat lyssavirus and Salmonella infection in reptiles are examples of infections which may pass directly from wildlife to humans
- The protozoa Cryptosporidia; a human pathogen that can be transmitted from humans to wildlife, resulting in wildlife disease
- white-nose syndrome (a fungal disease of microbats in North America) has been inadvertently spread by humans visiting caves; the global spread of chytrid fungus, a disease of frogs has been linked to the trade of amphibians for pet, medical and food purposes.

In order to manage these risks we need to understand both the hazard (in this case, the disease) and the options available for minimising risks. Wildlife workers need to be informed about the possible diseases that are associated with the animal species with which they work. They need to be able to identify the hazards, assess the risk associated with each hazard, and implement appropriate control measures to ensure both animal and human health risks are properly managed.

The National Wildlife Biosecurity Guidelines are intended to be used by all people who work with, or interact with wildlife, including wildlife managers, researchers, veterinarians, carers, and others who handle or interact with wildlife. They are designed as a tool to help organisations and individuals working with wildlife to gauge their own biosecurity requirements and to assist them to develop a biosecurity plan suitable for their particular circumstances.

What is covered by the guidelines?

The guidelines provide over-arching information on infection and disease control, how to assess and manage biosecurity risks and how to implement appropriate hygiene and work practices. Information is laid out in sections explaining:

- Why is wildlife biosecurity important?
- Principles of disease transmission
- The concept of inputs and outputs
- Pathways of disease transmission relevant for Australian wildlife
- Principles of risk management
- What do we mean by risk management?
- How do we apply risk management to managing biosecurity risks?
- Assessing and managing transmission pathway risk
- How do we proceed if little is known?
- Veterinary and other professional input into wildlife biosecurity
- Recognising, recording and reporting wildlife biosecurity and disease issues

- The importance of recognising signs of disease in wildlife
- Recognising and managing sick and diseased individuals
- Recognising signs of disease in wildlife populations
- Reporting animal disease or deaths for investigation
- Wildlife and Emergency Animal Disease response
- General approaches to managing biosecurity risks in wildlife
- Basic biosecurity practices
- Higher level infection prevention and control
- Hygiene practices
- Personal protective equipment
- Isolation
- Workflow practices to manage biosecurity risk
- Recognising and managing higher biosecurity risk situations
- Lower biosecurity risk situations
- Zoonotic disease risk management
- Zoonotic disease risk for wildlife workers
- Managing biosecurity risk associated with bites, scratches and other injuries
- Reporting and responding to suspected zoonotic disease
- Wildlife zoonosis risk to the public
- Managing wildlife in care
- Reasons for maintaining wildlife in temporary care
- Types of wildlife care facilities
- Who cares for wildlife?
- General facility construction and management
- Managing risk associated with pest, feral and domestic animals
- Orphaned, injured and sick wildlife undergoing treatment and rehabilitation
- Managing biosecurity risk associated with specific wildlife situations
- Managing biosecurity risk associated with wildlife translocation and release
- Managing biosecurity risk associated with confiscated wildlife
- Managing biosecurity risk associated with sick animals
- Managing biosecurity risk associated with biological samples, animal carcasses and waste
- Managing risks of inappropriate use of antimicrobial medications
- Appendices include Relevant state, territory and national regulations, legislative bodies and agencies, and useful documents and websites, Recommended operational protocols to be developed and maintained by organisations working with wildlife, Cleaning, disinfection and personal protective equipment protocols, Animal identification, record keeping and reporting, Zoonotic risk management, Planning for wildlife procedures - recommended checklists, Staff training and operational practice protocols and Infectious diseases of concern in Australian wildlife

Links and references to more detailed and specific information are included.

Acknowledgements

WHA would like to thank all the groups and individuals who have contributed to this important project, including the federal Department of Agriculture and Water Resources, who provide key funding support.

WHA is very keen to hear from anyone with an interest in wildlife health and biosecurity. If you would like to provide feedback on the National Wildlife Biosecurity Guidelines or if you can help us spread the word about the guidelines and the importance of biosecurity for all who work with Australian wildlife, please contact us at admin@wildlifehealthaustralia.com.au.

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Innovative ways to enhance early detection of significant diseases

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Introduction

The early detection of new, exotic or emerging pests and diseases of national significance is critical to their effective containment and eradication. Early detection mitigates the size and impact of an outbreak of pests and diseases on animal and plant populations, the economy and the environment.

A partnership approach for surveillance to enhance early detection of pests and diseases is particularly important in the light of current and emerging risk factors such as urbanisation, increased global movement of people and goods, intensification of agriculture and climate change. Additionally the data collected in a comprehensive surveillance partnership supports the maintenance and development of international markets for Australian food and fibre.

Background

The Improving General Surveillance in Australia Working Group Report 2013 highlighted that the estimated time to detection of an Emergency Disease event presented a vulnerability to Australia's preparedness and response capacity. The General Surveillance Analysis Tool identified activities such as the routinely observing stock, producer recognition of emergency disease clinical signs and likelihood of reporting to a veterinarian, as activities that provided the greatest opportunity to improve time to detection. Additionally enhancement of veterinarians skills in livestock emergency disease recognition and submission of appropriate samples to the laboratory were considered to influence time to detection.

The Department of Primary Industries and Regional Development (DPIRD) piloted a number of novel approaches through the Boosting Biosecurity Defences Project (Early Detection of Emergency Animal Diseases) and also through a Department of Agriculture and Water Resources and DPIRD partnership on developing regional surveillance champions to minimise the time taken to detect an EAD.

Current Situation

The early detection of emergency animal diseases (EADs) project aimed to reduce the amount of time it would take to detect an outbreak of an EAD in WA, such as foot and mouth disease (FMD), by:

- Building the capability of livestock producers, veterinarians and other regional livestock stakeholders to recognise and report potential EADs;
- Strengthening regional disease surveillance networks, including providing a financial incentive for livestock producers to report disease; and
- Implementing a biosecurity surveillance information system to facilitate recording, analysis, mapping and reporting of livestock disease in Western Australia.

1. Biosecurity Intelligence Platform

A new livestock biosecurity information and surveillance system, the Biosecurity Intelligence Platform (BIP) was designed and developed, then integrated with other DPIRD systems. BIP is a platform for recording biosecurity information, including that relevant to early detection of animal diseases for WA. The overall intent is for WA to have the capacity for more efficient analysis of biosecurity information, resulting in potential earlier detection of an EAD. The system also provides the capability to produce multiple reports to analyse disease patterns and detect new or emerging diseases.

2. Subsidised Disease Investigation pilot program

A financial incentive pilot program, the 'WA' SDI pilot program, was developed and implemented (over 3 years) to subsidise the veterinary cost to livestock producers of investigating signs of disease in their animals. The SDI pilot program offered a subsidy of $300 towards the cost of veterinary fees plus mileage (limit 200km) for producers to engage veterinarians to conduct a comprehensive animal disease investigation. A further $800-$1200 per investigation was allocated to laboratory testing of samples collected for each investigation, through the DPIRD's Diagnostic Laboratory Services (DDLS).

The purpose of encouraging a greater number of comprehensive animal disease investigations and submissions to specific standards is to:

- Enhance specimen collection and information provided for 'reportable disease exclusions', to support market access and trade negotiations for WA.
- Increase producer understanding of biosecurity issues by engaging veterinarians;
- Increase the likelihood of producers recognising signs of significant disease in their stock and reporting them to a veterinarian.
- Strengthen the relationship and trust between producers and veterinarians to increase the likelihood of producers reporting unusual signs of disease.
- Increase the quality of each disease investigation by ensuring the quality of the samples, history and epidemiological information supplied.

The WA SDI pilot program is an extension of an existing subsidy program; co-funded between the National Significant Disease Investigation (NSDI) and DPIRD Livestock Biosecurity Disease Surveillance Program. Benefits of the extended SDI pilot program relative to the existing co-funded program were:

- Both producers and vets were encouraged to initiate the SDI, whereas the existing co-funded scheme is vet-initiated only.
- An increased number of comprehensive disease investigations conducted per year (300 per year was the target for the SDI pilot program).
- The criteria to qualify for the SDI pilot program was expanded.

A comparison of the total number of diagnostic SDIs submitted before and during the SDI pilot program shows a 5-fold increase; from 226 in the 3-year period before the program started to 985 in the 3-year period that the program was running (see Fig 1.).

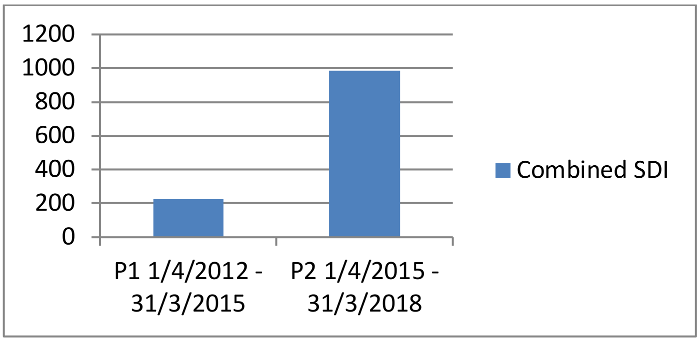

Figure 1: Comparison of the total number of diagnostic SDIs submitted in the consecutive 3-year periods before and during the SDI pilot program timeframe (SDIs from the SDI pilot program plus the existing co-funded program combined)

The SDI pilot program had a target of 300 SDIs and 100 reportable exclusions. Of the 481 SDI pilot program cases conducted, 308 resulted in reportable exclusions.

The increase in reportable exclusions from SDIs is likely to be partly attributable to engagement activities with private veterinarians, which reinforced awareness of signs and symptom of disease and the need to submit complete sample sets to laboratories when conducting necropsies. Additionally the laboratory actively ran exclusions for reportable diseases where appropriate. A comparison of the total number of FMD reportable exclusions resulting from notified cases before and during the 3 year timeframe showed a 16-fold increase (see Figure 2).

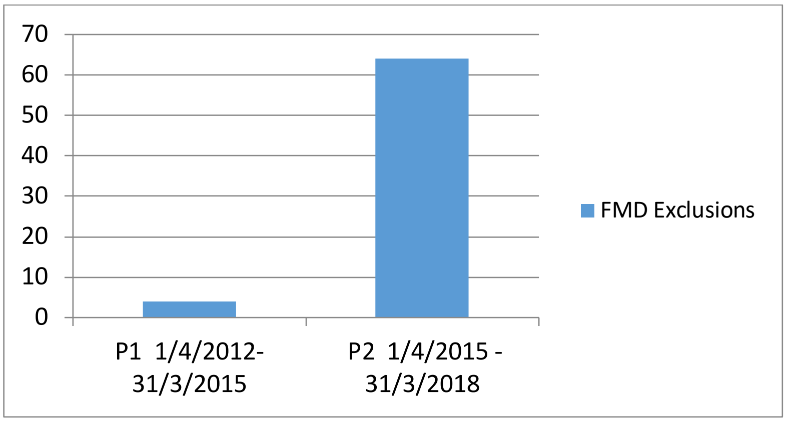

Figure 2: Comparison of the number of FMD exclusions conducted in the consecutive 3-year periods before and during the Early detection of EADs subproject timeframe.

A comprehensive analysis of the SDI program is currently being undertaken.

3. Engagement and promotion for early detection of EADs

While producers and veterinarians are key stakeholders for early detection of EADs, the project actively engaged a broader range of stakeholders, including saleyard, abattoir, livestock transport workers, livestock sales agents and tertiary institutions.

Producers were engaged through 43 workshops, field days or one-on-one meetings and 9 field day displays, reaching just over 1000 producers directly. An additional 3600 producers were reached via displays and interaction at large agricultural days. A bulk mail-out containing the early detection of EAD message was sent to 16,000 livestock producers.

Activities to increase veterinarian awareness of the WA SDI pilot program and to raise private veterinarian skills and awareness of the signs and potential impact of EADs included 12 group pathology workshops reaching a 130 veterinarians and visits to 42 veterinarians.

Stakeholders at congregation points were also targeted with 11 WA abattoir visits, communication outlining the early detection messages was sent to all WA abattoirs. Based on information gathered 2 state-wide abattoir EAD training workshops were developed and delivered. The objectives of the workshops was to strengthen inter-agency networking and improve participant awareness and preparedness around EADs, including requirements for sample submission. DPIRD provided sampling kits to each abattoir to further encourage submission of laboratory samples for reportable diseases. Following the delivery of the abattoir workshops there was a dramatic increase in submission of samples from abattoirs (figure 3).

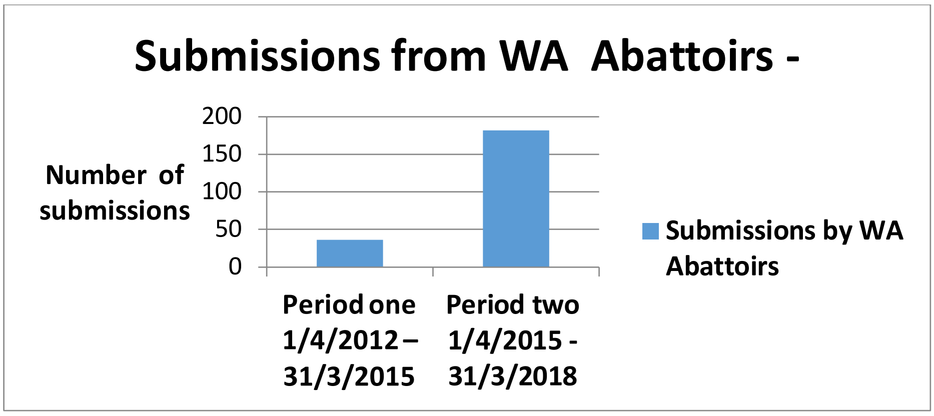

Figure 3: Comparison of the number of general tests performed on abattoir submissions in the consecutive 3-year periods before and during the Early detection of EADs subproject timeframe.

Targeted communications for saleyard personnel was seen as critical with about 1.2 million sheep and 250,000 cattle transiting yards in 2016. 20 information sessions were delivered to saleyard workers to raise awareness of EADs, potential economic impact, importance of early detection, clinical signs and how to report. The sessions reached a total of 72 saleyard personnel with an increase in the level of understanding of the risks and impacts of an FMD or BSE/Scrapie outbreak in WA.

Great Southern Cattle Surveillance Network

The Department of Agriculture and Water Resources and DPIRD are trialling an innovative cattle surveillance network in the Great Southern that uses SMS technology. The pilot has attracted more than 150 cattle producers as well as the support of cattle vets and agents.

DPIRD veterinarian Andrew Larkins and an industry advocate are coordinating the pilot project, which will initially run until April 2019.

The network aims to provide local producers with up-to-date information about syndromes occurring in cattle in the region, while also strengthening WA's ability to detect new, emerging or exotic diseases more rapidly.

Network members participate in a short series of SMS text messages each fortnight asking if they have seen any signs of illness in their cattle, they then receive monthly reports outlining cattle illnesses that have been occurring in the area, what the common causes were and options for prevention and management. Members who report signs of illness in their cattle can request a follow-up phone call from DPIRD. This might include services such as subsidised disease investigations and post-mortem sampling to obtain a definite diagnosis or it may be to refer them to their local private vet for assistance. The pilot will be evaluated once it has concluded.

CONCLUSION

The General Surveillance Assessment Tool (GSAT) was developed for evaluating the efficacy of the whole General Surveillance system across Australia, and uses Foot and mouth disease (FMD) as a case study. The outcomes of the GSAT modelling are for single farm sensitivity and estimates the time to detection of FMD for each of 12 regions in Australia; with WA one of those regions. Data for WA was first collected and run for the state of WA in 2011 by DPIRD veterinarians. Collection of a new set of 'expert elicitations' for WA was conducted in May 2018, by researchers contracted to Animal Health Australia.

The mid-2018 (post-subproject) results will be compared against the original values derived by Livestock Biosecurity in 2011. This will provide an indication of potential impact of the early detection project activities on estimated time to detection of FMD.

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Larkins, A and Dups, J, Great Southern Cattle Surveillance Network

Acute phase proteins in small animals

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Introduction

The research on acute phase proteins (APPs) and their applicability in clinical settings has greatly increased in the last decade. This increase has been the result not only of increased knowledge in the field, which is needed for interpretation of measurement results, but also of the increased access to appropriate assay systems for detection of relevant APPs. This paper will address the biology and kinetics of the major APPs, their diagnostic and prognostic role and the role in monitoring therapies.

Biology and kinetic of APPs

The APPs are blood proteins, synthesised in the liver in response to release of pro-inflammatory cytokines as part of the acute phase reaction (APR).[1-3] The term acute phase reaction describes a series of pathophysiological events that occur in animal exposed to infection, inflammation, trauma or other stimuli. APR begins within inflammatory sites, where cells involved in the innate immune response (i.e. macrophages and, to a lesser extent, neutrophils) produce and release pro-inflammatory cytokines (mainly IL-6, IL-1 and TNF-α).[4] These cytokines influence organs involved in homeostasis, such as nervous system and endocrine glands to establish a rapid and intense protective and reactive response. Cytokines are also responsible for the common clinical signs observed during systemic inflammation, e.g. fever, lethargy and anorexia.[4] The acute phase response also includes changes in the concentrations of plasma APPs, some of which decrease in concentration (negative APPs; e.g. albumin or transferrin) and others of which increase in concentration (positive APPs; e.g. C-reactive protein, serum amyloid A, haptoglobin, etc.).[1-3] Therefore, the APPs can be used to assess the innate immune system's systemic response and to differentiate local and systemic inflammatory diseases. In any given species, particular APPs demonstrate 'major', 'moderate' or 'minor' responses.

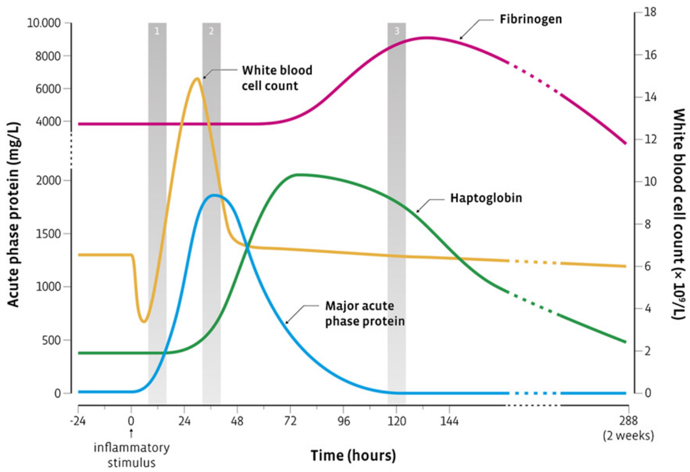

Figure 1 Kinetics of different APPs in domestic animals. From: Kjelgaard-Hansen and Jacobsen (Clin Lab Med 31:51-70; 2011)

A major APP 'responder' has a low serum concentration in healthy animals that rises dramatically by 100-1000-fold on stimulation, peaking at 24-48h and the declining rapidly during the recovery phase.[2] Moderate responders increase 5-10-fold on activation, peak after 2-3 days and decrease more slowly than major responders (Figure 1).[2] Major APP in cats is serum amyloid A (SAA) and moderate APPs are α_1 acid glycoprotein (AGP) and haptoglobin (Hp). Major APP in dogs are C-reactive protein (CRP) and SAA and moderate APPs are Hp and AGP.

Diagnostic and prognostic role of APPs

Acute phase proteins are highly sensitive, particularly those with major responses and increase soon after an inflammatory stimulus. APPs are considered a pre-diagnostic tool and are used as first line parameter during the initial laboratory screening. On the contrary, APPs are poorly specific because they increase secondary to numerous stimuli (e.g. infectious, immunologic, neoplastic, traumatic, etc.). Therefore, they are not useful to identify the source responsible for the acute phase reaction. However, APPS are extremely specific for inflammation: only the release of pro-inflammatory cytokines is responsible for acute phase protein synthesis in the liver and there are no storage sites. Thus, increased APPs should always interpreted as an ongoing systemic inflammation and should lead the clinician to investigate the site, type and severity of the inflammation (by means of clinical findings, diagnostic imaging, other laboratory tests, etc.). An exception is represented by feline infectious peritonitis (FIP), because measurement of APPs in effusions can help to differentiate FIP from other inflammatory diseases with a similar clinical presentation (e.g. body cavity effusions) (Figure 2).[5]

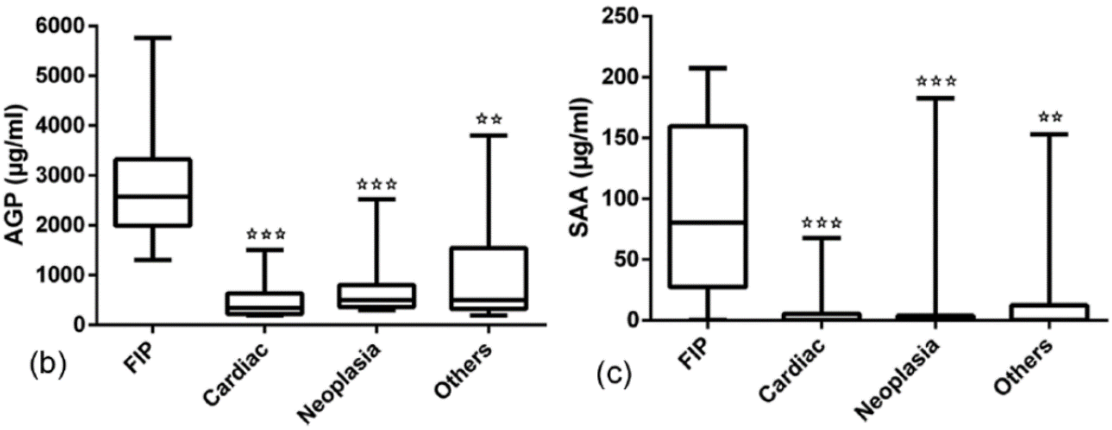

Figure 2 AGP and SAA concentration in effusion of cats with FIP and other diseases. From: Hazuchova et al. Journal of Feline Medicine and Surgery 19(8):809-816; 2017

A single measurement of the pre-operative or peak concentration of canine CRP is currently of limited value in the assessment of post-operative inflammation and thus the prognosis for the patient.[6-8] In dogs, the magnitude of increase in CRP, SAA and Hp is not correlated to the disease severity. On the contrary, SAA concentration in cats has been demonstrated an independent prognostic marker in cats with various diseases.[9] As has been documented for canine sepsis,[10] meningitis[11] and polyarthritis,[12] changes in CRP concentration over time may be more useful for monitoring of, and prognostic prediction in, surgical patients. Similarly, time-course monitoring of SAA in a cat with pancreatitis suggest that SAA was useful for evaluating disease exacerbation.[13] Unfortunately, there is a lack of consistency along studies on CRP baseline and pick pre- and after-surgery. Therefore, further studies are needed in order to further explore the usefulness of sequential measurements of APPs as predictors of infectious complications after surgery.[14]

The utility of APPs in the assessment of treatment response

One of the main advantage of the APPs is their 'real-time' secretion due to cytokines-dependent pathway. Because APPs are not storage, as soon as the inflammatory process is in regression, the APPs progressively decreased up to the baseline value. For that reason, APPs are extremely useful to assess the treatment response, bearing in mind that several treatments are symptomatic. Specifically, most of the clinical symptoms are mediated by prostaglandins and several therapies work interfering with phospholipase and COX pathways. In example, FANS or low dose of corticosteroids administration reduce prostaglandins release only, without any interference on the inflammatory pathway. A higher dosage of steroid will induce also a reduced cytokines release, thus a decrease in APPs synthesis. Therefore, the improvement of clinical symptoms not necessary is related with the resolution of the inflammatory stimulus and APPs are the ideal marker to evaluate the real-time evolution of the inflammation. Both CRP and SAA concentrations decreased rapidly after initiation of antimicrobial treatment in dogs with bacterial pneumonia.[8] When normalization of serum CRP is used to guide the duration of antibiotic treatment, treatment duration is significantly decreased without increasing the number of relapses.[8] In naturally retroviral infected cats undergoing interferon-ω therapy, SAA and AGP are useful to monitor the innate immune response potentiated by the drug.[15]

In conclusion, acute phase proteins are highly sensitive and specific, which could be use as first line marker to investigate inflammation. Their role in prognostication is limited without information about baseline values and the time points for further reassessments. APPs are extremely useful to monitoring patients once the therapy is started and to have a closer monitoring of the follow up.

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Antimicrobial Use and Resistance in Western Australia Dairy Herds: A pilot study

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ABSTRACT

This paper describes the results of a pilot study designed to provide evidence of the most common pathogens associated with subclinical mastitis in Western Australia dairy herds. A total of 162 isolates were recovered from bovine milk samples using MALDI-ToF mass spectrometry. The most common mastitis pathogens detected were Coagulase-Negative Staphylococci (n=42), followed by Corynebacterium spp. (n=31), Streptococcus uberis (n=27) and Staphylococcus aureus (n=19). A total of 104 isolates resulted in no growth/ no ID. The most common CNS species was Staphylococcus haemolyticus (n=16), followed by Staphylococcus chromogenes (n=12), Staphylococcus epidermis (n=5) and Staphylococcus xylosus (n=4). A total of 12 bulk milk samples were examined for the detection of contagious mastitis pathogens in bulk tank milk in WA. *Mycoplasma bovis* and *Streptococcus agalactiae* were not identified from the bulk milk samples. *Staphylococcus aureus* was identified in 7 out of 12 herds. CNS have become the most common mastitis pathogen in many countries. CNS species were the most common mastitis pathogens detected from chronic high somatic cows in this pilot study.

INTRODUCTION

Mastitis is an economically important disease and the leading cause of antibiotic use on dairy herds worldwide. Mastitis is an inflammation of the mammary gland that commonly originates from intramammary infections (IMI), most often caused by bacteria such as Streptococci, Coliforms, and Staphylococcus. The disease appears in 2 forms: either clinical, characterized by visible symptoms, sometimes general illness, and a long lasting negative effect on milk production; or subclinical, without visible symptoms but with an increase in somatic cell count (SCC) and suboptimal milk production

In Australia, its estimated that over $130 million per year ($200 per cow per year) is lost by the dairy industry due to poor udder health, mainly associated with mastitis (Dairy Australia, 2011). Furthermore, the subclinical form of mastitis accounts for almost two-thirds of this economic loss due to its long-term effects on milk yields. Thus, mastitis continues to be one of the most significant limiting factors, if not the most significant, to profitable dairy production in the United States and throughout the world.

Mastitis is the leading cause of antimicrobial use (AMU) on dairy farms and a risk factor to emerging antimicrobial resistance among mastitis pathogens. Pathogens resistant to various antimicrobial agents have emerged as a major concern of human and veterinary medicine. The emergence of antimicrobial resistant bacteria may be attributed to overuse and misuse of antimicrobial agents in humans and animals. Selection of antimicrobial resistant zoonotic bacteria in animals can lead to transmission of these bacteria to people by the consumption of contaminated animal products or by direct animal contamination.

To understand and manage the public health risks associated with the use of antimicrobials in food animals, quantitative assessment of antimicrobial use (AMU) in dairy herds is imperative for determining antimicrobial resistance (AMR) bacterial epidemiology. The availability of AMU data can aid in interpreting patterns and trends of AMR, serve as a basis of risk assessment of AMR, as a basis of decision-making for control measures, and to evaluate the effect of interventions for controlling AMR (WHO, 2003). Data on AMU in food animals are, therefore, becoming increasingly important for developing national and international policies to contain AMR. In addition to this, it can aid in improving treatment success rates by giving veterinarians access to contemporary resistance data.

In Western Australia (WA), data on AMU and AMR in dairy cattle is limited. The lack of AMU and AMR data may represent a short-term sustainability/ risk for the Australian dairy industry. Therefore, this pilot study was designed to investigate the most common pathogens associate with with subclinical mastitis in Western Australia dairy herds, and subsequently investgating the AMU and AMR in the herds.

MATERIAL AND METHODS

Study area and population

Data collected for this cross-sectional study originated from 12 dairy herds in Western Australia, between June 2018 and December 2018. Herds were included in the study based on their willingness to participate (convenience sample). Herd inclusion criteria included the participation in a DHI program in WA on an annual basis with an interval of 4 to 6 weeks between test days. All herds provided written consent to participate in the study. All procedures were previously approved by the Animal Ethics Committee at the Murdoch University (permit number R3003/17)

Sample collection

Individual cow milk samples were collected from 266 lactating Holstein and Holstein x Jersey cows. Milk samples were collected from chronic high somatic cell count cows, SCC >200,000 cells/ mL in two consecutive sample tests. Approximately, 10 mL of milk were collected from individual infected quarters and placed in a 30 mL screw cap sterile plastic tube. Samples were transported at ~5C to the AMR Laboratory at the Murdoch university for analysis.

Bulk milk samples were collected from 12 herds. Prior to collection, the milk from the bulk tank was agitated for 5 to 10 minutes, then, approximately 10 mL of milk was taken from the top of the bulk talk using a sterile pore free sampling scoop with rod. The sample was placed in a 30 mL screw cap sterile plastic tube and transported at ~5C to the AMR Laboratory at the Murdoch university.

Laboratory analysis

Laboratory followed standardized protocols for bacteriological culture. Pure cultures where identified using a MALDI-TOF and identified samples where frozen at -80^{o} for resistance testing. A milk sample from which 3 or more different species were cultured was considered contaminated, although colonies of Staph. aureus identified in these samples were enumerated. Sample sets with contamination in the composite sample were excluded from all analyses

MALDI-ToF Mass Spectrometry Identification

All isolates were characterized using the Bruker microflex MALDI-ToF MS system and Biotyper 3.0 software (Bruker Daltonik GmbH, Bremen, Germany). Samples were analyzed in duplicate using the direct transfer method as follows: bacterial cells from a single colony were transferred onto a stainless steel target, the spot was air dried at ambient temperature, was overlaid with 1 µl matrix HCCA (α-cyano-4-hydroxy-cinnamic acid) diluted in a solution of 50% acetonitrile and 2.5% trifluoroacetic acid, and allowed to air dry a second time at ambient temperature. The target plate was subsequently introduced into the microflex MALDI-ToF mass spectrometer for automated measurement of mass spectra and comparison to the reference database (Biotyper v3.0). Identifications scores ≥2.0 were required for confident species identification; scores <2.0 and ≥1.7 were considered confident genus identification;

isolates with scores <1.7 were re-analyzed in duplicate, and if they failed to achieve a score ≥1.7 on the second round, they were classified as unidentified.

Bulk Milk Tank PCR Collection and Identification

Milk samples DNA was extracted according to the PureLink® Genomic DNA Kit User Manual 3 (Blood Lysate). Samples where analysed using a PCR machine. Primers used were based on previous literature. Detection for S. aureus used a forward primer sequence (5′–3′) nuc GCGATTGATGGTGATACGGTT and reverse primer sequence (5′–3′) nuc AGCCAAGCCTTGACGAACTAAAGC 4. Detection for Strep agalactiae used SagroEL2 Forward 5′-GCAAGTTTTAGGACAGTCTGCT-3′ and SagroEL2 Reverse 5′-AGTTTCAGTGCCGCTACTTT-3′ 5

RESULTS

MALDI-ToF Mass Spectrometry Identification

Identification of 162 isolates were recovered from bovine milk samples using MALDI-ToF mass spectrometry. The most common mastitis pathogens (Table 1) detected were Coagulase-Negative Staphylococci (n=42), followed by Corynebacterium spp. (n=31), Streptococcus uberis (n=27) and Staphylococcus aureus (n=19). A total of 104 isolates resulted in no growth/ no ID. CNS isolates are listed on Table 2. The most common CNS species was Staphylococcus haemolyticus (n=16), followed by Staphylococcus chromogenes (n=12), Staphylococcus epidermis (n=5) and Staphylococcus xylosus (n=4)

Table 1. Prevalence of mastitis pathogens in chronic high SCC cow milk samples using MALDI-tof (n=167)

Mastitis pathogens	n	%
Klebsiella spp	1	1%
Pseudomonas spp	1	1%
Pasteurella spp	2	1%
Serratia spp	2	1%
Streptococcus spp (other)	3	2%
Streptococcus dygalactiae	3	2%
Enterobacter spp	4	2%
Bacillus spp	8	5%
E. coli	8	5%
Other	16	10%
Staphylococcus aureus	19	11%
Streptococcus uberis	27	16%
Corynebacterium spp	31	19%
Coagulase (-) Staphylococcus	42	25%

Table 2. Coagulase (-) *Staphylococcus* species isolated from chronic high SCC milk samples using MALDI-tof.

Coagulase negative *Staphylococcus* species	n	%
Staphylococcus hyicus	1	2%
Staphylococcus sciuri	1	2%
Staphylococcus equorum	2	5%
Staphylococcus simulams	2	5%
Staphylococcus xylosus	3	7%
Staphylococcus epidermis	5	12%
Staphylococcus chromogenes	12	29%
Staphylococcus haemolyticus	16	38%

Bulk Milk Tank PCR Collection and Identification

In total, 12 samples were examined for the detection of contagious mastitis pathogens in bulk tank milk in WA. *Mycoplasma bovis* and *Streptococcus agalactiae* were not identified from the bulk milk samples. *Staphylococcus aureus* was identified in 7 out of 12 herds.

DISCUSSION

The outcome of this pilot study provides a benchmark for the Western Australia dairy industry regarding the prevalence of subclinical mastitis pathogens in chronic high somatic cell count cows and bulk milk samples. Coagulase-Negative *Staphylococcus* (CNS) was the most common mastitis pathogens detected from in high somatic cows in this study, followed by *Corynebacterium spp*., *Streptococcus uberis* and *Staphylococcus aureus*. Coagulase-negative *Staphylococcus*, along with other agents such as *Corynebacterium spp*., are commonly considered to be minor mastitis pathogens, as opposed to major pathogens such as *Staphylococcus aureus*, *Escherichia coli*, *Streptococcus uberis*, and *Streptococcus agalactiae*. CNS is are the most common prevalent bacteria in herds using currently recommend mastitis control measures.

The high prevalence of CNS widely recognized but its importance remains a topic of debate [1-6]. Some authors consider CNS a main cause of subclinical and persistent mastitis [5,7], whereas others suggest that CNS have a protective effect against major pathogen IMI [8]. Additionally, studies have found no effect on milk production or decreased milk production associated with CNS IMI have also been reported [9,10].

The contrasting findings among researchers regarding the effect of CNS on udder health and milk production could be the result of regarding the CNS as one group[11]. CNS are a large and heterogeneous group [12], and it is known that differences exist among CNS species regarding their interactions with the host and the environment; consequently, they may have variable effects on udder health and milk production[13,14]. For example, IMI with *Staphylococcus chromogenes*, *Staphylococcus simulans*, and *Staphylococcus xylosus* have a greater effect on SCC compared with IMI with other species, such as *Staphylococcus cohnii* and *Staphylococcus sciuri* [7,15]. Some species, such as *Staphylococcus chromogenes* and *Staphylococcus epidermidis*, seem to be host adapted, whereas others, such as *Staphylococcus haemolyticus*, act as opportunists [16].

Herd prevalences were reported in this pilot study for *Mycoplasma bovis, Staphylococcus aures and Streptotococcus agalactea* are apparent prevalence. True herd prevalence, defined as herds that have contagious pathogen-infected udders, can only be determined if the sensitivity and the specificity of testing BTM are known. The sensitivity of a single BTM culture is low, between 21% and 54% for *S. aureus* and *S. agalactiae* and between 33% and 59% for mycoplasma. However, if consecutive samples are taken sensitivity will increase. The specificity of the test for the 3 pathogens can be assumed to be close to 100%. True prevalence is therefore probably higher than the apparent prevalence reported here.

The *Staph. aureus* herd level prevalence is this study was in agreement with earlier studies in North America and Europe, where herd level prevalence ranged from 31% to almost 100%[17-20]. Sampling bulk tank milk only once for Mycoplasma bovis may give an underestimation of the prevalence, due to intermittent shedding [21]; therefore, multiple sampling should be performed. Sensitivity of a single culture of bulk milk samples for *Mycoplasma* spp. ranges from 33% to 59% [22].

CONCLUSION

CNS have become the most common mastitis pathogen in many countries. CNS species were the most common mastitis pathogens detected from chronic high somatic cows in this pilot study. Further studies are necessary to elucidate the most common causes of clinical cases of mastitis and the true prevalence of Mycoplasmas spp in WA dairy herds.

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Alternative engagement methods for improved peri-urban smallholder livestock health

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The NSW Peri Urban Biosecurity Program

The NSW Peri Urban Biosecurity Program was established in August 2015 to address biosecurity risks within the peri-urban sphere. The program was initially piloted in the Greater Sydney region with Local Land Services (LLS) as a key program partner.

Peri Urban in the context of this collaborative biosecurity program refers to the urban-rural interface or urban fringe. It is a dynamic area or transitional zone with mix of urban and rural activities, including a complex mix of commercial, rural-residential and varied agricultural uses. It incorporates more than just smallholders and also covers the biosecurity risks associated with our gateways or ports. Peri urban regions in Australia comprise < 3% land use but responsible for 25% total gross value of agricultural production (GVAP)[1].

The NSW Peri Urban Biosecurity Program covers all biosecurity threats including animal and plant pests and diseases, weeds and pest animals. The program has enabled NSW Department of Primary Industries (NSW DPI) and the LLS to better understand peri-urban smallholders, their attitudes and behaviours in relation to biosecurity. These insights have been used to deliver targeted engagement and capacity building efforts to achieve improved biosecurity outcomes.

NSW Peri Urban Biosecurity Program rationale

Greater Sydney LLS region was selected as the Program's pilot location as it has long been recognised as a high biosecurity risk area for NSW. The risks are growing for this region as well, due to the large number of international travellers, major ports and a high density of smallholders, increased urbanisation, globalisation, climatic changes, changing demographics, cultural diversity and the increase of emerging diseases.

Greater Sydney is the gateway to NSW and as a result, is pivotal in securing market access for the $17.5 billion NSW primary industries sector[2]. NSW's economy, valuable primary industries, environment and community are all dependent on the effective management of biosecurity threats. Effective management of the biosecurity risks in Greater Sydney will protect and improve the whole of NSW's agricultural economy.

The Greater Sydney region has the largest peri-urban landscape in size and population density in NSW with approximately 5.1 million people. Its agricultural production industry is worth some $14.5 billion a year (GVAP) with poultry and horses the primary livestock enterprises[3]. Based on the LLS rateable land size classifications, the number of smallholdings <10ha and based on property identification code (PIC) data has increased seven-fold to around 31,000 holdings in Greater Sydney over the 10 years from 2007 to 2017. Other coastal regions have reporting similar growth of smallholdings, though to a lesser extent. It is expected this figure of smallholdings in Greater Sydney is conservative, as it doesn't include vegetable/horticulture enterprises and those who keep livestock but do not have a PIC.

Tools for engaging peri-urban smallholder livestock owners

Since its inception, more than 20 projects have been undertaken as part of the Program, including the development of four online education programs for veterinarians, hobby farmers and school-aged children; delivery of some 25 seminars and workshops; engagement with more than 50 stakeholder organisations and 250 individuals.

To establish the foundations of good engagement, the Program undertook a series of investigations seeking to better understand the motivations, practices and attitudes of peri-urban smallholders within the Greater Sydney region. Much of the research presented was undertaken with funding from the Australian Government's Agricultural Competitiveness White Paper under the government's plan for stronger farmers and a stronger economy. Through these initiatives, we sought to understand smallholders' biosecurity attitudes and behaviours, engage people whose livestock have names, engage smallholders using livestock champions and small farms networks as a communication tool and support non-English speaking background (NESB) producers via community service providers and cultural communication channels.

Key findings of our investigations suggest that smallholders generally do not feel as though they are part of any formal industry networks and look to alternative pathways to seek information and a sense of connection.

Veterinarians are seen as a key trusted source of animal health information by smallholders. Understanding the key drivers and motivations of smallholders can help in building vet/animal health-smallholder relations and trust by finding the "hook", (such as animal welfare, food for home consumption and family health) for continued engagement.

In addition, the Program has had success in engaging and influencing smallholders:

- via trusted local producers and community leaders who act as a "champion" or for those who are new to livestock or less experienced
- via service providers such as butchers, produce supply stores and saleyards/abattoirs. This has been particularly effective in engaging non-English speaking background (NESB) smallholder producers.
- utilising known social media networks such as Facebook, Instagram, Twitter and YouTube and cultural networks such as WeChat and Kakao Talk. These channels have been particularly effective in recent campaigns to highlight the risks of swill feeding for the introduction of African Swine Fever (ASF) into Australia.

Supporting veterinarians working with smallholders in peri-urban areas

Based on our understanding of the attitudes and behaviours of smallholders in peri-urban areas and the potential biosecurity risks faced in these areas, NSW DPI teamed up with the Australian Veterinarian Association (AVA) and Animal Health Australia (AHA) to develop an online training module. The module improves the knowledge and confidence of veterinarians and animal health specialists who service hobby farms or smallholdings in:

- investigating livestock and wildlife diseases (especially notifiable diseases)
- engaging, collaborating and knowing when to get help from government veterinarians
- knowing where to find biosecurity and communication resources for smallholders
- understanding their role within Australia's animal surveillance system for reporting and investigating notifiable diseases.

In addition to these online training modules, the Program worked with the Livestock Biosecurity Network (now part of AHA) to produce a YouTube video to educate smallholders on the biosecurity risks associated with purchasing stock online. The risks of this practice are growing, as electronic trade of livestock is increasing rapidly and smallholders are willing to travel further to purchase stock. Other resources for veterinarian and animal health professionals are available on the NSW DPI website.

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Australia and the global context

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Introduction

One health refers to the collaboration of multiple disciplines, sectors and groups working locally, nationally and globally to attain optimal health for people, animals and the environment. None of the challenges faced by human kind or indeed those brought to bear on other species and the environment, due to our actions, exist in isolation. It is the complex interplay and web-like dynamics of all living things that shape our experiences and the challenges we face.

The importance of one health

Globally we are facing unprecedented challenges requiring global one health responses. Humans have had an unrivalled impact upon the environment, climate and global biodiversity which is rebounding upon human and animal health. Understanding and interpreting the complex interplay between human, animal and ecosystem health, is critical to recognising and managing disease issues into the future.

The global context – Antimicrobial resistance

Antimicrobial resistance (AMR) is an issue that cannot be progressed without a one health approach. The drivers of AMR are multifaceted and interconnected and include the use and misuse of antimicrobials, inadequate governance, fragmented regulation around antimicrobial use and a general lack of awareness of the risks of AMR. These drivers have been amplified by population growth, mobility and demographics and the demand for antimicrobials in food production and health.

The global context – Food security

Food security is of growing concern to many countries and is yet another issue where a One Health approach is required to fully understand and tackle the problem. Not only do animal and human health experts need to be involved but economists, social scientists and other related disciplines are necessary to help understand the complex factors that underlie food availability to consumers.

Australia's position

Australian agriculture has ambitious goals for the future. In 2018 Australia's National Farmers' Federation (NFF) announced their vision to increase farm gate output to $100 billion by 2030,[1] a 67% increase on today's production value. The NFF's 2030 roadmap includes targets such as halving food waste, 50% renewable farm energy sources and trending towards a carbon neutral agriculture sector by 2030.[2] The NFF believe their vision of a $100 billion industry is within reach if the economic, social and environmental policy settings are right and have recognised the importance of a collaborative approach to both overcome the challenges mentioned above and to make the most of the opportunities.

Given the unprecedented global challenges we face in terms of food security, emerging infectious diseases, decreasing biodiversity and climate change, it will be essential for Australian agriculture to employ a one health approach in order to reach the identified targets. Australia has begun to recognise and harness these opportunities and some examples are discussed.

Conclusion

The One Health concept is not new however its modern application is, in my view, still in its infancy. Both in Australia and across the globe, our global institutions are still predominantly organised in sectoral terms resulting in differences in organisational cultures, missions, priorities, funding, and direction. We especially need to convince our leaders to step outside their traditional approach and move to managing an organisation where interconnectedness, synergy and a pursuit of the 'greater good' is both valued and rewarded. So, there are many challenges, but also significant opportunities, for today's leaders and practitioners to forge new One Health approaches to both old and emerging problems.

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A review of facial acupuncture points with anatomical relation to cranial nerves and the skull in the horse

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Introduction

There is a lot of focus on diseases of the equine head in western medicine and some of the pathologies have shown to be difficult to treat conventionally. TMJ, hyoid pain and dysfunction, eye problems, larynx pathology, headshaking with all its possible diagnosis such as photo sensibility, trigeminal neuralgia, infraorbital nerve neuralgia, fascial nerve paralysis, and tooth pain are just some examples. These are pathologies for which many clients seek holistic treatment to try and solve due to the limited success in conventional medicine.

From a holistic point of view, we will of course look at the whole patient and come up with a whole horse approach so we can treat the underlying cause with distant points but it is also important to use local points. To do so properly knowledge of the underlying head anatomy is important especially the cranial nerves and the skull with its foramina and sutures which is the purpose of this paper.

The 12 cranial nerves

The cranial nerves are well-known but what I will put emphasize on here is through which foramina they leave, their function and which local acupuncture points they are close to.

The first cranial nerve **n. olfactory I** is not described to commonly produce symptoms in the horses, so I will leave it for now.

The following five cranial nerves leave the cranium in the same area, fissura orbitalis. Fissura orbitalis is in the back of the orbit in the triangle between the pre- and basis sphenoid bones (which has sutures dorsal and caudal to the temporal bone), the ventral-caudal part of the frontal bone and the caudao-lateral part of the pterygoidea bone in the back of the orbit (1).

The fissure contains five foramina where the following nerves leave the cranium: N.opticus II through canalis opticus, N.oculomotorius III, N.trochlearis IV, N.trigeminus V (except from the mandibular branch which passes through the foramen lacerum) and N.abducens VI (2).

The second nerve **N.opticus II** is a tract of the brain and it has a sleeve of dura mater surrounding it which blends with the sclera. This means that an excessive cerebrospinal pressure can create a pressure on the nerve and might be the cause of photo sensibility and other less serious disturbances of the eye. It also means that a tension of the dura mater in humans can show up in difficulties in turning the eye laterally, if it also is true for animals is unknown. But in a lateral flexing test of the upper cervical vertebrae, some horses tend to avoid turning the eye laterally as if they cannot perform the test. The optic nerve passes through the optic canal and lies protected between the muscles of the eye.

The function of the third cranial nerve **N.oculomotorius III** has both somatomotor and parasympathetic fibres. It controls the muscles of the eye, eye lid and the pupil (1).

The fourth nerve **N.trochelaris IV** innervates the muscles of the eye with somatomotor fibres (1).

The big fifth cranial nerve **N.trigiminus V** carries somatosensitive fibres from skin and mucos membrans of the head and somatomotor fibres to the masticatory muscles. It has many branches. The three main branches are: n.opthalmicus, n.maxillaris and n.mandibularis. **N.opthalmicus** carries sensory fibres from the eye, eye lids, conjunctiva, and the skin around the eye and on the forehead as well as part of the nasal mucosa. **N.maxillaris** has sensory fibres from a small part of dura mater, the zygoma and temporal bones on the lateral side of the head, the palatine and also the nasal mucosa. The continuation of n.maxillaris is n.infraorbitalis that leaves the orbit through canalis infraorbitalis and exits through for.infraorbitalis. It innervates the skin of the lower part of the face from the middle of the head, the maxillary teeth, gingiva and the nasal region. **N.mandibularis** exits through for.lacerum (for.ovale) below the ear. It has mixed fibres that innervates the chewing muscles as masseter and temporal muscles, the mandibular part of the head, the cheeks, ear and parotid gland (1).

The sixth nerve **N.abducens VI** is also serving muscles of the eye with somatomotor fibres (1).

The seventh cranial nerve **N.fascialis VII** exits the cranium through the foramen stylomasteoideum caudal to the caudal part of the pars petrosal. It carries both motor and parasympathetic fibres to the face, hyoid muscles, ear and eye and taste fibres. It has a branch; ramus buccalis dorsalis which merges with the infraorbital nerve (2).

The eighth nerve **N.vestibulococlearis VIII** carries fibres of the vestibular organ for balance and for hearing (1).

The next three cranial nerves exit the cranium below the ear through foramen lacerum which is larges in the horse (absent in ruminants and carnivores). It is a fusion of more foramina such as for. ovale, for. spinosum, for. jugulare, fissure petrooccipitalis and canalis caroticus, so the different parts of for. lacerum still carries these names but with incisura instead of foramen (2). It is limited by the following three cranial bones: the occipital bone ventral, pars petrosal of the temporal bone dorsal and lateral, and the sphenoid bone rostral. The sphenoid-basilar suture is in the rostro-ventral parts of the foramen.

The ninth cranial nerve **N.glossuspharyngus IX** passes through for.jugulare (cranial part of for.lacerum) and carries motor, sensory and parasympathetic fibres to the pharynx, tough, tympani and carotid sinus (1).

The tenth nerve is the very important **N.vagus X** which passes through for.lacerum. It has motor, sensory and parasympathetic fibres. It merges with some of the other nerves: n.glossupharyngus, n.fascialis (part of pinna) and n.accessorius. It innervates the larynx by the laryngeus recurrens nerve, pharynx, inside of pinna, and all the inner organs through truncus vagosympaticus (1).

The eleventh cranial nerve **N.accessorius XI** passes as already mentioned also through for. lacerum and innervates part of the shoulder muscles (trapezius, brachiocephalic and omotransversarius mm.)(1).

The last and twelfth nerve is the **N.hypoglossus XII** which leaves through canalis n.hypoglossi dorsal to for.lacerum. It is solely a motor nerve and it innervates the tongue and some of the hyoid muscles (1).

This means that the two most important areas where these cranial nerves pass through the cranium is behind the orbit in fissure orbitalis and below the ear and pars petrosal of the temporal bone through for.lacerum. This means that the points around the eye **BL1, ST1, GB1** and **JING-SHU** where the needles are inserted into the connective tissue between the sclera and the lining of the orbit possible can have effect not only of the eye CN II, sight and dura mater but also on the CN III to VI.

The closest access to the for.lacerum and the CN VII to XII seem to be through GB2 which is placed in the fossa at the caudal aspect of TMJ ventral to the tragus of the ear (4), TH17 which is located in the depression between the mandible and mastoid process, ventral to the horizontal ear canal (3) and KAI-JIN-ZHUI (3). This point is located in the depression just dorsal to the caudodorsal aspect of the coronoid process of the mandible.

The innervation from the n.maxillaris which as mentioned before have sensory fibres from small part of dura mater, the zygoma and temporal bones on the lateral side of the head which then covers the area of GB2,TH17 and KAI-JIN-ZHUI might also connect these points to CN V passing through fissure orbitalis. The indications for TH17 and GB2 are similar; fascial paralysis, brain and cervical problems and for KAI-JIN-ZHUI it is TMJ problems, cervical stiffness and SI problems can well be explained by their anatomical placement. The effect on the SI joint works most likely through the dura mater.

More peripheral accesses to the CN nerves can be gained through superficial foramina. From for.infraorbitale the nerve of the same name (part of CN V) emerges and merges with a branch from N. fascialis (VII). The new position of IVAS ST2 is here and ST3 from Schoen (4). The point is found just above a line between the upper bony part above the nostril at the nasal bone and the rostral end and the fascial crest. The indications are eye problems, fascial paralysis and trigeminal neuralgia which fit with the innervation.

BAO-SAI is overlying the ramus dorsalis of the n.fascialis in the large depression in the masseter muscle, midway on a line from the lateral canthus of the eye to the ventral aspect of the mandible (3). The indications are swelling of the face, myofascial pain of the masseter muscle and fascial paralysis.

EAR SHEN-MEN on the inside of the ear are innervated by the n.fascialis with a branch to n.vagus which fits well with its indications: calms the mind and relieves anxiety (3).

LI18, SP9 and SP10 are all overlying the truncus vagosympaticus in the jugular sulcus along the ventral part of the neck. Some of the indications are idiopathic laryngeal hemiplegia, pharyngeal problems and stomach ulcer all connected to n.vagus.

The two important areas for the exits of the CN are very important in the cranio-sacral therapy (CST). The fissure orbitale are in a suture cross point of the parietal, frontal, sphenoid, temporal and pthyroid bones in the orbit which means that any tension between these bones might influence the cranial nerves II to VI. The author has experienced triggering of headshaking symptoms by treatment of the supraorbital area (the parietal, frontal, sphenoid, temporal area). One horse had had an injury to the frontal bone and showed excess redness of the nasal mucosa and was headshaking. Treatment with CST of the frontal-temporal suture, parietal-temporal suture and the sphlenoid-occipital suture (sphleno-basilar suture) releases tension of the orbit and thereby of fissure orbitalis and it passing structures, as might treatment of the eye points.

In the cranial motility there is an external rotation of the arcus zygomaticus of the temporal bone which then provides an internal rotation of the pars petrosa of the temporal bone. It can then put tension to the structures passing through the for.lacerum as a.carotis internus and CN VII to XII and through the other foramen close by. From a CST point of view soft treatment of the sulcus between the pars petrosal and the proc. paracondylare and the whole temporal bone will affect the area the most. The points mentioned above around the TMJ can possibly also release the tension in this area.

AP points overlying cranial sutures

There are some classical points and points of the GV whish are overlying the sutures of the cranium. These points can possibly affect tension of the cranium as dealt with in CST.

Just between **GV20** and **GV24** are the junction of the phalx cerebri, phalx cerebellum and the tentorium of the meningeal membranes. GV20 is located on the midline in the depression at the highest point of the poll (3), just rostral to the nuchal crest. The indications are prolapse of any inner organ, brain, eye, and ear problems, unconsciousness, shock, anxiety and immune stimulation. GV24 is the main point of **DA-FENG-MEN**'s three points, it is situated where the external sagittal crest of the parietal bones joins on the midline at the rostral base of the forelock. The two other points are located 1 cun latero-rostral to GV24. Indications are anxiety, rhinitis, convulsions and other brain problems (3). These indications correspond well with the close proximity to the meningeal membranes.

LONG HUI/YIN-TANG at the parietal- frontal suture which is named BREGMA in CST (5) just above where the septum between the frontal sinuses are ending and the attachment of the phalx cerebri. Indications are sinus problems, cervical problems, anxiety, seizures and other brain problems (3).

TONG-TANG is located in the depression at the junction of the frontal bones, at the level of the medial canthi (3). It is overlying the frontal-nasal suture which is named NASION in CST (5). The indications are sinus problems, cervical problems, anxiety and brain problems (3).

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Fascia – the forgotten tissue

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Connective tissue

Myofascia is for many the “forgotten” or even unknown tissue. When I followed the anatomy course at the veterinary school we dissected the entire fascia away so we could expose the muscles, vessels and nerves – “the real stuff”. We were not taught that fascia had much biomechanical function.

Fascia is part of the connective tissue that arises from the mesoderm in the embryo. On the 17th day of gestation the mesodermal cells start to develop and divides in the paraxial mesoderm which later shapes the axial somatic structures and the intermediate mesoderm that builds the visceral and parietal layers covering the viscera and the body cavities. Intermediate mesoderm can be viewed as a double back. From the mesoderm the mesenchymal cells develop and specialise into many types of cells such as blood stem cells, myoblasts, fibrocytes, osteocytes, chondrocytes, masts cells, fat cells, reticulum cells and endothelial cells. They form different connective tissue types like adipose tissue, soft interstitial tissue and muscles as well as dense connective tissue like fascia, tendons, ligaments, aponeurosis, bone and cartilage. Connective tissue consists of fibrocytes and fibroblasts in an extracellular matrix containing collagen, elastin and reticulin fibres (in variable amounts) as well as interfibrillar proteins such as glycosaminoglycans and proteoglycans. Collagen itself shows up in many molecular and architectural variations, in fascia interwoven collagen fibres are the major component. In general, the different types of connective tissue are woven into all the tissues of the body as a large continuous web (1).

In the muscle the single muscle cell is covered with a layer of connective tissue, the endomysium. Around bundles of muscles fibres perimysium forms a fascicle and peripheral to this around the muscle body lies the epimysium which is closely connected to the myofascia. The intramuscular connective tissue unites at the muscle end and form tendons or an attachment to the outermost elastic layer of the periosteum. At the inner part of the periosteum collagen fibres insert into small channels of the compact bone (2).

At muscle/tendon attachments and intersections such as at the coxal tuber or calcaneus it is possible to release the connective tissue from the bone by blunt dissection and still maintain the myofascial connections. This shows that a bony attachment is not a final “station” for the muscle/tendons but functions as an anchoring point and a connection point to other structures from where the forces can continue. The fascia sheets surrounding the muscles also continue into fascia sheets from other muscles and distribute strain and forces in several directions -and seen in a larger perspective- keep the whole body together (2). Myers 2009 (2) refers to the muscles as “one big muscle in 600 bags” describing the interstitial connective tissue as a big web-like bag. From this point of view the anatomical reductionist way of describing a muscle and its function with origin, insertion and specific single function is outdated. It is convenient for explaining single structures but not for describing body locomotion.

The myofascia which surrounds the muscles and the body acts as sleeves and can become tight due to the fact that fascia has a contractile property. Fascia has long been thought to show some plasticity but still to be inelastic and non-contractile. Myofibroblast cells – a” middle ground” between smooth muscle cells and fibroblast cells can provide contractile forces in fascia. They can provide long-duration, low-energy consuming contraction without nervous stimulation but induced by a) mechanical tension in the tissue b) pharmacological components such as histamine, mepyramin and oxytocin (contraction) and nitric oxide (relaxation) together with cytokines. Also, the pH of the tissue influences the contractility where a low pH induces contraction. The contraction happens very slowly over 20 – 30 minutes and lasts for more than one hour and the fascia get stiff and tense due to the myofibroblasts pull on the matrix components (2).

Tensegrity

To further understand this continuous connective web the concept of tensegrity is important. It is described by the designer R. Buckminster Fuller. The word is a composition of “tension integrity” meaning a construction or structure that can hold its shape (integrity) by a combination of tension and compression forces in balance. A tensegrity model can be built of pins (compression structures) and elastic band (tensional structures). The compressed members are not touching each other and the tensioned members delineate the space. The structure collapses if the compression members do not push out and hold up, and it falls apart if the tensioned members fail to pull inward and hold together. When force is applied to one area of a tensegrity structure the forces are distributed to the whole structure and in that way the structure adapts to different changes in context and pressure of the surroundings.

A lot of natural structures such as cells, water molecules, carbon atom and even living beings have tensegrity architecture. It is described by Ingber and Levine using the word biotensegrity (2). When we look at the body as a tensegrity structure with the connective tissue as the tensional members, it is easy to understand that injury or overstrain in one area can cause symptoms in another. An injury which may happen to one area spreads due to the tensional forces through the whole body and symptoms can show up at the body’s weakest link. Put into a TCM concept the injury is an external excess and the weak link is an area of deficiency. Pain (excess) can arise in one area but the cause of it comes from another place (the deficient area).

This understanding of the fascinating three-dimensional fascia network through the whole body and its properties, provide us with an anatomical foundation for arguing for the importance of holistic medicine.

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Fascia – the anatomical basis for acupuncture points and meridians in the equine back?

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As a continuation of the myofascial kinetic line dissection study, there is a close anatomical relationship to many of the 12 main acupuncture meridians (1) where the Superficial Dorsal Line (SDL) lies close to the Bladder (BL) meridian, the Superficial Ventral Line (SVL) lies close to the Stomach meridian, the Lateral Line (LL) to the Gallbladder (GB) meridian, the Deep Ventral Line (DVL) to the Foot-Yin meridians, Front Limb Protraction Line (FLPL) to the front limb Yang meridians and the Front Limb Retraction Line (FLRL) to the front limb Yin meridians. The two helical lines, the Spiral Line (SP) and the Functional Line (FL) do not relate to acupuncture meridians (1). Further dissections have been conducted and fascial connections to acupuncture points have been found. The work of Langevin & Yandow, 2002, has been the inspiration to this study (2). The Bladder meridian and Governing Vessel points of the back will be presented as well as fascia – acupuncture point relationships at BL39, BL 40, Kidney (KI) 10, KI27, Lung (LU) 1, and GB 20.

Langevin and Yandow (2002) (2) investigated the hypothesis that acupuncture points are situated over fascial planes. They marked locations of acupuncture points and meridians on a human upper arm and made serial cross sections, each section measuring 1 cun and then they described the underlying tissue. More than 80% of the points and 50% of the meridian intersections appeared to coincide with intermuscular or intramuscular connective tissue planes.

Fascial planes along the back in the equine

In order to look at the fascial planes in relationship to acupuncture meridians of the back of horses, the back of a Norwegian Fjord from Th4 to sacrum was frozen and sliced transverse at the level of each vertebral body. A 5 year old Arabian, a pony and an Icelandic Horse, 20 years old, were also dissected in this study. Photos were taken to show the fascial planes between the different muscles and the spine at the level of the inner and outer branch of the Bladder meridian (BL) and the Governing Vessels (GV), Du Mai both in the transverse and longitudinal plane. The anatomical description and the fascial planes were compared to the two meridians. The following point where also dissected: BL39, BL40, KI10, KI27 and LU 1 in order to look at the fascial planes in or near them.

The literature was searched for the placement of the GV and BL meridians. There was some meridian location disagreement (3 – 5). For instance, the distance from GV to the inner branch of the BL meridian varies between authors and its anatomical positioning is debatable.

Results

From the dissections, it was clear that the medial BL-meridian follows the intra-muscular fascia septa in the muscle longissimus dorsi (6) which is not in the longissimus-iliocostal groove. The outer/lateral branch is 5-6 cun from GV and in the longissimus-iliocostal muscle groove.

It was noticed how the angle of the needle makes a difference, if the needle follows the fascia or is inserted into the muscle. An approximate angle of about 35 to 45-degrees will direct the needle into the thick dividing fascia.

The Governing Vessel running in the dorsal midline

Bai- Hui or “Point of 100 Meetings” is placed over the lumbosacral (LS) space. The needle is inserted through the supraspinous ligament and into the interspinous ligament. At a transverse section of the area it can be seen how a needle also will pass through the thoracolumbar fascia, the dorsal sacroiliac ligament, the fascia between the tail muscles and the multifidii muscles. This will have an effect distally in association with the iliosacral joints and the L-S facet joints in close relationship with dura mater. So, this is indeed the “Point of 100 Meetings”.

GV14 is located in the dorsal midline between C7 and T1. An inserted needle will pass the nuchal ligament, go into the funiculus of the nuchal ligament which attaches to all the cervical vertebrae and into the fascia dividing the different layers of the neck muscles. The indications are cervical or back problems, respiratory and brain problems.

The following three points are according to the International Veterinary Acupuncture Society (IVAS) (3) almost lying on a transverse line:

BL 39 is located in the lateral end of the popliteal fossa just lateral to BL40. Indications: Edema and pain in the hindlimb, especially the hock area and urinary problems (3).

BL40 is situated in the midpoint of the transverse crease of the popliteal fossa between the bicep femoris muscle and semitendinosus muscle. It is the Master Point for the caudal back and hip region. He-Sea and Earth point. Indications: Local stifle problems, caudal back and hip area, gastrointestinal, urogenital and skin problems, fever and heat exhaustion (3).

KI10 is located in the depression between the semitendinosus and gracilis mm. at the level of the popliteal fossa. He-Sea and Water point. Tonifies Kidney Yin, expels Dampness from Lower Burner. Indications: Local problems in the stifle area. Urogenital problems (3).

These three points are all situated in the popliteal fossa on a transverse line. A transverse section of the area shows very clearly, how the fascia between the mentioned muscles aim into a large area of fascia in the popliteal region, where the tibial nerve, which is divided from the sciatic nerve, passes through, just caudal to the femerotibial joint. The popliteal lymph node is also situated in this fascial area. Superficially, just under the skin at BL39 is a branch of the fibular communis nerve. The fascia from KI10 is slightly more medial and from BL39 it is more lateral. It gives a clear indication of the connection between the Bladder and Kidney meridians.

KI27 is located 1 cun ventral and lateral to the manubrium (3). The point is nicely located in the fascial intersection between the sternomandibular and descending pectoral muscles next to the manubrium which is close to the 1st sterno-costal junction. The location fits the indications of asthma, cough and chest pain.

LU1 is located in the deep depression in the center of the muscle belly of the descending pectoral muscle, at the level of the third (not first rib) intercostal space and LU2 at the triangle formed by the sterno-mandibular, brachiocephalic and the descending (superficial) pectoral muscles directly ventral to the cephalic vein (6). The dissections of the area show no particular fascial structures at the level of LU1 in the descending pectoral muscle which is overlying the subclavian muscle. But the skin seems to be more tightly attached to the muscular fascia on a line between LU1 and LU2. Under LU2 there is a very clear area of fascia between the mentioned muscles close to the cephalic vein and close to the origin of the ascending pectoral muscle. This area is overlying the ventro-medial part of the shoulder joint. Medial to the shoulder joint the fascia complex engaged the brachial plexus and the axillary artery. So, a rotation of the needle in this point can affect the ventral neck muscles to the upper neck and mandible, the pectoral muscles, shoulder joint/front limb and sternum. From a fascia point of view LU2 seems to be able to affect a greater area than LU1.

Conclusion of the other dissected points

The BL39, 40 and KI10, the KI27 and LU2 had very close relationship to fascial planes. LU1 did not show clear fascia relationships in the muscle.

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Can Myofascial Kinetic Lines be an Anatomical Foundation for Acupuncture Meridians?

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Introduction

This is an introduction to the concept of myofascial kinetic lines that are believed to be deeply involved in the functional part of the locomotory system. The lines have now been verified by dissection of 50 horses and 35 dogs at University of Copenhagen. In the following text we hope to provide a holistic, biomechanical understanding which can be compared to the philosophy of TCM, to give an overview of the most current knowledge of connective tissue in general, fascia in particular, the concept of tensegrity and normal spinal biomechanics. It will also be shown how myofascial kinetic lines are anatomically closely related to acupuncture meridians in horses, the eight extra meridians will be viewed in relationship to the lines and finally different treatment approaches will be discussed.

Langevin and Yandow (1) investigated the hypothesis that acupuncture points are situated over fascial planes. They marked locations of acupuncture points and meridians on a human upper arm and made serial cross sections, each section measuring 1 cun and then they described the underlying tissue. More than 80% of the points and 50% of the meridian intersections appeared to coincide with intermuscular or intramuscular connective tissue planes. They write: ”These findings suggest that the location of acupuncture point, determined empirically by the ancient Chinese, was based on palpation of discrete locations or “holes” where the needle can access greater amount of connective tissue.” And “ On the basis of these findings and our previous experimental results (Langevin et al. 2001b.2002) we propose that acupuncture charts may serve as a guide to insert the needle into interstitial connective tissue planes where manipulation of the needle can result in a greater mechanical stimulus”.

Darsher (2) has made a computerized visualization comparing Anatomy Trains with the 12 Principal Meridians and found that 8 out of 9 (89%) lines has a substantial overlap and he concludes:” The strong correspondence of the distribution of the acupuncture and myofascial meridians provides an independent, anatomic line of evidences that acupuncture Principal Meridians likely exist in the myofascial layer of the human body”. It must be remembered that the myofascial lines are not thin lines but wider surfaces.

According to this way of thinking the superficial lines correspond to the Yang meridians and the deep lines to the Yin meridians. We find that the Superficial Dorsal Line (SDL) corresponds very well to the Bladder (BL) Meridian and the Superficial Ventral Line (SVL) to the Stomach (ST) Meridian. The starting point around the eye lies on fascia of the face. The ST meridian follows the SVL nicely along the sternomandibular muscles and the abdominal wall according to van den Bosch (3) and it ends over the insertion of the extensor tendon on the hind limb in the stomach ting point ST 45. The SDL follows the BL meridian over the semispinal and longissimus muscles of the neck and head to the erector spinae muscles: longissimus dorsi, iliocostalis and spinal muscles. The two branches of the BL meridian lie in the interstitial space between these muscle groups. Over the croup the bladder points lies on the gluteal muscle but the SDL follows the sacrotuberous ligament of the pelvis below that muscle so here is a slight deviation. The meridian continues in the space between the caudal head of the biceps of the thigh and semitendinosus muscles. Both of these muscles are part of SDL. From BL39 the meridian deviates laterally between the lateral gastrocnemius and the Achilles tendon both structures of the SDL which continues over the calcaneus to the flexor tendons onto the plantar surface of the toe so here is also a slight deviation.

The Gallbladder (GB) meridian follows the Lateral Line (LL) very well although it starts at the 3rd fibular muscle and not above the hoof. The different branches of the 3rd fibular muscle enclose the hock. GB40 lies on the lateral branch which inserts on the 4th metatarsal bone. Its muscle belly lies under the interstitial space between the two lateral extensor muscles where the meridian passes over. Both LL and the GB meridian run over the lateral part of the stifle, which the fascia lata of the tensor muscle covers. At this point the GB seems to continue more caudally than the LL but it is still over the same fascia. At the tuber coxae they take the same path following the abdominal muscles along the side and over the ribs to the neck. Here LL has two pathways over the brachiocephalicus and splenius muscles and the meridian follows the interstitial space between them. The myofascial line ends at the mastoid process just caudo-ventral to the ear which is very close to GB2.

The meridian system has no helical lines that cross the midline, the only transverse meridian is the girdle vessel or Dai Mai and it cannot be compared to the helical lines in course or in function. Darsher (14) finds that the Spiral Line (SL) is a combination of the ST and BL meridians which we partly agree on but they do not explain the crossing over the Governing Vessel (GV) and the Conception Vessel (CV).

The name and the course of Deep Ventral Line (DVL) points to the Foot Yin meridians; Kidney (KI), Liver (LIV) and Spleen (SP) which all pass over the medial aspect of the hind limb and enter the pelvis. The descriptions of the internal branches of these meridians are not so precise and can be a little hard to compare with the three pathways of DVL. Here it is necessary to remember the embryological origin of the serous membranes already mentioned. With one continuous net covering the inside of the thorax and abdominal cavities and the surface of the viscera, so maybe they are hard to distinguish. The internal branches of the SP meridian reach the oesophagus and end in the centre of the tongue which put it closes to the middle DVL pathway. The KI meridian also seems to follow the middle pathway but curves ventrally and ends at the root of the tongue, which is closer to the ventral pathway. The LIV meridian ends in the eye and continues to the GV so here it is closest to the dorsal branch. It is also possible to look at the Eight Extra Meridians to explain the abdominal/thoracic part of the DVL. The Governing Vessel (Du Mai), Conception Vessel (Ren Mai) and the Penetrating Vessel (Chong Mai) is according to Maciocia (4) considered as three branches of the same vessel. The vessel originating from the space between the kidneys from where it flows down to the perineum before they separates. They are thought to affect the energy on a deep constitutional level. The CV follows the abdominal midline, GV the midline of the back and the Penetrating Vessel the KI meridian, but it is only correct if the GV follows the ventral part of the spine.

We have not found it possible to identify superficial (Yang) and deep (Yin) front limb lines but we find that the retraction line placed on the palmar aspect of the limb includes the Yin meridians as the Yin Ting Points are on the more palmar surface of the toe and the meridians are continuing into the chest. It is also possible to argue that the Yin function is being more flexion. Opposite to that we find that the protraction line represent the Yang meridians as it is situated on the dorsal part of the toe, just as the Yang Ting Points are, and it continues to the neck and head as the Hand Yang meridians. Also it can be argued the Yang function is extension.

Treatment

The empiric experience we have had until now using the knowledge of these lines has provided a possible method to trace the primary cause of somatic pain and dysfunction and also to relate it to the viscera. In combination with TCVM knowledge it seems to be a strong diagnostic tool and is also useful for treatment. E.g. I have over a longer period found a consistent pattern by my osteopathic examination of horses with longer standing (deeper) problems. I find that they have dysfunction and/or pain at the cranium, hyoid bone, the thoraco-lumbar junction, diaphragm and the sacroiliac joints. These are all structures relating to the DVL.

When looking at the myofascial layers as sleeves that cover, protect and hold together it is understandable that if the sleeves are too tight, they need to be addressed by treatment. Normal function might not be regained if only the joints and muscle tissue within it are treated and released. Due to the different mechanisms of contractile action in muscles and fascia it is likely that the same manual therapy methods cannot bring release to both types of tissue, saying that traditional muscle massage techniques releasing muscles may not work on the all the fascia. Facing the fact that many of the human manual fascia techniques are quite painful, they are not very practical in animals.

Seen in the light of Langvin and Yandow's results acupuncture therefore seems to be a good choice. It is obvious to treat the meridians that correspond to the kinetic lines as described but finding specific acupuncture points that can influence the lines is an attractive option. My colleague Dr.Tove Due, DVM has found points, which seem to relax tension in the lines. They are not described acupuncture points but, in some places, very close to.

For the DVL the Eight Extra meridians offers treatment of several inner organs and the diaphragm through the Conception Vessel and Yin Steeping Vessel (Yin Qiao Mai) with LU7 and KI6 and through the Penetrating Vessel and Yin Linking Vessel (Yin Wei Mai) with SP4 and P6 (4).

The osteopathic functional indirect technique (earlier lectured on at the IVAS congresses by Patricia Kortekaas, PT) is a technique that addresses all the tension in soft tissue around the joints, and also seem to have good effect on fascia. According to Myers the bones rebalance if the soft tissue regain a tensional balance which is the same line of thinking.

The circular TTouch® from Linda Tellington-Jones might be known to some of the audience as a very effective treatment approach. If the effect is due to the myofascial tissue release it might be effectively explained. It is a technique that can be recommended to let the owner use on the patient between treatments.

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Myofasciale Kinetic Lines – a dissection study.

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Different writers have described myofascial kinetic chains or lines as rows of interconnected anatomical structures in the locomotion system that describes motion (1). Thomas Myers the author of "Anatomy Trains" is to my knowledge the first who has dissected human bodies to show these kinetic lines. His results are not published in previewed scientific papers but the DVD following the book shows the dissections very well. He describes 10 lines, 5 from head to toe; Superficial Back Line (SBL), Superficial Front Line (SFL), Lateral Line (LL), Spiral Line (SL) and Deep Front Line (DFL), 4 on the arms: Superficial Back Arm Line (SBAL), Superficial Front Arm Line (SFAL), Deep Back Arm Line (DBAL) and Deep Front Arm Line (DFAL) and one from forelimb to hind end: Functional Line (FL).

Different motions can be described by activity of single kinetic lines, but it is important to remember that no line works on its own; the lines are still interconnected according to the description above (1).

Elbroend and Schultz (2015) (3) conducted a dissection study in equines in order to verify related kinetic lines, 24 horses were dissected, and the lines followed. The guidelines for the dissection followed thus described by Myers (2) in humans: the depth of the tissue, the fiber direction and the function were to be similar. The tendon, muscle and ligament attachment were released from the bony attachments and could be continuous followed by connective tissue to the next muscle or soft tissue structure.

The kinetic lines were renamed to relate to quadrupeds as follows: Superficial Dorsal Line (SDL), Superficial Ventral Line (SVL), Lateral Line (LL), Spiral Line (SP), Functional Line (FL), Profound Ventral Line (PVL), Front Limb Protraction Line (FLPL) and Front Limb Retraction Line (FLRL). The kinetic lines in horses followed the human lines very well, although only two front limb lines has been published so far. Four deep kinetic lines has later been verified and in preparation for publication.

The SDL starts at plantar surface of P3 on the hind limb, follows the flexor tendons into the caudal thigh muscles, into the broad ligament of the pelvis, along the erector spinae muscles, into the long neck muscles, onto the occipital crest ending in the temporal fascia and muscle and on the mandible. It extends the spine and hip and flexes the lower limb.

The SVL originates at the dorsal surface of P3, follows the extensor tendons into the straight thigh muscle, to the hip joint, along the straight abdominal and thoracic muscles to sternum and forward through the sternomandibular muscle into the masseter muscle on the mandible. It flexes the spine and hip and extends the lower leg. SDL and SVL acts as antagonists and has to be in balance so the spine can move freely.

The LL starts at the lateral surface of the hind limb and splits in a superficial part and a deep part. The superficial part act when the spine is in flexion and runs through the cutaneous trunci and colli muscles into the brachiocephalicus muscle and attaches to the mandibular process at the temporal bone under the ear. The deep part acts when the spine is in extension and follows the superficial gluteus muscle onto tuber coxae cranially through the abdominals and the intercostal muscles, under the scapula and into the splenius muscle and terminates at the same process as the superficial part. The two parts lateral flexes the spine in flexion and extension, and they have to be in balance as well. Correct tensional balance between the right and left LL make it possible for the horse to be aligned straight.

The rotation of the spine is controlled by the two helical lines FL and SL. FL starts on the medial side of humerus in the latissimus dorsi muscle runs through the thoracolumbar fascia and crosses the midline into the superficial gluteus muscle, through the lateral quadriceps of the thigh and the lateral patella ligament, crossing under patella into the medial patella ligament and the medial quadriceps. From here it follows the gracilis muscle and has fascia cross-over at the pubis bone, into the straight abdominal muscle and ends in the ascending pectoral muscle and its fascia connection to latissimus dorsi. The dorsal part extends and rotates the spine and the ventral part work antagonistic by flexing and straightening the spine.

The SL is the most difficult and three-dimensional line which crosses the midline three times. It starts at the mandibular process under the ear in the splenius muscle and crosses over the midline at the level of C7-T1 into the rhomboid muscle on the other side under scapula. It continues into the ventral thoracic serratus muscle, into the external abdominal muscle and in the white line into the internal abdominal muscle and onto to tuber coxae. From here it runs on the lateral surface of the thigh surrounding the hock and turns dorsal again to the tuber ischia. From here it follows the sacral ligaments and crosses the midline for the third time over tuber sacrale. It then follows the same pass way as the SDL to the neck. The ventral part controls spinal flexion and rotation and the dorsal part act as an antagonist by straightening and extending the spine (3)

There are two main kinetic lines of the front limb – a protraction line and a retraction line which work around a pivot point at the level of the proximal third of the scapula (medial side) around which the front limb pendula (1). The protraction line has a deeper part that adduct and external rotate the limb and the retraction lines deeper part abduct and internal rotate the front limb (in preparation for publication).

The DVL starts in the deep medial hind limb muscles and enters the pelvis where it divides in three pass ways. One follows the ventral surface of the spine and ends through the longus capitis muscle on the base of the cranium and another follows the inner wall of the abdomen over sternum and ends via the hyoid muscles on the rostral end of the mandible. The middle pass way runs through the sartorius muscle into the iliopsoas fascia and muscle which relates closely to the crus of the diaphragm and comprises also the abdominal organs. It continues via the esophagus, lungs, heart and trachea to the larynx/pharynx and ends on the base of the cranium. This line connects the somatic body and the organs and flexes the spine.

The last line is the Deep Dorsal Line which is not described in humans. It starts in the dorsal tail muscles and continues through the supraspinous ligament into the nucheal ligament and ends at the occipital crest and through the multifidus muscles into the suboccipital muscles also ending on occiput. This kinetic line extends the spine and controls its smaller movements. (Elbrønd and Schultz, 2019, in preparation for publication).

The understanding of these kinetic lines can provide the anatomical basis for the importance of looking at the whole body to trace the cause of biomechanical dysfunction, organ problems or poor performance in horses.

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Do you know what the dog is trying to tell you? Reading body language 101

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Introduction

Reading canine body language should be the first thing veterinarians are taught at University. It is the most important form of communication between veterinarians and their canine patients.

Yet many vets graduate without the necessary skills to assess whether their patients are stressed, anxious or fearful so that they do not appreciate what can be done to help their patients and their owners. Additionally, by not being able to read canine body language correctly many veterinarians and their staff get bitten leading to significant WHS issues for the people and welfare issues for the dog.

So, does a wagging tails mean the dog is friendly? Do raised hackles mean the dog is aggressive? What can sniffing and urination mean? Can vets confidently explain body language changes to clients without referring to "dominance" to help them with their dogs?

Canine Communication

Dogs are social animals and have evolved communication systems that tell others how the individual is feeling at any particular time. They can also communicate how they may react to a particular stimulus or a challenge. Dogs are in fact communicating their emotions. While the signals given by a very frightened or an aggressive dog should be very familiar to most people, it is often the signals given by an unsure or internally conflicted dog that are missed. Reading the body language of a frightened dog as well recognising the signs of a truly relaxed dog is equally important.

When evaluating body language, it is important to look at the whole dog. That means looking at the eyes, the ears, the facial expression, the position of the tail, the stance, the hair position, body tension etc. It means looking at each individual aspect not in isolation but together. But just as importantly it means looking at the context. For without the context it is impossible to tell the emotions of the dog and predict its motivation let alone any outcome of any interaction.

The way an animal behaves at any given time depends on three key factors, its genetic predisposition, what it has learnt from previous experiences and the particular environment it is in at the time. None of these factors acts in isolation so all need to be taken into consideration. In general when we see an animal that is anxious or fearful response we see will be one of the four F's- Fight, flight, freeze or fiddle. These correspond with the physiological and emotional responses of the dog. Recognisng the signs of each is important.

So given that these signals given by a dog is a form of communication about how it feels at any particular moment in time it is imperative that veterinarians are equipped to read the signals. It does not necessarily predict what may happen if the context changes nor how the dog may react the next time in a similar context, but it may be a good indicator.

While most people are familiar with the signals given by a threatening or aggressive dog or a really terrified dog, the signals of an unsure or fearful dog can be more subtle. Dogs under stress also react according to their personality and their previous experiences in similar situations. It is important to recognise that aggressive dogs are really scared dogs and are not behaving in that manner because they are being dominant, naughty or nasty.

The signals of an unsure animal can be very subtle and ambiguous. Displacement behaviours (normal behaviours that are seem out of context, such as grooming, sniffing the ground or scratching) should never be ignored. These are performed because the animal is in a state of internal conflict. Dogs may also display what are sometimes called cut-off signals. These are used to turn off threatening behaviour from another animal or person. They send the message "I am not interested in fighting or interacting with you."

Common Canine displacement behaviours include:

1. Yawning
2. Lip licking
3. Panting
4. Paw lifting
5. Sniffing
6. Scratching
7. Sleeping

Canine cut off signals include:

1. Avoiding eye contact
2. Turning the head away
3. Turning the body away
4. Lowering the body
5. Sniffing the floor

However, the behaviours that are of most welfare concern are those of the freeze behaviours. This is dog that, when being examined by the vet, appears to accept or tolerate the process. People often assume the dog is feeling okay, when in fact it is "frozen" and actually terrified. Continuing to examine this dog although "easy" and possible may have a significant detrimental effect on the emotional health of that dog. Although the dog may not respond with aggression on this visit there is no guarantee that this will not happen in future.

Conclusion

Veterinarians, and their staff, should be able to read canine body language and educate owners about their dog's responses so that fearful or anxious dogs are not put into situations which will make their fear or anxiety worse.

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Noise sensitivity or noise phobia? What is the difference and why it matters

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Noise Sensitivity

The hearing of dogs is thought to be at least 4 times more acute than that of humans. Therefore, it is not surprising that many dogs can be very sensitive and therefore reactive to noises.

Noise sensitivity in people is a recognized problem. Hyperacusis is a hearing disorder that makes it hard to deal with everyday sounds. It means that the people who have been diagnosed with it find some sounds unbearably loud even though other people don't seem to notice them. Sounds like the sound of a running tap, the refrigerator or dishwasher or even people talking loudly can be problematic to people.

It appears that some people are only mildly bothered by these sounds, while others have severe symptoms such as a loss of balance or even seizures. However, hyperacusis is rare and it affects about 1 in 50,000 people. These people may also suffer from tinnitus. Interestingly a lot of people who have it also have normal hearing.

Ears detect sounds as vibrations but with hyperacusis, the brain confuses or exaggerates certain vibrations so the brain reacts differently to them than people who are not noise sensitive. People aren't usually born with hyperacusis -- it's usually caused by certain diseases or health issues. The most common ones that may be relevant to animals are head injury, damage to one or both ears because of medication, taking valium regularly, certain kinds of epilepsy, post-traumatic stress disorder and autism.

Being around a loud noise also can cause hyperacusis. Something like a single loud gunshot can trigger the condition. But it also can come from being near loud noises over a long period.

Treatment will depend on what caused it. In some cases, like with injuries to the brain or ear, the sound sensitivity might get better on its own. If it doesn't, sound desensitization may help. It can take 6 months to a year or more to get the full benefit of this therapy.

In dogs noise sensitivity has been associated with musculoskeletal pain and avoidance of certain places so veterinarians need to be on the lookout for signs of pain or stress.

Noise Phobia

A phobia is defined as an irrational, intense, persistent fear of certain situations, activities, things, or people. The fear (or panic) response is out of proportion to the stimulus and is maladaptive. Animals with phobias do not habituate to the stimulus even after very many harmless contacts and the response does not decrease with time.

It is most commonly exhibited in response to sounds like thunderstorms and fireworks. However, it may generalise to other sounds like cars backfiring, gunshots or even sausages sizzling!

It can develop from a single exposure (one event learning) or from continued exposure to the fearful stimulus.

Sadly, noise phobias in cats are not recognised by most owners (or veterinarians) and therefore they are often left untreated despite the distress the animal may be experiencing.

Treatment of noise phobias involves three key areas: environmental management, behaviour modification and medication. Monitoring the response to the treatment programme is essential to assess progress.

Although ideally noises should be avoided this is not easy or even possible in many cases. However, every attempt should be made to minimise the dog's exposure to noises that concern the dog.

The visual stimuli can be minimised by confining the dog to a brightly lit room with light proof shutters or shades. Sometimes, increasing the distance from the stimulus or finding some relatively sound-proofed room, playing music etc might also help with the process.

In some cases providing a specific place to which the dog can retreat when frightened is useful. Enclosed spaces such as shower stalls or cupboards can be useful to provide security for some dogs.

Ignoring the pet when it is anxious may actually increase the pet's anxiety. However reassuring the pet may decrease the pet's anxiety.

If the pet will eat at these times then it should be given its favourite treat so that over time the pet associates the noise it now fears with the treat it most desires.

Punishment should be avoided as it is not effective in changing behaviour for the better in anxious pets. It serves to further increase the anxiety as well as impede learning of non-anxious behaviour and should be avoided.

Desensitisation is not possible with true noise phobias. Therefore, medication is often needed. Medications that influence serotonin metabolism, such as the selective serotonin reuptake inhibitors- SSRIs (eg fluoxetine) and the tricyclic antidepressants -TCAs (eg clomipramine), have been used to help these pets. Additional anxiolytic medication has also proved useful in some cases in combination with TCAs and SSRIs, especially if the pet has panic attacks.

Acetylpromazine (ACP) is contraindicated for noise phobias as it can lead to dogs becoming more noise sensitive and unpredictable.

Conclusion

Although noise sensitivity and noise phobia are often used interchangeably perhaps it is time to rethink the way veterinarians not only classify but also treat these very debilitating conditions.

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Things you should consider when anaesthetising older patients

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Introduction

Senior pet care is an increasingly important component of modern veterinary medicine. As pets age they may require anaesthesia for many reasons. They may require anaesthesia for major surgery or minor surgery. Regardless of the reason for the anaesthetic there are several considerations from a behavioural perspective that veterinarians need to consider when discussing anaesthesia with their patient's owners.

The senior dog or cat may also have specific behavioural changes that occur with age and it is important to recognise and discuss these before anaesthesia. However, age is not a disease!

Anaesthesia

"Anaesthesia refers to the practice of administering medications either by injection or by inhalation (breathing in) that block the feeling of pain and other sensations, or that produce a deep state of unconsciousness that eliminates all sensations, which allows medical and surgical procedures to be undertaken without causing undue distress or discomfort." http://www.anzca.edu.au/patients/what-is-anaesthesia

There are many agents that are used to anaesthetise animals and veterinarians should be very familiar with their use and the advantages and disadvantages of each of them that they use.

Cognitive Decline syndrome (CDS)

CDS is a multifactorial syndrome. The most common signs attributable to CDS include:

- loss of learnt behaviours such as toilet training;
- confusion/disorientation such as staring into space or getting lost in corners;
- not being as interactive socially with owners;
- compulsive behaviours such as vocalisation for no apparent reason;
- aggression / irritability;
- change in sleep wake cycles such as increased sleeping during the day and restlessness at night;
- change in appetite.

Questions to ask before anaesthesia of elderly patients

A thorough physical and behavioural examination should ideally be carried out prior to admitting the pet. This should include complete blood work, urinalysis as well as asking clients about all medications that the pet is on. This should include not only those prescribed by the veterinarian but also all supplements, additives etc that the owner is administering. All may influence not only the anaesthetic but also the outcome of the anaesthetic.

For elderly patents veterinarians should consider asking clients to fill out a cognitive dysfunction syndrome (CDS) questionnaire the general anaesthetic (GA). Why?

Anaesthesia Risks

In elderly humans a GA has been shown to be associated with post-operative delirium (POD) and post-operative cognitive decline (POCD).

In humans- these risk factors have been identified for POD

- Visual and hearing impairment
- Cognitive impairment
- Sleep deprivation
- Immobility
- Dehydration

These factors would also be commonly be seen in elderly dogs and cats.

In people these are recommended to help prevent POD

- Orientating information
- Cognitive stimulation activities
- Exercise
- Feeding and Fluid assistance
- Non-pharmacological sleep aids

For veterinary patients the following should also be considered:

• Orientating information	Owner's scent
• Cognitive stimulation activities	Puzzle toys
• Exercise	Short walks, physiotherapy
• Feeding and Fluid assistance	Helping them eat and drink
• Non-pharmacological sleep aids	Massage, quiet times, music

General Anaesthesia is associated with higher risk of developing POCD

Clients will know that something is not quite right and they will note signs such as:

- Disorientation
- Vagueness
- Not recognising the owner
- House soiling
- Irritability

If these things cannot be put down to pain or don't improve with pain control- think POCD

There are a number of medications and diets that may be indicated in helping these older animals pre and post GA.

Other considerations:

Many older pets have anxiety and this should be treated prior to a GA. If the dog or cat is already on medications such as selective serotonin reuptake inhibitors (SSRIs) or tricyclic antidepressants (TCAs) then these should NOT be stopped prior to a GA. However, drug interactions should be considered.

Conclusion

Age is not a disease. However, care should be taken when considering a GA in older pets. Prevention of POD or POCD should always be a consideration.

Perhaps it is time to rethink the way veterinarians think about older pets and the consequences of anaesthesia. By administering CDS questionnaires pre and perhaps even post anaesthesia would give us more information. But better still implementing some preventative measure may be even better for the welfare or dogs and cat and their owners.

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Understanding Emotional Development in Dogs and Why it Matters

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Introduction

What happens from the time of conception in the womb to 12 weeks of age affects the way the puppy develops not only physically but also emotionally. The period is characterised by intense development and change within the central nervous system.

Early experiences affect the animal's resiliency or coping capacity. The ability to cope with life's stressors varies with genetic predisposition, learning from previous experiences and the current environment. Early identification of individuals who are not developing emotionally normally or have little resilience allows early interventions to be implemented. Early intervention can alter brain development. Interventions may take the form of environmental management, behaviour modification and often medication.

Environmental management involves creating a complex but stable and predictable environment for the puppy. Interaction with complex environments results in denser brain development. A stable environment allows the puppy to develop expectations or "rules" for predicting the outcomes of interactions and so develop coping strategies.

Behaviour modification helps shape acceptable and desirable behaviour. More importantly, behaviour modification encourages and rewards calm, quiet behaviour and relaxation. It allows the cognitive part of the brain to put a "dimmer switch" on the emotional or reactive part of the brain. This way the puppy learns more coping strategies to deal with what life will present.

Medication can be used to normalise brain neurotransmitter function and also to maximise neuronal connections through the release of brain neurotrophic factor. New connections are encouraged along more desirable pathways.

Appropriate early interventions can affect brain development, thus helping more normal growth and behaviour to occur.

Canine Development

There are five generally recognised periods of development in dogs, each having characteristic behavioural and physiological changes. These periods are not rigidly fixed but influenced by the individual animal's genetic predisposition, their experiences and the environment. These periods are the neonatal, transitional, socialisation, juvenile and adult. However, it is important to note that the prenatal period is also significant and more emphasis has been placed on this period recently. For example, how the bitch is handled during her pregnancy will influence the behaviour of her pups. Bitches that are gently handled are more likely to have calmer or less reactive pups.

Neonatal: Birth - 2 Weeks

The neonatal period starts at birth and lasts until approximately two weeks of age. During this time puppies can grunt and suckle. Vocalisations appear to be solely in response to physical discomfort and separation. As puppies are born neurologically immature, movement is limited to a slow crawl and both the eyes and ears are closed. Perineal stimulation is necessary for urination and defecation.

Approximately one third of the puppy's time is spent feeding and the majority of the rest, sleeping. On electro-encephalographic examination there is little to distinguish sleep and awake periods initially. During this period puppies are totally dependent on the mother for survival.

Transitional: 2 - 3 Weeks.

In the transitional period, which occurs between two and three weeks of age, rapid physical and behavioural changes occur. The puppy starts to ingest solids, the eyes and ears open, and the auditory "startle" response can be elicited. The puppy becomes more independent, learns to walk, and can crawl both forward and backward. Interaction with littermates and early play fighting and growling are observed. The puppy starts to leave the nest to urinate and defaecate and does not need maternal stimulation of the anogenital area to eliminate.

Socialisation: 3 - 12 Weeks

The socialisation period follows and is thought to last from approximately three weeks to about 10-12 weeks of age. The timing of this period is thought to vary according to the individual dog, the breed and experiential factors. The puppy is more independent and acquires bladder control. This is usually the time that a puppy might go to its new home. This period has been extensively studied as it appears that experiences during this time have profound effects on later behaviour.

Juvenile: 12 Weeks - Sexual Maturity

The juvenile period is considered to last from 10-12 weeks until sexual maturity. Although the basic behavioural patterns do not change during this time there are gradual improvements in motor skills and male puppies begin to lift their leg to urinate. It has been argued that the puppy's learning capacity becomes fully developed by the beginning of this period.

Adult: sexual maturity - Death

The adult period is characterised by the onset of sexual maturity and continues until the end of the life cycle. Recently, emphasis has been placed on the senior dog when again behavioural changes associated with aging may occur.

Sexual maturity usually occurs between 6-14 months depending on the individual and the breed. Generally, the larger the dog, the later the onset of sexual maturity. with the exception of the Basenji, oestrous occurs approximately every six months after sexual maturity.

Social maturity is thought to occur between 18 and 36 months of age and is often associated with the onset of many behaviour problems such as aggression or anxiety related problems. This probably more correctly identifies the beginning of the true adult period.

Senior

The behavioural changes that occur in the ageing dog are now being recognised and increasingly studied. The start of this period is thought to occur at varying ages depending on the size and breed of the dog. It is characterised by the onset of physical and physiological changes and continues until the end of the life cycle.

Conclusion

Understanding the periods of development of dogs allows us to best manage their behavioural and emotional development and hence help them grow into the pets that owners want. Recognising if there are issues and then intervening early is important. But before any intervention is implemented then recognizing what is "normal" or typical is essential.

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What to Do when Training Does Not Help

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Introduction

We can and often do many things to and with the animals that share our lives. But is what we do always "good" for the animals? Do we always consider the impact of what and how we do what we do, on the animals - physically and even more importantly, emotionally?

If we do not consider these things then the animal's welfare may be comprised or at least be sub-optimal. Animal welfare used to be considered mostly in terms of the physical health of the animal and its environment (provision of shelter, feed and so on) but now more focus is also on the emotional well-being of the animals.

People and Dogs

Dogs have been selected through domestication and breeding for ease of forming social affiliations and relationships with people. They are an obligate social species.

Yet many people do not understand dogs and their behaviour. If there are issues with the dog's behaviour the first person that owners generally reach out to is a dog trainer. Unfortunately, training does not always help these dogs and the type of training may actually be detrimental to the dog emotionally. Anxiety disorders are common in both people and pets. It thought that at least 20 % of dogs have anxiety disorders. Anxiety disorders are mental health (behavioural) issues (diseases) so the welfare of the pet can be severely compromised if these issues are not recognised and appropriately addressed. Veterinarians play a key role in not only recognising and diagnosing diseases but also treating them.

Training problem or Behaviour Problem?

Many people offer behavioural advice and it can be hard for the general public (and vets) to know where to seek help. A search on the web for 'dog behaviour' brings up many different businesses and "professionals". Some of these people have some qualifications and clear experience, and some have none at all. Others are simply 'dog lovers'.

Unfortunately, there are no clear definitions within the pet industry and the word 'behaviourist' is used very loosely and often isn't in reference to a veterinary behaviour specialist or any person with any specialised qualifications or training.

Anyone, apart from registered veterinarians, can call themselves a canine behaviour specialist or a behaviourist as there are no laws governing the use of these words in most jurisdictions to date. This means that they don't need to have any qualifications whatsoever. Someone who calls themselves a 'behaviourist' or 'behaviour specialist' may not necessarily have any formal training.

Veterinarians, however, need to have additional qualifications to call themselves a behaviourist (ie a specialist). Veterinary behaviourists are specialists. They are veterinarians who have extra qualifications in behaviour (Fellowship or Diplomate) and have done extensive study in this field.

While training is useful for teaching basic manners, it won't assist with medically based behaviour issues (disease). That is why now in some countries these veterinary specialists are recognised as veterinary psychiatrists.

Dog trainers primarily deal with training problems. The focus of training is to teach dogs good manners and help with any training issues. The most common training issues include pulling on the lead, jumping up on people, digging, basic manners such as sitting and staying and toilet training. To modify these issues, a good training program should include rewards for desirable behaviours and redirection from the ability to perform undesirable behaviours where possible. Punishment (or even yelling at a dog) can make the problem worse and it does not teach the dog what is actually the behaviour desired of the dog. Punishment should NEVER be part of any training problem but especially for those dogs that are ill (have a disease).

Reward based training that is, rewarding the desired behaviours rather than punishing unwanted behaviours, is considered the best method of training as well as the optimal way of improving animal welfare. Trainers that advocate using forceful methods, are "balanced" trainers or base their recommendations on being dominant, assertive or the pack leader have not familiarised themselves with the current scientific research.

Medically based behavioural issues such as anxiety, noise phobias, aggression, and obsessive compulsive disorders such as tail-chasing, shadow-chasing or checking behaviours will not be able to be resolved by training. This is much the same as sending a dog to a trainer to resolve polydipsia, diabetes, hypothyroidism etc.

So what if training does not help?

The first step for any patient that presents with a behavioural issue is a thorough physical examination. Why? Because pain is a major contributor to behavioural changes. Pain is both a psychological and physiological experience and therefore the subject is both complex and controversial. Pain may be acute, chronic, localized, generalized, physical, emotional, adaptive, maladaptive and an individual may experience several types of pain concurrently.

The behavioural signs of pain may be overt or covert and vary with species, gender, age, previous experience as well as the current situation in which the animal finds itself. This adds to the difficulty in interpreting how pain affects animals.

Pain is a sensation (a feeling) which is very aversive and therefore it may elicit motor and vegetative reactions, may cause emotional reactions and may modify behaviour either in the short term, medium or long term or in fact all three. It may involve fear and lead to anticipation of pain and therefore lead to anxiety. Being able to distinguish pain, fear and anxiety is key to not only being able to recognise pain but also manage and treat pain.

This is something that veterinarians must be able to do.

Once the pain has been treated /managed then it is time to treat the other diseases (eg anxiety). The use of medications, behaviour modification techniques and environmental management will be essential.

Ongoing monitoring of the patient is important. This is no different from monitoring the diabetic or hyperthyroid patient.

Conclusion

Dog trainers and veterinarians deal with very different issues however the distinction isn't well understood by the general public (or even vets or dog trainers at times). Many people still think that if the dog is behaving "badly" then training is the answer. Unfortunately this is not always the case. Additionally many training methods utilise punishment and this can increase the anxiety and sometimes border on abuse.

While some methods appear attractive to humans as they provide what appears to be a quick fix the focus should always be on the effect it has on the pet – on not only the short term outcome but also the short and long term welfare. This why training is not always the solution and when referring to a trainer it is important to know who you are referring to and be very familiar with their training methods. Good trainers always refer to veterinarians when they know they are dealing with a medical issue (anxiety, pain etc).

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Coxiella burnetii seroprevalence in Australian Veterinary Workers

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Background

Q fever is a disease of people caused by the zoonotic pathogen *Coxiella burnetii*, a small obligate intracellular gram-negative bacterium. A range of non-specific symptoms may occur following exposure, with some patients experiencing severe symptoms such as pneumonia, hepatitis, obstetric complications, and persistent endocardial, vascular and osteoarticular infections. Post Q fever fatigue syndrome (QFS) may persist for years with debilitating consequences.

Many production, companion and wild animal species harbour *C. burnetii*, shedding bacteria into the environment in placental tissues, faeces, urine, and milk. Cattle, sheep and goats have been implicated as the source of infection in most human Q fever outbreaks globally; however other species including cats and dogs have been associated with disease in people. Environments may remain contaminated for many months, with bacterial spread occurring via wind and animal transport. Inhalation is the primary route of infection for people.

Approximately 74% of all veterinarians in Australia have sought vaccination for Q fever[1]. However, veterinary nurses report lower vaccine uptake (29%) for a variety of reasons, including a perception that they are not at risk of exposure to *C. burnetii*[1, 2]. A better understanding of *C. burnetii* exposure among the Australian veterinary workforce is required to assist workers and medical practitioners in making informed decisions regarding the prevention of Q fever, particularly with regards to Q fever vaccination. This study aimed to achieve this through investigating *C. burnetii* seroprevalence in these workers.

Methods

A cross-sectional serosurvey of the Australian veterinary workforce was undertaken. Veterinary workers were opportunistically recruited during the Australian Veterinary Association (AVA) national conference in Perth in 2014, the AVA New South Wales (NSW) divisional conference in Goulburn in 2014, and the AVA Pan Pacific Veterinary Conference in Brisbane in 2015. Additional participants included research, clinical, and administrative staff from within the veterinary departments at the University of Sydney Camperdown and Camden campuses in 2015. Only workers who had not been previously vaccinated for Q fever were included in the study.

Participants provided a blood sample and completed a questionnaire about their demographic, work, and Q fever disease and vaccination history. Blood samples were sent to the Australian Rickettsial Reference Laboratory (ARRL), Victoria, Australia, for *C. burnetii* serology via indirect immunofluorescence assay (IFA). Participants returning an anti-phase II or anti-phase I IgG titre ≥1/50 were considered positive. Multivariable logistic regression modelling, adjusted for age and sex, was undertaken to identify factors significantly (*p<0.05*) associated with a positive serology result.

Findings

Coxiella burnetii seroprevalence among the study cohort was 19% (36/192; 95% CI 14-25%). This finding confirms that veterinary workers in Australia have a higher risk of exposure than general populations, for which seroprevalence reports range from two to seven percent[3-5]. A positive serological result was significantly associated with (1) workplace remoteness ($p = 0.025$) and (2) the percent of total career spent working with ruminants (sheep, cattle, goats) ($p = 0.009$). These associations are consistent with similar studies in veterinary populations around the globe [6-10].

Those returning a positive *C. burnetii* serological result were most likely to be currently working within outer regional / remote areas of Australia (odds ratio [OR] 6.2; 95% confidence interval [CI] 1.9– 20.8; reference = major cities), and most likely to have spent more than 50% of their total career working with ruminants (OR 4.8; 95% CI 1.7 – 13.5; reference = 15% or less). Seroprevalence among veterinary workers currently working in outer regional / remote areas was 53% (9/17; 95% CI 31-74%), compared to 13% (16/123; 95% CI 8-20%) among metropolitan workers and 21% (9/43; 95% CI 11-35%) among inner regional workers. Seroprevalence among workers who had spent more than 50% of their total career working with ruminants was 38% (12/32; 95% CI 21-54%), compared to 11% (13/118; 95% CI 7-18%) among workers who had spent 15% or less of their total career working with ruminants.

Four participants reported having been medically diagnosed with Q fever, confirmed with laboratory testing. Three of these patients were veterinarians with varied animal exposures, all having worked in the veterinary industry for 35 years or more. The fourth was an administration worker within a small animal veterinary clinic in a major city, who had worked in the industry for only 6 years and reported no direct occupational animal handling in that time.

Conclusion

These findings confirm that veterinary workers in Australia have an increased risk of exposure to *C. burnetii*, supporting the Australian Government recommendation for Q fever vaccination of all veterinarians, veterinary students, and veterinary nurses. This recommendation should be extended to cover broader veterinary support staff, such as kennel hands, farm hands and administration workers. Importantly, this study also highlights that four out of five currently unvaccinated veterinary workers may be eligible for, and could benefit from, Q fever vaccination.

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FMD Training in Nepal 2018

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Foot and Mouth Disease

Foot and Mouth Disease (FMD) is a highly contagious viral disease of cloven hooved animals that causes vesicles in the mouth, between claws and on teats. The virus can also cause death in young animals due to effects on the heart. FMD is a challenging disease to manage as it is highly infectious, shedding starts before clinical signs and large amounts of virus can be excreted. Whilst the disease is quite debilitating in the short term, it has a low mortality rate except in very young animals.

It is estimated to have a global cost each year of US$6-US$21 billion through loss of production and trade. There are also welfare and societal effects to consider.

There are several differences in the epidemiology and clinical presentation between susceptible species. Cattle are commonly infected after inhalation of the virus. The main clinical signs are excess salivation, oral vesicles, reduced milk production, lameness, fever and lethargy. Cattle can also harbour the virus and FMD has been detected up to 12 months after infection in the oropharynx. Pigs usually become infected after ingesting the virus. The main clinical sign seen is lameness but there can also be lesions in the snout and mouth. Pigs can play an important part in spreading FMD as they excrete large amounts of virus in respiratory aerosols, which can travel over considerable distances. Sheep and goats could also play an important role if an outbreak occurred in Australia. Sheep and goats generally have fairly mild symptoms with FMD. These are oral lesions and lameness. These mild signs could go unnoticed leading to a more widespread outbreak.

FMD in Nepal

FMD is endemic in Nepal and there are around 200 outbreaks per year. This is estimated to cause 20% reduction in milk production and a 10% reduction in meat production. This leads to an estimated monetary loss of US$66 million per year due to FMD. Nepal's livestock industry is a quarter of the size of Australia's.

FMD Virus

FMD is a small virus in the picornoviridae family.

It is very resistant to the environment and will survive

- 3 days in soil in summer

~14 days in dry faeces

- 39 days in urine

- 20 weeks in hay/straw

- 6 months in winter slurry

It is however susceptible to high and low pH

FMD could enter Australia in many ways;

Due to the resistant nature of the virus, FMD poses a serious risk to Australia's FMD free status. It could enter the country via tourists, animal products (illegal or legal), returning Australians or agro-terrorism. Government estimates have put the potential cost of an outbreak between $6 and $50 billion depending on the distribution and length of the outbreak. An outbreak would decimate Australia's livestock industries and these are worth $18 billion per year in exports alone.

Australia's FMD Plan

After the 2011 Mathews report on Australia's preparedness to an FMD outbreak, a National FMD Action Plan and Taskforce was put in place. If an FMD outbreak occurred in Australia, testing would be carried out at the Australian Animal Health Laboratories in Geelong. Once confirmation of the disease is made AUSVETPLAN is enacted. AUSVETPLAN is a group of policies that detail Australia's response to a group of exotic diseases. There is an AUSVETPLAN for FMD. In the event of an outbreak the Government pays 80% of costs while industry pays 20%. A national 72-hour stock standstill would be put in place. During this period tracing of animal and vector movements would occur to determine the source of disease and at risk premises. Infected animals would be destroyed and surveillance and testing carried out. Vaccination may be used to control disease. After the last case there would be extensive testing in order to declare Australia FMD free. It takes a minimum of 3 months to be declared FMD free without vaccination and 6 months if vaccinations have been used.

Early recognition of FMD is crucial

For example, in the UK 2001 outbreak:

FMD was confirmed 20 February 2001 after being present in the country for approximately 3 weeks. By then 57 premises were infected. The cost to the United Kingdom of this outbreak was UK 6.7 billion pounds. This highlights the importance of having veterinarians trained in Australia who are able to recognize FMD to enable a rapid response to limit the spread of the disease.

FMD Real Time Training

"Foot and Mouth Disease Real Time Training" is a course run by the European Commission for Control of FMD and United Nations Food and Agriculture Organization. Funding is provided by the Australian Government to train Australian vets in early recognition and management of FMD cases. There have been over 300 Australian vets and 100 Nepalese vets complete the course thus far.

The aim of the course is to speed recognition of the disease and have vets trained to be ready to respond to an outbreak in FMD free countries. Training focuses on recognition of clinical signs, diagnostic testing, epidemiology of the disease (trace back and trace forward to identify high risk premises) and biosecurity.

Training of Nepalese vets aims to reduce the prevalence in Nepal to reduce risk of spread to free countries.

What to do if you have a suspicious case:

If you see a possible case of FMD, ask some questions – how many affected, since when, introductions, visitors etc. This can help to determine if high or low risk situation. The next step is to notify your DVO or the Emergency Disease Hotline on 1800 675 888.

They will give advice on sampling and biosecurity. Their response from here will depend on the possible risk.

Lessons Learnt:

FMD Real Time Training is a fantastic opportunity for Australian veterinarians to learn about FMD. During this training, the ability to see the disease should enable personel to be better able to recognise the disease. It also highlights the severity of the disease and the massive economic impact on infected premises.

The training also highlights the possible modes of introduction into Australia and the devastation it could cause. It shows the importance of border biosecurity and the need for strong border control. Education is also a critical part of prevention in reducing the risk from people such as overseas backpackers who often work on farms. When travelling people should be mindful of the ability of the virus to survive on them and their clothing and the safest option is to leave high risk items overseas and to not come into contact with animals immediately after returning to Australia.

Most importantly we need to be observant of stock and report suspicious or unusual diseases immediately.

Emergency Animal Disease Hotline: 1800 675 888

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NUMNUTS® - providing pain relief for lamb marking

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Introduction

The majority of lambs produced in Australia are marked (castrated and tail docked) between the ages of 4 and 12 weeks. Although surgical methods are available, use of constricting rubber rings is increasingly popular, particularly when lambs are non-mulesed. The constricting rubber ring leads to ischaemic necrosis of the tissue which is subsequently sloughed, meaning that the procedure is bloodless. However, the process is acutely painful, leading to physiological changes and display of acute pain behaviours such as rolling and writhing on the ground, abnormal standing postures, and kicking or foot-stamping [1-4]. Local anaesthetics have been shown to alleviate this acute pain response [5-8], but to date delivering local anaesthetic to individual lambs on a large scale as would be required on Australian sheep farms has been impractical due to the precise injection technique required. The NUMNUTS® tool has been designed to allow safe, consistent, repeated delivery of local anaesthetic to the target tissues at the time of rubber ring application. The NUMNUTS® tool is also a more ergonomic design than the current ring application tool, reducing the risk of repetitive strain injury to the wrist and forearm. During development of the NUMNUTS® tool, a series of research studies were carried out to assess pain relief efficacy.

Study methodologies

All studies were carried out under the authority of the Animal Ethics Committees with oversight at the relevant institute at which each study was conducted. Efficacy studies focused on lamb behaviour as the measure, and used 2% Lignocaine hydrochloride (Ilium Lignocaine 20, Troy Laboratories, Australia). A preliminary study on tail docking was in the format of pen studies, each pen containing a group of lambs with their mothers, and behaviour video recorded and analysed offline against a defined ethogram [9]. It incorporated a positive control group (lambs which received ring application with no local anaesthetic; RING); a negative control group (lambs that were sham handled and no ring applied; HANDLE); and a NUMNUTS® group (lambs that had the ring applied and local anaesthetic delivered at the site of ring application RING+LA). Treatments were mixed within pens. In a final study, lambs were assessed under field conditions. Two iterations of the field study were carried out, one in VIC and one in NSW. Each iteration contained five treatment groups: Sham (lambs that were sham handled and no ring applied); FRR (female lambs that were ring tail-docked with no local anaesthetic); FNN (female lambs that were ring tail-docked with local anaesthetic delivered at the site of ring application using NUMNUTS®); MRR (male lambs that were ring castrated and ring tail-docked with no local anaesthetic); and MNN (male lambs that were ring castrated and ring tail-docked with local anaesthetic delivered at the site of each ring application using NUMNUTS®). Acute pain behaviours were counted for one-minute blocks at 5 min, 20 min, 35 min and 50 min post procedure.

Results

Pen study, tail docking: Active pain behaviours in ring tail docked lambs without local anaesthetic decreased over time in the first hour. Active pain behaviours were significantly greater in ring tail docked lambs without local anaesthetic (RING) than in NUMNUTS®

(RING+LA) or sham HANDLE lambs at 5, 15, 25 and 30 minutes post procedure. NUMNUTS® lambs did not differ from HANDLE lambs at any time point (Figure 1).

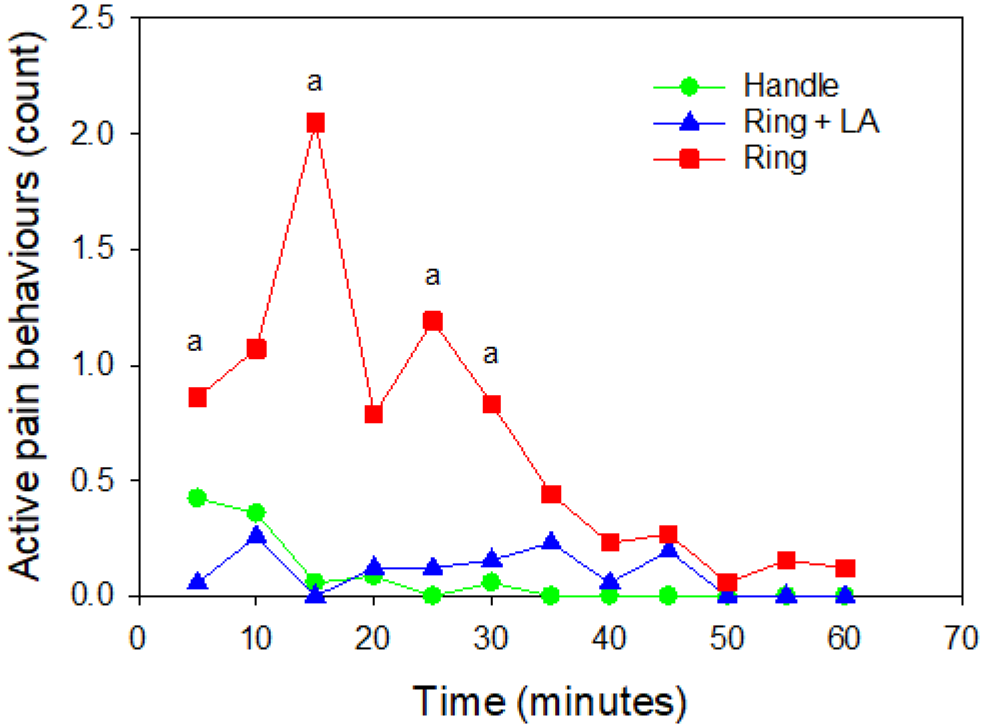

Figure 1: Pen study on tail docking. Acute pain behaviours expressed by lambs in the first hour post tail docking. a, b: Means within a time interval with different superscripts are significantly different (P < 0.05)

Field Study: Active pain behaviours in ring marked lambs initially increased, then decreased over time in the first hour. RING and NUMNUTS® treatments were significantly different from SHAM at all time points. In males, NUMNUTS® was significantly different from RING at 5 and 20 min post marking. RING and NUMNUTS® did not differ at 35 or 50 min (Figure 2), while in females, NUMNUTS® was significantly different from RING at 5, 20 and 35 min post marking (Figure 3).

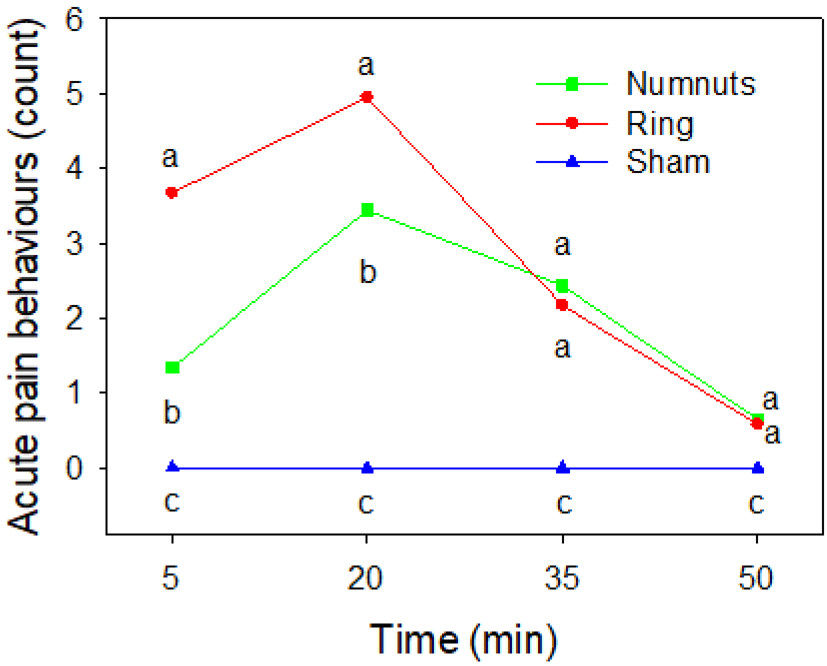

Figure 2: Field study on lamb marking. Acute pain behaviours expressed by male lambs, data pooled across both trial sites. a, b, c: Means within a time interval with different superscripts are significantly different (P < 0.005)

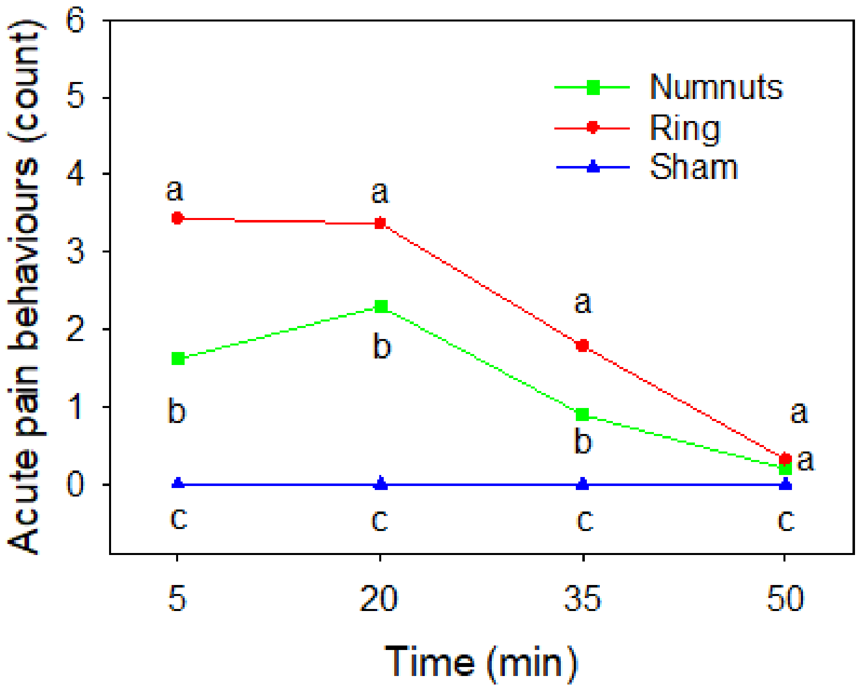

Figure 3: Field study on lamb marking. Acute pain behaviours expressed by female lambs, data pooled across both trial sites. a, b, c: Means within a time interval with different superscripts are significantly different (P < 0.005)

Conclusions

Using NUMNUTS® at lamb marking reduced the expression of active pain avoidance behaviours in the first 30 min post procedure, the time at which the behavioural pain response is at its greatest. The duration of efficacy is limited by the pharmacokinetics of the agent used, and is likely to be improved if a longer-lasting agent or combination of agents is used.

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What is equine chiropractic treatment and how can it help?

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What is equine chiropractic treatment and how can it help? – integrating chiropractic care into performance horse management

Introduction

Simply stated, equine chiropractic treatment is a manual therapy that evaluates and treats abnormal or restricted motion of the joints of the axial skeleton and extremities. Chiropractic treatment can help restore normal motion and relieve associated muscle spasm, pain and dysfunction of movement. With international regulation of certifying organisations and delegates, the training of equine veterinarians in animal chiropractic is more commonplace. Many now recognise the value of this therapy for poor movement and less specific signs of pain during work. The following discussion describes what chiropractic assessment and treatment is, and the indications for integration of this therapy into lameness and poor performance examinations in the equine athlete.

Who is an equine chiropractor?

Animal chiropractic techniques evolved following owner and veterinary interest in the benefits of personal human chiropractic care for management of muscle pain, stiffness and immobility in the back and body. Different physical therapy disciplines use joint mobilisation or adjustments (manipulation) to induce articular movements for musculoskeletal rehabilitation to restore joint function. Mobilisation is lighter repetitive joint movements within the normal physiologic range of joint motion. Joint mobilisation techniques are used by chiropractors, osteopaths and physical therapists to assess and treat joint restrictions. Chiropractic treatment of restricted joints uses small amplitude thrusts that are manually applied to specific joints or anatomical regions to induce therapeutic responses through induced changes in joint structures, muscle function and neurological reflexes. Joint mobilisation and adjustments (manipulation) induce different physiological responses, chiropractic adjustments seem to provide a larger clinical effect in humans and horses.

Australasian-trained veterinarians do not receive formal training or education in chiropractic principles or its clinical application, and conversely, human chiropractors do not have formal training in comparative equine anatomy, physiology, pathology and certainly clinical equine experience. This led to the establishment of primary organisations in North America and Europe to provide post graduate training of registered veterinarians and human chiropractors in animal chiropractic care. International certifying bodies provide accreditation of trained practitioners following assessment, with annual continuing professional development required to maintain accreditation. It is worth noting that internationally not all jurisdictions permit use of the term equine chiropractor, despite ‘chiropractic’ treatments techniques being employed, as such other terminology such as ‘veterinary medical manipulation’, ‘veterinary medical manipulation practitioner, or ‘animal biomechanical medicine practitioner’ are used by alternate training bodies. In Australia the Box Hill Institute offer a Graduate Diploma of Animal Biomechanical Medicine which is open to veterinarians, chiropractors and osteopaths. There are also schools offering training in osteopathy (International School of Equine Osteopathy) and certification in veterinary spinal manipulation therapy. Many of these techniques and therapies rely on the principles of assessment and treatment of impaired joint movement or function in the axial skeleton, and have value as physical therapy in equine practice, however the different terminology adds to confusion and scepticism in the equine veterinary community.

The historical paucity of education, training, and accreditation in equine chiropractic care has afforded a period of time for lay therapists, claiming to be equine chiropractors, to use techniques that do not adhere to chiropractic principles and are often a misapplication of the modality. Typically, uneducated discussions are had with owners about problems that may exist in their horse such as: being 'out in the poll or withers' or diagnosing 'sciatica', the 'pelvis is out', 'back is subluxated', or there is a 'bone out of place'. Longer-lever manipulations may be performed that have potential to injure the patient, or even worse these manoeuvres are performed with the horse sedated or anaesthetised where there is no inherent reflex muscular protection during axial skeleton manipulation. These discussions and practices are outdated and inaccurate and do not reflect or acknowledge the modality of chiropractic care as a science and a profession. This has led to confusion, misinformation and scepticism in the equine and veterinary community. Clinicians are consequently reluctant to engage or become educated in the equine chiropractic discipline. As such their clients may be missing out in the potential benefits offered in diagnosis and management of musculoskeletal pain or movement complaints. It is important that if horses are being treated by chiropractic techniques, that they are under the primary care of a veterinarian to ensure conditions that should be diagnosed and managed by traditional medical treatment have been identified and addressed, such as lameness or serious pathology of the musculoskeletal system.

What is equine chiropractic medicine?

Equine chiropractic medicine includes the diagnosis and treatment of abnormal or restricted movement in the joints of the axial and appendicular skeleton. These commonly lead to muscle tension, pain and dysfunctional movement. It provides expertise in evaluating vertebral column disorders and can provide additional means of diagnosis and early treatment options in certain types of gait abnormalities or performance problems.

In order to practice and understand equine chiropractic techniques and principles it is essential to have knowledge of equine musculoskeletal anatomy, physiology, biomechanics and pathology. The axial skeleton is commonly the focus; however, assessment involves evaluation of the whole horse as a biomechanical unit. This entails evaluating the horse during movement, static palpation of musculoskeletal system, assessment of passive and active range of movement of the vertebral segments of the axial skeleton, and motion palpation of individual joints to determine focal or regional restrictions in movement. Regions of abnormality are identified by muscle hypertonicity, local or regional pain, or restricted movement of specific joints upon motioning. Once abnormal locations of movement are identified manual techniques are used to re-establish normal motion of the joints involved. This is primarily through application of low amplitude, high-speed forces at an angle specific to that particular joint.

Why do chiropractors use the word subluxation?

The term 'vertebral subluxation complex' or VSC is an historic chiropractic definition describing joint dysfunction in two adjacent vertebrae and the associated soft tissues that bind them. This VSC is diagnosed by identification of loss of or asymmetrical joint motion, lowered pain thresholds to pressure in adjacent paraspinal tissues and osseous structures, abnormal paraspinal muscle tension, and visual or palpable signs of active inflammation in superficial tissues. This is NOT a subluxation as defined in medical terminology, and this has been the source of confusion and scepticism for many equine veterinarians. For this reason, rather than using the term 'subluxation complex' to describe abnormal joint motion and associated local tissue changes, it is more readily described in veterinary terminology as a 'restriction' in motion or 'vertebral segment dysfunction'. The basic elements of joint dysfunction include altered articular neurophysiology, biochemical alterations, pathological conditions of the joint capsule, and articular or cartilage degeneration.

What is an adjustment?

An adjustment, as described by chiropractors, is a short-lever, low-amplitude, high-speed force that is applied manually to a joint at a specific angle to re-establish movement in a motion segment. By stimulation of receptors in the joint capsule and local tissue, improved proprioceptive input is achieved and local muscle spasm can be overridden which results in reduced hypertonicity and pain in the local region allowing re-establishment of normal movement. Adjustments are applied to previously identified regions of restricted motion, most commonly in the vertebral segments of the axial skeleton. The joint or vertebral segment is evaluated for restriction in specific directions (determined by the local anatomy and range of movement) by using gentle but progressive pressure to take the joint to the end of the passive joint range and determine the 'end feel'. If this passive joint range is restricted the adjustment can be applied. Because the adjustment is a low amplitude motion the joint is stimulated, but not taken outside the anatomical limit. Importantly this differentiates this technique from long-lever methods used by some, typically under anaesthesia, where normal anatomical limits are exceeded with the potential for long term damage.

How does chiropractic therapy work?

The goal of chiropractic therapy is to reduce pain and muscle tension, restore joint motion, and stimulate neurological reflexes. The exact mechanisms by which therapeutic effects are elucidated are not certain. Concurrent muscle spasms restrict joint motion and may contribute to further development of joint stiffness. Following chiropractic treatment palpable changes and reduced tension in paraspinal muscles is achieved. Chiropractic therapy can improve restricted joint motion to reduce the harmful effects of joint immobilisation. By motioning or adjusting restricted vertebral segments it is thought that mechanoreceptors (golgi tendon organ, muscle spindles, joint capsule) are affected and induce reflex inhibition of pain and reflex muscle relaxation to correct abnormal movement patterns. Additional modalities used in people and horses include stretching and massage to relax hypertonic muscles, strengthening of weak muscles and re-education of movement patterns through exercises and physiotherapy. This is an area where the owner can become engaged in the rehabilitation, and advice in this area is often received enthusiastically.

What are the indications of equine chiropractic evaluation and treatment?

Chiropractic treatment is commonly sought for horses with "sore backs", but the indications and benefits of chiropractic evaluation are much broader than this. It does expand the diagnostic and therapeutic arsenal of the equine veterinarian, providing evaluation and treatment in horses where a specific diagnosis or pathology has escaped traditional evaluation techniques. It is important to note that pain in the axial skeleton can arise from restricted movement in the spine with associated paraspinal muscle spasm rather than pathology of the osseous structures. That is, non-specific back pain in most cases is due to a functional impairment and not a structural disorder. Chiropractic treatment can be very effective in relieving pain in the back or cervical spine in these situations.

Approximately 75% of horses that have signs of back pain have concurrent limb lameness, and approximately 25% of horses that have limb lameness also have back pain. Therefore, veterinarians focusing solely on limb lameness may disregard the influence of back pain on altered gait patterns in a large proportion of their patients. The treatment paradigm that management of distal limb lameness will resolve back pain is outdated and inappropriate for a lot of horses. A multifaceted approach should be used where all identified pain regions are addressed to enable a more rapid and prolonged return to work through normalisation of movement patterns.

Distal limb injuries can cause alteration in the carriage of the affected limb and altered gait, which subsequently can overwork or injure proximal limb musculature and the paraspinal musculature. Similarly, vertebral column injuries can produce gait abnormalities, increased concussive forces, and distal limb lameness. The diagnostic dilemma is deciding whether the limb or the vertebral column is the primary or initial cause of the horse's clinical problem.

Regardless, abnormal movement in any part of the musculoskeletal system due to pain is likely to have compensatory or secondary effects in other locations. The clinician should keep an open mind to this possibility, particularly if the response to treatment of one region is poor or incomplete. The aim of chiropractic treatment is to re-establish normal movement in regions of dysfunctional movement that has occurred due to local trauma or secondary to other problems.

Chiropractic evaluation and treatment is indicated primarily or as part of an integrative approach for any horse that has non-specific pain of the axial skeleton, and vague lameness or performance complaints, where veterinary examination has identified and treated or ruled out relevant pathologies. Chiropractic evaluation of the axial skeleton can provide insightful information into diagnosis of functional or structural abnormalities of the spine and can assist and direct veterinarians in their diagnostic and therapeutic approach. Chiropractic treatment is contra-indicated in acute episodes of arthritic pain or impingement of dorsal spinous processes, severe articular changes, joint subluxations, and most acute soft tissue injuries. It is not a 'cure-all' for all back problems and is not indicated for treatment of fractures, infections, neoplasia, metabolic disorders, neurological disease and non-mechanical joint disorders. It can be very beneficial in the rehabilitative phase to help re-establish normal movement and joint mobility. For these reasons, close communication and a team approach to chiropractic and veterinary care is recommended for management of performance problems in horses.

Prophylactic vs therapeutic chiropractic care: when and how often?

Horse owners familiar with, and invested in preventative health care of their horses as athletes, commonly request chiropractic evaluation and treatment at the start of a training campaign, or within a training campaign to allow early detection and intervention of minor restrictions in movement that may not yet have led to a clinical problem. Such horses are often treated every 2-3 months or if subtle alterations in their behaviour or movement occurs. The benefits of such management as an equine veterinarian with chiropractic training is early identification of any abnormalities of the musculoskeletal system, which can be addressed or treated before learned muscle memory associated with compensatory movement becomes established. Additionally, it affords the clinician a close relationship with the horse by means of sequential assessments over a time period so subtle changes are more likely to be detected. This provides a positive veterinary–client–patient relationship rather than crisis management of a serious problem or lameness that veterinarians often face.

When using chiropractic techniques to treat horses for problems of stiffness or pain of the back, neck or sacropelvic region, or as complementary treatment to a diagnosed lameness issue, there is no set formula to the number and frequency of treatments. This is dependent on the use and age of the horse, the duration of pre-existing or detected abnormalities, concurrent medical or orthopaedic problems, and the number of sites of restricted movement identified in the examination.

Horses that have long standing chronic problems or restricted joint motion through the axial skeleton (due to lameness or previous traumas) are more likely to require several treatments at 2-3-week intervals, with gradual improvement noted on each exam. It is very difficult to effectively and specifically diagnose and treat restricted movement in vertebral segments when a horse is tense, anxious or painful. Increased force should not be used in these scenarios, rather regional mobilisation, soft tissue therapies, and occasionally sedation may be indicated. If sedation is administered the clinician should be mindful of using reduced force in motion palpation and adjustments.

If the abnormalities in movement or gait due to stiffness, pain or lameness are recent but promptly addressed by chiropractic and veterinary treatment (if required) then the restricted areas and resolution of the problem is likely to be rapid and long lasting without recurrence. The reason that chronic problems take a longer time to normalise, and recent problems are rapidly corrected is due to 'muscle memory'. Chronic pain or stiffness results in new movement patterns being learned and adopted by the nervous system in attempt to reduce pain or discomfort. Long after the initial injury has healed, adaptive or secondary movement patterns may continue to persist that predispose to additional joint or muscle injury. Established movement patterns in muscles are more difficult to return to correct movement thus multiple treatments and additional therapies such as massage, stretching and acupuncture can be useful.

If there is pre-existing musculoskeletal pathology or external influences (saddle or rider) that are contributing to altered gait and back movement or stiffness, chiropractic treatment will only provide temporary relief and recurrence of the problem will occur within days to weeks. If underlying causes are resolved, normal movement is established, and treatment of restrictions effective, then repeat treatment is unlikely to be required for several weeks to months depending on daily activities.

Post treatment recommendations for actively training horses usually include rest for one day to allow the musculoskeletal system to respond to the treatment without immediate re-exposure to potential inciting factors of the vertebral dysfunction. If stiffness or soreness occurs, light gentle exercise is recommended for a further day before resumption of training.

Integration into of chiropractic techniques into equine practice

Integrating knowledge, techniques and these skills can afford many benefits to evaluating lameness and movement disorders in horses. Importantly these skills should be acquired through accredited training and certification. Functional anatomy of the equine axial skeleton and diagnosis of dysfunction, pain and stiffness in this area is lightly taught in undergraduate veterinary degrees and poorly understood in equine practice, but integral to understanding lameness and biomechanics in the horse.

Veterinarians are typically called when there is overt lameness or gait problems and many clients that have already spent a lot of money on lay practitioners that may delay diagnosis and early intervention of clinical problems. Training and education in equine chiropractic and spinal assessment therapies allows veterinarians to be able to offer increased value to their examination and become involved in preventative care rather than being the last resort that owners turn to when pathology is already well established.

In a profession where veterinarians are becoming more reliant on technology and advanced imaging modalities for identification of pathology we are at risk of focusing on specific areas of pain or imaging abnormalities without appreciating or questioning the clinical significance or the impact that occurs on the entire biomechanics and movement of the horse. Owners are becoming more invested in preventative health care for their horses with increased education on the benefits of appropriate training and early intervention to prevent lameness remaining undetected until significant pathology exists. Interest in animal welfare and pressure to reduce traditional medications administered to equine athletes close to competition has also increased owner interest in alternative measures to maintain soundness of their animal.

Chiropractic techniques can provide specialised evaluation and treatment of joint dysfunction and conservative treatment of neuromusculoskeletal disorders that currently lack treatment in traditional veterinary medicine. Integration of these techniques offers the equine clinician improved scope for diagnosis and treatment of lameness, pathology, and gait abnormalities in the equine athlete.

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Examination of the 'sore-backed' horse – a practical approach

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The complexities of a horse with a 'sore back'

A horse with a 'sore back' is a common presenting complaint in equine practice. The specific localisation and diagnosis of pain or pathology is often difficult, and consequently frustrating for both clinicians and clients. The basis of this difficulty stems from a poor knowledge of functional anatomy and movement of the axial skeleton, coupled with a difficult examination, in part due to the large musculature overlying the bony elements, and a range of non-specific signs reported by owners or riders. Veterinary advice is typically sought at a late stage, well after owners have exhausted other avenues, including saddle fitters and lay body workers, who they believe are more qualified and effective than veterinarians to manage back pain in their horse. Symptomatic relief may be achieved for a period of time by such techniques, they may contribute to a delay in identifying the underlying cause of back pain and any genuine pathology that may be present.

Horses can present with a variety of clinical signs that are not necessarily specific to back pain, and these clinical signs can originate from a variety of pathologies not necessarily isolated to the back or axial skeleton. Secondary behavioural changes add another layer of complexity to determining the underlying cause. For these reasons resolving all clinical signs in a horse with a sore back can be challenging.

Lameness versus back pain?

Lameness presents as obvious asymmetry in movement, and commonly originates from a focal region or structure of the distal limbs. Clinicians can specifically diagnose the problem by using palpation, diagnostic anaesthesia and diagnostic imaging. In contrast, horses with a sore back or pain in the axial skeleton can present with non-specific generalised regions of pain, or restriction in movement which is difficult to localise to a specific structure or pathology. There can be primary muscle pain or muscle tension that is secondary to underlying osseous, joint or ligamentous pathology. Adverse but varied behavioural changes are commonly noted when the horse is worked. Lameness of the hind limbs can cause secondary or compensatory restricted movement of the joints and muscles of the sacropelvic and lumbar axial skeleton, which can further lead to spasm, tension and pain of thoracolumbar musculature.

Regardless of the underlying primary cause, pain in the axial skeleton or distal limbs can cause compensatory altered loading patterns leading to transition of pain to other regions. A thorough clinical examination is imperative to ensure that the cause(s) of pain are identified and their relevance ascertained by the appropriate diagnostics and imaging. Such examinations can be time consuming and occasionally unrewarding in the initial instance, but patience and addressing the abnormal findings one at a time will usually result in successful outcomes. It is important to inform the owner that there may be a primary underlying problem but several secondary problems may also need to be identified and addressed before normal movement and function is restored.

The aim of the examination is to peel back the layers of the compensatory or secondary pain regions (or behaviour that indicates pain) in order to identify what may have been the inciting cause. Owners often ask, 'what was first, the chicken or the egg?' when discussing lameness, muscle spasm, behaviour and back pain. This question is not always easily answered, but pain anywhere in the musculoskeletal system can result in the clinical presentation of back pain, adverse behaviour or poor performance. The cause of back pain is therefore often multifaceted, so an open mind and a thorough examination is essential to increase the success of obtaining a diagnosis that is relevant and allows effective interpretation of imaging and advice on treatment and management.

Anatomy and function

The thoracolumbar spine of the horse typically includes: 18 thoracic vertebrae (T1 to T18), 6 lumbar vertebrae (L1-L6, occasionally only 5 lumbar vertebrae) and 5 fused sacral vertebrae (S1-S5). T3 is the first palpable dorsal spinous process at the cranial aspect of the withers. The dorsal spinous processes of T3-T10 are elongated and project dorsocaudally. T13-15 are the transitional vertebrae that have a gradual inclination of the DV (flexion/extension) angle of the articular facets from 45 degrees to vertical at the anticlinal vertebrae (T16). The anticlinal vertebrae (T16) is identified by a spinous process that is perpendicular to the vertebral axis. The majority of the lateral flexion and rotation in the thoracolumbar spine occurs in the mid thoracic region, whereas the majority of flexion and extension in the thoracolumbar spine occurs in the lumbar regions, mainly at the lumbosacral joint. The spinous processes of L2 to L5 are often higher therefore when epaxial muscle atrophy or weight loss occurs the horse can develop a 'roached' appearance in this region. The spinous process of L6 is not commonly palpable in well-conditioned or well-muscled horses because it is shorter and sits just cranial to the tuber sacrale of the ilium. The lumbosacral joint is made up of the articulations of the vertebral bodies and articular processes of L6 to S1 and the intertranverse joints between the caudal margin of the transverse processes of L6 and the cranioventral aspect of each side of the ileum.

The bony prominences of the thoracolumbar vertebrae and ribs allow attachments of ligaments (supraspinous, interspinous, intertranverse, ventral and dorsal longitudinal ligaments) and muscles (intrinsic spinal muscles, epaxial muscles and hypaxial muscles) to stabilise and control transfer of movement through the axial skeleton to create desired motion. This motion results from generation of force within the muscles (mostly of the hindlimbs) leading to efficient transfer of kinetic energy through the axial skeleton to create forward movement. The generation and conservation of kinetic energy is furthered enhanced by the 'bow and string' effect of the vertebral column and hypaxial and epaxial musculature. For this reason, pain free unrestricted movement of the soft tissues and bony elements of the axial skeleton is important for correct movement in performance.
Important joints of the thoracolumbar and sacral axial skeleton:

- Intervertebral articular facet joints (left and right)
- Intervertebral discs
- Costotransverse joints (rib articulations with respective vertebral bodies)
- Intertranverse joints (L6 transverse process to ilium)
- Lumbosacral joint
- Sacroiliac joints

Important ligaments of the thoracolumbar axial skeleton:

- Supraspinous ligament
- Interspinous ligaments
- Intertransverse ligaments
- Dorsal and ventral longitudinal ligaments

Thoracolumbar epaxial muscles – induces extension of spine, lateral flexion and some rotation in unilateral contraction

- Spinosus muscle (inserts on spinous processes)
- Longissimus dorsi (strongest muscle)
- Iliocostalis (erector spinae caudally)

Thoracolumbar intrinsic muscles – small muscles that lie against vertebral bodies and are important in proprioceptive adjustment of the spine

- Multifidus

- Intertranversarius

Thoracolumbar hypaxial muscles – ventral to vertebral bodies and induce flexion, unilateral contraction induces lateral flexion and some rotation.

- Psoas major and minor (mainly act to flexor lumbar sacral junction and lumbar spine).
- Rectus abdominus (inserts pubis, sternum and ventral ribs, therefore overall thoracolumbar flexion).
- Rectus oblique (lateral flexion and rotation due to insertion onto tuber coxae and ribs).

Innervation of the thoracolumbar vertebrae, intervertebral joints and axial muscles:

- Segmental thoracic and lumbar spinal nerves that exit each intervertebral foramen.
- Normal function and movement at each vertebral segment are therefore important for normal functioning of spinal nerve afferent and efferent input and output to produce accurate movement in the axial skeleton.

The clinical signs of back pain

The majority of the clinical signs of back pain reported by owners are likely 'behavioural expressions' of musculoskeletal pain anywhere in the body. The consulting veterinarian is required, through a thorough physical examination, to determine if back pain is present, the site or sites of back pain, and the potential lesions responsible for the pain.

Common complaints of a horse with a sore back and numerous, and include but are not limited to: "girthiness", "cold-backed" behaviour, bucking under saddle, head tossing, tail swishing, resistance to transitions or lateral movements, tension in the lumbar back muscles, roached back, loss of topline, poor impulsion, stopping at fences, unwilling to engage, stiffness under saddle or reluctance to bend, facial expression changes, head position changes, crookedness in work, hurrying, resisting, choppy gait, difficulty fitting a saddle or finding a saddle to suit the horse, and general poor performance or behavioural changes consistent with reduced willingness to perform. These signs are most commonly observed and noted while the horse is being ridden, and a thorough examination should include assessment under saddle.

The physical examination

The horse should be evaluated for lameness, particularly low-grade hind limb lameness which over time can cause tension and muscle pain in the lumbar musculature. If lameness is detected, diagnostic blocking and imaging should occur prior to or concurrently with examination of the back.

Posture and conformation: Assess the horse for symmetry or atrophy of the thoracolumbar and sacropelvic musculature. Atrophy of the epaxial musculature is an indication of reduced movement of the back, which is likely due to pain. Particular attention should be paid to the spinal contour and deviations, such as lumbar kyphosis, thoracic lordosis, lateral flexion/deviation, prominent or asymmetrical bony processes, and focal thickening of the supraspinous ligament or asymmetry. The horse should be standing square and evaluated on a firm flat surface. Spinal alignment and muscle shape and symmetry can be better appreciated from an elevated surface behind the horse. Preferred hind limb positioning by the horse, such as being 'camped out' or 'camped under' should also be noted.

Static palpation: When palpating the back region, start with a light touch to ascertain muscle tone and tension and allow the horse to habituate to your presence and palpation. A systematic approach is recommended by the author, starting at the cranial wither region and progressing caudally just off midline toward the ipsilateral tuber sacrale. Identify regions of increased tone or pain in response to light palpation. Extend laterally over 2-3 passes on each side of the spine until you have reached the iliocostalis muscle over the angle of the ribs. Next palpate from the tuber sacrale towards the tuber coxae, and the spinous processes of the sacrum laterally to the greater trochanter and caudally to the ischium, then down the semimembranosus and semitendinosus. Compare responses on the left and right sides. This is a general palpation scan, not looking for deep pain but for lightly detecting increased tension or a pain response to light palpation. Texture of the skin and subcutis can be palpated during this palpation scan. The skin should be supple and freely mobile over the underlying fascia and musculature. Subtle superficial changes in hair texture, swelling, or hair loss may indicate poor fitting tack or rider position. Look for repeatable pain responses rather that deliberately eliciting a pain response at an expected region. Sensitivity, tightness of skin, a quivering or strong panniculus response detected during light palpation usually reflects anticipation of pain, or focal pain due to external trauma such as a rubbing saddle blanket, or poor fitting saddle/tack, rather than deep muscular, osseous or ligamentous pain.

The next step is to examine for deeper pain or tension within the epaxial muscles. Allow the horse to relax cutaneous musculature so you can palpate to a deeper layer, particularly the to the underlying longissimus dorsi which is the most common muscle to develop tension when there is pain in the back region. In order to achieve this start with gentle pressure in one spot with the flats of your fingers of both hands, then slowly increase the pressure and again move from cranial to caudal. The horse will usually tense then relax if no abnormalities are present, or may tense firmly and spasm in areas of pain. Diffuse generalised tension or resentment in the muscles may indicate compensatory muscle pain due to 'guarding' which is seen in hindlimb lameness or from pain associated with sacroiliac or lumbosacral joints, whereas focal regions of tension and pain may more likely represent primary deeper axial skeleton pathology. These are generalisations, but it is important to note that each horse has different tolerances to this type of examination depending on temperament, training, breed and what has led to the presentation of back pain.

The supraspinous ligament is evaluated by direct palpation to detect pain, abnormal thickening, or asymmetry. The supraspinous ligament becomes remnant from L5 to S1 so is less readily identified in this region and corresponds to the ventral 'dip' in the topline observed just cranial to the tuber sacrale, particularly in horses that have epaxial muscle atrophy. From an elevated surface it is easier to apply direct and firm palpation over each of the dorsal spinous processes to determine closeness and pain on motioning in a ventral direction which may be consistent with impingement of spinous processes. Gentle ventral traction of the tuber coxae and axial compression of the tuber sacrale can be performed to determine if there is a pain response of the sacroiliac joints.

Range of movement and mobilisation assessment: The range of movement of the spine should be assessed. Pain responses may be reflected by a 'crouching' posture to avoid pressure, change in facial expressions or tail swishing, muscle tension, grunting or resistance to induced motion. Assessment of thoracic, thoracolumbar, and lumbosacral flexion, extension and lateral flexion can be achieved by using firm pressure strokes with a hard instrument in a cranial to caudal direction either side of the spine, and over the gluteal muscles. The horse should be observed for free relaxed and repeated movement through the spine while this is performed. Resistance or guarding responses may be consistent with pain in the affected regions. Light palpation of muscles with one hand during these induced movements can determine if muscle spasm occurs. Gentle oscillations in a ventral to dorsal direction can also be performed from an elevated surface with hands placed on the individual spinous processes. Focal regions of evasion may indicate pain within those vertebral segments.

Assessment of spinal motion in movement and under saddle: The horse should be assessed in hand at the walk and trot, and lunged at the trot and canter if possible. This is imperative to assess not only pain and resistance, but functional disorders and reduced regional intervertebral mobility. At the walk in a straight line the left and right tuber coxae should rotate in a figure-8 motion (pubis central) when viewed from behind. Walking in small circles should show willing lateral flexion of the spine. At the trot on a lunged circle the back should again show symmetrical lateral flexion in each direction and hind limbs should show adequate engagement and propulsion. The canter on the lunge affords the observer the opportunity to view the spine for active flexion and extension during hindlimb protraction and propulsion respectively. The lumbosacral spine has more movement in flexion and extension than the thoracolumbar spine. Resistance, tightness across the back, reduced spinal movement and poor hind limb impulsion may indicate back pain.

Viewing the horse under saddle may allow the clinician to determine how tack and weight of the rider changes the horses gait, back motion, or behaviour, and can provide valuable insight to the regions of pain, possible causes and underlying pathology. It is important to note that back pain is readily induced by poor fitting tack, poor riding, or mismatched rider to horse weight ratios. These subjects can be difficult to diplomatically discuss during the consultation but involving the owner in the discussions during the examination and enlisting the support or advice of the owner or rider's friend or coach can be invaluable. Videoing the movement of the horse without tack and rider, with a surcingle only, with the saddle only, and with the rider and saddle may allow the rider to objectively reflect on the horse's responses. If examination under saddle is not possible, the 'surcingle test' can be used whereby a surcingle with padding can be placed and fastened firmly. Observe for adverse behaviour or muscle responses on tightening, and for changed spinal and gait biomechanics when assessed during movement as above.

At the completion of the above physical exam, the clinician should be left with a general impression of where the pain is originating from and what might be contributing.

Diagnostic anaesthesia

Local anaesthesia is useful to determine the significance of overriding dorsal spinous processes that may have been identified on radiography. A high proportion of horses that have radiographic evidence of impinging spinous processes **do not** necessarily have back pain. Direct infiltration of mepivacaine in or adjacent to the interspinous space is recommended to help determine the clinical significance of radiographic abnormalities. Depending on the number of sites affected 60-80ml of mepivacaine can be injected with or without ultrasound guidance using 4cm needles. The horse should be re-assessed for all pain responses at 15-20 minutes, including behaviour and movement under saddle. Local anaesthesia infiltrated into deep epaxial musculature if primary muscle pain is suspected, or under ultrasound guidance into the dorsal articular facet joints can be performed, but the specificity of response is reduced due to the close proximity of the spinal nerves to these joints and the likelihood of diffusion over time. A general improvement in the exam findings may help confirm the region of pain even if specific pathology is not confirmed.

Pathology commonly identified in the thoracolumbar region

- Overriding dorsal spinous processes
- Supraspinous ligament desmitis
- Dorsal articular facet osteoarthritis
- Lumbosacral joint disease
- Vertebral spondylosis
- Lumbar transverse process ankylosis
- Muscle spasm/pain restricting movement of vertebral segments
- Primary muscle pathology – rhabdomyolysis, muscle tear, direct trauma

Diagnostic imaging of the thoracolumbar region

Radiography, ultrasonography, nuclear scintigraphy and thermography are all commonly applied to evaluate the soft tissues and osseous structures of the thoracolumbar spine. Radiography can be used to identify impingement of the dorsal spinous processes, and to evaluate the vertebral bodies and the articular facet joints (synovial intervertebral articulations). A high-output X-ray machine is necessary for imaging the vertebral bodies and facet joints effectively. Ultrasonography can be used to examine the supraspinous ligament, interspinous ligament, and closeness or overriding of adjacent dorsal spinous processes. The transverse processes and the dorsal articular facets can be imaged in paramedian scans and can aid in detecting irregular or proliferative margins of facet joints, suggestive of osteoarthritis, and in performing ultrasound guided facet joint injections. Evaluating the lumbosacral joint and the intertransverse lumbar and lumbosacral articulations can be evaluated by per rectum ultrasonography. Nuclear scintigraphy can provide valuable information to differentiate pathology in the osseous structures of the axial skeleton by identification of abnormal radiopharmaceutical uptake. Active bone remodelling of the dorsal spinous processes, synovial intervertebral articulations, vertebral body pathology and spondylosis can be identified, but this is still not necessarily indicative of a definitive diagnosis. The usefulness of thermography is limited to superficial muscle injuries or demonstrating the effect of poor fitting tack or riding to the owner, however images lack sensitivity and specificity.

Treatment

In depth treatment and management strategies are beyond the scope of this discussion however basic principles and options are detailed for reference. Treatment of back pain should be multimodal and aimed at addressing the ‘pain-muscle spasm’ cycle, to facilitate early mobilisation and rehabilitation. Rest is rarely indicated unless acute osseous or soft tissue pathology has occurred. Treatment of the primary problem(s) depends on the diagnosis; commonly more than one abnormality is detected, and all should be treated if possible. Treatment of identified pathology can include, but is not limited to: treating concurrent lameness, intralesional interspinous injections (impingement of dorsal spinous processes), ultrasound guided intra-articular injections of facet joints (osteoarthritis of dorsal articular facet joints), mesotherapy (primary muscle pain), shock wave therapy (osteoarthritis of dorsal articular facet joints, impingement of dorsal spinous processes, chronic supraspinous ligament desmitis), administration of bisphosphonates (mature sport horses only), and monitored use of nonsteroidal anti-inflammatories. Secondary or other integrative therapies that have been shown to be beneficial in managing back pain and muscle tension include acupuncture, chiropractic motioning, soft tissue therapies such as massage, myofascial and Bowen, and Class IV cold laser.

Rehabilitation

Rehabilitation and re-establishment of normal spinal movement and muscle function is imperative to maintaining soundness and pain free performance in horses. The type and progression of exercise is determined by the underlying cause, however, is generally aimed at gradual increments of controlled range of movement exercises and strength acquisition before resumption of normal training. All movement should be aimed at achieving correct and complete muscle activation of the epaxial, hypaxial and intrinsic spinal muscles through ‘long and low’ work, lateral flexion exercises, walking over poles or obstacles, lunging with correct engagement, and ensuring correct fitting and well tolerated tack before riding commences. Complementary and integrative therapists and personnel with expertise in these areas are beneficial for the rehabilitation phase. Regular medical rechecks on ongoing treatment may be required.

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Enteropathogens in Cats and Dogs

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Introduction

Gastrointestinal disease in cats and dogs is one of the most common clinical disorders presented to the veterinarian, with the clinical sign of diarrhoea being frequently reported. Diarrhoea can be of small and/ or large intestinal origin and be due to many conditions including dietary indiscretion, hypersensitivity/ inflammatory disorders, neoplasia, infection by bacterial, fungal, protozoal, and viral organisms, and helminthiasis, to name a few. Attempting to diagnose the cause for diarrhoea can therefore be challenging, even more so if infectious microorganisms are suspected to be the cause as many enteric pathogens can be part of the resident gastrointestinal microbiome in normal individuals.

Enteropathogenic bacteria causing diarrhoea

Known bacterial enteropathogens of cats and dogs include *Clostridium difficile*, *Clostridium perfringens*, *Campylobacter jejuni* and *C. upsaliensis*, *Salmonella*, and *Escherichia coli*. *Clostridium difficile* and *C. perfringens* are Gram-positive, anaerobic spore-forming bacilli which inhabit the gastrointestinal tract of humans and animals. Both organisms may cause diarrhoea as a result of enterotoxin production. Whilst the pathophysiology of *C. difficile* infection is poorly understood, infection by strains which produce toxin A (TcdA) and toxin B (TcdB) will cause disease. In most cases, both strains are produced together.

C. perfringens organisms are divided into 5 biotypes based on the presence of one or more toxin genes including alpha (α), beta (β), iota (ι) and epsilon (ε) genes and each of these biotypes may also produce subsets of other toxins including enterotoxin (CPE) and beta2 (β2) toxin. The majority of enterotoxigenic strains of *C. perfringens* belong to Type A strains. Diagnosis of *C. difficile* and *C. perfringens* infection can be challenging as isolation of the organisms by culture and detection by PCR methods can be problematic due to the normal carriage of these organisms in normal non-diarrhoeic animals. Detection of faecal toxins by ELISA-based methods, cytotoxic culture methods and latex agglutination assays is considered the most reliable mean to diagnose infection however some of these may be costly or are not readily available and they also have limitations in their positive and negative predictive values. Ideally, these methods should be used in combination with other organism detection methods such as PCR, antigen ELISA, and culture.

Campylobacter spp. are Gram-negative, micro-aerophilic curved motile bacilli. Many species and subspecies of this organism are known with the majority of these being non-pathogenic. Known pathogenic species include *C. jejuni* and *C. upsaliensis.* Reports have shown that carriage of *Campylobacter* is high in non-symptomatic normal animals and those with diarrhoea and prevalence in younger animals tends to be higher than that in adults. Co-infection with other organisms is also common, particularly in younger animals. Dogs and cats housed in shelter or crowded conditions or fed home-cooked diets and table scraps are also more likely to have *Campylobacter* detected in their faeces. Since carriage of *Campylobacter* is high in normal animals, detection by a combination of methods such as culture and PCR are advised for a diagnosis and results should be interpreted in conjunction with the appropriate clinical signs.

Salmonella spp. are Gram-negative, motile, non-spore-forming facultative anaerobic bacilli which have the potential to cause acute and sometimes chronic gastrointestinal disease in dogs and cats. Because the prevalence of *Salmonella* in healthy animals is similar to that in diarrhoeic dogs and cats, the diagnosis of salmonellosis can be challenging. This is further complicated by the fact that not all strains of *Salmonella* will cause diarrhoea and many infections may also be subclinical. Dogs and cats housed in shelter conditions or fed raw food diets have been found to have higher prevalence. Diagnosis of Salmonella infection is best made using a combination of culture methods and PCR. Culture methods utilise various selective culture media and enrichment broth to optimise isolation of the organism. Ideally, PCR positive faecal samples should be cultured using selective enrichment media. *Salmonella* isolates can then be serotyped, usually by specialized laboratories.

Escherichia coli are Gram-negative, non-spore-forming bacilli that form a significant component of the normal intestinal microbiome. Under certain conditions, such as diminished local/ systemic immunity and the acquisition of bacterial virulence factors, they can become pathogenic and cause diarrhoea. Various pathogenic *E.coli* types exist, and these have been isolated from healthy and diarrhoeic animals. Pathotypes include: enteropathogenic *E. coli* (EPEC); enterotoxigenic *E. coli* (ETEC); enterohemorrhagic *E. coli* (EHEC); necrotoxigenic *E. coli* (NTEC); enteroinvasive *E. coli* (EIEC), enteroaggregative *E. coli* (EAEC); and adherent-invasive *E. coli* (AIEC), which has been implicated in histiocytic ulcerative colitis (granulomatous colitis) observed in Boxer dogs.

Protozoal organisms causing diarrhoea
Giardia lamblia (= *G. duodenalis*) is an anaerobic flagellated protozoan parasite which can cause diarrhoea in dogs and cats and humans. It is considered the most prevalent intestinal parasite infecting cats and dogs with variable reported prevalence, depending on the method of detection used. Prevalence of up to 20.6% and 15.2% in dogs and cats, respectively have been reported. Clinical signs associated with infection are not specific and can vary, ranging from asymptomatic to severe gastrointestinal disease. Because of its zoonotic potential, diagnosis of infection is particularly important. Detection of *Giardia* organisms can be achieved using various techniques including faecal floatation, examination of faecal smears, ELISA-based methods to detect coproantigen, PCR testing, and direct immunofluorescence (IFA) methods. Direct IFA microscopy is considered the gold standard test for *Giardia* detection with reported sensitivity and specificity of 100%. ELISA-based methods to detect coproantigen perform well with some assays having test sensitivity of 91.2% and specificity of 99.4%.

Tritrichomonas foetus is a single-celled flagellated protozoan parasite which infects and colonises the large intestine of cats, causing prolonged and often intractable diarrhoea. Infection is acquired by the fecal-oral route from an infected individual to an uninfected one. *T. foetus* is not considered to be part of the normal fecal microbiome, therefore its isolation/ detection in faecal samples either by culture methods or PCR testing is considered significant. It is important to note that *T. foetus* organism can be isolated from healthy cats due to subclinical shedding. When present, clinical signs are reported to persist for 5 to 24 months from the time of diagnosis. Prevalence varies considerably with up to 42.4% being reported in cats housed in cattery conditions. Young age (<1y) is considered a risk factor for *T. foetus* infection as well as breed (Abyssinians, Siamese, Bengal and Norwegian Forest cats). Diagnosis of infection can be achieved by various methods including: examination of a saline-diluted faecal smear for the presence of trophozoites (14.7% sensitivity); culture (26.4% sensitivity); culture using the InPouch™ TF medium (58.8% sensitivity); or by PCR. When undertaking testing to diagnose *T. foetus* infection, faecal samples should be diarrhoeic and free from litter as formed faeces rarely tests positive.

Enteric viral infections

Canine parvovirus (CPV) is a member of the Parvoviridae family. Infection with CPV type 2 (CPV-2) is an important cause of haemorrhagic enteritis in dogs and three variants of CVP-2 exist in Australia, CPV-2a, CPV-2b and CVP-2c. Infection with CVP-2 is associated with high morbidity (100%) and mortality up to 10% in adult dogs and 91% in pups. The virus is highly contagious, and infection is generally acquired by the faecal-oral route through contact with faeces from infected dogs or contaminated surfaces. Severe clinical disease most often occurs in dogs younger than 6 months of age, although adults with insufficient immunity may also be affected. In infected dogs, virus shedding commences a few days prior to the commencement of clinical signs. Virus shedding typically lasts for 7-10 days but long-term excretion may also occur. Since infection is associated with high morbidity and mortality, rapid antemortem diagnosis is important. There is no gold standard method to detect parvovirus in faecal samples. Various faecal antigen-based assays are available and are useful rapid screening tools for determining the presence of CPV-2 infection in clinically suspicious dogs. In general, these tests have high specificity, however reported sensitivities vary considerably. If a negative test result is obtained in a dog with haemorrhagic diarrhea, PCR methods should be employed. Although faecal PCR methods have a higher sensitivity and specificity than faecal antigen tests it is important to note that positive PCR results for CPV may be seen in dogs without signs of gastroenteritis or even in dogs recently administered an attenuated live virus vaccine.

Feline coronavirus (FCoV) is a positive-stranded RNA virus that is common in cattery-confined cats and pet cats with reported prevalence of 80-100% and 20-35%, respectively. The virus is typically shed by healthy individuals and transmitted to other cats via the faeco-oral route and primarily infects enterocytes. Outcomes of infection are variable, and many cats are often infected without any consequence. Infected cats often present with mild diarrhoea. The virus has minimal clinical significance until it mutates into the highly virulent feline infectious peritonitis virus (FIPV). FIPV is a systemic disease typically occurring in young cats (<3y) and there is no effective prevention or treatment. Diagnosis of FCoV infection can be achieved through serological and molecular methods. Measurement of FCoV antibodies is made using direct IFA techniques and a positive result may indicate infection or previous exposure. Young cats housed in shelter and cattery conditions may demonstrate very high antibody titres and it is considered that high antibody titres correlate with high fecal shedding of virus. Molecular methods such as PCR is a sensitive tool to detect FCoV however detection does not always prove causation, since FCoV can be detected in the faeces of asymptomatic cats.

Prevalence of intestinal microorganisms in diarrhoeic dogs and cats in Perth, Western Australia

A retrospective analysis of the prevalence of intestinal microorganisms in a cohort of dogs and cats with diarrhoea in Perth, Western Australia was recently undertaken. Faecal samples from domestic pets and shelter animals were submitted to Vetpath Laboratory Services between July 2014 to August 2015 and analysed by multiplex tandem RT-PCR methods. For dogs, the PCR panel included: *Campylobacter* spp., *Clostridium perfringens* alpha-toxin gene, *Salmonella* spp., CPV, *Giardia lamblia*, *Cryptosporidium parvum* and *C. hominis*, canine coronavirus (CCoV), and canine distemper virus (CDV). For cats, the panel included *Campylobacter* spp., *Clostridium perfringens* alpha-toxin gene, *Salmonella* spp., feline panleukopenia virus, Tox*oplasma gondii*, *Tritrichomonas foetus*, *Giardia lamblia*, *Cryptosporidium parvum* and *C. hominis,* and feline coronavirus (FCoV). In this cohort of diarrhoeic dogs and cats, *Campylobacter* spp. and *C. perfringens* were most commonly detected and additionally in cats, FCoV.

A total of 289 feline faecal samples, from domestic and shelter animals ranging from 25 days to 20 years of age, were analysed. Of the samples assessed, 57.4% had more than one organism detected. *C. parvum* was not detected in any sample. Five samples were positive for *Salmonella* (1.7%) and *C. perfringens* was detected in a total of 235 samples (81.3%): as a single detected organism in 77 cats and as co-presence with other organisms in 158 cats (68.2%). *Toxoplasma gondii* was detected in 3 samples (1.0%) and all had co-presence of *C. perfringens*. *Tritrichomonas foetus* was detected in 14 samples (4.8%) and all of these had co-presence with other organisms. *Giardia* was detected in 32 samples (11.1%). Of the viral organisms analysed, FCoV was found to have 39.5% prevalence and feline panleukopenia virus 6.5% prevalence. No breed predisposition was detected for any infectious agent.

Of the 80 shelter cats assessed with diarrhoea, 6 individuals had no microorganisms detected. Most commonly identified were *Campylobacter* (42/80,), *C. perfringens* (65/80), FCoV (43/80). No *Salmonella* were found in any shelter cat faecal samples. *Giardia* and panleukopenia virus were occasionally detected (7/80 and 11/80, respectively).

A total of 405 canine faecal samples were analysed, seven of which were from shelter animals and 2 from racing greyhounds. The remainder of the submitted samples were from domestic pets. 35 samples (8.6%) had no pathogens detected and, similar to that observed in cats, no canine faecal samples were positive for *C. parvum*. *C. perfringens* was present in 85.4% of samples: and as a single organism in 180 samples (52%) and as co-presence in 166 samples (48%). *Campylobacter* spp. was detected in 147 samples (36.3%) and *Salmonella* was detected in 22 samples (5.4%). *Giardia* was detected in 25 samples (6.2%). Of the viral organisms analysed CPV, CCoV and CDV were detected in 9.4%, 4.7% and 1.5% of samples, respectively. No breed predisposition was detected for any infectious agent.

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Significance and outcome of tarsal fractures on pre-sale yearling radiographs

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Weanling survey radiographs are obtained by vendors to identify horses which may need treatment of musculoskeletal disease before sale. Later, a defined set of repository radiographs are obtained within 42 days of public yearling auctions. Veterinarians are required to interpret the significance of radiographic findings for sale, soundness and future racing performance. Concerning tarsal radiographs, much has been reported on osteochondrosis, yet other tarsal lesions occur. These include incomplete (and occasionally complete) slab fractures of the third (T3) and central tarsal bone (Tc), incomplete fracture extending from the dorsolateral aspect of the tarsometatarsal joint (TMTJ) into the third metatarsus and fragmentation of the talus at the dorsolateral margin of the proximal intertarsal joint (PITJ).[1,2]

The purposes of our study were to determine (1) the prevalence and radiographic appearance of T3 and Tc fracture in juvenile Thoroughbred horses before entering race training, (2) whether radiographic evidence of T3 or Tc fracture affected purchase price, (3) whether radiographic healing occurred despite continued pasture turnout and (4) whether horses in which healing was complete raced successfully as 2- and 3-year-olds.

Results

Fractures were identified in 186 tarsi (184 T3 fracture only, one Tc fracture only, one Tc and T3 fracture) of 157 horses (126 unilateral T3, 29 bilateral T3, one contralateral Tc and T3, one unilateral Tc and T3) from 7,676 radiographic examinations (weanling or yearling pre-sale series). The prevalence of T3 and Tc fracture was 2.4% (95% CI 2.07, 2.76) and 0.04% (95% CI 0.01, 0.11) of radiographic examinations. The true prevalence of these tarsal fractures may have been higher than we report.

T3 and Tc fractures were identified by survey examination at 11.1 +/-1.3 months in 85.7% horses. Fractures occurred in the dorsolateral aspect of T3 in a dorsomedial to plantarolateral orientation and were most readily seen on the D55-65°MPLO view within 0.65cm of the dorsolateral articular margin and in the sagittal plane.

At initial diagnosis, 84.3% T3 fractures appeared incomplete and involved the distal articular surface. All horses were managed by continued pasture turnout. For horses that had radiographic examination at weanling and yearling age, it was evident that fracture healing progressed with pasture turnout yet overall there was some increase in dorsal modelling of T3 and Tc ($p<0.001$) and osteoarthritis (OA) score increased in the distal intertarsal joint (DITJ) ($p<0.001$) but not the TMTJ between survey and repository examination. Fractures healed by repository examination in 71.9% tarsi if there was >6 months between examinations.

There was no difference in sale price, and horses with T3 fractures had fewer trials when 2 and 3-years-old ($p=0.023$) yet no difference in other parameters of racing success when 2 or 3-years-old compared to controls.

Discussion

Horses with T3 fracture subsequently had fewer trials when 2 and 3-years-old compared to their peers yet it is unclear whether this is causal and potentially due to a delay in onset of race training or lameness due to tarsal injury in these cases or merely an association. There was no difference for other performance variables including number of starts and total prize money. However, the lack of reduction in racing success associated with T3 fracture in juvenile horses should be confirmed in a larger study before a firm conclusion is made. Only two horses had Tc fracture and neither raced. Although we suspect that the prognosis for Tc fracture is more guarded than for T3 fracture, this is an insufficient number of Tc fractures to make a conclusion regarding prognosis and the impact of Tc fracture in juvenile horses on future racing potential remains to be determined.

A factor that may be important in determining whether tarsal slab fracture adversely effects future performance may be whether OA of the distal tarsal joints occurs. In general, radiographic lesions of OA are most commonly seen on the dorsomedial or dorsolateral aspects of the TMTJ and DITJ. Dorsomedial lesions would be superimposed on the D55-65°MPLO view used in the study and therefore radiographic signs of OA could have been underdiagnosed as only standard repository views were obtained. Osteophytes and enthesiophytes of the distal tarsal joints (particularly the DITJ) are the only tarsal condition evaluated previously that may be associated with a reduction in 2- and 3-year old racing success.[3] In the current study, although the fracture usually involved the distal articular surface, there was an increase in OA score in the DITJ but not the TMTJ between survey and radiographic examination suggesting that loading conditions that caused fracture may also result in some degree of OA that may or may not be of clinical significance, particularly since fracture was not associated with reduced racing success. A limitation of our study was that outcome was racing ability and whether any horses developed lameness due to OA of the distal tarsal joints that was treated and if this affected the outcome is unknown.

As the duration of fractures when survey radiographs were obtained was unknown it is difficult to be certain of the time required for fracture healing, however, it appears that to ensure fracture healing in most horses, a minimum of 6 months is needed following diagnosis. Whether surgical repair and or a period of stricter confinement would reduce the time required for healing remains to be determined.

In other studies, T3 fractures have been diagnosed most commonly in Thoroughbreds during their first year of race training, in a repeatable location through the dorsolateral aspect of the dorsal facet of T3, not associated with a specific traumatic event, and have therefore been considered stress fractures.[4] Given our findings, the possibility of pre-existing incomplete T3 fracture in horses diagnosed during their first year of training warrants further investigation. None of the horses in our study were reported to be lame at the time of radiographic examination and there was no reported history or signs of external trauma. Fractures had a similar predictable configuration to those in adult horses,[4] occurred in the dorsolateral aspect of T3 and were invariably incomplete, and thus may also be considered stress fractures with increased cyclic loading during pasture turnout the proposed mechanism for these fractures.

Others have found that subchondral bone modelling in T3 and Tc, evident by around 2 years of age, also mainly affects the dorsolateral aspect of the tarsus.[5] Pasture exercise alone appears to provide enough biomechanical loading to increase bone density within T3 in foals: a similar increase in T3 bone density was present at 5-months of age in foals that had only exercised at pasture and in foals that were confined to boxes and had an imposed sprint training program as in unexercised foals.[6] We propose that the cause of tarsal fractures in juvenile horses is repeated high loading of the dorsolateral aspect of the tarsus during pasture turnout and that increased body mass or malalignment are possible contributing factors. We have also seen fracture, extending from the TMTJ into the third metatarsal bone, within 0.65 cm of the dorsal articular margin, and fragmentation adjacent to the dorsal aspect of the distal talus, distal to the lateral trochlear ridge in some juvenile Thoroughbred horses. We presume these conditions are also likely to be caused by increased cyclic loading on the dorsolateral aspect of the tarsus. A limitation of our study was that body weight or condition score and limb conformation was not recorded and any history of incomplete ossification of the tarsal bones following birth was unknown, yet these factors may influence whether excessive loads are placed on the tarsal bones. Outward rotation of hind limbs which improves as musculature develops during growth is common in yearlings,[7] yet the impact of this on risk of injury is also unknown. We also observed an increase in the proportion of horses with tarsal fractures per year over the study period. Although it is possible that veterinarians evaluated radiographs more closely in later years for evidence of these fractures, further investigation is required to determine whether paddock surface conditions, for example surface hardness or changes in hardness with variable seasonal conditions, influence the frequency of these fractures.

Conclusions

Incomplete, and occasionally complete slab fractures of the T3 occur in juvenile Thoroughbred horses during pasture turnout yet are uncommon. Tc fractures are comparatively rare. Fractures are typically initially detected on survey radiographs of weanlings that are not presented because of lameness. Fracture healing progresses with continued paddock turnout and is complete in around one third of tarsi within 6 months and in a little over two thirds of horses if more than 6 months pasture turnout is allowed after diagnosis. Horses with healed T3 fracture performed as well as their peers during the 2 and 3-year-old racing seasons. However, it may be incorrect to assume that T3 fracture never impacts on a horse's ability to race and a larger study is needed before making a firm conclusion regarding the prognosis for racing, the clinical significance of radiographic changes suggesting OA of the DITJ and long-term soundness, particularly for complete fractures that were poorly represented. The impact of Tc fracture on racing career remains to be determined. Although further investigation is also needed to determine factors that may contribute to the occurrence of tarsal fracture in juvenile horses, consideration should be given for the management of juvenile horses including the volume of exercise and body weight/condition score to reduce musculoskeletal injury, recognising that recommendations may need differ for individual horses.

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Land clearing: the costs borne by wildlife

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Introduction

Land clearing is a significant environmental issue both globally and nationally; in Australia it is an area of active legislative reform.[1] The premise of this paper, and that of our 2017 manuscript, is: "that the deaths, physical injuries, other pathological conditions, pain and psychological distress experienced by individual wild animals during and after land clearing constitute a form of *harm* that is of sufficient intrinsic value to warrant broad consideration in environmental decision-making, including in assessments of applications for permits (or other authorisation) to clear native vegetation, assessments of planning or development proposals that will require land clearing, and strategic planning initiatives in which land clearing is contemplated."[1] Currently, the harm caused by land clearing to the *welfare* of individuals is either ignored, or is merely a peripheral consideration, with the threat to/loss of individuals from a proposed clearing action solely assessed based on the modeled consequent population-level effects.[1]

We argue that as land clearing is both an animal welfare issue *and* causes substantial mortality, it is imperative that the harm that land clearing causes to *individual* animals, as well as populations, is identified and evaluated in decision-making relating to land clearing.[1] This paper and presentation will, using both national and global examples, address the propositions underlying this premise, namely (a) why land clearing is an animal welfare issue, and (b) the fact that land clearing causes substantial mortality, such that it is one of the biggest drivers of national and global biodiversity loss. Although this paper focuses on harm to mammals, reptiles and birds, the issues are broadly applicable to other vertebrates (e.g. amphibians), as well as to invertebrates – noting, however, the relevant differences across taxa in terms of pain perception and their experience of psychological distress.[1]

(a) Land clearing is an animal welfare issue

(i) Land clearing causes deaths that are physically painful and psychologically distressing because of their traumatic and debilitating nature.

Land clearing involves the removal of some or all of a site's native vegetation, as well as the destruction of any burrows, middens and termite nests also present, and commonly employs various types of machinery.[1] Hanger and Nottidge (2009) described the traumatic injuries and issues of entrapment that may arise when native vegetation is cleared in the following terms: "Animals injured directly in the process of vegetation clearing generally suffer from major crushing, deceleration or fall related injuries. Arboreal species may suffer from trauma associated with falling from a tree and/or crushing and avulsive injuries associated with boughs falling on or beside them. Such injuries include severe internal bleeding and organ disruption, multiple bone breaks, [and] eye and head injuries. Animals resting in hollows, similarly, may receive crushing injuries if the hollow bough disintegrates, or suffer internal organ injuries and tearing as a result of rapid deceleration (deceleration injury). Ground dwelling animals, such as bandicoots, echidnas, snakes and lizards, most commonly suffer from crushing and avulsive injuries (such as traumatic limb amputation), or may be buried alive during earthworks. Highly mobile species such as birds and macropods may avoid direct injury by machinery, but may suffer injuries by running into fences, motor vehicle strike or other misadventure. Injuries suffered by animals during land clearing vary from mild to severe and fatal, but these animals are only rarely presented to wildlife hospitals or shelters. This is primarily because they are less likely to be discovered by members of the community and are more usually buried or confined in piles of debris during the process of clearing, which are then subsequently burnt or chipped."[2]

As we stated in our 2017 manuscript: "Possible outcomes include death arising from traumatic injury, non-drowning asphyxiation as a result of suffocation, as well as pain and shock. Forms of traumatic injuries that animals may experience as a result of land clearing include compression injury, penetrating injury, laceration, degloving injury, amputation, fracture, joint luxation (displacement of a bone from a joint) or subluxation (partial dislocation), and blunt force injury to the skeleton, soft tissues, and central nervous system and internal haemorrhage. Those injuries may be sustained through contact with vegetation (e.g. as it is felled or shifted after felling), soil, machinery, motor vehicles, or containment barriers."[1] Whilst these quotations clearly demonstrate the physically painful, psychologically distressing, traumatic and debilitating nature of the injuries sustained by wildlife in an acute to subacute timeframe, any individuals that survive beyond this period may still succumb to chronic disease, thus experiencing suffering and harm of chronic duration.

(ii) Land clearing causes physical injuries, other pathological conditions, pain and psychological distress over a prolonged period as animals attempt to survive in the harsh and unsuitable environment of the cleared area or in the environments they are displaced to; these chronic stressors can lead to maladaptation and chronic disease.

Land clearing significantly alters a habitat's features, e.g. abiotic environmental conditions, availability of non-food and food resources, and the biotic and social environment (e.g. prey presence/absence and abundance, presence of and interaction with predators and conspecifics, interspecific interactions with new species including potential disease vectors or reservoirs);[1,3,4,5,6,7,8,9,10,11,12] such changes often then being factors that adversely impact upon any surviving individuals, with the potential to cause harm.

The harms that may occur include, but are not limited to: pain, predation, temperature-related injuries, secondary infection and septic shock arising from injuries, misadventure, exertional myopathy, nutritional disease, dehydration, increased likelihood of infectious disease transmission, as well as maladaptation and chronic stress-related pathology (e.g. adverse effects on reproduction, adversely affected immune function, suppression of growth).[1] The progression of most of these harms are common sense; however the development of maladaptation and chronic stress with respect to adverse health effects and the permissive role they play in disease warrants discussion.

(b) Land clearing causes substantial mortality and is one of the biggest drivers of national and global biodiversity loss

As we stated in our 2017 manuscript: "Whilst some individuals of certain species may disperse to other habitats (if available) when land is cleared, most, and in some cases all, of the individuals present will consequently perish, either immediately or in a period of days to months afterwards."[1,6,11,13] On the basis of clearing rates (current in 2017), more than 50 million mammals, birds and reptiles are likely to be killed *each year* due to land clearing in just Queensland and New South Wales.[1] A global study by Allan *et al* (2019) states: "The biggest drivers of global biodiversity loss are hunting, harvesting, and the *conversion of natural habitats for agriculture, urbanisation, and other industrial activity*."[14]

Australia is far from immune – more than 120 Australian vertebrate species are now on the national threatened species list[15] and at least 1000 species of plants and animals are facing extinction[16] largely due to bulldozing of bushland habitats. In fact, eastern Australia is one of the world's top 11 deforestation hot spots - alongside the Amazon, Borneo, and the Congo; with excessive land clearing worst in NSW's central west and north west, with pockets on the north coast, Hunter Valley and south west.[15,16,17] Research by the Nature Conservation Council and WWF-Australia has found koala habitat the size of 14 football fields has been bulldozed *every day* in just one part of NSW since the government weakened the laws.[18] Indeed, land clearing almost tripled in the north-central region around Moree and Collarenebri (northern NSW), up from 2,845 hectares to 8,194 hectares in just one year *despite* the Koala having been listed as Vulnerable under Federal laws in 2012 (and currently remaining so in QLD, NSW, and the ACT – although a recent report suggests that their numbers have declined in QLD and NSW to the stage they should actually be listed as Endangered[19]).[18] At this rate, Taylor *et al* (2017) suggest that koalas face extinction in NSW by 2050.[18,19] Although another report utilising passive acoustics published in 2018 found that koala *occupancy* in 171 sites in the NSW hinterland was up to ten times higher than previously thought,[20] a population estimate could not be provided using the methodology; and whilst the study showed that sites that had previously experienced native forest timber harvesting retained occupancy to varying degrees, the harvesting practices are not as invasive as full scale land clearing.

National as well as international illustrative examples that will be drawn upon during the scheduled presentation include:

- the in excess of 50 million mammals, birds, and reptiles estimated to be killed annually due to land clearing in QLD and NSW (based on rates current in 2017);[1] including the 210 threatened fauna species (in QLD alone), for which land clearing has been identified as a threat[21]
- the plight of koalas in parts of Queensland and New South Wales, where clearing is estimated to have killed in excess of 5,000 koalas between mid-2012 and mid-2016,[22]
- the Tapanuli, Sumatran, and Bornean Orangutan, all officially listed as critically endangered by the IUCN, and
- various wildlife species caught up in the now scrapped Roe8 Highway Project in Perth, Western Australia, where around 35 hectares of land was cleared in a matter of months following clearing commencing December 6th, 2016.

It is worth remembering that [in Australia] even if animals are recovered from/around clearing sites, approximately only a third of those that are rescued and treated recover sufficiently to be rehabilitated and released;[15] it is likely that the rescue/rehabilitation/release rates are even lower in less developed nations.

Conclusion

The word 'harm' is deliberately used to describe the deaths, physical injuries, other pathological conditions, pain and psychological distress that animals may suffer due to land clearing, for two reasons. Firstly, the term 'harm' connotes physical injury and deliberate intent.[1] As previously discussed above in (a, ii), of those individuals that initially survive, most, and in some cases all, will perish in a period of days to months afterwards. That forgone conclusion is an important basic consideration for decision-making because it means that any decision to clear land of its native vegetation (or allow it to be cleared) ergo is also a decision to kill most or all of the individuals inhabiting that vegetation (or allow them to be killed).[1] Therefore, suffering is the inevitable consequence of a decision to clear land (or allow it to be cleared).[1] As we stated in our 2017 manuscript: "The relevant question for decision-making is not *if* death, injury and other pathology will occur when land is cleared, but *how much* of that harm will occur, *how severe* it will be, and *whether it ought to be avoided*. If such harm is, nonetheless, deemed necessary, then the question is *how the harm to be imposed could be minimised*."[1]

The second reason for deliberate use of the word 'harm' is to establish a link between the harm caused by land clearing and the concept of harm that underlies Australian animal welfare legislation, given several Australian animal welfare statutes include definitions for 'harm'.[1] As we stated in 2017, "Further work is needed to develop appropriate statutory and policy-based mechanisms to identify and evaluate the harms caused by proposed land clearing activities and to allow for the effective consideration of those harms in decision-making relating to land clearing."[1] Ultimately, law reform including strengthening of laws to stop excessive land clearing is urgently required.

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- The Beeliar Group - professors for environmental sustainability (https://thebeeliargroup.com).
- Rehabilitating Roe8 (https://rehabilitatingroe8.org).
- Dr Jon Hanger (Director), Endeavour Veterinary Ecology (http://www.endeavourvet.com.au).
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Adrenal Surgery

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Summary

Fascinating yet complex, the adrenal glands situated just cranial to the kidneys produce two main substances : Adrenal medulla produces catecholamines, adrenaline and noradrenaline: Adrenal cortex produces corticosteroids and sex hormones in small quantities. Adrenal glands can malfunction due to either a centralised pituitary tumour or from a primary adrenal tumour/s, occasionally some patients will have tumours of mixed composition either bilateral or unilateral, so a diagnostic challenge. Diagnostic investigations incorporating routine blood profiles, adrenal function testing, blood pressure recording and ultrasonography form a solid baseline to work from if adrenal disease is suspected. With more skilled ultrasonography becoming available in general practice, we are finding more patients with incidental adrenal asymmetry or a well established adrenal masses. This raises a dilemma, as to operate is not without potential fatal risk to what may genuinely be an asymptomatic patient. Should we adopt a surveillance approach to those patients? Advanced imaging is extremely helpful to determine if there is invasion of the adrenal mass into the phrenicoabdominal vein (PAV) or caudal vena cava (CVC). Evidence of invasion brings another dimension of risk into the decision making equation of whether to operate – do you have the skill set? Do you have the surgical equipment, blood products, intensive care support that may well be required for these more complex patients?

Surgery is contemplated most commonly for the following conditions:

- Adrenal dependent hyperadrenocorticism (HAC)
- Phaeochronocytomas (PC)
- Adrenal tumours secreting excess aldosterone Conns syndrome in cats
- Adrenal gland over producing sex hormones in Ferrets

Important practical facts about adrenal glands and adrenal tumours

- **Normal adrenal size**: Dogs 1-5cm long 0.2-1.2cm wide Cats 0.45-1.37cm long 0.3-0.53cm wide
- **Depth of adrenal location :** Have in mind that the adrenal glands in all canine breeds are not in an easily accessible location, situated in the abdominal paravertebral gutters, you need good sustained exposure for surgery with surgical assistance and optimal theatre lighting
- **Close association with CVC** : The right adrenal gland is often underneath the CVC and interwoven into the adventitial covering of the CVC
- **Neovascularisation** : Under the influence of neoplastic angiogenesis, multiple fragile vessels form to connect the CVC, aorta, kidney, abdominal wall to the abnormal adrenal gland.

Ten interesting facts about adrenalectomy surgery

- With the advancement in preoperative supports, haemostasis and surgical techniques in general the mortality rates are between 13-22% in dogs
- Most common adrenalectomy complications are uncontrolled haemorrhage, pulmonary thromboembolism, acute pancreatitis, renal failure, hypoadrenocorticism
- Patients confirmed with HAC by endocrine testing benefit from pre operative Trilostane medication
- Patients confirmed or suspected of a PC may benefit from Phenoxybenzamine (PBZ) an alpha-adrenergic antagonist, 2-3 weeks pre operatively. First reported in 2008 to reduce mortality rates from 48% untreated to 13% treated with PBZ
- Incidental adrenal masses : Ultrasound finding in 4% dogs, CT finding in 9% dogs
- Cats with HAC, 50-80% of those patients have concurrent diabetes mellitus. Very careful patient selection is required if surgery is contemplated and provision of post operative enteral feeding support an essential consideration in HAC cats
- Any patient having a bilateral adrenalectomy will need long term corticosteroid and mineralocorticoid support.
- Cat with Conns syndrome treated by adrenalectomy can afford a disease free interval of 5 years and benefit from pre-stabilisation of potassium, spironolactone and amlodipine.
- Ferrets having adrenalectomy afford a 98% 1 year survival and a 70% 5 year survival rate.
- Local invasion PAV or CVC: Adenocarcinoma 11% PC 39-71% : two recently published reports in 2018 and 2019, document the practicalities of such interventions. Mortality rates 21-25%

Key Learning Objectives

- Revise adrenal anatomy and physiology
- Understand the different adrenal presentations
- Learn to plan, prepare and action surgical tips for adrenalectomy surgery

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Advanced Biliary Surgery – when the bile stops flowing

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Summary

The two most common conditions we encounter for biliary tract surgery in the UK are cats with extra hepatic biliary tract obstruction (EHBTO) and dogs with biliary mucocoeles. Formulating the best treatment plan still remains a challenge. In EHBTO patients that are not medically responsive, what are the surgical options? The latter is very much dependent on where in the biliary tract is the problem located:

- Gall bladder
- Cystic duct
- Common bile duct (CBD)
- Duodenal papilla

The exact location and severity of the problem is not always realised until the surgical exploration is complete and only then can you finalise and action a surgical plan. One thing is for certain is that biliary tract surgery will always require careful and protracted retraction on neighbouring viscera . The following instrumentation and accessories are vital to have at hand :

- Good theatre lighting
- A surgical assistant for additional retraction
- Balfour retractors
- Soaked swabs
- Surgical clips : Mediplus SLS clips small and medium
- Variety of urinary catheters / feeding tubes 3.5-10fr, soft to pass into CBD but with some stiffness (ideally not rigid plastic or with stylet)
- Debakey forceps 15-18cm long
- Polydioxanone 4/0 1.5m : 5/0 1m: 6/0 0.7m
- Oesophagostomy feeding tubes : Infusion Concepts 10Fr- 20Fr

In human beings with EHBTO, the majority are treated with endoscopic placed bile duct stents, choleliths are dealt with by lithotripsy or laparoscopic cholecystectomy, converting to open surgery if required. Although these techniques are filtering into the veterinary world, they are far from main stream treatments yet for the majority of our patients. The most common surgical procedures we still consider are:

Procedure	What is this?	Reason
Cholecystotomy	Opening up of GB	Remove inspissated bile
Choledochotomy	Opening of a dilated CBD	Remove stone in CBD
Duodenotomy	Open proximal duodenum in vicinity of duodenal papilla DP	Catheterise the CBD, ensure patent, gently aspirate biliary sludge and lavage
Choledochal stenting	4-8cm stent of soft feeding tube between CBD and DP	Encourage bile flow, allow inflammation to resolve. Stent is defeacated out.
Cholecystostomy	Place foley catheter into GB via abdominal wall	Allow temporary decompression of GB whilst inflammation / pancreatitis resolves
Cholecystectomy	Removal of GB	GB stones or mucocoele
Cholecystoduodenotomy	Attach the GB to the duodenum via 3cm stoma	Divert bile flow due to non patent CBD

You will optimize a patients outcome by delicately handling the biliary tissues, preoperatively having a knowledge of their ability to clot, supplementing with Vit K pre operatively as required and providing enteral support. However, particulary with feline EHBTO patients undergoing biliary diversion surgery, there is an overall high mortality rate of 40% in those patients with an underlying inflammatory condition. These patients may not have suffered substandard surgical technique but often experience recurrent clinical signs of cholangiohepatitis, pancreatitis and chronic vomiting which precludes a long term quality of life. Patients with neoplasia of the biliary tract have a very poor prognosis, except cats with benign biliary cystadenomas which can be amenable to surgery even though large.

Key learning objectives

- Reconsider the biliary tract anatomy
- Realistically what are the surgical options in practice
- Must have accessories to maximize a successful outcome

Useful reading references

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- Monticelli et al JFMS 2016 1-6
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C-section : Optimise your approach

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Summary

There is always a buzz in the air with the news that a 'caesar ' or C-section is on its way into the practice, with the expectation that we will safely deliver some healthy puppies or kittens and all will be well. But like a number of situations in every day practice, despite this being classed as a 'routine' surgery, there could be a number of pitfalls and numerous questions suddenly come to mind as you start to assist the patient. Does this patient need and warrant a C section ? Should I give a premed? When should I give analgesia to the bitch or queen? What if the placenta will not come away? How do I optimally close a uterus? What is the best way to revive a neonate?

- Greater than 60% of cases of dystocia in the bitch and queen require caesarean section.
- Determination of the need for C-section are based on the assessment of the dam, the progression of labour and foetal heart rate. A reduction in foetal heart rate to 150 beats per minute, primary uterine inertia, secondary iterine inertia, obstructive dystocia, uterine rupture and uterine torsion are indications for surgical intervention.
- Note that no drugs are licensed for pregnant bitches / queens or neonates, so a clear verbal discussion and written consent from the owner / breeder must be acquired before admission.
- Anaesthetic protocols should be based on minimizing the time from induction to delivery of neonates, maintaining materal airway and blood pressure, supporting uterine blood flow. There is no one ideal anaesthetic protocol as many anaesthetic agents cross the placental barrier. Alpha-2 agonists, ketamine and thiopental are best avoided.
- Analgesia is vital for the welfare of the bitch / queen and can be given once the last puppy or kitten has been removed from the uterus.
- Uterine closure can be a single or double layer using appositional sutures
- Two causes of foetal depression associated with C-section include hypoxia associated with dystocia and depression from medications given to the dam as part of the anaesthetic protocol. Signs of hypoxia in the neonate include slowing of the heart rate and respiratory rate and reduced movements. Resuscitation should involve warming the neonate and supplementing oxygen to tissues. Doxapram at 1 to 2 drops under the tongue can be useful as it is a respiratory stimulant, but it is only effective when used in conjunction with supplemental oxygen. Acupuncture can be used to stimulate respiration by inserting a 25-gauge needle into the nasal philtrum until it contacts bone and turning the needle. Neonates with persistently slow heart rates may benefit from lateral chest compressions to stimulate heart rate.

Key learning objectives

- Decision making as to when to perform a C-section
- Anaesthesia protocols appropriate for a C-section
- Pre, intra and post operative considerations for a C-section

Useful reading references

- BSAVA manual of canine and feline reproduction and neonatology 2010

Feline tail pull injury

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Summary

A sudden traction injury to the tail is thought to occur as a tail gets caught underneath a car tyre. As the car moves in the opposite direction to the tail, a sacrocaudal fracture or luxation occurs which results in varying degrees of trauma to the sacral spinal segments and cauda equina. The most devastating sequel to this traumatic event is urinary dyssynergia with sometimes concurrent anal sphincter dysfunction. The innervation to the bladder is complex.

- Pelvic nerve is responsible for the detrusor muscle contraction
- Pudendal nerve contracts the urethral striated muscle
- Hypogastric nerve contracts the urethral smooth muscle and relaxes the detrusor muscle.
- Both the parasympathetic pelvic nerve and the somatic pudendal nerve originate from the spinal cord segment S1-3. Although the sympathetic hypogastric nerve originates much further cranially at L1-4/5, it merges caudally with the pelvic nerve at the pelvic plexus. So, you can appreciate that a sudden traction to the sacrocaudal region will have potentially catastrophic effects of the delicate neural control of urination.
- Urinary dyssynergia can be defined as a lack of coordination between the detrusor muscle and the urethral 'sphincter' muscles.

Owners may report their cat does go outdoors, had become quiet in demeanour, seems sore around the tail / bottom, they may have noted the immotile tail, they may or may not have witnessed any stranguria. A full clinical examination should always be performed, particularly where there is a high index of suspicion of a traumatic incident. 84% of cats with tail pull injuries have concurrent injuries. Neurological examination should include testing the segmental reflexes. The perianal reflex which is where the anus twitches and the tail flexes in response to forceps stimulating either side of the anus is often absent or reduced. To test the actual pain response / nociception around the perineum, if mosquito haemostatic forceps are closed on the skin around the perineum, the cat should consciously respond. Gentle palpation around the pelvis and spine and sacrocaudal junction may identify an area of malalignment and even the point of luxation. It is important during that initial examination to palpate the bladder and get an appreciation of how full it is of urine. Quite often the cats are presented a number of days after an unknown trauma, so the urinary dyssynergia has set in and the bladder will be turgid upon presentation. Observation of a cats ability to urinate is important. If they do void, then immediately afterwards, check the size of the bladder to see how fully they are voiding or if they have voided only a small amount, you will find that there will be a lot of residual urine within the bladder indicating dyssynergia. Plain radiographs in conjunction with the suggestive clinical signs of a tail pull injury may be all that is needed to make a diagnosis.

So, does the urinary dyssynergia come right? It can, but it does take time. The best prognostic indicator we know of for the return of actual urinary continence is the conscious recognition of a painful stimulus around the perineum or tail base. Two separate studies found that if cats had perineal or tail base sensation at the initial presentation then within 30 days from injury 75-100% of cats had regained their urinary function. Urinary function had been defined as urinating purposefully in a litter tray, Importantly, however, we do see cats that do not have these perineal or tail base sensation at initial presentation and 50-60% of those cats will also regain urinary function within 30 days, they will just do so more slowly. A poor prognosis should be given to cats that within 2 weeks of the trauma still have no perineal or tail base sensation, a flaccid anal sphincter and no clinical signs of regaining conscious urinary function. Many cats have ongoing urinary dyssynergia despite having an progressing level of continence. Urinary dyssynergia can certainly improve though with time and medical management.

Management of our tail avulsion cats should incorporate analgesia, bladder function modifying drugs, bladder expression, or initial urethral catheterization or temporary cystostomy tubes. The use of accessories for urinary diversion allows the medications below to take effect and allows some of the initial contusions to resolve or at least improve. Tail amputation is advised once we know if the cat is regaining urinary control. Surgery to remove the flaccid tail could be performed sooner if the tail is degloved to improve pain control.

Medications that can modify bladder / urethral function

Drug	What does it do?	How much?	Possible side effects
Diazepam	Skeletal muscle relaxant	1-2.5mg tid	Sedation ataxia Muscle weakness Excitement Appetite stimulant Fulminant hepatic necrosis
Dantrolene	Skeletal muscle relaxant	0.5-2mg/kg bid	Sedation Weakness Gastrointestinal upset Hepatotoxicity Pleural effusion
Prazosin	Smooth muscle relaxant	0.25-1mg bid or tid	Hypotension Sedation Weakness
Phenoxybenzamine	Smooth muscle relaxant	0.5-1mg/kg bid	Tachycardia Miosis Hypotension
Bethanechol	Increases detrusor tone	0.1mg/kg tid	Note concurrent urethral muscle relaxants must be used. Vomiting Diarrhoea Cramps Anorexia

Note : The author rarely uses Bethanechol due to the unwanted side effects

Key learning objectives

- Recognition and assessment of a tail pull injury and the need to give clear guidance and expectations to the owners from the outset of treating their cat.
- Enable you to formulate a thoughtful analgesia, bladder medication, bladder decompression plan for the cat affected with a tail pull injury
- Recognise the need for ongoing urinary voiding observations and to adjust the medical plan accordingly.

Useful reading references

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Feline urethral obstruction and rupture

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Summary

Feline urethral obstruction is usually due to feline lower urinary tract disease (FLUTD). Male cats are most commonly affected and the obstruction comprises of crystals, mucous, uroliths and urethral plugs either alone or in combination. Urethral spasm is always present during and after an obstruction is relieved and should never be underestimated in its ability to cause ongoing discomfort and signs of dysuria. Once a blocked cat has received analgesia and appropriate stabilisation, then sedation or preferably a short general anaesthesia should be performed to allow attempts at urethral catheterization. Anaesthesia allows a more complete relaxation of urethral musculature, so reducing the chance of iatrogenic urethral trauma by catheterization attempts.

The male feline urethra has mainly smooth muscle cranial to the prostate gland but mainly striated muscle surrounding the remainder of the penile urethra. Urethral spasm may be better responsive to skeletal muscle relaxants such as Dantrolene 0.5-2mg/kg bid.

The most ideal catheter for unblocking a urethral should have the following properties

- End hole
- Small diameter 3.5Fr
- Sufficiently strong ideally without a stylet

Hydropulsion remains the key to dislodging most urethral obstructions and this can take time and repeated flushing to be successful. The penile tip may be swollen, oedemaous, contused, so catheter introduction has to be done gently, once into the tip, try not to keep replacing – the obstruction should be relieved by flushing through the catheter not by the action of the catheter itself pushing the obstruction along the urethra.

Not all urethral catheters are kept as indwelling post obstruction relief, for instance those cats with a relatively easy mucosal plug to relieve and with perhaps a larger component of functional urethral spasm are less likely to benefit from a catheter sat against the urethral mucosa, no matter how refined the material of that catheter.

For indwelling catheters they are secured with 4 x skin sutures into the prepuce, 4/0 1.5m nylon. Intermittent drainage of a closed system can sometimes work better than a cat being attached to a continual drainage system, particularly if the cat is ambulatory. Open drainage is not favoured by the author, the patients quickly become urine soaked and the open ended catheter may act as a conduit for infection. Antibiotics are not currently recommended as prophylactic measure with the use of urethral catheters.

What are the options if we cannot pass the urethral catheter?

- Cystocentesis
- Cystostomy tube
- Time, analgesia, urethral relaxants and then try again to catheterize
- Perineal urethrostomy
- Pre pubic urethrostomy
- Euthanasia

Trauma to the urinary tract can arise due to various situations, the most common being a road traffic accident, but a blunt kick, a sudden frenzied biting attack or iatrogenic damage by passing a urethral catheter can have devastating consequences.

The urethra does have a capacity to heal within an average of 7 days if ;

- there is some urethral mucosal integrity
- if we can prevent further urine extravasation.

A patient with a urethral rupture may still be capable of passing some urine, do not assume these patients are voiding normally. If they have pelvic fractures, demonstrate some dysuria or stranguria and have a subtle oedema (urine extravasation) to their perineum you should investigate the intregrity of the urethra.

A pelvic or perineal urethral tear needs to heal around a catheter. This catheter can be placed retrograde or ultimately normograde via a cystotomy. If initially urethral catheterisation is not possible in a retrograde manner then a temporary cystostomy tube is placed to divert urine into a collection bag until a treatment plan can be finalized and the patient in a more stable condition. When urethral catheterization is not possible, surgery for pelvic urethral tears can be considered in the form of a pre–pubic urethrostomy, trans-pelvic urethrostomy or even a vaginourethroplasty. However, where the situation is complex such as a patient with a pelvic urethral tear that is debilitated with concurrent injuries, then euthanasia should also be discussed. Bite trauma to a perineal urethra could be resolved by a perineal urethrostomy providing there is no evidence of urethral stricture rostrally beyond the level of the caudal pelvic brim region. The prognosis for cats with traumatic urethral rupture is favorable, with well over 50% of cats with iatrogenic urethral rupture achieving a good clinical outcome.

Key learning objectives

- Really think about the delicate feline urethral anatomy
- How to relieve a urethral obstruction or manage a urethral rupture
- Importance of short term but ongoing analgesia and skeletal urethral muscle relaxants

Useful reading references

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Oesophageal FB: scope or operate

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Summary

Oesophageal foreign bodies can be one of the most satisfying clinical presentations to resolve and generally carries a good prognosis – that is providing the diagnosis is prompt and the necessary equipment is to hand to remove the foreign body effectively and safely.

Non-surgical removal is possible in 64–92% of cases: fish hooks have the lowest success rate. Endoscopic extraction requires a rigid or flexible endoscope capable of air insufflation. Monitor oxygen saturation, blood pressure and respiratory rate/depth in case insufflation causes tension pneumothorax if perforation occurs. To remove, try one of the following:

- Grasp with rigid forceps, gently rotate to free and orientate any sharp points caudally then withdraw
- Withdraw with basket forceps
- Withdraw with a balloon catheter positioned caudal to the foreign body
- Grasp fish hooks at the neck, gently free from the mucosa then withdraw close to and with the endoscope, ideally with the barb pointing caudally
- Alternatively, try to advance the object into the stomach and either leave to digest unless they cause clinical signs or if indigestible retrieve via gastrotomy
- After extraction, inspect the oesophageal lining for ulcers or perforation.

Surgical removal is indicated if:

- Withdrawal or advancement fails
- The foreign body has perforated the oesophagus
- The oesophagus or great vessels may be lacerated during non-surgical removal

Foreign bodies between the heart and diaphragm may be removable via gastrotomy. Otherwise, approach the oesophagus via one of the following:

- Ventral midline cervical approach
- Cranial median sternotomy
- Intercostal thoracotomy at the right 3rd to 5th or left 7th to 9th intercostal space.

Complications include oesophagitis, ischaemic necrosis, dehiscence, leakage, infection, fistula, stricture formation and fatal perforation of the aorta or pulmonary artery by the foreign body during removal.

Key learning objectives

- Understanding of the options and techniques for nonsurgical and surgical removal of oesophageal foreign bodies.
- Understanding of when surgical removal is required
- Knowledge of the main potential complications of oesophageal foreign body removal.

Useful reading references

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- Binvel M et al JSAP 2017 (59) 45-49
  Endoscopic and surgical removal of oesophageal and gastric fishhook foreign bodies in 33 animals
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  Removal of oesophageal foreign bodies comparison between oesophagoscopy and oesophagotomy in 39 dogs.
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- Rousseau A et al JVetECC 17 (2) 159-163
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  Oesophageal foreign bodies in dogs : 34 cases

Supporting the trauma patient

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Summary

There are many ways we can assist a patient that has been involved in a traumatic accident. During this lecture we will look at 3 aspects: thoracic drainage, enteral feeding and open wound management.

Thoracic drainage

Trauma to the thorax can cause a haemo-pneumothorax due to damage to intrathoracic organs, particularly lung laceration, and/or damage to the thoracic wall. Pneumothorax may also occur without trauma (spontaneous pneumothorax) due to pulmonary bullae/blebs or other pulmonary disease (pneumonia/tumour). Haemothorax may occur without trauma due to coagulopathy (particularly rodenticide intoxication).

In patients presenting after trauma, the findings of thoracic auscultation will generally be abnormal in the presence of pleural space disease. Lung and heart sounds sound more dull and distant than expected for the degree of respiratory effort. Needle thoracocentesis is generally a very safe procedure and can be performed prior to thoracic radiography as both a diagnostic and therapeutic step. It is much safer to radiograph a patient after improving their respiratory function by thoracocentesis, rather than restraining a severely dyspnoeic animal for radiography.

Thoracocentesis (repeated if necessary) alone normally provides adequate management of traumatic pneumothorax. Most traumatic lung lacerations will seal spontaneously within 48 hours without the need for a thoracic drain or surgery. A thoracic drain is indicated if needle thoracocentesis is required on more than 2-3 occasions, or if it is not possible to remove all of the air from the pleural space (continuous leak – consistent with a tension pneumothorax).

Traditional thoracic drain placement can be performed using a “non-surgical” trochar technique or a surgical technique. More recently, thoracic drains placed by Seldinger “over-the-wire” technique have become widely used. These drains are smaller diameter than traditional thoracic drains, and so are suitable for air and non-viscous fluids. They are much more easily placed and better tolerated than larger drains. Thoracic drains can be drained intermittently, connected to a one-way valve (Heimlich valve) for patients over 5kg or connected to a continuous drainage system (3-bottle system). Patients with thoracic drains in place require close monitoring to ensure detection of any complications relating to the drain (e.g. patient interference leading to open pneumothorax).

Surgery is rarely required for traumatic pneumothorax and is reserved for patients where air continues to accumulate several days after trauma. In contrast, surgery is almost always required for spontaneous pneumothorax.

Significant bleeding into the thoracic cavity will generally be detected because of its cardiovascular effects alongside respiratory compromise. Haemothorax following trauma is generally mild and does not require surgical treatment. Where haemorrhage is severe, it may well be fatal before surgical intervention can be attempted. If severe haemorrhage in the thoracic cavity is identified post-trauma then early median sternotomy following resuscitation with crystalloid fluids/blood would be indicated to arrest the bleeding.

Thoracocentesis tips:

- Equipment: Butterfly needle, 3-way tap, syringe or a quick aspirating device with a one way valve, kidney dish
- The preferred site for thoracocentesis is between the seventh and the ninth intercostal space (IS).
- If fluid and air are present in the pleural cavity, the needle is inserted approximately halfway up the thoracic wall. If only fluid is present, the needle should be inserted in the ventral third of the chest wall or in the dorsal third if it is a pure pneumothorax.
- To avoid the intercostal vessels and nerve, lying on the caudal aspect of each rib, the needle should be introduced close to the cranial costal border. To avoid pulmonary trauma, the needle should be inserted at a 45-degree angle. Once the needle is carefully advanced in the thoracic cavity, with the bevel facing the lung, an assistant can aspirate the fluid or air.

Thoracic drain placement tips:

- The use of small bore wire guided chest drains are an alternative to larger trocar drains. These are 14-gauge polyurethane, 20 cm long (or 12-gauge, 30 cm long) multi-fenestrated thoracostomy drains placed using a modified Seldinger technique.
- Placement of small-bore chest drains using the Seldinger technique in veterinary patients is usually possible in compromised conscious patients or with the use of additional local anaesthesia.
- Guide wire drains are not skin tunnelled prior to thoracic cavity insertion
- Guide wire drains come with secure clips that are sutured to the skin, no need for a finger trap suture
- Always know the location / number of the fenestrations, so that all drain fenestration are within the thoracic cavity

Enteral feeding

These accessories often provide a vital lifeline as a means to supply nutrition in severely traumatised patients. Of particular relevance to those small breed dogs and cats with multiple or extensive injuries necessitating higher doses of analgesia or frequent sedations and dressing changes, essential management, but not inductive to resuming a patients appetite. The general rule is to feed from as high up the tract as possible to facilitate the usual physiological processes of digestion. Naso-oesophageal tubes whilst useful for patients that are not stable for anaesthesia, tend to block easily, do not stay patent or in place for more than a few days and generally are unpleasant for the patient. The authors preference in most patients is therefore an oesophagostomy tube.

Equipment:

- Silicone oesophagostomy tube. Long enough to reach from the mid-neck region to the heart base with at least 10 cm extra
- Cats + small dogs <10kg : 14Fg   Dogs 15kg + : 20Fg
- No. 11 scalpel blade
- Long-handled curved haemostats
- Non-absorbable suture material (my preference 2/0   polypropylene)
- Povidone–iodine impregnated gauze (Inadine)
- Sterile absorbent dressing with adhesive border and stockinette (Surgifix)

Oesophagstomy tube placement:

- The patient is anaesthetised and placed in right lateral recumbancy
- The left side of the neck is clipped and prepared as for surgery – from the angle of the jaw to the front of the shoulder
- The tube is premeasured and marked at the correct insertion length
- ☐An assistant passes the closed forceps into the mouth of the patient with the curved tip pointing upwards
- The tip should be advanced down the oesophagus to the mid region of the neck and then angled upwards so that the point is evident under the skin. The jugular vein and carotid artery should be avoided
- Keeping upwards pressure on the forceps, the clinician incises through the skin down on to the tip of the forceps – just an incision large enough to accommodate the tube
- The tip of the forceps is pushed through the incision and the forceps jaw opened. The distal tip of the oesophagostomy tube is captured in the tip of the forceps
- The forceps are drawn back into the oral cavity with the tube
- The tip of the forceps and tube are advanced down the oesophagus, past the incision until the premeasured mark on the tube is at the level of the skin incision
- Tube position is checked by thoracic radiography
- The tube is sutured in place using a finger-trap suture Placing the iodine dressing around the incision reduces stoma site infections.

Open wound management (OWM)

The power of OWM never ceases to amaze. It is the authors belief that given support in the form of creating the most appropriate environment dependent on the stage of wound healing that pretty much all wounds eventually heal without definitive or extravagant surgical techniques. Keeping things the treatment plan simple, developing the plan as the wound bed changes, involving the owners in the treatment and managing their expectations from the outset is the key to success.

Analgesia, lavage and debridement still remain the first line approach and it is key to remember that the point of lavage is to remove debris, necrotic material and to reduce bacterial load. Once a wound bed starts to visually improve, often within a few days, then further lavage has little purpose and a conscious patient often is not tolerant of this sensation. Recommendation of optimal lavage solutions have been plentiful over the years, often without much scientific or in vivo data. Recent data from 300 human triage patients found that the use of tap water on traumatic lacerations did not increase the rate of infection when compared to the use of sterile saline. The implication of this study and the anecdoctal practice of the author being that perhaps we can confidently use tap water lavage for our veterinary patients.

We are fortunate, yet perhaps over whelmed with the choice of topical products available for OWM. There are a number of products, albeit used historically that have come back into popularity and with good reason, they are locally anti-microbial and assist wound healing. The advent of different dressing formulations of Manuka honey, silver, alginates and hydrocolloids means that with a bit of imagination we can invent a dressing plan to suit most wound bed locations. Is there a need to take a swab for bacteriology culture and sensitivity of traumatic wounds at initial presentation? Typically not, by the time you have your swab result, it is out dated when compared to the progression of wound healing. The initial lavage, debridement, potential use of wet to dry dressings and then topical products are often well on the way to making a marked improvement in the wound bed state. It is rare the author uses any systemic antibiotics for patient with a traumatic wound bed as the topical products with antimicrobial properties are usually more than capable of dealing with any infection. The use of systemic antibiotics increases the chances of a multi resistant drug resistant (MDR) bacteria taking hold on a wound bed. In a recent study 35% of dogs in the above scenario developed an MDR bacteria. The author advocates taking a swab for bacterial culture if the wound bed is deteriorating or static, if the patient is becoming unwell or if the patient has been on previous antibiotic courses prior to your command of the case.

There will of course be times during the healing process when wound beds become static and we have to explore other options to stimulate and kick start the healing process. For some wound beds, this is merely a change in topical product or allowing more oxygen to circulate. The use of lasers, negative pressure wound therapy and punch grafting are amongst the techniques that are more readily available in our veterinary practices for those stubborn wound beds. Negative pressure wound therapy can also be considered as a more first line approach for extensive and deep cavity wounds.

Key learning objectives

- Know your vital accessories
- Recognise how best to manage them
- Feel confident with open wound management

Useful reading references

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Surgical haemorrhage: arrest the bleed

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Summary

When faced with surgical haemorrhage, it is up to you to resolve the problem in a safe and efficient manner, such that you know 100% you have control and are not concerned about on going blood loss through poor haemostatic techniques. The risks and complications associated with operative bleeding are reduced through preoperative assessment to identify bleeding disorders. Patients should go into surgery with ideally a normal coagulation profile and optimal PCV. Alarm bells should ring for those surgical procedures that are likely to cause haemorrhage and extra preparations made accordingly.

Methods of intraoperative haemostasis

- **Pressure**
- **Vasconstrictors** such as topical adrenaline use.
- **Ligatures and Torniquets**
- **Disposable vascular clips** are also available. The clips are employed for the occlusion of blood vessels. The advantage of using vascular clips is the speed of the application.The application of multiple clips is recommended during closure of a blood vessel of significant size. Before applying vascular clips, the vessel should be well isolated from the surrounding tissues, with the vessel inside the limits of the clip, so that the clip locks into itself. In addition, positioning of the clip should leave sufficient vascular segment length to prevent any slipping once the vessel has been severed.
- **Surgical staplers** which deliver staggered rows of staples, such a the thoracoabdominal (TA) stapler or the automated ligating dividing stapler (LDS) which can be useful for splenectomies. The use of an LDS on the large splenic artery and vein still though warrants caution.
- **Monopolar and bipolar electrocautery** can be used for haemostasis. Monopolar electrocautery, however, should be used with caution, as the current can pass to tissues out of the visual field, leading to iatrogenic thermic injury. Bipolar electrocautery is safer because it is usually of lower voltage and the electrical current is passed only between the tips of the bipolar forceps used.
- **Vessel sealing devices** are a more recent addition to some of our veterinary practices.These devices simultaneous seal and cut tissue. They work through pressure exerted on tissue when the tissue is crushed between the jaws of the device, followed by application of diathermy or ultrasonic energy (harmonic techno-logy) to the tissue. This process leads to denaturation of collagen molecules in the vessel wall, with permanent sealing. It has been demonstrated that the use of vessel sealing devices can significantly decrease surgical time and surgical bleeding, therefore shortening anaesthesia duration. Two bipolar diathermic sealing devices in the UK are the LigaSure and the Enseal. A second-generation LigaSure device is known as the Force Triad. Both devices have tips that are indicated to seal veins up to 7 mm in diameter. The harmonic sealing devices are indicated to seal vessels up to 5 mm in diameter. Although sealing devices are safe, care must be taken when they are used in proximity to neurovascular or other vital structures.

- **Topical haemostatic agents** are used in surgical procedures as an adjunct to help control capillary bleeding and oozing haemorrhages when ligation or other conventional methods of control are not practical. Topical haemostatic agents include absorbable gelatin/collagen powder or sponges, oxidized regenerated cellulose polymer and the newer fibrin sealants. Collagen and cellulose are biocompatible materials that can be absorbed by the body within approximately 3 weeks as a result of phagocytosis and enzymatic degradation. Collagen provides faster haemostasis than oxidised cellulose and can be combined with antibiotics.
- **Autotransfusion**
- **Anti-fibrinolytic agents** : Tranexamic acid and E-aminocapric acid

Key learning objectives

- Consider the risk factors to look for pre operatively, the patient and procedure factors, enabling you to anticipate a complication
- Decision making to improve a patients ability to clot or abort an elective procedure
- Know your haemostasis options and be confident in their usage

Useful reading references

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Distinguishing between hypomotility and obstruction in the inappetant rabbit

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Introduction

Inappetence in rabbits is a common presentation with several possible differential diagnoses. As some of these differentials have vastly different pathophysiologies, and therefore treatments, it is important to be able to differentiate between the main gastrointestinal causes of hypomotility and intestinal obstruction early in the diagnostic process. This paper will address modalities for diagnostics and treatment plans in these two causes of inappetence.

Pathophysiology

Hypomotile inappetence in rabbits is often a secondary condition, though identifying the primary disease process can be challenging. Environmental and husbandry factors such as stress, ambient temperature, interaction with conspecifics and diet can all contribute to the development of gastrointestinal hypomotility in rabbits. Equally, pathological processes such as dental disease, traumatic injury, diarrhoea, and renal disease are also common causes.[1] It is still important to treat the hypomotility whilst diagnosing and treating the primary cause. For dental-related problems, a dental float under general anaesthetic will often be required to allow the patient to eat normally, and so a calculated decision must be made as to when is most appropriate to anesthetize the unwell patient. The patient may need a period of hospitalization prior to any dental work being done.[2]

The differential diagnoses for gastrointestinal obstruction are much simpler. The most common cause is a trichobezoar[3]. Rabbits are fastidious groomers and many pet rabbits are heavy seasonal shedders. As rabbits are unable to vomit due to the structure of their cardiac sphincters, ingested hair cannot be regurgitated and must pass through the gastrointestinal tract.[4] Trichobezoars commonly consist of fur and ingesta but may also include ingested fibres from the environment such as carpet or clothing fibres. Other causes include true foreign bodies such as seeds and small pebbles, or masses of neoplastic or parasitic origin. Enterotomies will negatively impact the prognosis of the patient and a conversation with the owner should be held regarding prognosis and quality of life, if indicated. Trichobezoars most commonly lodge in the proximal duodenum or the ileocaecal junction.

History-taking

The owner must be relied on to report inappetence in their pets, and so it is important to educate owners on the severity of inappetence during routine consultations. If owners are unsure if their rabbit is inappetent, a “treat food test” has been a reliable indicator of appetite for the author. The owner can offer the rabbit a highly palatable treat food that the rabbit has preferred in the past; if the rabbit eats it normally, then the situation may be considered less urgent; however veterinary attention should still be sought.

The history can help to elucidate the primary cause of inappetence. If the rabbit has recently undergone a stressful event, or if there are potentially other pathological signs such as polyuria and polydipsia, this can help guide the diagnostic process. Rabbits that are moulting their haircoat heavily are at an increased risk for both hypomotility and obstruction.

Presenting signs

Inappetent rabbits will commonly present with some degree of dehydration. They will often have a hunched posture, and be reluctant to move around. Most healthy rabbits will be curious about the clinical environment and display behaviours such as sniffing and ear cocking when first removed from the pet carrier. In contrast unwell rabbits will generally be apathetic despite being in a new environment.

The most important aspect of the physical exam in an inappetent rabbit is the abdominal palpation. It will be difficult to identify individual structures of the gastrointestinal tract in the healthy, appetent, and well hydrated rabbit. In dehydrated animals, the stomach and caecum will often be firmer to the touch, and have a texture similar to Play-Doh. Inappetent patients often have reduced or absent gut sounds on auscultation. However, this must be interpreted with caution as many healthy rabbits that come in for routine vaccination will have reduced gut sounds due to the stress of travel and being in an unfamiliar environment.

In the hypomotile patient, the gastrointestinal tract will have reduced fill, and the stomach and caecum will be easier to identify due to dehydration of their contents. In obstructed patients, the stomach will often be firm and tympanic and the patient will resent palpation. In both healthy and hypomotile rabbits, the author has found that the stomach can often be comfortably palpated bilaterally to the point where the tip of the contralateral finger can be felt through the body and stomach wall. This is not possible in the obstructed rabbit.

The obstructed rabbit may not always be straightforward to diagnose on physical exam. A rabbit in the earlier stages of obstruction may not present with the classical gastric dilation, particularly if the obstruction is distal. However, a rabbit with a proximal duodenal or pyloric obstruction should present with severe gastric dilation.

Diagnostics

Whilst it is best to obtain as much information on the patient as possible, there may not be enough time or resources to obtain a full range of diagnostics. The author has found that the most helpful modalities in distinguishing between hypomotility and obstruction in the rabbit are blood glucose and abdominal radiographs. Both diagnostic options are also quick and easy to perform. Blood glucose readings can be obtained using a portable glucometer, and the blood sample can be obtained via the marginal auricular vein, the cephalic vein, or the lateral saphenous vein. A blood glucose of >20 mmol/L has been associated with poorer prognostic outcomes. In one study, rabbits with a gastrointestinal obstruction had a mean blood glucose reading of 24.7 mmol/L, whereas hypomotile patients had a mean reading of just 8.5 mmol/L.[5]

Abdominal radiographs are useful for assessing the size and contents of the gastrointestinal tract.[6] Rabbits suffering from hypomotility will have a smaller stomach that rarely extends ventrally beyond the level of the sternum. The contents often appear to be a heterogenous soft tissue opacity with a surrounding ring of gas. There may be small pockets of gas within the small intestine, rather than an obstructive pattern with dilated continuous loops of intestine. In the obstructed patient, the stomach is commonly filled with a homogenous soft tissue opacity with a bubble of gas, rather than a ring. The size will be markedly larger, and may extend ventrally to touch the body wall. In severe cases, the stomach may push the body wall ventrally.

Abdominal radiographs that indicate free gas in the abdomen often indicate a gastrointestinal rupture. This will be corroborated by a patient with a severely depressed demeanour that is painful on abdominal palpation. Unfortunately, euthanasia is indicated for these patients.

Treatment

The treatment options for hypomotility and obstruction are fundamentally different; however it is vitally important in both situations that the patient is aggressively rehydrated. A lateral auricular vein intravenous catheter is generally easy to place and well tolerated by most patients. Catheters in the cephalic and saphenous veins can also be placed if the auricular vein is not suitable, such as in Netherland Dwarf rabbits that have short pinnae. A 24-gauge catheter is suitable for the majority of rabbits and the fluid rates that they will require. Rates of up to 4x surgical rate (10 ml/kg/hr) can be used initially, but frequent monitoring of breath sounds and hydration levels is necessary. A lignocaine or lignocaine-fentanyl constant rate infusion is useful in managing visceral pain, and should be considered for any painful hypomotile or obstructive patient.

In hypomotile patients, the main focus is to restore normal gastrointestinal tract motility and correct any underlying or primary conditions that may be contributing to the episode of stasis. In patients that are not admitted to hospital, subcutaneous fluids can be administered. It is important that the fluids are warmed to body temperature. The author's preference is to use 60 ml/kg of warmed Lactated Ringer's solution. Rabbits generally tolerate the administration of subcutaneous fluids well. Due to the large volumes being administered, it is preferable to use an 18-gauge needle to reduce the administration time. Oral syringe feeding is also an integral part of treatment, and a commercial preparation such as Oxbow Critical Care or Burgess Excel Dual Care can be used at a rate of 5 ml/kg/2 hours for the completely inappetent rabbit. For patients that are starting to eat independently, the rate and frequency of the feeding can be decreased until the patient is eating 100% of their normal without assistance.

Medical therapy is also useful in encouraging the gastrointestinal tract to return to normal. Prokinetic agents are commonly and successfully used. Ranitidine at a rate of 2-5 mg/kg BID orally has a dual effect of enhancing motility as well as being a H_2 blocker, and is the author's main prokinetic drug of choice. Metoclopramide is commonly stocked by veterinary clinics, but the general consensus in the exotics community is that it has a low efficacy as a prokinetic in rabbits. Cisapride appears to be a more powerful prokinetic agent at a rate of 0.5 mg/kg orally every 8 hours, but concerns have been raised regarding the necessity of administering cisapride when the patient is already on ranitidine, especially given the potential of cardiac side effects.[7] The author is comfortable using cisapride judiciously in more severely affected rabbits with hypomotility. Pain relief with agents such as meloxicam (0.5 mg/kg BID orally) and tramadol (10 mg/kg BID orally) is an important part of the treatment process, but the risks of side effects with these drugs must be accounted for. The renal side effects of meloxicam do not appear to be as severe in rabbits as it is in dogs and cats, but it is still recommended to ensure the patient is euhydrated prior to administration.[8] Opioids can be used prior to achieving an adequate hydration status, but the hypomotility effects of opioids must be taken into account, and it is preferable to switch to meloxicam once the patient is normally hydrated.[9] A single dose of buprenorphine can be administered to the patient instead of oral tramadol. At this stage, the efficacy of tramadol as an analgesic in rabbits has not been formally studied as it has in canines.

Surgical exploratory laparotomy to relieve gastric dilation by milking the obstruction distally into the caecum or rectum is frequently the treatment of choice for obstructed rabbits. Medical management with aggressive fluid therapy, opioid-based pain relief, and attempted gastric decompression with a nasogastric tube can be attempted, but progression to surgery must always be kept as an option. The size of the nasogastric tube often precludes successful decompression, and in many cases the tube will continuously block with ingesta and fur. Some obstructions may gradually pass through the gastrointestinal tract, and the patient will often display a waxing and waning pattern of depressed demeanour and abdominal pain. Sequential radiographs to monitor the gastrointestinal tract are an important factor in managing these cases medically.

Exploratory laparotomy in the rabbit is similar to that in a dog or cat. Gentle tissue handling is of utmost importance. It is usually easiest and least traumatic to exteriorize the enlarged stomach first, as it will be acting as a space-occupying lesion in the abdomen. After the stomach is exteriorized, the rest of the gastrointestinal tract including the caecum should be exteriorized. The obstruction can then be located by examining the tract visually and by palpation. Trichobezoars may be gently compressed and milked through to the caecum or rectum. Care must be taken not to overly traumatize the intestine and mesentery. It may be beneficial to use heparinised saline for flushing and moistening the gastrointestinal tract and abdominal cavity during surgery to reduce the risk of adhesions; however the efficacy of this in rabbits has not been formally studied.[10] For ingested foreign bodies, a decision needs to be made intra-operatively as to the location of the enterotomy. In some cases, it is preferable to milk the foreign body into the stomach, then remove it via a gastrotomy. It is difficult to omentalize enterotomies due to the small size of the rabbit omentum. It is best to avoid incising into the gastrointestinal tract when possible. Obstructions due to neoplasia or masses in the gastrointestinal tract are surgically challenging. Resection and anastomosis is possible, but drastically reduce the patient's prognosis. Post-operative monitoring for pain and evidence of failure of the anastomosis is paramount. In patients with traumatized gastrointestinal tissue, IV metronidazole during surgery and post-operative hospital care can be useful. The patient can be switched to oral metronidazole after discharge.

Post-operative rabbits should be kept hospitalized until eating and defecating in small amounts. Prokinetics can be used carefully after surgery to promote return to normal gastrointestinal function. Syringe feeding can be delayed until some gastric emptying has been achieved. Non-steroidal anti-inflammatories and opioids can be used in combination during the recovery period to achieve multimodal analgesia.

Prognosis

Rabbits suffering from hypomotility of the gastrointestinal tract generally have a good prognosis with appropriate treatment, as long as the underlying cause does not preclude recovery. It is expected that these patients will eat and defecate normally within 72 hours of initiating treatment.

The prognosis of rabbits after an obstructive lesion is much more varied. Rabbits with minimal gastrointestinal trauma and that recover well after surgery tend to have a good prognosis, as do rabbits that are able to pass an obstruction with medical therapy. Rabbits with gastrointestinal trauma as evidenced by erythema and discolouration of tissue in surgery can have a variable prognosis, with some patients recovering without further incident, and some patients passing away up to 14 days post-surgery. Repeated diagnostics and radiographs can help monitor the progress of a patient.

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Practical Aspects of Sperm Morphology & Motility

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Introduction

Assessment of sperm morphology and motility are critical components of the bull fertility examination. Sperm motility predicts the ability of the sperm to travel to the egg and sperm morphology predicts the ability of the sperm to fertilise the egg. These parameters can reflect inherent poor sperm quality, disease, environmental or nutritional stressors but may also be influenced by collection, handling storage and measurement techniques.

Sperm motility

The ACV uses a scale of 1-5 to describe the range in gross motility (mass activity) from no swirl to fast distinct swirl with continuous dark waves. Mass activity is dependent on 3 factors: concentration, % of progressively motile sperm and speed of progression. By example, semen of fair concentration but 80% progressive motility may have no wave motion, whereas, highly concentrated sperm may only have 50% motile sperm and have a slow wave motion. There is not a good relationship of mass activity to fertility at natural service. It is more of a screening test to describe whether a representative sample was collected.

Sperm are immotile in the cauda epididymis. They are dependent on the pH, temperature, osmotic balance and adequate ions and nutrients in seminal fluid to become motile. Seminal fluids do not maintain sperm viability for very long and sperm quickly lose their vigour of forward progression. Motility is highly sensitive to time, temperature and pH. pH < 6.5 and >8 results in suboptimal motility of bull sperm (Contri, 2013)

Progressive motility describes the % of individual sperm progressing forward. Definitions of the minimum velocity of progression have varied from one sperm head length to one full sperm body length per second. Additionally, reports of bull sperm head length again vary from 8 to 14 μm (Gravance 1998, Ciftci 2010).

Progressive motility may be approximated subjectively using a microscope or using an in lab CASA (Computer Assisted Semen Analysis) machine or infield using a portable iSperm semen analyser. Microscopy requires the dilution of most samples to be able to visualise individual sperm. iSperm recommends the dilution of concentrated samples when >500 million sperm/ml. Dilution presents challenges. Although physiological saline is osmotically balanced, it is not a suitable diluent, as the pH may be as low as 5.5. Phosphate buffered saline, does not contain anti-microbials or nutrients and must be stored carefully once made up and sperm assessed rapidly once semen is diluted. Semen extenders are the ideal diluent for motility assessment. Regardless of diluent type, semen, diluent and equipment in contact with semen must be maintained before and during assessment at 37C.

As semen is not 100% uniform, accuracy of motility calculations will increase with repeated tests. Repeated slides will increase accuracy of the subjective assessment to a point where the motility decreases due to reduced sperm viability. Laboratory CASA machines vary between 3 and 5 sample chambers measured simultaneously. iSperm uses a single chamber, but up to 4 areas can be assayed and automatically averaged from the single chamber in minimal time to increase accuracy.

Sperm morphology

Sperm morphology is performed using a DIC microscope at 1000x magnification on a preserved sample or by CASA machines. Similar to motility, sperm morphology may be influenced by collection and handling. Collection technique may be lacking, and the sample not representative of a true ejaculate. Samples of pre-ejaculate will contain “stale” /”rusty” sperm with increased loose heads and swollen acrosomes. Samples where preservation has been delayed post collection may also exhibit high number of loose heads and swollen acrosomes. Fresh samples collected at 37C and then preserved into cold preservative will experience “cold shock”, which may results in reflex tails or mid pieces without droplets. Bacteria, white blood cells, urine and red blood cells may contaminate the samples, changing the pH and osmolarity, resulting in swollen acrosomes. Unbuffered or osmotically unbalanced preservative can also result in swollen acrosomes and reflex tails may be seen in hypotonic mediums. Some samples are too concentrated and sperm clump together making it difficult to view sperm in their entirety.

Interpretation needs to be in light of technique limitations. Microscopic evaluation of morphology is limited by counts of 100 or 200 sperm, which is a very small subset of an ejaculate. CASA machines tend to count larger numbers of sperm but are limited in their ability to detect vacuoles in the same manner as DIC microscopy. Only sperm heads or whole sperm with heads are counted in the spermiogram, as a result, loose heads in a sample can falsely decrease the count of non- head defects such as proximal droplets, coiled tails and midpiece defects. Acrosome defects may also hide small apical vacuoles. Sperm with multiple >4 defects will be classified as teratoid and may not accurately represent individual categories.

Sperm may have multiple defects and the resulting category percentages are not mutually exclusive. Common examples are proximal droplets and pyriform heads, swollen acrosomes and loose heads, large vacuoles and midpiece defects. The sum of individual defect percentages will often exceed 100.

Spermatogenesis, the process of replication and development of spermatogonia to spermatozoa takes 60 days and the spermatozoa then pass through the epididymis for a further 10 days. Cells differ in their susceptibility to insult depending on their stage of development at the time of insult. Thus defects may appear in semen in overlapping stages, up to 70 days post a single insult (and even longer if insult has been ongoing). A single sample only represents one point in time. Hence all bulls which have unsatisfactory morphology should be retested after 2 months to assess if defects are transient.

Current thresholds as per Bull Reporter are loosely based on compensable vs non compensable defects. Defects that are considered to be compensable can be compensated for by the addition of more sperm. These are defects which will affect the sperms ability to reach the egg such as midpiece defects, tails and loose heads and acrosome defects that will prevent the sperms ability to start the binding reaction at the egg’s zona pellucida. Thresholds of 30% are allowed for these defects. Non compensable defects cannot be compensated for by the addition of more sperm and only 20% are allowed. These defects may bind and start the egg reaction but fail to result in viable embryo such as vacuoles. The 20% threshold also applied to proximal droplets (PDs). Presence of PDs indicates failure of their removal by the epididymis and when >20% PDs are associated with reduced binding of all the sperm to the egg. There is some crossover with these categories, for example, a teratoid sperm may not reach the egg and therefore could be considered compensable. The acceptable levels for compensable sperm should also consider the concentration and volume of the ejaculate/ straw to calculate the total number of sperm. Percent normal sperm >70 % is associated with increased fertility of female and male offspring, therefore, the bulls purpose as a terminal sire vs stud sire should be considered. Single sire or multi sire natural mating or collection also come into consideration when making recommendations based on motility and morphology results.

Conclusion

Sperm motility and morphology values are 2 of the most valuable indicators of male fertility. They need to be considered with an understanding of their significance and limitations.

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Animal Welfare and Farmer Depression and Anxiety:

How to broach the elephant on the farm?

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Introduction

It is not uncommon when called out to a sick animal or to do a post mortem, that while looking over the whole flock or herd you wonder if this is only the tip of the iceberg. Most large animal vets are competent in progressing from dealing with one animal to discussing the whole flock, or to the whole farm; but what about recognising the farmer's mental health and its implications? What if they are suffering from depression or anxiety?

Background

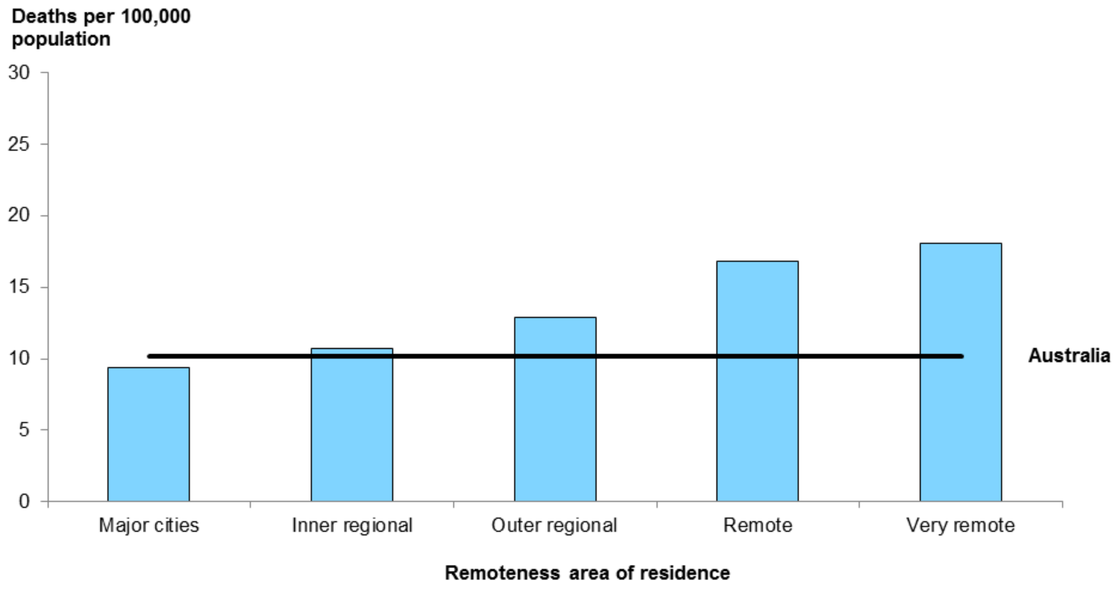
Decision making is well recognised as one of the hardest tasks when depression hits. Asking for help is another. It is easy to believe that even if you give the correct advice, farmers who may be suffering from depression may not act on it, and this is not just a possibility but a probability.

It may be best in this situation to give advice in small chunks. For example you may have diagnosed a parasite issue in the animals, then let's treat this and then look at what to do next. It would not be great in such a case to leave with recommendations for a full Faecal Egg Count Reduction Test (FECRT), reducing stocking rate, altering grazing management, weaning early and introducing a new feeding regime. In the case where the farmer can barely think of one thing at a time, such a suite of advice, however excellent, will not be acted upon. It may be preferable to step them through these stages slowly, one at a time. Alleviate the animal welfare issue first, then work towards preventing it recurring. Hopefully it will be easier for the farmer to deal with if they know the crisis is at least over for the time being, removing one major stressor for them. This may require you to be the proactive one and follow up to see how they are managing each of the steps and leading them down the path to the next one.

Obviously, depression is complex. Whilst stressors like their animals' welfare will of course contribute, clinical depression occurs due to a whole range of factors including the predisposition of the individual, and an accumulation of many long and short term stressors and bad experiences.

In farmers depression is fuelled by isolation on the farm (71% of farmers do not employ anyone[1]), and with the remoteness of the person's place of residence[2]. Other factors such as loss of respect in the general community for farmers and the continual cost price squeeze also obviously contribute. Without doubt this is further exacerbated when a crisis occurs such as drought, floods, fires or disease outbreaks.

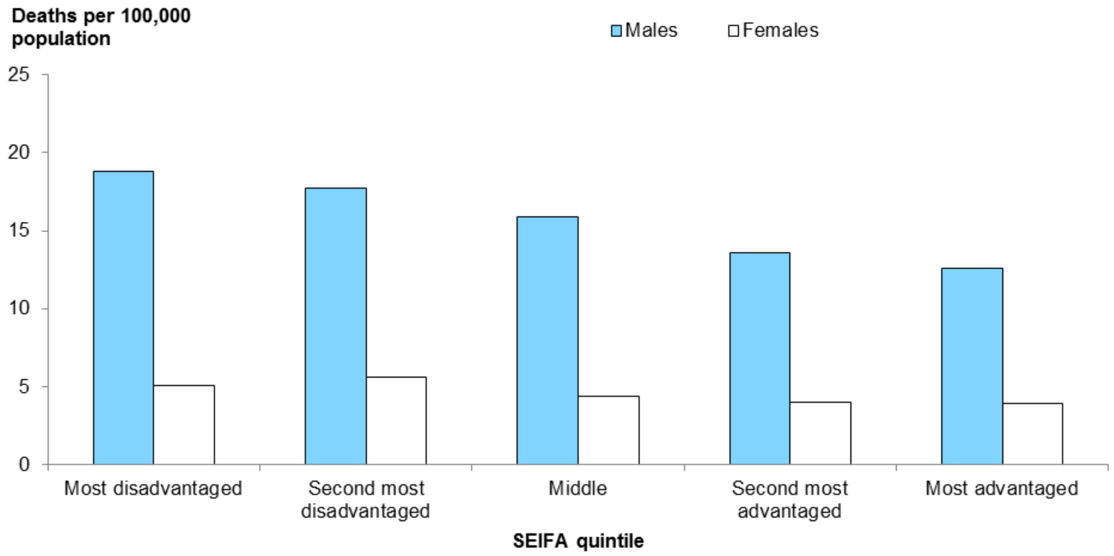
The reluctance of politicians and government workers to even call a drought or acknowledge the impact of climate change adds further frustration. By using terms such as "unseasonal dry conditions" this downplays the issue and can make farmers feel alone in dealing with the severity of these problems on their farm.



Source: AIHW National Mortality Database.

Fig 1. Suicide rates, by remoteness area of residence, Australia, 2010–11

Farmers are often asset rich and income poor. Farm income is notoriously complex to measure and hence to compare to other groups, but there is no doubt that the huge extra cost of feed in a drought or other crisis, coupled with a drop in production and often the lower value of animals is a large drain on income and cash reserves. There is also a correlation between rates of depression and suicide and Socio-Economic Indexes for Areas (SEIFA) so no doubt the financial pressures *per se* let alone the relentlessly depressing feeding of hungry livestock plays a huge role as well in stressors contributing towards depression.



Source: AIHW National Mortality Database.

Fig. 2 Suicide rates, by SEIFA quintiles, by sex, Australia, 2010–11

There is also an issue with farmers being able to access help with mental health issues when required. The reported prevalence of mental illness in rural and remote Australia appears similar to that of major cities. However, access for farmers to mental health services is substantially more limited than in major cities[4]. The National Rural Health Alliance quotes figures reflected in Medicare data when compared with major cities, per capita, Medicare expenditure on mental health services in rural and remote areas in 2015-16 was, respectively, 74 per cent and 21 per cent.[5]

Approach

As difficult as it may be to broach such a personal issue with the farmer it is important that we recognise the high incidence of farmer depression and suicide, consider if there are any issues with the farmer you are dealing with, and try to broach the subject if you think there is a problem. Even if it starts with the question RUOK.

What's the worst that can happen if we do try and discuss this with the farmer? There is good evidence that discussing suicide does not increase the likelihood of it occurring so we can't really make matters worse by being aware and caring. It may help to relate the state of mental illness to the illness of the animals. It's not the animal's fault they are ill and similarly mental illness is also an illness; it not the farmers fault, it is a medical condition. Like a cold it can be mild or it can develop into pneumonia and potentially kill you.

Recognising anxiety or depression in farmers can be helped by listening to the language that they are using. Markers for depression may include "If only I had done…" whereas language around anxiety is often based around "what if ….. happens". Obviously, anxiety and depression are not black and white but are a spectrum. Understanding these basic things to listen for might help trigger how we approach the discussions with the farmer.

As vets we are not professional mental health carers, nor are we expected to be. However we can try to make a difference in this space and help farmers with the difficult decisions before an animal welfare case develops. It may be that you are the one person that they have reached out to and that you might help them make that first step to receive help.

It is worth having knowledge of assistance that can be sought such as off farm labour, bale runners, Blaze Aid, financial advice, rural financial councillors and general councillor services. Carrying a pamphlet with a list of resources that might be available to the farmer might be well accepted and can be an easy way to lead the farmer into seeking assistance[8.] You should know your local hospital number which can then give you the number for your states Mental Health Hotline, your local council will be aware of local services in the area. If you are concerned for the immediate state of a farmer you can call the police and ask for a Welfare Check, which means the police will come and ensure that the farmer is safe.

Suicide call back service is available throughout Australia 1300 659 467, and other services such as Beyond Blue are also there to help.

There are also some excellent technical guides for drought feeding and management of beef cattle or of sheep available on line or in hard copies from Agriculture Victoria [6,7] or other similar resources in other states and it could be good to be familiar with these and even hand them out when developing plans for destocking and feeding, and to be aware of the issues arising during droughts.

Discussion

I believe there is an intrinsic link between the cycle of farmer's depression and anxiety and the health and welfare of the animals in their care, especially if there is a crisis such as drought. As a profession working in the animal health area we need to do all that we can to help understand this and work within the constraints this creates to resolve the problem we have been asked to answer.

Traditionally the role of animal welfare regulators is to police and prosecute animal welfare cases, and animals were often destroyed or sold on in the process. Whilst this rapidly resolves the animal welfare issues, it is surely not the best compassionate response and would do nothing to help the farmer when the issue is exacerbated and allowed to develop because of farmer depression. I think there is a huge role for experienced vets or livestock consultants to lead teams of volunteers that coud help farmers for a period of time while they take a much needed break, but equally important to make the essential tough decisions and do the tough tasks like going through and culling stock, and finally to develop and document a long term sustainable solution, or even to make the decision to leave the farm. Organisations such as Blaze aid already exist in agriculture and could become a model for this.

In doing this it could be possible to achieve rapid resolutions to animal welfare cases, but also to help the farmers who are battling with their own welfare issues as well.

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Dog Attack- Decision Making in the Critical Trauma Patient

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Introduction

Dog bites are common in veterinary practice with some studies showing they account for 2.5% of all canine cases presenting to general/primary care practices and 10-15% of all canine and feline trauma cases.

Dog bites and dog attacks may involve familial pets or non-familial pets. In the authors experience when injuries occur between members of the same household or family there will often be a recurrence unless the owners take steps to prevent or avoid the inciting conflict or situation. During the initial consult owners are often distraught and are generally not considering the long term repercussions of why the injury may have occurred and how to prevent recurrence. As part of the initial assessment and treatment, veterinarians should raise with the owners that they should consider what steps may need to be taken to prevent the pet being injured again in the future. In some situations awareness of the potential for recurrence may help owners decide between euthanasia and treatment of a severely injured pet, especially if the owners don't think they can prevent a recurrence.

Medicolegal considerations

When dog bites and dog attacks occur involving animals owned by non-family members, there are often medicolegal considerations that the veterinarian may need to take into account. The owners of injured dogs and cats should be guided to contact the local rangers and lodge a complaint or incident report. Within Australia the rangers are responsible for investigation and when required prosecution under the relevant dog acts.

Dog attack is well described legally. The following extract is from the 1976 WA dog act, and updated in 2017:

" In this Act, unless the context otherwise requires —

***attack**, in relation to the behaviour of a dog, does not include behaviour which was an immediate response to, and was induced by, provocation, but includes —*

(a) aggressively rushing at or harassing any person or animal; or

(b) biting, or otherwise causing physical injury to, a person or an animal; or

(c) tearing clothing on, or otherwise causing damage to the property of, the person attacked; or

(d) attempting to attack, or behaving in such a manner toward a person as would cause a reasonable person to fear physical injury,

unless the owner establishes that the behaviour was justified by a reasonable cause; "

Depending upon the state, the fines under the dog act can be quite high and some council rangers are becoming proactive in pursuing prosecution of the owners of attacking dogs. As professionals we have a responsibility to adequately document injuries so that there is sufficient evidence that irresponsible owners can be held to account for the injuries their dogs cause; in the hope this acts as a deterrent for future dog attacks. The owners of injured dogs may also elect to pursue civil claims against the owner of the attacking dog to reimburse them for the medical costs involved.

Below is an example for some of the potential fines that are legislated in WA for dog attacks:

"33D. Dog attacks etc.

(1) If a dog attacks or chases any person or animal and physical injury is caused to the person or animal that is attacked or chased, every person liable for the control of the dog commits an offence. Penalty:

(a) for an offence relating to a dangerous dog, a fine of $20 000, but the minimum penalty is a fine of $1 000;

(b) for an offence relating to a dog other than a dangerous dog, a fine of $10 000.

(2A) If a dog attacks or chases any person or animal without causing physical injury to the person or animal that is attacked or chased, every person liable for the control of the dog commits an offence. Penalty:

(a) for an offence relating to a dangerous dog, a fine of $10 000, but the minimum penalty is a fine of $500;

(b) for an offence relating to a dog other than a dangerous dog, a fine of $3 000."

Veterinarians should document all injuries and take photographs of both unclipped and clipped wounds to prove the injuries occurred and to highlight the severity if the case goes to court. Photographs are essential both in case of a report being requested by local rangers and also helpful to document the wounds in case there are later queries from the pets owner about why wounds may have broken down and required repeated surgery or later treatment.

When asked to write a report for council rangers the following format may provide a useful example written on your hospitals letterhead:

To whom it may concern,

My name is (*Veterinarians full name*) and I am registered veterinarian with the following qualifications (*BVSc/DVM etc*)

On the (*day*) of (*month), (year*) I was working at the (*Veterinary hospital name and address*) when I examined and treated a (*age), (sex*), *(breed*) (*species i.e. dog/cat*) known as (Pets *name*) and belonging to (*Owners name*) of (*owners address*).

In my professional opinion the injuries which (*Pets name*) sustained on the (*date*) are consistent with having been repeatedly bitten by a *small/medium/large sized dog(s). (select appropriate*)

Dog bite wounds are more severe under the skin than the superficial puncture wounds visible on the skin. Dog bite wounds are a mixture of penetrating wounds, crushing injuries and tearing injuries. The wounds which (*Pets name*) sustained were *mild/moderate/severe/life threatening*. (*select appropriate*)

The following wounds were found in the skin- *location, size and description of puncture wounds*.

During surgery to clean, explore and repair the wounds the following internal injuries were found:

1) *First injury*
2) *Secondary injury*
3) *3rd injury*
4) *Etc*

There is no possibility in my professional opinion that (*Pets name*)'s wounds could have been sustained from a motor vehicle accident or a stabbing injury such as a knife attack.

Pictures of (*pets name*), taken within a couple of the injuries and presentation to (*hospital name*) have been included below.

(*Pets name*)'s treatment at (*Vet hospital name*) included intravenous fluid therapy, intravenous antibiotics, pain relief, surgery, repeated bandage changes *etc*. The final cost to the owner of (*pets name*) was…………

Yours sincerely,

Dr……………………………………

Veterinary Surgeons Board Registration:………………………………………………………

General pathophysiology of dog bite wounds:

The force involved in dog bites has been reported to range between 150 to 450psi. What is externally visualized with dog bite wounds is classically described as 'the tip of the iceberg', with skin injuries often looking deceptively innocuous. The injuries occurring under the skin are nearly always much more severe than can be visualized externally and the area of deeper tissue injury is often significantly greater than the overlying wounds.

Bite wounds are a combination of puncturing, cutting/shearing, avulsing, and crushing injuries. Because of the mobility of dog and cats skin internal injuries may be at a distant location to the visible skin puncture wounds. Due to the elasticity and recoil of dog and cat skin, even without penetrating skin wounds, there is potential for deeper internal crushing, tearing, and avulsion injuries. Larger dogs will also cause injuries from shaking of smaller dogs and cats. Thoracic, abdominal, and cervical wounds are associated with a higher risk of mortality.

In some situations cutting/shearing injuries with canine teeth may produce tears in the skin and underlying tissue with minimal additional deep tissue devitalization.

Avulsion injuries occur when skin is compressed into the wound and the canine teeth rake through the deeper tissues and lift up the deeper tissues and organs. It can result in avulsion of internal organs from their blood supply (i.e. splenic, renal, gastrointestinal, and bladder avulsions), lifting up and tearing of muscle, and body wall herniations. The author has seen intestinal, splenic, and renal puncture wounds and avulsion injuries associated with a fairly innocuous looking body wall puncture wound.

Crushing/compressive injuries occur also in deeper tissues and may result in fractures, bleeding from internal organs such as the liver, spleen and kidneys, large areas of tissue ischemia and tissue necrosis, and also pneumothorax from lung injuries. In some situations these injuries can occur without a penetrating wound i.e. only skin bruises are present.

Avulsing and crushing injuries are at an increased risk of developing infection because of the disruptive forces/energy that the tissues have been exposed to.

Dog bite wounds are generally contaminated with bacteria with a reported incidence of 48% to 95% of initial cultures growing bacteria. Studies with lower rates of bacterial growth often included animals which had already received antibiotics. Polymicrobial organisms are also common with a range of anaerobes, oral and skin flora being detected.

Dogs and cats often have their own hair pushed into the wounds which acts as a nidus for infection. Additionally during the struggles associated with a dog attack, dirt and vegetation can be forced into the wounds. The combination of contamination and compromised blood supply, dead space, and avascular tissues provides an ideal environment for infection.

Infection of bite wounds is determined by invasion and multiplication of bacteria in the wounds and the bodies response. The incidence of confirmed infection is determined by the time period post bite, the amount of ischemic and devitalized tissue in the wounds, dead space, and the presence of foreign bodies such as dirt, debris, and hair within the wounds. The greater the crushing forces inflicted on wounds the greater the tissues ability to resist infection is compromised. The longer the delay between the bite and veterinary assessment and cleaning of the wounds, the greater the likelihood that the wounds will be infected.

Necrotic and infected tissue, large hematomas, and potential foreign bodies which remain within the wound, will continue to perpetuate the inflammatory response and infection. Additionally these will interfere with penetration of antibiotics and impair the repair phase of wound healing by interfering with the growth of new blood vessels, fibroblasts and macrophages.

The argument is sometimes made that bite wounds should not be surgically explored for 24 hours to allow the full extent of tissue injury and tissue necrosis from crushing and avulsion injuries to be visualized. The author thinks this a flawed argument because this provides time for contaminated wounds to become infected wounds which may have been avoided by pre-emptive exploration and thorough lavage and decontamination of the wounds to decrease the bacterial load. Additionally this time delay exposes open wounds to increased risk of colonization by environmental bacteria.

Patients are also at risk of developing systemic inflammatory response syndrome (SIRS) secondary to the severe inflammation associated with the injured tissues. Sepsis and septic shock can develop as complications of infection. Secondary organ damage reported after severe dog bite wounds includes disseminated intravascular coagulopathy, cardiovascular dysfunction and hypotension, acute respiratory distress syndrome and hypoxemia, renal dysfunction and azotemia, hepatic dysfunction and pancreatitis. Care should be taken to consider the effects on the whole body with regards to treatment choices and medications.

Potential injuries and specific concerns by bite location:

In addition to concerns about infection and subcutaneous fat and muscle trauma, the location of bite wounds also helps to determine what other underlying injuries could potentially occur.

Over 50% of animals who are presented for bite wounds will have wounds in more than one body location especially smaller animals. Therefore clinicians should take care to examine the whole body to minimize the chance of missing additional bite wounds.

Head

With head injuries fractures of the skull and mandible can occur. Osteomyelitis of the skull and delayed presentation of brain abscess have also been reported. Antibiotics should be considered for any penetrating head wound especially if it may involve the cranium. If there is epistaxis, ear canal hemorrhage, or oral hemorrhage then also consider the potential for the patient's own oral, nasal, aural or pharyngeal organisms to cause infection even if external skin wounds are not detected.

Patients which are unconscious with head injuries should be intubated and ventilated to minimize worsening brain injuries secondary to hypoxia or hypercapnia. Care should also be taken to ensure the patient remains normotensive to ensure adequate brain perfusion.

Cervical

In one UK study cervical bite wounds were associated with a 25% incidence of damage to vital structures including tracheal tears, tracheal avulsions, trauma of the larynx, recurrent laryngeal nerve injury, laryngeal fractures and avulsions, hyoid bone fractures and epiglottic trauma, jugular vein laceration, injury to salivary glands, pharynx, oesophagus, ear canal, skull, thyroid, parathyroid, and spinal fractures. Significant deep injures may occur without penetrating bite wounds. Mortality for cervical bite wounds has been reported between 4 and 20% in various studies.

Temporary tracheostomy may be required if there is marked pharyngeal swelling which may occur initially from hematomas and later potentially from abscesses. Subcutaneous emphysema should always prompt concerns that there has been tracheal injury (puncture, laceration, avulsion). If the subcutaneous air swelling dynamically changes with respiration then a large airway rupture, which requires primary surgical closure, may be present. Static subcutaneous emphysema may occur from a tracheal puncture and simple tracheal punctures may heal without primary surgical repair.

Ventral cervical soft flocculent swellings should raise concerns that the jugular vein or other large vessel has been lacerated. Pressure bandages can be temporarily applied to slow bleeding while assessing the remainder of the injuries. If dyspnea develops after placement of a compression bandage then it must instantly be removed.

Except for the most minor of cervical puncture wounds, radiographs should be recommended to assess for orthopedic injuries such as spinal fractures and tracheal avulsion. However there is always the potential that tracheal lacerations and other injuries may be missed on radiographs. If available CT will provide significantly more information about injuries.

When inducing for anesthesia remember to assess laryngeal function because of the potential for recurrent laryngeal nerve trauma.

Thoracic

Occlusive dressings should be immediately applied because of the potential for extension of wounds into the pleural cavity and secondary pneumothorax.

There is a higher risk of mortality associated with thoracic wounds because of the likelihood of significant internal injuries and it is reasonable to recommend radiographs in every patient with thoracic bite wounds. Studies have shown that animals with thoracic bite wounds who present with respiratory distress and dyspnea are highly likely to have radiographic abnormalities. However one study also found that 22% of thoracic bite wound patients who presented without respiratory distress still had at least one thoracic radiographic abnormality. Overall 77% of dogs and 100% of cats in one study of thoracic bite wounds had radiographic abnormalities detected.

Another study of thoracic injuries from dog attacks found that 56% of patients without penetrating bite wounds had a flail chest. Common injuries include rib fractures, intercostal muscle tears, pulmonary contusions, pneumothorax, and traumatic lung injuries. 20% of thoracic bite wounds extend into the pleural cavity.

Penetrating thoracic bite wounds should be explored and lavaged with sterile saline to remove any hair or dirt packed into the wound and also assess for evidence of intrathoracic injuries. Consideration should be given to the ability to ventilate patients (manually or mechanically) during anesthesia in case the wounds do communicate with the pleural cavity. If surgical drains are required then closed suction drains (Jackson Pratt, vacuum assisted) should be utilized. Because of the potential for subcutaneous injuries to extend into the thoracic cavity and cause a pneumothorax, Penrose drains should be avoided.

Abdominal

There is a higher risk of mortality with abdominal bite wounds in all studies. Body wall disruption and herniation is relatively common and can occur without overlying penetrating injury. Paralumbar herniation is common and easily missed. There is also the potential for crushing and avulsion injuries to internal abdominal organs. As bacterial peritonitis is best treated as soon as possible and ideally prevented, a wait and see approach to abdominal bite wounds is not really justifiable and surgical exploration should be considered in all cases. A midline laparotomy approach to surgery enables examination of all internal organs and if abdominal wall hernias have occurred then the internal abdominal muscles can be closed in situ and reinforced with an omental patch as required. Once internal abdominal injuries have been surgically corrected then the abdominal incision can be closed and a standard surgical approach is made to explore the bite wounds around the abdomen from the skin surface.

Perineal wounds

A rectal exam should be performed to assess for rectal tears or hemorrhage into the rectum. Rectal trauma needs to be surgically closed because of the high bacterial load associated with faeces. Perineal urethral trauma is possible in male animals if bite wounds are close to this region.

Because of the increased cutaneous bacterial load in the perineal region, these wounds are at high risk of becoming infected and should be surgically managed as soon as possible.

Spinal

Bite wounds may cause vertebral fractures and can penetrate around the vertebra into the abdominal or thoracic cavity. Spinal fractures are generally closely localized to the site of overlying bite wounds. Neurological deficits should always raise concerns that vertebral fractures are present.

Limb

Crush injuries may compromise distal blood flow and there is a potential for fractures to occur. Delayed sequelae include septic arthritis, osteomyelitis, diffuse skin necrosis and severe localized infections which can result in a need for amputation.

Initial Assessment:

Intravenous access should be obtained as rapidly as possible in cases with severe bite wounds.

Analgesia

Except for the most minor of bite wounds, severe pain is likely to be present. It is reasonable to give an injection of a pure mu agonist opiate such as fentanyl or methadone at presentation. NSAID administration should be delayed until it is confirmed that the patient has no evidence of shock and the wounds are minor and unlikely to require anesthesia and surgery as the presence of NSAIDs together with hypotension under anesthesia increases the risk of developing acute kidney injury or failure.

Airway

Assess for evidence of airway compromise which may require emergency intubation or emergency tracheostomy.

Breathing

Patients with respiratory distress and cyanosis should be provided with oxygen. When thoracic wounds or bruises are present and the patient has respiratory distress, immediately cover any open wounds with an air impermeable adhesive dressing (or sterile lube and gladwrap/plastic wrap) and either do a quick thoracic ultrasound to assess for pneumothorax or perform a thoracocentesis. There are minimal risks associated with careful thoracocentesis in this situation. If there is an obvious penetrating wound into the thoracic cavity, place a temporary drain directly through the wound into the pleural space and temporarily close the wound with staples and a sterile adhesive air impermeable dressing, and drain the pleural cavity to improve respiratory function. Angiocaths (14G x 5 inch), sterile feeding tubes and IV extension tubing can all be used for thoracic drainage in this situation.

Bleeding

If major bleeding is suspected, such as jugular vein laceration, then firm pressure wraps should be applied to the area and shock treatment rapidly started. The patient is prepared for immediate emergency anaesthesia and surgical exploration. Arterial bleeding can be controlled by occluding the artery with haemostats.

Cardiac

Assess for evidence of shock. If not already done, place an intravenous catheter, collect baseline PCV/TS and blood samples if possible through the catheter and start a fluid bolus of crystalloids if there are any concerns that shock is present.

Shock boluses of 10 to 20ml/kg of crystalloids such as Hartmanns are rapidly infused over 5 minutes in dogs and 10 minutes in cats and repeated to effect to treat cardiovascular instability and hypovolemic shock. If pulmonary contusions are suspected then conservative fluid rates should be used i.e. smaller 5ml/kg boluses at half this rate and repeated as required until there is signs that the shock is improving. Remember that any case suspected to have pulmonary contusions may actually have a tension pneumothorax which will require thoracocentesis to correct the shock and improve venous return through the vena cava.

Intravenous antibiotics effective against anaerobes, oral, and cutaneous flora should be administered at this stage.

Disability

Assess if there is any evidence that the patient may have a spinal injury or an unstable fracture which may require specific support or additional care in moving the patient. Ideally recumbent patients who potentially have a spinal injury are not rolled or turned. However in a dog attack situation there may be severe injuries on the dependent side of the animal and a controlled lift and turn of the patient with several coordinated members of staff may be required to confirm that there are no other severe injuries on the patients dependent side. Take care to keep the spine in a normal alignment and avoid twisting or bending the spine.

A neurological exam may be significantly affected by shock and cardiovascular instability i.e. patients in shock may be comatosed to stuporous and yet have normal mentation once the shock is corrected. Peripheral pain sensation responses are often suppressed by severe shock. Final decisions on neurological dysfunction should wait until shock has been corrected.

Wound assessment

Thoroughly examine all of the patients skin and hair to look for saliva, blood, or bite wounds and clip every potentially bitten area (including areas where just saliva is present). Clipping should be extensive (>5cm past each area of injury or bruising) to detected further small wounds and also to look for teeth marks and bruises in order to identify the extent and severity of the bite wounds or crushing injury and the amount of area affected. Assess whether the far side of the limb or body area has evidence of involvement i.e. is a bite pattern extending around the thorax or abdomen. Examination of bruises and teeth patterns can help to determine the likelihood of internal injuries. Additionally part long hair coats to look for areas of bruising and clip any which are found.

Open wounds should have sterile gel placed into them to minimize clipped hair contaminating the depths of the wounds.

As wounds are found they should be immediately covered with a sterile adhesive dressing to avoid iatrogenic contamination with potentially resistant hospital bacteria while a thorough clip is being performed to ensure that every bruise and every wound is found.

Query owners on the size of the attacker or other dog and what areas of their pet were bitten or grabbed i.e. was their pet picked up by the neck, a hind leg, or rolled over and bitten over the ventral thorax or abdomen, was the injured pet shaken?

Because the skin of dogs and cats is so mobile it is possible for the cutaneous bite wounds to be well separated from an area of internal muscle injury or cavity penetration or a crushing injury. Blunt probing of the wounds commonly misses injuries and is of minimal benefit in the conscious patient.

Once all areas of injury have been identified then a plan can be made to determine the likelihood of internal injuries.

Grading of dog bite wounds

The grading systems commonly reported vary between 3 and 4 grades. However the grading system doesn't take into account the huge range of severity within the highest grade.

Grade I- partial skin thickness (dermis not penetrated)

Grade II- full thickness laceration (>10mm length)

Grade III- full thickness puncture (<10mm)

Grade IV- full thickness puncture or laceration with underlying tissue avulsion and dead space.

Potentially there should be additional grades to cover the vast ranges of size and severity of bite wounds. For example grade IV bite wounds when a small terrier bites a large dog are often significantly less severe than when a large dog bites a small terrier or cat.

Diagnostics:

Ultrasound

Serial thoracic and abdominal focused assessment with sonology for trauma (TFAST and AFAST) exams can be used as an initial screening test to assess for any evidence of internal injuries. Scanning the abdominal wall and assessing the abdominal musculature can also help to identify potential areas of herniation.

Radiographs

Radiographs are indicated for grade III- IV wounds and bruises over the cervical, thoracic, or abdominal cavity to help assess the involvement of deeper structures and organs. Over 60% of traumatic bite wounds resulting in a body wall hernia will have radiographic evidence of herniation, indicating that radiographs are useful test but may miss some hernias. Many of the hernias which occur from dog bite wounds are paralumbar body wall hernias. At least 30% of bite wounds which result in traumatic abdominal hernias or thoracic flail areas are associated with intra-thoracic and intra-cavitary injuries, therefore surgical exploration of the thoracic or abdominal body cavity is indicated whenever abdominal wall hernias or an area of thoracic flail are detected.

CT

If available CT is ideal for major dog attacks when multiple areas of the body have been injured. CT provides significantly more information than radiographs as it enables examination of bones and soft tissues including muscle, fat and all internal organs. If available whole body CT's for trauma can rapidly investigate multiple injuries and help with surgical planning.

Blood tests

When severe injuries are present ideally also obtain baseline CBC, biochemistry, electrolytes, and coagulation testing as multiple organ dysfunction can occur and this does affect prognosis. One study of bitten dogs admitted to an ICU found that the development of a single organ dysfunction was associated with 9% mortality which increased to 67% mortality when 4 organs were dysfunctional. Obtaining baseline bloods tests enables later recognition of the severity of changes when blood tests are repeated if the patient is not doing well. For example a progressive rise in creatinine even if it remains within the species reference range should raise concerns that acute kidney injury is developing and a progressive marked decrease in platelets together with the development of mild increases in clotting time tests indicates that disseminated intravascular coagulopathy is probably developing.

Baseline and follow up serial PCV/TS can be used to determination of the severity of blood loss.

Antibiotics:

Many studies on dog bite wounds have stated that the use of antibiotics is controversial. However all veterinary studies that the author found utilized antibiotics during treatment of grade III to IV wounds and some reported culture results based on samples taken after antibiotics were administered. In human medicine antibiotics are standard of care for dog bite wounds with a 5 day course of amoxicillin-clavulanate recommended as a first line treatment for non-infected wounds, if there are no contraindications such as penicillin allergy. Metronidazole plus either trimethoprim sulfa or ciprofloxacin are recommended for patients with penicillin allergy. Australian recommendations for infected bite wounds in humans are 14 days of intravenous antibiotics.

In the authors opinion intravenous antibiotics should be considered at presentation for all except the most minor of bite wounds to rapidly obtain high blood levels of antibiotics and help prevent contaminated wounds becoming infected. SC antibiotics and oral antibiotics can also be used however absorption may be impaired when patients are in shock.

Grade I (partial thickness) wounds are unlikely to require antibiotics but care should be taken to assess for small missed punctures which could have introduced bacterial into ischemic tissues under the skin.

Antibiotic sensitivities vary in studies with every study published finding that no single antibiotic will be effective for every infected dog bite wound. Wounds which, at presentation, are already infected (as opposed to contaminated) or which develop evidence of infection during treatment, despite antibiotic administration, should have aerobic and anaerobic cultures performed. Differing studies have found that the following antibiotics are suitable for the majority of wounds- amoxicillin-clavulanate, trimethoprim -sulfa, 1st and 3rd generation cephalosporins, ampicillin + either a fluroquinolone or aminoglycoside. Because anaerobes are commonly present in dog bite wounds the author prefers to utilize an antibiotic which covers for anaerobes and uses amoxicillin-clavulanate as a first option (an IV form manufactured by Juno is now available in Australia) and in very severe or infected wounds the author also utilizes both amoxicillin-clavulanate and enrofloxacin.

Analgesia:

Analgesia should start with pure mu agonists as bite wounds tend to be severely painful- a fact which should be understood by any veterinary professional who has sustained a penetrating bite wound themselves. Post-operatively if the patient is stable and has not recently been through a period of shock then non-steroidal anti-inflammatory drugs can also be administered.

Anaesthesia:

Anaesthesia is required to flush and clean most wounds which are generally too painful to manage conscious, or with local anaesthesia and sedation. Routine anaesthetic protocols can be used for small uncomplicated wounds.

The following discussion on anaesthesia relates to major dog attacks:

Anaesthesia should occur as soon as the patient is stable, ideally within a few hours of injury however with some serious injuries surgery will be required to stabilize the patient which necessitates anaesthetizing an unstable patient. The principles of balanced anaesthesia should be utilized with attention paid to decreasing pain in order to minimise the vasodilatory effects of gas anaesthesia. Infusions of fentanyl, repeated doses of methadone, and use of additional analgesics such as ketamine and lignocaine or a morphine/ketamine/lignocaine infusion will help to decrease the concentration of isoflurane or other gas anaesthesia.

The doses of induction agents such as propofol and alfaxalone can be minimized by use of co-induction agents such as midazolam and diazepam at 0.1 to 0.2mg/kg IV. When inducing with propofol or alfaxalone give the induction dose slowly to effect, taking into consideration the delay between injection of the drug and it reaching the brain. The author's preference is to start an infusion of fentanyl, remifentanil or morphine/ketamine prior to surgery and then co-induction with propofol/midazolam- giving 1mg/kg propofol IV followed by 0.1 to 0.2mg/kg midazolam and then 1mg/kg propofol every 30 to 60 seconds until the patient is able to be intubated and placed on gas anaesthesia. In severely unstable patients induction can be achieved using 2ug/kg fentanyl and then 0.2 mg/kg midazolam which may be sufficient for intubation or if required 1mg/kg propofol. The author avoids giving a benzodiazepine prior to fentanyl or propofol because of the small risk of disinhibiting the patient.

Hypovolemia secondary to internal blood loss can be present and will contribute to anaesthetic instability and 5 to 10ml/kg boluses of crystalloids may need to be administered during anaesthesia. In some situations a blood transfusion may be required.

When a major dog attack requires surgery, the veterinarian should be prepared to ventilate the patient during anaesthesia if required and also to use vasopressors such as dopamine or noradrenaline if hypotension occurs.

The *dopamine* dose rate is 5 to 20ug/kg/min. Start dopamine at 5ug/kg/min and increase by 1-2ug/kg/min at 10 minute intervals until blood pressure is adequate.

The *noradrenaline* dose rate is 0.05 to 1.0ug/kg/min. Start noradrenaline at 0.05ug/kg/min and if there is no response in 10 minutes then increase to 0.1ug/kg/min and then increase by 0.1ug/kg/min every 10 minutes until blood pressure is adequate.

Hypothermia can become a major issue during anaesthesia especially when large amounts of hair will be clipped off and wide areas of skin will require surgical scrubbing and wound lavage. Every effort must be made to keep the patients temperature normal during anaesthesia.

Surgery:

17% of bite wound patients in one study were found to require more than 1 surgery. With some wounds repeat surgery may need to be planned for or expected to be likely to occur.

If the chest or abdominal cavity are potentially involved then consideration should be given to the resources available in the veterinary practice and whether the patient should be referred to a suitable 24 hour facility for surgery or for post-operative care taking into account also the travel distances involved and the likelihood of patients reaching a referral facility alive as well as the owners financial resources. Placement of chest tubes and temporary closure of wounds may be required to stabilize a patient for transfer and definitive surgical management.

Surgical exploration is indicated for all penetrating bite wounds and should also be strongly considered when there is a potential crushing injury over the thorax or abdomen. When wounds are present over the thorax and abdomen the surgical clip and prep should be wide enough to enable an exploratory thoracotomy or laparotomy to be performed if required. A sterile water based lubricant should be applied to wounds and then they should be clipped and prepped.

Lavage of wounds

Wounds should be copiously flushed, ideally at 7 to 8 mmHg of pressure. This can be achieved by using a bag of saline or lactated ringers pressurized to 300mmHg and an 18 to 22G needle on the end of a giving set. Though dilute aqueous iodine or chlorhexidine has been recommended in some studies these solutions have been shown to adversely affect fibroblasts in vitro and therefore the author prefers to use sterile saline and rely on the basic principle of 'dilution of pollution'.

If there is gross contamination visible in the wounds then rather than having nurses attempt to flush the wounds during surgical prep consider whether it will be better to have the surgeon resect contaminated and devitalized tissue and then copiously lavage the wounds during surgery.

With all wounds traumatised fat must be removed as it is likely to become ischemic and a medium for bacterial growth.

Thoracic wounds

The thorax should be widely clipped and prepped in case exploration of the wounds reveals a need for thoracotomy. If the pleural cavity is involved then it should be explored to look for foreign material such as hair which may have been driven into the wound. If a pneumothorax is present and there is uncertainty about leakage of air from a lung lobe then once a major airway tear has been ruled out as the source of the leak, the thoracic cavity can be filled with sterile saline and assessed for bubbling while the patient is being ventilated. A rare complication of thoracic lavage during a thoracotomy is atelectasis of lung lobes which will necessitate positive end expiratory ventilation (PEEP) to re-expand the lungs. If the pleural cavity is involved then a chest tube should always be placed to manage post-operative pneumothorax and pleural effusion.

The chest wall can be reconstructed by placing encircling sutures around adjacent ribs in a staggered pattern, preserving any viable intercostal muscles and also using the superficial muscles and the skin to produce an airtight seal. JP drains with a bulb suction device can be used to help manage dead space. Penrose drains should not be placed in thoracic wall wounds if there is any chance that a tissue may devitalize and cause a tract between the skin and the pleural cavity which could result in a sucking chest wound and a life threatening pneumothorax.

Vacuum assist devices have been utilized to manage thoracic wounds where it was impossible to obtain primary closure and thereby seal the pleural cavity from the atmosphere.

Abdominal wounds

If there are puncture wounds or bruises over the abdominal cavity this indicates the attackers bite range was large enough to have crushed internal organs and an exploratory laparotomy is indicated to assess for intra-abdominal injuries. Care must be taken to examine the entire abdomen including the diaphragm, retroperitoneum, the abdominal wall, liver, spleen, kidneys, ureters, bladder, pancreas and the entirety of the gastrointestinal tract for puncture wounds, avulsion of organs from their blood supply, and for contamination.

Abdominal wall tears often appear as shredded muscle and should be sutured together in layers to try to close hernias. Omentum can be used to help patch shredded abdominal wall and minimise the risk of herniation of abdominal contents during recovery. Once the exploratory laparotomy has been performed and the abdominal wound closed then the patients external abdominal skin/wall wounds should be explored.

Other wounds

Puncture wounds should ideally have the skin edges excised and opened with sterile haemostats to enable visualization and to determine the extent of the underlying injury. If a large volume of dead space is present then the wounds should be opened up surgically for exploration and repair. Hair and foreign material should be removed from the wound. Traumatised fat and necrotic tissue should be removed en-block when possible and the wounds lavaged with sterile saline under pressure (as described above) to decrease the bacterial count. Where wounds are relatively clean and there is an adequate amount of viable remaining tissue then primary closure can occur if it can be done without excessive tension. Care must be taken to allow for adequate drainage utilizing either stab incisions or Jackson Pratt (JP) drains. JP drains will decrease the volume of any dead space by application of negative pressure. Passive drains such as Penrose drains can be used but are considered less than ideal because of bacterial colonization.

Where excessive tension on the wound occurs or is expected, or there are concerns about the remaining tissue viability, or evidence of infection or such severe contamination of the wound that it is impossible to render the wound clean for primary closure, then the wounds should be bandaged with either manuka honey (authors preference), hypertonic saline dressings or wet to dry dressings with bandaging. Tie over dressings are often ideal for bite wounds enabling wound coverage and visualization for strike through. Such wounds initially are managed by the author with twice daily lavage with sterile saline and redressing.

To perform tie over dressings the author places very loose individual sutures all around the wounds. These sutures should be loose enough to place a finger between the knot and the skin. The wounds are coated with manuka honey, sterile lap sponges are a useful absorbent material to place into a wound which still requires some debriding. Alternatively apply a non-adherent dressing such as melonin over the wound and then use lap sponges or swabs to allow absorption of fluid. Sterile plastic wrap is placed over the internal dressing and sutures or umbilical tape ties are used in a shoe lace pattern tied back and forth to hold the dressing in place. When using manuka honey care must be taken to rapidly place the dressings over the honey as it becomes extremely runny once it warms to body temperature.

Later primary closure or skin flaps can be used once a healthy wound bed is present.

Because of the increased risk of wound breakdown the author prefers not to remove skin sutures or staples any earlier than 10 and ideally 14 days.

Injured humans:

Though we are not responsible for the medical care of humans, it is common for the owners of attacked dogs and cats to be bitten while attempting to protect their pet. In this situation dog bite wounds often occur to the hands. Hand injuries in humans can have profound effects on the individuals ability to work and maintain employment and studies show a higher risk of serious infection with hand bites than other areas of the body. Veterinarians should actively encourage injured and bitten humans to immediately seek urgent medical treatment ideally at the local human hospital emergency department, as intravenous antibiotics and also surgical lavage and debridement are often recommended. Over 1/3 of hand bites will require surgical exploration within 24 hours and for the majority intravenous antibiotics are administered in Australia.

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GDV- Great surgery is only half the battle

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Introduction

Gastric dilation and volvulus (GDV) is a classic acute surgical emergency which is common in emergency practices and afterhours. This condition has been extensively researched and there are hundreds of journal articles investigating predisposing risk factors, pathophysiology, surgical options, perioperative complications and recurrence rates.

A GDV case is an excellent test of a veterinary practices ability to manage a peracute surgical emergency, especially after hours when resources may be limited. Currently the survival rates of GDV patients which underwent surgery has been reported to be 80% in UK first opinion emergency practice and over 90% in referral practices. This lecture provides a balanced approach to GDV cases and many of the points raised are applicable to other acute surgical abdomens.

Incidence

GDV is a relatively common after hours surgical emergency, with a reported incidence of 0.64% in first opinion emergency after hours UK practices. Rarely GDV occurs during normal working hours.

There have been many environmental and genetic factors postulated to be associated with an increase in the risk of GDV developing, some of which have been supported by small studies and then contradicted by larger studies. The main risk factors for GDV are large or giant breed dogs (lifetime risk of GDV in at risk breeds varies from 4% to 40%), a first degree relative with GDV, increasing age and increased thoracic depth to width ratio. Feeding large volumes of food per meal is associated with an increased risk hence recommendations to feed at risk dogs twice or more daily. Potentially once daily feeding and increasing age may result in a gradual stretch of the hepatogastric ligament predisposing to volvulus when the stomach is full and heavy with food. Gastric foreign bodies may also be an increased risk factor. Other factors such as food particle size and food composition variation have had inconsistent results in studies and no diet has been proven to prevent GDV.

Previous splenectomy may slightly increase the risk of GDV development, potentially because of a sudden increase in the available abdominal space. However a patients current anaesthetic state and other concurrent risk factors for GDV such as breed and age should be taken into account before recommending gastropexy routinely every time a splenectomy is performed.

Other potential predisposing risk factors which require further investigation include motility disorders, inflammatory bowel disease and a change in the gut microbiome which may predispose to fermentation, gas production and gastric stasis.

As a breed Great Danes are universally overrepresented in GDV case studies. A UK study of causes of death in purebred dogs found that GDV was the cause of 2.5% of deaths in all breeds and was reported to occur in 65 breeds including include Akita, Alaskan Malamute, Bassett hound, Bloodhound, Borzoi, Boxer, Bull Mastiff, Chow Chow, Curly Coated Retriever, Dalmation, Deerhound, Dobermann, Hungarian Vizsla, Irish Wolfhound, Mastiff, Neopolitan

Mastiff, Newfoundland, Old English Sheepdog, St Bernard, Standard Poodle, Weimeraner. German shepherds also frequently develop GDV. Occasionally GDV is seen in small dogs and there are rare reports in cats and other species. GDV has been reported in captive wolves also which in the authors opinion raises the question that rather than breeding large breed dogs towards a risk of GDV, has the risk of GDV been bred out of small breeds?

Prognosis:

The GDV patients prognosis has significantly improved over the past few decades. Prognosis also appears to vary with the veterinary teams experience of GDVs with 90% or more of cases taken to surgery expected to survive in experienced 24 hour facilities and a lower prognosis in less experienced hands. The overall prognosis is likely to be more guarded if there are prolonged periods of time between onset of signs and surgery, very high lactate at presentation, hypotension or hypothermia. However there is no single marker which can accurately predict which GDV patient will not survive with surgery and appropriate intensive care.

Pathophysiology:

GDV most commonly occurs after meals but can occur unrelated to feeding. The pylorus rotates from the right side of the abdomen, across to the midline and then continues dorsally along the left abdominal wall and towards the spine. As the pylorus moves the pyloric antrum and body of the stomach also rotate. This rotation causes twisting and obstruction of the cardia and the pylorus and obstruction of initially venous and then arterial blood flow to the stomach, resulting in vascular compromise, ulceration and sloughing of the gastric mucosa and ischemia and necrosis of the gastric wall.

The spleen because of its close attachment to the fundus of the stomach is commonly involved, with tearing of the short gastrosplenic arteries at the head of the spleen, splenic torsions and also splenic thrombi occurring. Blood loss occurs from torn gastrosplenic arteries.

Gastric dilation occurs as gas production increases within the stomach and eructation is prevented by the torsion obstructing the cardia. It has still not been proven whether dilation or torsion occurs first and descriptions of owners are suggestive that either may occur first i.e. some dogs bloat and then show signs of abdominal pain, others show signs of abdominal pain and then bloat.

Gastric wall necrosis can occur very rapidly in some GDV patients, occasionally within as little as 2 hours of the onset of clinical signs, though for many patients it may take many hours before gastric necrosis occurs and in rare situations patients will present after 12 or more hours of distension and not develop gastric necrosis. Overall the longer the period of clinical signs and marked distension the more likely it is that gastric necrosis and secondary complications will occur.

GDV patients commonly present with a combination of obstructive, distributive, hypovolemic, and cardiogenic shock. In rare cases when significant cranial displacement of the diaphragm impacts on tidal volume then hypoxia is also present.

Gastric distension and rotation together with concurrent rotation of the spleen, causes obstructive shock by compressing the caudal vena cava, splenic and portal veins and splanchnic vein and decreasing venous return to the heart. Engorgement of the spleen commonly occurs and this sequestration of blood also contributes to the decreased venous return.

Hypovolemia occurs secondary to intragastric fluid losses and intra abdominal haemorrhage from avulsed short gastric and gastroepiploic arteries.

Cardiogenic shock occurs primarily from myocardial ischemia and secondary arrhythmias (ventricular tachycardia and ventricular premature contractions) which are worsened secondary to global hypoperfusion and increased circulating inflammatory mediators including endotoxins. Cardiac troponins have been shown to be elevated in GDV cases and the elevation of markers of cardiac ischemia relates to the severity of arrhythmias.

In rare cases gastric pressure and distension are so great that it impacts on diaphragmatic and respiratory function resulting in cyanosis and hypoxia. Additionally a small proportion of patients will have aspirated prior to presentation.

Hyperlactatemia is common and relates to the severity of the shock, global hypoperfusion and gastric wall necrosis. GDV patients develop global hypoperfusion which sets the stage for the development of secondary multiorgan dysfunction and failure. Disseminated intravascular coagulopathy (DIC), acute kidney injury, hepatopathy, pancreatitis and acute respiratory distress syndrome may all occur as complications of GDV. Overall the prognosis worsens with delays in treatment however the significance of this cannot be accurately predicted for an individual patient.

Post operatively gastric ileus occurs secondary to prolonged overdistension of the stomach and damage to the muscularis. Gastric ulceration occurs because of the high energy demands of the gastric epithelial cells and the energy deficit which occurred during the period of ischemia during the torsion and dilation. Gastric perforation occurs perioperatively due to death of the muscularis and post operatively from progression of gastric ulceration.

Disseminated intravascular coagulopathy occurs secondary to diffuse microthombi and consumption of clotting factors related to both the gastric necrosis and also the global hypoperfusion. Myocardial ischemia occurs secondary to increased cardiac work during severe shock and this results in the development of ventricular arrhythmias. Acute kidney failure occurs secondary to prolonged hypoperfusion and hypotension and involvement in generalized inflammation and microvascular thrombi. Acute lung injury occurs secondary to activation of neutrophils and inflammation. Pancreatitis occurs due to decreased pancreatic blood flow during the period of torsion.

Clinical signs:

The more rapidly GDV cases are recognized and treated, the better the outcomes are likely to be. In order to optimize outcomes, owners of at risk breeds need to be informed of the potential risk and the clinical signs of GDV and this should happen during routine vaccination and preventative health consultations. This is also an ideal time for general practitioners to discuss the potential benefits of prophylactic gastropexy.

Receptionists need to be trained to recognize a potential GDV or acute abdomen case over the phone, so that whenever owners of any large breed dog call about an acute onset of vomiting or retching they should be instructed to seek immediate veterinary assessment. Occasionally we see GDV patients who had been told over the phone by their regular vet practices receptionists to try nil per mouth for the next few hours and if the vomiting doesn't resolve to then come down for a consultation. Time delays in seeking treatment often result in a significantly more complicated and expensive clinical course for the patient and in some cases a post mortem diagnosis.

GDV dogs are distressed and from owners reports they initially may pace and appear restless though later as shock worsens they will become collapsed. They have unproductive retching or regurgitate thick white ropy or frothy saliva. Some dogs will have borborygmi reported by owners. Owners will commonly say on the phone that their dog is vomiting or coughing and rarely report unproductive retching unless they are aware of GDV risk in their dogs breed. Dogs also occasionally show bizarre behaviours such as digging holes and attempting to vomit in them or repeatedly looking at their stomach or standing with their head and neck outstretched while groaning. Gastric distension is common but not always recognized by owners and in some breeds with a long thorax such as standard poodles, gastric distension can mainly occur under the rib cage making it difficult to visualize.

On physical examination common findings are tachycardia, slow capillary refill, weak pulses (in advanced cases) and gastric distension. The stomach is normally palpably large and tympanic and it can feel as though there is a well inflated basketball palpable in the cranial abdomen.

On physical examination most dogs will be walking however more advanced cases may be recumbent, gums may be hyperaemic to pale , CRT is slow, pulses normal to weak, heart rate is generally elevated significantly and there is palpable cranial gastric distension and cranial abdominal tympany. Occasionally the spleen may feel like a firm mass overlying the stomach. In some dogs with very long rib cages the gastric distension occurs under the caudal thoracic ribs and is difficult to palpate. Tachycardia is normally present and is related to shock, pain and sometimes ventricular tachyarrhythmias. When recumbent patients present with a 'normal' heart rate then decompensated shock is likely to be present.

As increasing age is known to increase the risk of GDV, it is important to question owners about significant co-morbidities or previous illness which may be present, as co-morbidities may adversely impact outcomes in geriatric patients who present with GDV. For example severe pre-existing conditions such spinal disease, severe osteoarthritis, kidney failure, neoplasia and congestive heart failure can all impact on the likelihood of an individual patient who presents with GDV having a good outcome, and for some geriatric patients who are already suffering a poor quality of life then GDV is a reasonable reason for euthanasia. However if there are not any significant co-morbidities present then geriatric GDV patients can have an excellent outcome with surgery and age alone should not be seen as a reason to recommend euthanasia over treatment.

Radiographic Diagnosis

A right lateral abdominal radiograph reveals a compartmentalized stomach or 'double bubble' or 'bum' or 'popeye' sign. The cranial border of the stomach is often distended more cranially than expected and positioning the cranial edge of the radiographic plate at the cardiac apex, helps to ensure this diagnostic tissue fold is visualised. For inexperienced veterinarians GDV is easiest to recognize if the radiograph is taken prior to gastric decompression. Post decompression both the right lateral and a correctly labelled DV or VD radiograph may be required to identify a tissue fold and a malpositioned pylorus. Care should always be taken to differentiate GDV from food engorgement which is not normally a surgical disease.

In geriatric animals whose history suggests a chronic co-morbidity may be present then thoracic radiographs should be considered to screen for metastatic disease.

Blood tests

At a minimum a PCV/TS should be obtained as this may help guide transfusion therapy later if required. However more comprehensive testing is ideal:

Blood gases and electrolytes: The most common acid base derangements are hypokalemia and metabolic acidosis however hypochloremic metabolic alkalosis and mixed disorders may also occur.

Lactate: helps to assess the severity of shock and can be rechecked to help determine the response to resuscitation and treatment. Historically high lactate (>6) was reported to be prognostic for gastric necrosis and increased risk of death, however further studies have shown this to have poor sensitivity and specificity for predicting gastric necrosis or risk of death. However failure of elevated lactate to significantly decrease (by >50%) within 12 hours of surgery suggests a poorer prognosis than patients whose lactate decreases by >50% or normalizes. Patients whose lactate increases post surgery warrant further investigation for complications.

CBC and biochemistry: are useful to assess for co-morbidities in geriatric animals and evidence of concurrent disease. Azotaemia if present may be pre-renal, acute kidney injury or chronic renal failure.

Coagulation tests: PT, APTT and platelet count can raise awareness of early DIC which may require fresh frozen plasma transfusions however the most important treatment for DIC is to resolve the inciting cause i.e. surgery to correct the torsion.

BMBT: should be performed for Dobermanns not previously screened for von Willebrands disease.

In the authors practice baseline bloods are collected at the time of intravenous catheterization and include PCV/TS, lactate/glucose/blood gases and electrolytes and ideally haematology and a general health profile in older or more critical patients. Collapsed patients should have more comprehensive testing including coagulation testing as complications including DIC and multiorgan dysfunction are more likely.

Co-ordinating emergency assessment and initial emergency treatment:
One of the primary goals of GDV treatment is to improve gastric wall perfusion as rapidly as possible to help avoid complete gastric wall necrosis and euthanasia on the table.

On presentation of a suspected GDV patient they should be immediately taken to the resuscitation area of the hospital. This is not the time for a slow and thorough examination and discussion in the consult room prior to deciding on treatment. Baseline vitals are obtained and a large bore IV catheter is placed and baseline bloods are collected through this catheter, then IV opiates (fentanyl or methadone) are administered concurrently with a 10 to 30ml/kg shock bolus (often simplified to 500ml to 1000mls Hartmanns infused via a pressure bag). An IV catheter should always be placed as it will either be used for fluids, anaesthesia and treatment or will be required for euthanasia.

For the rare patient who presents collapsed, cyanotic and agonal, the very first step in treatment is immediate percutaneous gastric trocarisation to decrease pressure on the diaphragm and improve respiratory function.

All other patients are taken straight for radiographs while a shock bolus is running and a right lateral cranial abdominal radiograph is obtained and then percutaneous trocarisation or orogastric tube or both is used to decompress the stomach. Gastric decompression is an essential step in managing GDV's as it helps to improve blood flow to the wall of the stomach. For clinicians who elect to refer their GDV's elsewhere for surgery, trocarisation of the stomach to remove as much air as possible and starting shock rate fluids and allowing them to run during transport can make the difference between a viable and non-viable stomach at surgery and minimize the risk of euthanasia on the surgery table.

Ability to place an orogastric tube does not rule out GDV!

Lignocaine at 2mg/kg IV bolus can be administered immediately prior to decompression and then continuing with a 50ug/kg/hour CRI for 24 hours as this may be beneficial in improving outcome either from its effects on ischemia and reperfusion injury or analgesia however prospective randomized trials are required to confirm pre treatment with lignocaine makes a significant difference to GDV outcomes as current studies are based on historical controls.

Percutaneous trocarisation: use a large bore 14 to 16G catheter (BD angiocath) or needle and place it into the distended stomach where gas is most likely to have risen to. Use percussion or ultrasound to confirm that the spleen isn't overlying this region and ideally clip and prep site. Once the stomach is trocarised remove the stylet if using a catheter and as the stomach deflates, press down with the catheter hub so that the abdominal wall is pushed down against the deflating stomach to avoid the stomach wall falling off the distal tip of the catheter before the majority of air has escaped.

Orogastric intubation: Place an entire bandage roll such as Vetwrap or Cohesive bandage into the patients mouth so that the front of the roll is clamped by the incisors with an assistant holding the dogs mouth closed around the roll. Pass a premeasured and lubricated stomach tube through the bandage roll and as it approaches the pharynx allow the patient to bend their head and swallow the tube to minimize the chance of it passing into the trachea. If the dog coughs or the tube feels like it is moving over corrugations then it may be in the trachea, pull out and restart. Once the tube approaches the cardia take care not to attempt to force the tube inwards because of the small risk of perforation. If it doesn't pass through the cardia easily then try blowing on the end of the tube or encouraging the dog to stand, sit or change position to try to pass the tube through the twisted cardia. Once the tube is through the cardia drop the end to allow air and fluid to empty out of the tube. Having an assistant palpate the stomach can help to remove more of the gastric contents. Sometimes if initial orogastric tubing was unsuccessful it will be successful on a second attempt after trocarisation has removed some gas and decreased the tension on the torsed cardia. The ability to pass an orogastric tube does not rule out volvulus and one study of confirmed GDV cases found that orogastric intubation was successful in 76% of cases attempted.

Surgical clipping can occur while the conscious dog is being stabilized to help to minimize anaesthesia prep times.

The aim is to have resolved shock and have the patient anaesthetized and being prepped for surgery within 1 hour of presentation. 10 to 20ml/kg boluses of replacement crystalloids such as Hartmanns are repeated as required until the patients clinical status has stabilized i.e. HR is less than 140 -150 and shock has resolved or significantly improved. If large volumes i.e. 80 to 90ml/kg are likely to be required then consideration should be given to the use of vasopressors to help stabilise the patient, and to rechecking the PCV/TS to assess for evidence of anaemia or hypoproteinemia developing. In very large dogs a second intravenous catheter should be placed prior to surgery in case large volumes of fluids or blood products are required during anaesthesia.

Anaesthesia:
Cardiovascular depression, arrhythmias, hypotension and gastric hypoperfusion and ulceration are common in GDV's. Therefore care should be taken with anaesthetic and analgesic drugs to minimize worsening these complications.

Analgesia is best provided by pure mu agonists initially. NSAIDS should be considered as absolutely contraindicated due to the already high risk of gastric ulceration and acute kidney injury in GDV patients as well as the presenting state of shock. Alfa-2 antagonists should also be avoided during anaesthesia of these cardiovascularly unstable patients, though at microdoses they may play a role for sedation in the post-operative period.

Currently the authors preferred anaesthetic induction is co-induction with fentanyl 2ug/kg IV+ propofol 1mg/kg IV + midazolam 0.1-0.2mg/kg IV and then an additional 1 to 2mg/kg propofol IV if required. Alfaxalone would be a suitable alternative to propofol and the author has used val-ket and thiopentone a long time ago for GDV cases. This is not a situation to try a new anaesthetic induction agent and the veterinarian should use induction agents they are comfortable with while remembering these patients are highly unstable and will require significantly lower doses for anaesthetic induction than a healthy patient.

The authors preferred anaesthesia maintenance is isoflurane/O2 + remifentanil 5-20ug/kg/hr CRI. Fentanyl can be used at similar doses. Alternatively methadone can be used for perioperative analgesia, morphine is normally avoided because it may stimulate vomiting. Mechanical ventilation if available helps to significantly smooth out the plane of anaesthesia during surgery especially prior to gastric decompression when tidal volumes are impacted by the marked gastric distension.

Ideally anaesthetic monitoring includes pulse oximetry, direct or indirect blood pressure, ECG, capnography and temperature. At a minimum blood pressure monitoring should be available as hypotension during anaesthesia is common. Ventricular tachyarrhythmias generally only require treatment if the rate is fast enough to impact on effective diastole and is associated with hypotension or causes significant pulse deficits i.e. a VPC rate of 180bpm or higher. Lignocaine (lidocaine) is the treatment of choice for VPC's and intravenous loading occurs with doses of 2mg/kg over 2 minutes to a maximum of 8mg/kg while assessing for a response. If the patient responds then a CRI of 75ug/kg/min (4.5mg/kg/hr) is started with a range of 50 to 80ug/kg/min for effective rate control.

Hypotension occurs frequently during GDV anaesthesia and vasopressors such as dopamine or noradrenaline should be available and are commonly required. In the authors experience GDV patients who have suffered hypotension under GA have a more prolonged hospital stay, increased complications and are more likely to die post operatively. It is important to remember that patients who are severely hypotensive under anaesthesia can still have palpably adequate pulses i.e. the ability to palpate femoral pulses does not rule out hypotension. When relying on oscillometric blood pressure during anaesthesia, if the patient is at a suitable plane of anaesthesia and yet remains tachycardic and the monitor is having difficulties measuring blood pressure then the most likely reason is that the patient is hypotensive. Hypotension during anaesthesia will also make accurate assessment of gastric wall viability nearly impossible.

Dopamine dose rate is 5 to 20ug/kg/min, start at 5ug/kg/min and increase by 2ug/kg/min in 10 minute intervals until BP is adequate.

Noradrenaline dose rate is 0.05 to 1.0ug/kg/min, start at 0.05ug/kg/min, if no response in 10 minutes then increase to 0.1ug/kg/min and then increase by 0.1ug/kg/min every 10 minutes until BP is adequate.

Perioperative intravenous antibiotics are given especially if trocarisation has occurred i.e. 22mg/kg cefazolin pre-op and repeated every 90 minutes.

Surgery:
The midline celiotomy incision should extend from the xiphoid to the caudal third of the abdomen and to the pelvis if the stomach is severely distended. The entire stomach should be easily visualised. The torsed stomach is normally covered with omentum and from the surgeons viewpoint the pylorus has traversed from the patients right side along the cranioventral aspect of the abdomen to the left cranial abdomen (180 degree rotation) and potentially dorsally towards the spine (270 degree rotation). A very distended, tympanic stomach is difficult to derotate and placement of a orogastric tube or trocarisation and suction of gas from the highest point of the stomach in surgery (through a healthy area of gastric wall) can make it easier to manipulate the stomach and pylorus. In rare situations the stomach rotates in the opposite direction which means the pylorus has moved from the right side of the abdomen and along the right dorsal abdominal wall. In this situation the stomach is unlikely to have rotated more than 90 degrees and omentum is not overlying the stomach. To derotate the classically rotated (omentum covered) stomach, the author stands on the patients right side reaches across the stomach and palpates the left craniodorsal aspect of the abdomen for the pylorus. Once the pylorus is identified the surgeon carefully pulls the pylorus towards the ventral abdomen, while using the other hand to push down on the left caudal aspect of the distended stomach and rotate the remainder of the stomach in a rocking motion so that the stomach gradually derotates. As soon as the stomach is derotated, an orogastric tube should be placed to allow evacuation of gastric gas and fluid and decrease the degree of gastric dilation. If the stomach repeatedly distends with gas after derotating, an orogastric lavage should be performed by the anaesthetist to remove gastric contents.

All aspects of the stomach including the cardia are examined for evidence of trauma and necrosis. The areas of greatest damage are normally the fundus and the cardia with extension to the body of the stomach in severe cases. Care should be taken to assess the dorsal aspect of the stomach also. A grey palpably thin gastric wall indicates loss of the mucosa and necrosis of the muscularis layer and should always be resected. Devitalised gastric wall often stretches and it is easy for surgeons to become convinced that the entire stomach has become necrotic. Reassess the area of viable stomach along the lesser curvature from the pylorus to the cardia to determine if there will be sufficient stomach remaining to form a tube from the cardia to the pylorus. Always ensure there is sufficient viable stomach at the cardia. Stomach wall colour and hence stomach wall perfusion can be affected significantly by hypotension and when concerned that gastric wall resection may be required, the gastric wall is best reassessed 30 minutes post derotation of the stomach before deciding upon resection. Significant improvements in stomach colour (from dark purple to red) and hence perfusion can occur in this time after derotation. Stomach wall which remains black should be resected.

If large areas of gastric wall requires resection a stapling device can be used to completely resect the necrotic stomach wall. However without a stapler, the author finds it easier to start at the caudal body of the stomach and excise necrotic gastric wall while concurrently performing a continuous appositional suture and gradually progressing cranially towards the cardia to help minimize the risk of peritoneal contamination with gastric fluid and ingesta. If performing an open gastric resection it is common to see large areas of black mucosa within the gastric lumen. This is a common finding however mucosal colour changes alone do not indicate the stomach wall has become necrotic or avascular. Only consider resecting the area of the stomach which has evidence of serosal and muscularis necrosis. If the outside of the stomach looks normal, there is no indication to perform a gastrotomy to check for mucosal injury as all GDV patients should be prophylactically treated for gastric ulceration. Gastric wall necrosis can be determined by the colour of the stomach from the serosal side, palpation of 'tissue slip' which indicates separation of the layers of the stomach wall, and overall thinning of the stomach wall as compared to more normal stomach wall. The final closure of gastric wall resection requires a double layer inverting suture pattern.

Whether or not a resection is required, the greater curvature and fundus should be examined to determine if there is active haemorrhage from the short gastric arteries which are often torn during the initial volvulus and dilation and may require ligation.

If there are large hard gastric foreign bodies present i.e. bones, raw carrots and there is significant bruising of the gastric wall and concerns about the viability of the fundus then it is the authors preference to surgically remove the foreign bodies as these patients appear to be at increased risk of delayed perforation possibly due to perpetuation of gastric ulceration from mechanical irritation. It is reasonable to open the compromised areas of stomach to remove the foreign bodies and then resect the compromised gastric wall.

Involution can be considered for small areas of necrosis but in the authors experience involution tends to be associated with ongoing ulceration, blood loss and complications including DIC. Gastric resection removes this inflamed and damaged gastric wall, and in the authors experience appears to have less post operative complications, than involution.

The spleen should be assessed for viability and to ensure it has not sustained excessive thrombosis or that there is uncontrollable haemorrhage at the hilus. If there is evidence of significant thrombi or uncontrollable haemorrhage then a splenectomy is performed.

A gastropexy must always be performed at the time of surgery and this can be an incisional gastropexy of the pyloric antrum, belt loop, modified belt loop or circumcostal gastropexy. Recurrence rates with these gastropexy techniques have been reported to be 0 to <5% compared to >54% to 80% in cases which have not had a gastropexy performed. Care should be taken to ensure that the stomach is definitely derotated and that the pexy occurs on the right side of the dogs abdomen and in the region of the pyloric antrum. The pexy site chosen should allow the stomach to lie as close as possible to its normal position when the dog is standing. To avoid incision of the diaphragm, the pexy site for an incisional gastropexy should be just caudal to the 13th rib and approximately ¼ to 1/3 of the distance from the midline to the dorsum.

Incisional gastropexy is the authors preference as it is simple, quick to perform, and is strong with no higher recurrence rates then the belt loop or circumcostal gastropexy and less risks of iatrogenic pneumothorax than occurs with circumcostal gastropexy. There appears to be a higher risk of recurrence with ventral midline gastropexy potentially as the antrum and pylorus remain mobile. Tube gastrotomy case reports appear to have a higher risk of serious complications and no advantages for the patient.

An incisional gastropexy is performed by pinching muscularis and serosa on the ventral surface of the pyloric antrum and allowing the gastric mucosa to slip down away from tissue fold. The 3 to 6cm incision is made through both the serosa and the thick gastric muscularis layers of the stomach but avoids cutting through the mucosa. The incision can be performed parallel to the blood vessel branches extending from the greater curvature of the pyloric antrum towards the lesser curvature or longitudinally between the greater and lesser curvature. The incised pyloric antrum is then lifted up to 'kiss' the internal transverse abdominal muscle, while being assessed for the best position and angle to incise the abdominal muscle to help avoid kinking the pylorus or pyloric antrum. An appropriately angled cut is made through the full thickness of the peritoneum and the internal abdominal muscle. A continuous 2-0 to 0 PDS suture pattern is used to suture together the cranial internal abdominal oblique to the cranial muscularis edge of the stomach. Another continuous suture is used to suture together the caudal aspect of the incisions. If the stomach appears to be under significant tension and looks to be trying to tear away from the abdominal wall then several stay sutures can be placed around the gastropexy site to minimize the forces on the pexy site as it heals.

Patients who do not have evidence of gastric contractions during surgery should ideally have a nasogastric tube placed during anaesthesia to help manage post-operative gastric ileus. Correct placement of the tube can be confirmed by the surgeon palpation the tube passing through the cardia.

The abdomen is suctioned and lavaged with sterile saline and a final check for bleeding vessels is performed. Routine abdominal closure is as per the surgeons preference. Abdominal drains (JP drains) are not required unless gastric rupture occurred prior to surgery resulting in severe peritoneal contamination.

Post-operative care:

Gastric ulceration commonly occurs in GDV cases and may be recognized when blood tinged fluid is evacuated via the orogastric tube. Delayed gastric perforation occasionally occurs in the first 2 weeks post GDV surgery. Proton pump inhibitors (pantoprazole, esomeprazole, omeprazole) are more effective at inhibiting gastric acid production than H2 blockers such as ranitidine. Proton pump inhibitors should be administered intravenously BID in hospital and then continued as oral medications at discharge. i.e. Pantoprazole 1mg/kg IV BID followed by omeprazole 0.7 to 1mg/kg PO BID. Oral sucralfate can be utilized if there is evidence of severe gastric ulceration. Nurses should be advised that sucralfate must not be placed down an NG tube if one is present as it will rapidly causes an irreversible obstruction of NG tubes. NSAIDs are contraindicated post operatively because of the high risk of ulceration in this syndrome.

Gastric ileus/paresis commonly occurs and tends to be more severe and long lasting in patients who have suffered prolonged periods of torsion and distension prior to surgery. Repeat screening ultrasounds will reveal a dilated atonic stomach with normal contractility of the intestines. The author expects to see at least one vigorous gastric contraction per minute in a normal post-operative patient and if this does not occur then ileus is present. Placement of a nasogastric tube may be required post surgery to prevent excessive gastric distension occurring. Prokinetics are often required and can be used in combination:
Cisapride 0.5 to 1mg/kg PO TID or 0.25 to 0.3mg/kg per rectal TID
Erythromycin 1mg/kg IV TID or 1 to 2mg/kg PO every 4 hours initially and then TID after a response is seen.
Metoclopramide 2mg/kg/day should be placed into maintenance fluids.

Nutritional support should start as early as possible once the patient has recovered from anaesthesia. If patients remain anorexic for longer than 12 hours post surgery then the authors preference is to start nutrition via a nasogastric catheter using Jevity or Ensure (veterinary specific liquid diets such as Clinicare are not available in Australia). The nasogastric catheter can also be intermittently suctioned to assess residual gastric volumes and the stomachs ability to cope with the volume of nutrition being infused. Care should be taken not to unnecessarily discard gastric residual volumes as this can predispose to hypovolemia and metabolic alkalosis.

Antibiotics: the author utilizes IV antibiotics (ie cefazolin 22mg/kg IV) perioperatively for a maximum 24 hours (in case of peritoneal contamination during trocarisation) and then discontinues them unless the stomach had ruptured or was opened during surgery and gross peritoneal contamination occurred.

Analgesia: pure mu agonists i.e. fentanyl or methadone are utilized perioperatively and then weaning down to buprenorphine or tramadol for discharge. Suitable adjunct analgesics if required in hospital include lignocaine and ketamine infusions. Non-steroidal anti-inflammatory drugs should be regarded as contraindicated because of the already high risk of gastric ulceration.

Arrhythmias are common post surgery. Most commonly these are ventricular tachycardia, ventricular premature contractions and ventricular ectopy. Not all arrhythmias require treatment but if the HR is consistently greater than 180 and is associated with marked pulse deficits and hypotension or there are multiform VPC's at a high rate or R on T phenomenon then lignocaine infusion should be started with 2mg/kg IV boluses every 2 minutes until conversion to an acceptable rhythm (maximum 8mg/kg IV) followed by a 50-80ug/kg/min infusion. Correct pain, hypovolemia, hypokalemia, hypomagnesemia, hypoxia or severe anaemia if present as all of these factors may increase the risk of ventricular arrhythmias developing.
Prophylactic lignocaine has had variable findings with regards to outcome with one paper finding lignocaine showed no improvement in survival but was associated with a longer period of hospitalization and another with an improved outcome compared to historical controls. If prophylactic lignocaine is being used, unless there are severe ventricular arrhythmias, it should be discontinued within 24 hours as it may contribute to nausea and inappetence.

Tachypnoea or hypoxemia should prompt thoracic radiographs to assess for evidence of aspiration pneumonia and if present post operatively then appropriate IV antibiotics should be started and oxygen provided if indicated.

Fluid therapy: patients should remain on intravenous fluid therapy and whenever possible intravenous medications until they are eating and drinking and gastric ileus has resolved sufficiently to rely on oral intake. Electrolytes should be rechecked daily to help optimize fluid therapy and guide appropriate additives ie potassium chloride. Hypovolemia in the post-operative period is associated with a poorer outcome and should be aggressively treated in a addition to determining if there is internal haemorrhage or other identifiable source of ongoing fluid loss.

Serial postoperative ultrasound screening can help to detect early gastric necrosis and perforation or splenic thrombosis. Increasing volumes of abdominal fluid are an indication to perform abdominocentesis and fluid analysis to assess for evidence of bacterial peritonitis.

Discharge advice:

All clients should be counselled about the potential for delayed gastric ulceration and perforation and should be advised to represent immediately if their dog starts vomiting, shows signs of abdominal pain or they are worried that they have deteriorated within 2 weeks of GDV surgery. Ideally recheck 2 days post discharge to assess requirement for ongoing promotility drugs and to confirm that the dog is continuing to improve.

Rest and minimize exercise for 4 to 6 weeks while the gastropexy adhesion is being formed. Initially recommend small frequent feeds of soft food and for long term feed 2 meals per day. Consider avoiding bones for life because of potential for mild kinking at the pylorus.

Medications for discharge:
The authors preference is:
2 weeks omeprazole BID if routine GDV, 4 weeks if surgical evidence of gastric necrosis.
Sucralfate 1g PO TID x 7 to 10 days if evidence gastric necrosis.
Prokinetics as required: Generally as a minimum 5 to 20mg metoclopramide TID and if there was significant gastric ileus then erythromycin 1-2mg/kg PO TID and cisapride 0.5 to 1mg/kg PO TID. Prokinetics are gradually discontinued depending upon the dogs appetite and response.
Analgesics if required- fentanyl or buprenorphine patches or tramadol.

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He ate what???

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Introduction

This lecture discusses a rational approach when the veterinarian is presented with an animal which has recently ingested a foreign body or a potential toxin, to help decide an appropriate first step to take. Options available to the veterinarian are induction of emesis or other methods of gastrointestinal decontamination, to use antidotes or symptomatic treatment, and in some situations surgery or endoscopy should be considered. The toxins and foreign bodies chosen to be discussed are based on calls for advice from referring veterinarians to the authors hospital. Specific supportive and antidotal treatment of individual toxicities is not discussed in this lecture unless there is not currently information easily available in the veterinary literature.

Emesis

Emesis may be used to remove gastric foreign bodies which are a risk for gastrointestinal obstruction to avoid the need for a later exploratory laparotomy, or for ingestion of life threatening toxins.

Patient factors which are considered a contraindications to emesis include any condition which may increase the risk of aspiration such as a decreased level of consciousness, severe tremors which may impact on pharyngeal co-ordination, seizures, laryngeal paralysis or megaoesophagus. Patients which have already vomited multiple times are unlikely to benefit from administration of emetics.

Ingested toxins for which emesis is contraindicated include any caustic (acid or alkali) because of the increased risk of oeophageal injury; detergents, hydrocarbons and volatile compounds because of the known increased risk of aspiration; rapid onset neurotoxins when clinical signs are already present and there is a known increased risk of aspiration or airway obstruction i.e strychnine which can cause a generalized muscle spasm during the act of vomiting or tetrodotoxin (blowfish/puffer fish) when severe paralysis is already present. Emesis is also contraindicated when the toxin is already severely affecting the level of consciousness.

Ingested foreign bodies for which the risk of emesis must be weighed up carefully include sharp foreign bodies which may lacerate the stomach or oesophagus during the act of vomiting, large foreign bodies which are unlikely to pass up through the oesophagus and linear foreign bodies which are already suspected to be anchored in the distal intestinal tract.

Emesis is generally recommended to be performed within 2 hours of ingestion of a toxin. However in the authors experience it can be successful used to remove toxins up to 4 hours later. The success of emetic administration later than 2 hours post ingestion is affected by the volume of toxin ingested, whether the animal had a full or empty stomach and if a slow release medication or toxin was ingested.

Emetics

Apomorphine

Apomorphine acts as a dopamine (D_2) agonist in the chemoreceptor trigger zone which is outside the blood brain barrier. Apomorphine will also bind to mu-receptors on the vomiting center which is inside the blood brain barrier and when these mu-receptors are bound vomiting may be inhibited. Apomorphine has been shown to have a 97% efficacy for inducing emesis in dogs. Doses vary from 0.03mg/kg IV to 0.04 to 0.08mg/kg SC and 0.04mg/kg IM. Alternatively part or all of a crushed 6.5mg tablet can dissolved in saline and instilled into the conjunctival sac and thoroughly washed out once emesis is successful. Conjunctival administration can cause secondary conjunctivitis.

Injectable apomorphine is available in Australia in 20mg 2ml vials. The costs of each vial is half the price of individual apomorphine tablets and therefore if used to treat more than 1 patient, the injectable form is more cost effective than the tablets. In the authors practice the contents of a vial are decanted into a syringe which is kept refrigerated and then individual doses are drawn out for individual patients. For non-emergency practices, who do not utilize emetics as frequently, the vial can be split into 0.1ml doses in syringes and stored in the freezer. With no cost benefit to the use of tablets, veterinarians who prefer to inject apomorphine should consider if they would be better to stock the injectable form than the tablets.

The emetic effects of apomorphine on the chemoreceptor trigger zone can be blocked by the mu-receptor effects of apomorphine on the vomiting center with higher doses. Interestingly the onset of vomiting is faster with SC administration than IM administration which may be related to the slower absorption that occurs with SC administration thereby stimulating the chemoreceptor trigger zone before inhibition of vomiting via the mu-receptor effects in the vomiting center occurs. Pretreatment with 0.04mg/kg naloxone IV 10 minutes before administering 0.1mg/kg apomorphine IV has been shown to reverse this effect on the vomiting center. If patients don't vomit in response to apomorphine, then administering dilute naloxone IV slowly to effect up to a maximum of 0.04mg/kg could be trialed to see if this reverse the mu effects and causes emesis.

Apomorphine studies have shown that between 10% and 87% of the total dose of recently ingested oral medications was recover in the vomitus.

Sodium carbonate. Washing soda crystals

Sodium carbonate decahydrate $Na_2CO_3.10H_2O$ are alkaline and produce emesis by irritation of the oral, oesophageal and gastric mucosa, and have been shown to have a 81% efficacy for inducing emesis in dogs. Only the crystals should be used as the powder has been associated with severe laryngeal and gastric mucosal injury in the authors experience, Probably due to the increased surface area of the powder rather than a crystal. The dose used by the authors practice is $1cm^3$ per 20kg i.e. a 5kg patient receives approximately ¼ of this dose and a 40kg patient double the dose.

Washing soda crystals should not be used for emesis of caustic or irritating chemicals because they could worsen gastrointestinal irritation and this could increase the risk of oesophagitis, oesophageal perforation, gastritis and gastric ulcers or perforation. Crystals need to be translucent to be effective. When washing soda crystals become opaque or cloudy they are no longer effective as an emetic. The crystals become opaque when they lose water to the atmosphere so should be kept in a sealed container without moisture absorbents.

Tranexamic acid

Tranexamic acid induces vomiting by stimulating NK_1 receptors. Emesis is more successful when there is some food in the stomach or the dogs were fed within 2 hours of emesis. One study found that 50mg/kg IV induced vomiting in all dogs (10/10) and 40mg/kg induced vomiting in most dogs (4/6). Dogs vomited a maximum of 2 times within 250 seconds. In a clinical case series 84.7% of dogs vomited after a 50mg/kg bolus of tranexamic acid. One dog seizured after a 50mg/kg tranexamic acid IV bolus (1.5% incidence) however this dog was emesed because of ingestion of a neuroexcitatory toxin which may have placed a role in the seizure. Of the dogs which didn't vomit with the first dose of tranexamic acid, 64.7% of the remainder vomited after a second 50mg/kg IV bolus within 5 to 10 minutes of the first dose.

3% Hydrogen peroxide

This used to be commonly recommended in the US as an emetic that owners could administer at home to dogs. It acts as a caustic irritant on the oral, oesophageal, gastric and duodenal mucosa. It has been associated with gastritis and gas emboli in dogs. It is considered relatively contraindicated in cats and there is one report of it inducing severe necroulcerative hemorrhagic gastritis in a cat. In dogs the standard oral dose of 3% hydrogen peroxide is 1-2ml per kg PO which can be repeated once if vomiting is not achieved with a maximum total dose of 45ml per dog.

One study investigated the effect of 3% hydrogen peroxide in normal dogs. This study involved repeated endoscopy to visually grade lesions and gastric and duodenal biopsies. This study found evidence of gastric and duodenal injury and ulcers in all dogs which were administered 3% hydrogen peroxide within 4 hours of dosing. The adverse effects of this were significantly worse at 24 hours and the ulcers induced were healing but still identifiable by 2 weeks post dosing. The results of this study and the findings of significant adverse effects on the oesophageal, gastric and duodenal mucosa associated with administration of 3% hydrogen peroxide make it difficult to justify veterinarians routinely recommending owners use this emetic at home, except in the case of a rapid onset severe life threatening toxicity where the chance of the patient reaching definitive veterinary care alive is unlikely i.e. ingestion of 1080 in dogs on a rural property several hours drive from the nearest veterinarian.

Emesis in cats

Vomiting in cats is induced by stimulation of alfa-2 receptors in the chemoreceptor trigger zone or gastrointestinal irritation. In the authors experience use of emetics in cats can be a frustrating experience which is not always successful. Current options for emesis in cats include xylazine, medetomidine, dexmedetomidine and washing soda crystals. The author has successfully used apomorphine in cats however there is a risk of CNS excitement occurring and apomorphine should not be used in cats if naloxone is not available in the practice.

Studies have compared xylazine to dexmedetomidine for inducing emesis. A study comparing an average dose of xylazine 0.44mg/kg IM to dexmedetomidine 10ug/kg IM found that approximately 51% of cats vomited and the number was slightly higher for dexmedetomidine than xylazine however this difference didn't reach statistical significance. 100% of cats in another study vomited after 1mg/kg xylazine however this high a dose may be associated with adverse cardiovascular and sedative effects.

Medetomidine can be used as an alternative drug for emesis at a dose of 5-20ug/kg IM or SC.

Atipamezole can be used to reverse the sedative effects of medetomidine, dexmedetomidine and xylazine. 25-50ug/kg IM atipamezole is recommended to reverse xylazine. For medetomidine and dexmedetomidine use the recommended atipamezole dose on the bottle.

If alfa-2 agonists are administered and appear to be unsuccessful then sometimes giving a washing soda crystal before the patient becomes very sedated can be sufficient stimulus to cause emesis.

Gastric lavage

Gastric lavage is only indicated for life threatening toxicities for which there is isn't an effective antidote and when there is likely to still be toxin present in the stomach; as there is a small risk associated with anaesthesia and also with aspiration during recovery from anaesthesia. For example gastric lavage cannot be justified for vitamin K antagonist rodenticides as there is a highly effective antidote available. Gastric lavage is contraindicated for caustics. During the procedure the patient needs to be anaesthetized and intubated and care must be taken to recheck that the ET tube cuff is forming an effective seal whenever the patients position is changed.

The authors method of gastric lavage is to utilize the largest size single bore tube that will fit down the oesophagus. Fill the stomach with gravity flow of warm water i.e. via a funnel, to ensure that the rugal folds are distended which helps to minimise toxin trapping in the rugal folds. Do not attach the lavage tube to a hose or tap as this can cause excessive pressure and gastric rupture. Once the stomach is palpably full then drop the oral end of the tube below the patient while gently bouncing the stomach, with a hand placed under the stomach, to help swirl the gastric contents into the effluent. Continue to fill and empty the stomach until the effluent runs clear. Turn the patient to the other side, recheck the ET tube cuff to ensure there is an adequate seal of the trachea and then repeat the process of filling, then emptying the stomach. Generally a 3 sided lavage is performed and to do this effectively takes a significant amount of time i.e. at least 30 minutes. Occasionally with large volume toxin ingestions the patient may need to have a 4 to 5 sided lavage. Before recovery from anaesthesia as much water as possible is emptied out of the stomach and the author gives 0.3mg/kg of metoclopramide IV. Deflation of the ET tube cuff and extubation should be delayed for as long as possible in dogs to minimise the chance of regurgitation and aspiration during recovery, especially if activated charcoal was administered prior to recovery, and swabbing or suctioning of the pharynx may be required. Because of the risk of laryngospasm in cats, delayed extubation is not recommended.

Activated charcoal

Activated charcoal is an adsorbent powder which is produced with a massive surface area (1000m2/gram) which can bind a variety of drugs and toxins. Certain compounds have been shown not to adsorbed by activated charcoal and this includes acids and alkalis, alcohols, ethylene glycol, metals and also hydrocarbons. Liquid forms of activated charcoal such as Carbosorb X and Carbosorb XS (which contains sorbitol) are less messy to make up than the activated charcoal powder but that is their only advantage. When using repeated doses of activated charcoal a formulation which does not contain sorbitol is recommended because of the risk of inducing hypernatremia from free water loss into the gastrointestinal tract associated with multiple doses of sorbitol or other cathartics.

Activated charcoal can be mixed with a small amount of food for administration, however this has been shown to slightly decrease its absorptive capacity. For some patients mixing the compound with food is the only practical way of safely administering it. Activated charcoal tablets have been shown in vivo and in vitro to be significantly less effective for drug absorption than the liquid forms and are not recommended for use in hospital. Do not administer activated charcoal to dehydrated patients until intravenous fluids have been started because of the risk of worsening dehydration and the small risk of charcoal causing a gastrointestinal obstruction.

Repeated doses of activated charcoal can be useful for slow release medications and drugs and toxins which undergo enterohepatic recirculation, thereby trapping the toxin within the gastrointestinal tract and reducing blood levels over time.

The standard dose of activated charcoal is 1 to 2g/kg (5-10ml/kg of Carbosorb liquid) or 4 to 10 times the dose of the drug ingested.

Intravenous lipid

Intravenous lipid can be used for specific lipid soluble toxicities such as permethrin in cats. It acts as a lipid sink so that lipid soluble toxins move into the microscopic lipid droplets in the blood and away from the tissues. It also postulated that intralipid can potentially act as a cellular energy source in non-lipid soluble toxins.

The dose used by the author is 15ml/kg over 1 hour. If there is a marked improvement in clinical signs then the infusion can be stopped before this dose is reached. In severe toxicities with animals close to death the author has given a 5ml/kg bolus over 1 minute and then continued the remainder of the dose more slowly. The potential adverse effects of intralipid include the potential for pancreatitis, anaphylaxis and temporary corneal lipidosis to occur. The author has administered intralipid to many patients with no adverse effects.

Drugs are considered potentially lipid soluble if their octanol/water partition co-efficient has a LogP >1.0 with marked increases in lipid solubility as the LogP increases. Permethrin has a LogP of 6.1 and intralipid has been shown to be beneficial for permethrin toxicity in cats and has also been shown to partition Permethrin into the lipid in an invitro study. The author uses the PubChem website to search for the LogP of toxins and drugs to determine their lipid solubility: https://pubchem.ncbi.nlm.nih.gov/ Search in compounds and then go down to Chemical and Physical properties section to find this information.

How to find information on novel toxins and drugs

For ingestion of novel toxinsor drugs the veterinarians at the authors practice use the following sources of information- VIN, poisons info or google searches utilizing the following search terms

Chemical brand name + MSDS

Toxin/drug name + LD50 + dog (or cat)

The use of the keyboard short cut Ctrl + F enables rapid searching of on-line documents for the species involved i.e. to search documents for 'dog' to rapidly find any information relevant to dogs.

Another useful search term is to look at the NOEL- the no observed adverse effect level. Dogs are often used in studies to determine what dose of chronic daily toxin ingestion was show to cause no adverse effects.

Foreign bodies

The decision on whether to induce emesis for foreign bodies depends upon the risk of harm occurring during vomiting and the risk of the foreign body causing harm if left in situ.

Emetics should never be used if the foreign body is within the oesophagus or the intestines or if the animal has already vomited multiple times which is normally predictive of failure of emesis. Care should also be taken before considering emesis with sharp foreign bodies because of the potential for causing perforation or laceration of the stomach or oesophagus. However, if the foreign body is small and the owners cannot afford alternatives such as gastrotomy or endoscopy then emesis should be considered as an option. The author also takes into consideration the size of the ingested object in comparison to the maximum diameter of a bolus of vomitus from a dog of that size. Remember that the oesophageal lumen when dilated by a bolus of vomit is wider than the pyloric lumen. Gastric foreign bodies occasionally can remain within the stomach for prolonged periods of time (up to years) so in asymptomatic dogs presenting 12 hours or longer post ingestion of a foreign body the author will still attempt emesis if there are no contraindications. Heavy foreign bodies tend to be less likely to be successfully removed by emesis. For sharp foreign bodies when gastrotomy or endoscopy is not financially an option or practical, the author ensures owners are informed of the potential risks and feeds a large volume of food before inducing emesis to try increase the chance of the foreign body being surrounded by a food bolus during emesis.

Recently ingested foreign bodies can commonly be removed via endoscopy however owners should always be counselled that endoscopy may be unsuccessful and surgery could be required.

Ant baits

Borax

Borax or sodium tetraborate decahydrate is the most common ant bait (Ant-rid) we received enquiries about. It has a wide margin of safety in mammals and requires large doses to cause toxicity. Dogs have been fed 6g/kg per day with no adverse effects apart from vomiting. However at higher doses there is a potential risk of renal toxicity.

Imidacloprid

This has minimal toxicity for dogs and cats. Studies have shown that dogs who chronically ingested 41mg/kg/day had no adverse effects. The clinical signs associated with higher doses are mainly gastrointestinal irritation i.e. vomiting and diarrhea.

Fipronil

This is also very safe in dogs and cats. It was found to cause neurotoxicity in dogs who were orally dosed with 20mg/kg/day after 5 to 13 days. There is limited data in cats. Rabbits and guinea pigs have been shown to have increased susceptibility to fipronil toxicity.

Other ant baits available for sale to the public in Australia may contain bifenthrin or permethrin.

Absorbent meat pads

The contents of the pad are not toxic however consideration should be given to bacterial and fungal growth in old pads and the potential for the pad to become an intestinal foreign body depending upon the patient size and the pad size and how well it was chewed. So if the meat pad was small and well chewed, no treatment may be required. Giving food before emesis can increase the probability of success.

Batteries

Disk/Button batteries are a high risk foreign body which should be removed as rapidly as possible post ingestion. Because the positive and negative terminals are close together when in contact with moist or wet tissues they cause a localized electrical current which results in the production of hydroxide ions and a rapidly progressive burn injury in adjacent tissues. This can quickly cause a full thickness perforation of the oesophagus, stomach or intestinal tract. In a an experimental study in a dog full thickness perforation of the oesophagus occurred within 30 minutes of contact with the oesophagus and the perforation had extended through to the trachea within 1 hour. The larger disk batteries i.e. 20mm 3V batteries produce injury more rapidly than the smaller batteries but ingestion of any size disk battery in dogs or cats should be regarded as an emergency. Radiographs should be taken to locate the battery and these batteries have a double halo sign on radiographs which can help differentiate between batteries and coins. Once the location of the battery is confirmed, then surgery or endoscopy should immediately be performed to remove the battery to minimise the risk of perforation and death. If the patient is asymptomatic and there is food and a battery within the stomach and ingestion was recent then emesis can be considered, however the owner should be counselled about the potential risk of perforation occurring.

Alkaline pencil batteries (i.e. AAA, AA, D sizes) if perforated when dogs chew on them, release their contents and can cause caustic oesophageal and gastric injury. If a chewed pencil battery has been ingested the battery should be removed via endoscopy or laparotomy and the patient should be aggressively treated for oesophageal and gastric ulceration with proton pump inhibitors. There are minimal risks apart from routine risks of obstruction if the patient hasn't chewed the battery, but of course the problem is asking the patient whether or not they chewed on it first. One case series found that the majority of batteries were passed through the gastrointestinal tract within 48 hours and conservative monitoring of pencil battery ingestion may be reasonable in patients which show no signs of gastrointestinal irritation.

Cockroach baits

The biggest concern from ingestion of the majority of cockroach baits is that the plastic bait station can cause an intestinal foreign body if consumed. The active ingredients in the majority of baits are very safe for dogs and cats.

<u>Indoxacarb</u>

Is minimally toxic, dogs have eaten 5mg/kg/day for 90 days with no adverse effects and the average cockroach bait contains less than 23mg indoxacarb.

<u>Chlorpyrifos</u>

Caused no adverse effects in dogs when fed to dogs at 3mg/kg/day for prolonged periods and acute ingestions of 10mg/kg in cats also showed no clinical signs however higher doses did cause signs of carbamate toxicity in cats. The average chlorpyrifos bait station contains 10mg.

<u>Imidacloprid</u> (see ant baits above)

Cooked bones

In the authors experience cooked and raw bones dissolve quickly in stomach acid and in the majority of times do not require removal even if they are quite large and appear spectacular on radiographs. The only indication for removal of gastric bones is if they are causing significant discomfort for the patient or obstructing the pylorus. Proton pump inhibitors should be avoided until the bone has dissolved because neutralization of gastric acid will slow bone digestion.

Oesophageal bone foreign bodies (or any oesophageal foreign body) in comparison is a major emergency and should be immediately removed because of the high risk of oesophageal perforation or oesophageal stricture formation as a delayed sequelae. If endoscopy is not available in the practice then these cases warrant immediate referral even after hours.

Corn cobs

In the authors experience small corn cobs can sometimes be successfully vomited up with emetics if they are still within the stomach. Endoscopy can be used to remove corn cobs depending upon the size of the corn cob and the size of the patient. If emesis is chosen first then ideally feed the dog a reasonable amount of a gruel diet such as Hills AD as the presence of food within the stomach can help to increase the efficacy of emesis. Then if emesis is unsuccessful the food can be lavaged out of the stomach prior to surgery or endoscopy. It can be very difficult to find gastric objects with endoscopy if the stomach has a large amount of chunky food within it.

Very big dogs may occasionally successfully pass corn cobs through the gastrointestinal tract but owners should be counselled about the high risk of obstruction requiring intestinal surgery. Because of the higher risk of dehiscence associated with intestinal rather than gastric surgery, the author always counsels owners about the risks associated with not removing the corn cob immediately if a wait and see approach is taken.

Dog leads

In the authors experience emesis is often successful in removal of dog leads if they are still in the stomach. If there is any chance that the lead is caught in the pylorus and forming a linear foreign body then surgery is indicated.

Fish hooks

If nylon is hanging out of the mouth it should never be tugged on as this increases the chance of the hook becoming imbedded in the oesophageal or gastric mucosa. Fish hooks shouldn't be left in situ to see if they pass because of the high risk of anchoring and the risk of a linear foreign body developing as the majority of ingested hooks have a length of fishing line attached to them.

Fish hooks in the mouth can be removed by driving the barb through the skin or mucosa, cutting off the barb with bolt cutters and then pulling the remainder of the hook back out. If the hook is multi-barbed then cut off the other barbs first to prevent them becoming imbedded in surround mucosa.

Radiographs are required to determine the location of the hook if it is not visible in the mouth. If there is evidence of pneumomediastinum, pleural effusion, pneumoperitoneum or peritoneal effusion then surgery will probably be required.

In the authors experience, oesophageal fish hooks normally can be removed via endoscopy and most will have the barb buried in the mucosa. If the hook is caught in the proximal cervical oesophagus of a small dog and an endoscope is not available it can sometimes be visualized by using 2 long bladed laryngoscopes to open up the oeosphagus and then the hook can be grabbed with long rigid graspers.

The author prefers to use alligator forceps or similar long rigid graspers for removal of the majority of oesophageal fish hooks and flexible endoscopic graspers for gastric hooks. However flexible endoscopic biopsy forceps can also be successfully used and is the preferred technique of highly skilled endoscopists and medicine specialists. If the majority of a large hook is buried under the mucosa then consider surgical removal because of the risk of damage to vital peri-oesophageal structures.

Whichever of the following methods of hook removal is utilized, care must be taken to ensure that the hook isn't driven deeper into the oesophagus. The hook may be grasped with endoscopic grabbers and driven forwards through the mucosa until the barb is visualized. The barb is then grabbed with the endoscopic grabbers and the remainder of the hook and eye is pulled through the mucosa while manipulating the tip of the scope to avoid twisting the hook and following the line of the path the hook is already taking. This will be less traumatic than pulling the barb backwards which causes more tearing of the oeosphageal mucosa. In the authors experience tearing the barbs backwards through the mucosa, if appropriate care is taken, only causes small oesophageal mucosal tears which heal without complication.

If the hook is difficult to manipulate out of the mucosa then another method of removal is to use a large bore lavage tube (of shorter length than the endoscope) which is placed into the oesophagus. The endoscope is then passed down through the tube and both the endoscope and tube are passed down to the hook. The lavage tube is used to catch the curve of the hook and force it caudally to tear the hook out of the mucosa. If the string is long enough to pass through the tube, then holding onto the string and applying tension can help keep the hook well positioned so that the eye isn't deflected caudally by the lavage tube during this procedure. Once the hook has been removed, the barb can be manipulated and pulled inside the lavage tube which acts as a sheath to protect the oesophageal mucosa from the barb.

If the oesophageal mucosa is torn during removal then the author prefers that the patient is kept nil per os for 12- 24 hours then allowed water only for another 24 hours before starting soft foods to enable the mucosa to form a seal.

Gastric foreign bodies are removed in a similar manner and the string can be used to help guide the endoscope to the hook if there is a large amount of food material within the stomach. If the hook appears to have penetrated a significant depth through the gastric mucosa or the string is going through the pylorus and passing into the duodenum then exploratory laparotomy is recommended.

Glad wrap

The risk depends upon the amount of glad wrap ingested, if a large wad in ingested then this may form a ball of foreign material that could obstruct the pylorus or intestines or potentially form a linear foreign body. Very small pieces will pass through most dogs uneventfully. Ingestion of glad wrap is more of a concern in cats because of their small size. Emesis should be considered if a large volume relative to the patients size and intestinal diameter has been ingested.

Kebab sticks

Kebab sticks are rarely visualized on radiographs and are perfectly designed to become a migrating foreign body through the abdomen or thorax. In the majority of situations emesis is too dangerous. However if there are financial concerns then emesis could be considered as an alternative to euthanasia only if the owner is extremely confident that the stick has been chewed into short lengths before being swallowed and the animal is showing no evidence of discomfort. Alternatively the stick could be left alone and the dog monitored for complications. The forceful muscle contractions associated with vomiting can drive the kebab stick through the gastric wall or the oesophageal wall. If emesis is elected for then the owner needs to be counselled that traumatic perforation of the stomach or oesophagus could occur with life threatening consequences. Endoscopy can be successful for the majority of recent kebab stick ingestions and enables confirmation that there is not a kebab stick within the oesophagus. If a large number of kebab sticks have been ingested then they can form a large tangled spiky ball of full length and partially broken sticks and meat within the stomach which cannot successfully be removed with endoscopy and requires surgery and gastrotomy in the authors experience. Therefore owners should be counselled that surgery may be required if endoscopy is not successful.

Hair bands/ scrunchies

Emesis is recommended with recent ingestion of hair bands in small patients and scrunchies in all patients. If this is not successful then endoscopy or surgery should be considered if the size of the hair band in comparison to the diameter of the patients pylorus and small intestine is likely to cause an obstruction.

Grass

Many dogs appear to enjoy a daily graze on small amounts of grass with no adverse effects. Dogs who have pharyngeal, oesophageal or gastric irritation or nausea for other causes occasionally eat large volumes of grass in what appears to be an attempt to self emese and vomit. The grass can forms a large tangled ball in their stomach which becomes too large for the dogs to vomit. Endoscopy is a frustrating and unrewarding endeavor in the authors experience of attempting to remove a grass ball. As dogs do not effectively digest grass, then surgery is required to remove large balls of grass within the stomach. During anaesthesia care should be taken to examine the mouth, pharynx and tonsillar crypts for embedded grass seeds. If there is no evidence of grass seeds or other cause of pharyngeal irritation found then consider whether investigation for other causes of nausea may be required. During surgery the entire gastrointestinal tract should be palpated to ensure that grass hasn't also impacted elsewhere in the intestines.

Cats also like to graze on grass and occasionally may present with retching, gagging and pharyngeal irritation because they have vomited a piece of grass up and one end of the blade has wedged in the nasopharynx with the remainder of the grass blade extending down the oesophagus. It is reasonable to examine the pharynx under anaesthesia if history suggests this is a potential cause of the cats clinical signs. If an endoscope is not available then the nasopharynx can be visualized with a pen light, a dental mirror and using a spey hook to reflect the soft palate.

Lego

Small pieces of Lego may successfully pass through the gastrointestinal tract of large dogs. Large pieces are a risk for obstruction. Often toys are more successfully emesed if there is some food in the stomach.

Mushrooms

Mushrooms depending on the species ingested vary from being edible to gastrointestinal irritants to potentially causing liver failure, kidney failure, rhabdomyolysis, hypoglycemia, hallucinations, muscarinic effects, hemolytic anemia, encephalopathy and coma.

Amanita mushrooms which include the death cap mushroom are found in some areas of Australia after being introduced. Death cap mushrooms cause no signs for approximately 6 to 24 hours post ingestion and then vomiting and diarrhea develops which is then followed by liver failure within a couple of days of ingestion.

Mushrooms are notoriously difficult to identify by non-experts and experienced mycologists rely on light microscopy to identify the species involved. The author, after treating patients with fulminant liver failure from suspected toxic mushroom ingestion, now recommends emesis and activated charcoal for all wild mushroom ingestions.

Sewing Needles

Radiographs should be taken to localize the sewing needle as the position will determine the most appropriate approach to take. Most commonly needles are located in the oesophagus and stomach followed by the oropharynx and intestines if the ingestion was recent. Sewing needles commonly have string attached which can cause a linear foreign body if the needle anchors. Sewing needles also commonly migrate out of the gastrointestinal tract. If a sewing needles is not known to have been recently ingested and is an incidental finding on radiographs, then the radiographs should be closely examined to ensure that the needle hasn't migrated into the liver or other solid organ where it may be difficult to locate at surgery. There is the potential for needles to pass through the gastrointestinal tract however endoscopy or surgery is recommended, especially as dogs and cats are unable to express that pain may have developed unlike adult humans.

Nicotine gum and cigarettes

These products may contain Xylitol or a non-toxic sweetener and normally the sweeter coats the outside of the gum. Nicorette gum contains 590mg of xylitol per gum. See xylitol section below for information on xylitol toxicity.

The nicotine is released from the gum when it is being chewed on therefore nicotine gum which doesn't contain xylitol rarely causes adverse effects in animals. If in doubt about whether the gum contains xylitol then emesis is recommended as the safest option.

Nicotine is highly toxic to dogs and cats. If more than 0.9mg/kg of nicotine has been ingested and is likely to be absorbed, then gastrointestinal decontamination is recommended. Nicotine can be absorbed by activated charcoal.

If cigarettes are ingested then it is important that veterinarians are aware that a 2mg cigarette contains 13 to 30mg of nicotine. 2mg is what the smoker receives after the smoke has been filtered. 5 to 7mg nicotine remains within the average smoked cigarette butt.

Paddle pop sticks

Paddle pop sticks commonly are ingested whole. In the authors hospital veterinarians report an approximately 50% success rate for emesis in medium to large sized dogs and that emesis is more successful if the dogs are fed before administering the emetic. If trialing emesis first then feed a gruel based diet such as Hills AD as it can be lavaged out of the stomach if emesis is unsuccessful and endoscopy or gastrotomy is required. If the stick has been chewed then perforation is a potential risk of emesis and endoscopy or gastrotomy would be a safer option.

Play dough

Ingestion of home made play dough is likely to cause hypernatremia and emesis with apomorphine should always be considered if the patient is asymptomatic and is likely to have ingested a toxic dose of salt. Recipes vary in the amount of salt used but normal ratio's are ¼ to ½ cup salt per cup of flour. A cup of salt weighs approximately 300grams. Severe hypernatremia can occur after ingestion of as little as 1.9 grams/kg of salt and clinical signs have been reported at levels of 0.5g/kg.

Shop bought Play-Doh is not toxic to dogs though other products will need to be investigated if ingested.

Sand

Dogs will sometimes eat large amounts of sand when it is tasty due to spillage of ice cream, cooking oil or other canine delicacies. Due to the weight of the sand, emesis and gastric lavage are generally unrewarding. Sand is easily identified on radiographs. If a small amount has been ingested it can be passed by encouraging ingestion of food and fiber and using oral laxatives such as lactulose. Large amounts of sand cause severe gastrointestinal irritation and dehydration and tend to become impacted resulting in severe illness and rarely signs of sepsis probably from the severe damage of the gastrointestinal mucosa which occurs from the traumatic passage of the sand.

This is a common problem treated in the authors hospital and is managed with aggressive intravenous fluid rehydration and administration of colon-lytely via a nasogastric tube at 40 to 60ml/kg over 3 hours. This dose can be repeated every 12 hours as required with the aim that large volumes of watery diarrhea will be passed. Serial radiographs are taken to monitor the effectiveness of therapy. Care must be taken to monitor electrolytes and hydration because of the potential to cause electrolyte disturbances such as hypernatremia and dehydration secondary to the repeated doses of colon-lytely.

Sodium monofluoroacetate (1080)

Because of the rapid onset of clinical signs and death occurring after ingestion of 1080 and the long distances between farms and veterinary practices this is one of the only times that the author will recommend at home emesis is attempted if the dog has recently ingested 1080 and is asymptomatic. The owners need to be informed of the potential risks associated with at home emesis. Most commonly the author recommends the use of salt at a dose of 1gram/kg be given per os. A measuring teaspoon of salt weighs approximately 5 grams. A tablespoon weighs 20-22grams. The salt is moistened with a few drops of water and formed into a ball which is given per os like a tablet.

Owners are also recommended to then immediately place their dog into the car and travel to the nearest veterinary hospital which has a veterinarian in attendance or on call because treatment for 1080 or salt toxicity may still be required. This is not a time that owners should worry about the dog vomiting in the car! Ideally 2 people should travel with the dog in case it develops signs of toxicity. Running seizures involve screaming, snapping and continued attempts to run around. The owners need to be counselled on how this can be managed if this occurs while driving. The owners should also be counselled to bring a container of water that the dog can drink from if required if salt toxicity develops. Inform owners they should also attach a lead to the collar in case they need to restrain the dog if it develops excitement on route. Obviously the dog should not be placed in the tray of a ute unless it can be caged in. If the dog has already developed excitement and running fits or seizures then it should immediately be brought to a vet hospital if one is close enough that survival is a chance or alternatively the owner, if a farmer, can consider shooting the dog. Anyone who has seen a dog with 1080 toxicity considers it a more horrific death than a quick death by being shot. The author's practice has successfully treated a couple of 1080 cases which developed running fits when the owners were 30 minutes drive of the veterinary hospital but has also had patients which died despite treatment.

Spectacles

The glass in spectacles breaks into pieces which are not particularly sharp. Emesis is often successful in removing parts of ingested spectacles. If concerned about the size of ingested frames then radiographs may be useful.

Sugar

Confirm that the sugar is not xylitol which is always an emergency. Large volumes of sugar (sucrose) when ingested can cause a spectacular osmotic diarrhea. Emesis can be successfully used if required but many dogs are fine. If severe vomiting or diarrhea develops and the patient is too nauseas to drink then intravenous fluids may be required.

Tampons

Tampons are perfectly designed to cause a linear foreign body and immediate emesis should be recommended. Inconveniently for veterinarians, dogs seem to prefer used tampons rather than unused tampons.

Toilet tablets cleaners

These tablets are alkaline corrosives. If dogs ingests the tablets then they should be immediately advised to drink milk before coming to the veterinary hospital. These can result in severe caustic burns of the oesophagus and stomach. Drinking the blue coloured diluted water in toilets normally only causes gastrointestinal irritation. In the authors limited experience dogs who ingest these are often vomiting and retching and showing signs of severe distress. Emesis and gastric lavage is contraindicated because of the increased risk of an alkaline caustic injury which can cause a liquefactive necrosis of tissues. In the authors practice cases have been treated by placement of a nasogastric tube into the stomach and repeatedly lavaging the stomach with milk and suctioning the contents out until there was no further discolouration of the milk. This resulted in significant improvement in clinical signs and no long term adverse effects. If sufficient volumes of milk was not available then water could be used. Treatment for oesophagitis with proton pump inhibitors and sucralfate is recommended afterwards and the owners need to be warned about the potential for oesopahgeal perforation, oesophageal strictures and gastric perforation.

Toys

Soft toys and cloth can often be successfully emesed many hours after ingestion. If there are clinical signs of nausea and vomiting then radiographs or ultrasound should be performed to check for evidence of gastrointestinal obstruction requiring surgery. Emesis is unlikely to be beneficial in dogs who have already vomited several times.

Wine glasses

Ideally ask owners to bring in any remaining pieces of glass so you can assess how much is missing and also how sharp the edges are likely to be. Radiographs are sometimes useful to find glass depending upon the radiographic density of glass and also whether there is food material present in the stomach. Assess whether the animal has any signs of pain, if pain is present then there is likely to be injury to the oesophagus or stomach and endoscopy is recommended. The author has seen large lethal lacerations of the oesophagus from ceramic and shattered glass on endoscopy. If the glass has shattered into multiple small pieces then it can be very difficult to retrieve via endoscopy. Gastrotomy surgery can be considered but hasn't needed to be done by the author. Feeding shredded cotton wool is an option. The author has in some cases induced emesis after feeding a large volume of food to asymptomatic dogs so that the food is coating the glass as much as possible and has removed the majority of pieces of glass in this manner. The rationale for this is that the pylorus has a smaller diameter than the oesophagus when it is distended during the act of vomiting a bolus of food. A careful and detailed discussion needs to be undertaken with the owners by the veterinarian regarding the potential risks and benefits associated with each approach or taking a wait and see approach to see if the glass passes.

Xylitol

Australian has lagged behind the US in use of xylitol. However we are starting to see case of xylitol ingestion from gums and also people who use it for cooking as they are trying to lose weight or are on keto diets. Hypoglycemia is reported at doses of 100mg/kg and liver failure at doses of 500mg/kg.

Emesis should be performed if ingestion is recent to try to decrease the total dose ingested. Activated charcoal is not effective for xylitol which is a sugar alcohol. Because this natural sugar is highly toxic to dogs, hospitalization and treatment for xylitol toxicity should always be recommended. Xylitol is only toxic to dogs and hasn't been shown to cause adverse effects in cats.

Zinc cream

Zinc oxide creams have the potential to cause toxicity, however the dose required to cause toxicity is reported to range between 100mg/kg to 1 gram/kg of zinc. Acute ingestions often cause severe gastrointestinal irritation which tends to effectively decontaminate most animals. Toxicity can occur from repeated administration to skin when the animal licks it off. Activated charcoal is not effective for zinc toxicity. If a toxic dose may have been ingested then monitor for evidence of hemolytic anemia.

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Should I give maropitant or meloxicam to the vomiting dog?

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Introduction

All veterinarians aim to 'do no harm' and try to act in the best interests of their patients while also trying to do the best thing for the owner. However owners need to understand the potential risks associated with treatment trials if the suspected diagnosis is not correct and that adverse effects may occur.

Treatment trials are a very reasonable approach to addressing illness in the majority of patients. However there are times when a treatment trial may result in delays in definitive treatment which can significantly impact on the outcome or survival of patients; or impact on future tests and the ability to obtain a definitive diagnosis of the illness if the treatment trial fails. Additionally this may result repeated in dispensation of incorrect medications with significant cost to the clients and client frustration if the suspected diagnosis is later found to be incorrect.

Owners may feel that risks of a treatment trial were not adequately explained if there is a poor outcome for their pet and in some situations this may result in litigation or a board complaint. No matter how often your clients decline what you believe is best practice medicine it is important to discuss during each initial consult for each problem, the pro's and con's of a treatment trial as apposed to trying to obtain a diagnosis and definitive treatment. Many your clients will elect for your diagnostic recommendations if the risks of not doing so are clearly explained. Some veterinarians will routinely offer clients several options including ideal diagnostics and treatment, minimal diagnostics and treatment or a treatment trial. If the potential risks of not obtaining a diagnosis are not clearly explained then clients will generally opt for the cheapest option, The author occasionally has consultations with clients who state that "Of course I would have done all the tests if my veterinarian had explained that my pet could die." As an increasing proportion of pet owners feel that their pets are family members, this will become a more common problem in veterinary practice.

This lecture discusses some of the potential adverse outcomes of utilizing maropitant, non-steroidal anti-inflammatories, frusemide and corticosteroids without a definitive diagnosis and things that veterinarians should consider before considering use of these drugs for treatment trials; in the hope that it helps prevent one of your patients ending up with a very expensive emergency bill and minimizes the need for a referral clinician or a second opinion veterinarian having to defend the rationale for previous treatment decisions.

Maropitant

Maropitant is a neurokinin 1 receptor antagonist which was introduced to the veterinary market in 2007 to prevent vomiting in dogs. Maropitant is also being used peri-operatively for its MAC sparing effects, management of chemotherapy induced nausea and for management of pain and inflammation in some conditions. This discussion only relates to its use for control of nausea and vomiting and the problems which may occur when such a potent anti-emetic is used without appropriate diagnostics. Some studies have also shown that clinically the anti-emetic effects of maropitant may be significantly more effective than its anti-nausea effects.

Maropitant has been shown to be highly effective at preventing vomiting in dogs induced by both apomorphine which stimulates vomiting by its action on the chemoreceptor trigger zone and also syrup of ipecac which stimulates vomiting by gastric irritation. The elimination half-life of injectable maropitant in dogs is 8.8 hours, with a duration of effect of approximately 24 hours.

Maropitant has also been shown to effectively prevent emesis in cats that were administered xylazine or dexmedetomidine 20 hours after administration of maropitant or saline. The anti-emetic effect of maropitant in cats is likely to last significantly longer than 24 hours due the 16.8 hour elimination half-life of maropitant in cats. Additionally in this study 1/3 of the treated cats did show evidence of nausea at the time xylazine and dexmedetomidine was administered but were unable to effectively vomit.

Initial clinics studies of maropitant in practice excluded dogs with suspected gastrointestinal foreign bodies or toxin ingestion from being enrolled. Currently because of its efficacy in suppressing vomiting it appears to be the most frequently administered anti-emetic in small animal practice. Maropitant is also frequently used in the authors hospital also but only if a gastrointestinal foreign body has been ruled out by imaging or the patient will have an ultrasound with a specialist radiologist the next day or another definitive cause of vomiting has been identified or the owners finances are such that surgery if required would never be an option for the patient and there is a low index of suspicion that a foreign body is present.

Anecdotal comments from veterinarians suggest that vomiting during treatment with maropitant has been recognized as potentially indicating the presence of a foreign body. The author's concern is that when another reason is presumed to be the cause of vomiting such as pancreatitis then vomiting despite maropitant may be presumed to be secondary to pancreatitis or another non-surgical disease.

Patients with a gastrointestinal foreign body or other acute surgical abdomen have been shown in one study to sometimes have a positive or high canine specific lipase (Spec CPL) measurement which may have occurred from secondary pancreatic inflammation. This study included 38 dogs presenting with an acute abdomen who had a confirmed diagnosis of their disease and had SNAP CPL performed and Spec CPL. In this study 11 patients were confirmed to have pancreatitis and of these 9 had a positive SNAP CPL. Another 27 dogs did not have pancreatitis and of these 11 were SNAP CPL positive. Of the 11 SNAP CPL positive dogs who did not have pancreatitis 8 had a surgical disease and of these 3 had intestinal foreign bodies as the primary cause of their illness. Six SNAP positive dogs which did not have pancreatitis had a Spec CPL >400u/L. In the authors opinion ultrasound imaging should be used to help confirm the presence of pancreatitis and also rule surgical causes of vomiting. If ultrasound is not available radiographs should be performed in cases of suspected pancreatitis to try to rule out other causes of vomiting.

Maropitant may inhibit gastrointestinal motility, with a study in mice demonstrating it did decreased intestinal motility. This has not been investigated in dogs or cats to the authors knowledge but should be considered if a patient on maropitant has significant gastrointestinal ileus.

The author's hospital commonly admits and treats emergency patients who have recently been treated with injectable maropitant and some have additionally been treated with a 4 day course of oral maropitant for suspected gastroenteritis or pancreatitis; and on further investigation in hospital some of these patients are diagnosed with a gastrointestinal foreign body or other abdominal pathology which requires surgery for definitive treatment. Some patients who have been treated with gastrointestinal obstruction will have profound gastric dilation and intestinal distension despite not vomiting or only occasionally regurgitating. This can result in the risk of severe hypovolemia not being recognized because fluid is third-spacing into the gastrointestinal tract.

Prolonged delays in the diagnosis of any cause of nausea which requires surgery for definitive treatment increases the financial costs of surgery and post-operative care as well as the incidence of patient complications. These complications include severe electrolyte and acid base abnormalities, prolonged hypovolemia, increased incidence of requiring intestinal resection and anastomosis, prolonged periods of gastrointestinal ileus post-surgery, septic peritonitis and death.

One final concern that the author has with indiscriminate medication with maropitant is that it may not alleviate nausea as well as it suppresses vomiting. Nausea is a real phenomenon in all species but in non-verbal species it is difficult to judge its severity. In veterinary practice subtle signs of nausea may be missed during a consultation. Therefore by administering maropitant a patient may potentially still be suffering from nausea because the clinical signs of vomiting are masked.

The use of a single dose of ondansetron or metoclopramide may be safer for symptomatic therapy of the patient which presents with acute nausea when acute gastritis is suspected. In the majority of cases of acute gastritis in dogs this is sufficient to control the short period of nausea and vomiting that occurs and then the client is happy that treatment has been administered. If the patient's vomiting recurs and the need for further diagnostics has been discussed with the owner then they may be more understanding of the need for further diagnostics.

Corticosteroids

Corticosteroids significantly improve the quality of life of many patients but can also have significant side effects associated with long term use including polyuria, panting, polyphagia, calcinosis cutis, iatrogenic hyperadrenocorticism and also an impaired immune system which can increase susceptibility to sepsis.

If corticosteroids are trialed for a potential immune mediated disease then the patient is committed to a long course of treatment and it is hard to justify this without a definitive diagnosis unless the owners finances preclude further investigation.

If an infection is present then administration of corticosteroids will inhibit the immune system and worsen the patients disease despite what often appears to be a temporary improvement in clinical signs.

Corticosteroids can also alter test results and interfere with diagnostics and in some situations make an accurate diagnosis nearly impossible to obtain.

Corticosteroids are no longer recommended for the treatment of shock with the exception of hypoadrenocorticism and potentially anaphylaxis. Corticosteroids are also not recommended for trauma cases including traumatic brain or spinal cord injuries or for disc disease. Clinical studies have shown significant adverse effects and minimal or no benefit when corticosteroids were administered in these situations.

Immune-mediated diseases

Immune-mediated polyarthritis may be a primary problem but can also be a secondary problem to another disease or an infection somewhere in the body. It is possible to have one infected joint and then secondary inflammation in multiple joints. Immune-mediated polyarthritis is a painful condition but not life threatening. If diagnostics can not be immediately performed then it is reasonable to treat the patient with opioids to manage their discomfort until the suspected diagnosis can be confirmed. If there is significant concern that bacterial arthritis is present and joint taps cannot be immediately performed, then patients can also receive antibiotics prior to confirmatory diagnostic testing.

Immunosuppressive doses of corticosteroids are a mainstay of treatment for confirmed immune-mediated polyarthritis which predisposes animals to significant weight gain among other complications. Anti-inflammatory doses of corticosteroids are sometimes administered as a treatment trial with the rationale that there will be less chance of doing harm however they will not cure the pathology which is occurring but will also result in significant weight gain which is not beneficial for the patient.

Meningitis and inflammatory brain diseases

Most non-infectious inflammatory brain diseases are treated with high doses of corticosteroids and other immunosuppressive treatments. Prior treatment with corticosteroids can affect the results of a CSF tap and significantly interfere with obtaining a definitive diagnosis.

Infectious diseases such as cryptococcus, toxoplasma, neospora and the rare bacterial meningitis or brain abscess do also occur and all of these conditions will worsen with immunosuppressive doses of corticosteroids after an initial very temporary improvement.

The brain as an organ is particularly unforgiving when inappropriate treatment is administered or appropriate treatment is delayed. Some patients will rapidly progress to coma and death. Clients should be counselled about the risks of delaying diagnostics whenever meningitis or inflammatory brain disease is thought to be a potential differential diagnosis. In the situation of a young dog with neck pain, who may just have sustained muscular trauma when unobserved, then consider a treatment trial of opioids and instruct the owners to call immediately if their dog develops any unusual behavior or appears to be deteriorating and arrange to recheck the patient within 24 to 48 hours for reassessment of its pain and to recheck it neurological status.

Addison's disease/ Hypoadrenocorticism

Corticosteroids can rapidly suppress adrenal function and patients need to be off corticosteroids for at least 2 to 3 weeks and in some situations significantly longer before an ACTH stimulation test can be performed to confirm primary Addison's disease is present and the cause of the patients illness. The exception to this is patients who have received long term corticosteroids which are suddenly discontinued and develop secondary Addison's disease.

Because patients with Addison's disease will require life-long therapy then an ACTH stimulation test should be performed immediately in suspected patients. Relying on a basal cortisol level alone to diagnose Addison's disease will result in some patients with normal adrenal function being unnecessarily treated life-long with corticosteroids. If Synacthen is not available then consider immediate referral for diagnostic testing to a hospital which can perform the test.

Most patients with hypoadrenocorticism will markedly improve with appropriate correction of the shock with intravenous fluid therapy. However if corticosteroids are required to stabilize the patient prior to performing an ACTH stimulation test then dexamethasone should be used (0.1mg/kg IV) and the ACTH stimulation test should be performed as quickly as possible as both pre and post ACTH stim results can be affected within 12 to 48 hours of dexamethasone administration. In this situation collect a serum sample before administering dexamethasone in case the test results of a later ACTH test are equivocal.

In house SNAP cortisol assays for diagnosing Addison's disease can be artifactually elevated by the presence of dexamethasone and samples are best sent to a reference laboratory if dexamethasone was administered prior to testing.

Non-steroidal anti-inflammatories (NSAIDs)

NSAIDs are commonly used to treat pain or fever and they are highly effective analgesics and antipyretics. However there are always alternatives to NSAIDs for the management of acute pain, such as opiates, tramadol and in hospital lignocaine and ketamine can also be utilized for acute pain.

Though the packet inserts don't specify that vomiting, diarrhea or dehydration are contraindications to the use of these drugs in dogs or cats, the majority of veterinary specialists who primarily treat dogs and cats consider pre-existing vomiting, diarrhea or dehydration to be an absolute contraindication to the use of NSAIDs. This is because we have had to deal with the complications of NSAIDS administered to patients with these clinical signs. Recent shock is also a contraindication for emergency and critical care specialists.

NSAIDs can cause gastrointestinal ulceration in normal patients and the risks in patients who already have gastrointestinal disease are considered to be significantly higher. Additionally patients with vomiting, diarrhea and dehydration may have hypovolemia and NSAIDs interfere with the production of protective prostaglandins that maintain renal blood flow and the gastric mucosal barrier. The final concern to consider is that patients with vomiting, diarrhea and dehydration may have liver or kidney disease as the cause of their clinical signs and this may be significantly worsened by NSAIDs.

NSAIDs effects on renal blood flow

Prostaglandins within the kidneys have a vasodilatory effect which helps to maintain renal blood flow and glomerular filtration, particularly when perfusion is impaired or dehydration is present. All NSAIDs interfere with the production of protective prostaglandins within the kidneys and this can result in acute kidney injury or acute kidney failure.

NSAID effects on the stomach and intestines

NSAIDs inhibit production of protective prostaglandins within the gastrointestinal tract leading to decreased secretion of bicarbonate, impairment of mucosal blood flow, inhibition of protective mucous production and epithelial cell repair and regeneration. During states of poor perfusion subclinical gastrointestinal injury commonly occurs even without administration of NSAIDs. Patients with vomiting and diarrhea already have inflammation and cell damage occurring within the gastrointestinal mucosa.

Pyrexia

Pyrexia is an extremely useful marker to assess the adequacy of treatment. Though pyrexia can make a patient feel miserable, masking the pyrexia without definitively treating the underlying cause could delay recovery of the patient.

In human medicine there is a legitimate concern that untreated severe pyrexia can cause a febrile convulsion in babies. This is a specific problem for the human species and should never be extrapolated to dogs and cats. To the best of the authors knowledge there has never been a reported case of a febrile convulsion in dogs or cats. The highest level of pyrexia that the author has seen was 41.6^{o}C and this resolved with treatment of the underlying disease and NSAIDs were not administered.

Pyrexia is an appropriate physiological response to infection and by increasing the hypothalamic set point for temperature, the body creates an environment less conducive to multiplication of infectious organisms. By indiscriminately treating the pyrexia with NSAIDs the veterinarian may inadvertently do more harm than good. Additionally the analgesic effects of NSAIDs may make it harder to identify a focus of inflammation within the body thereby interfering with the ability to obtain a diagnosis.

However if the cause of the fever has been definitively identified and appropriately treated i.e. by lancing a cat bite abscess and starting antibiotics then administration of a single dose of NSAIDs can be completely appropriate if none of the above contraindications are present.

Frusemide

In dogs and cats the only indication for use of frusemide in the majority of patients is to treat congestive heart failure via its diuretic effects which increase urinary excretion of sodium and water. In very rare situations it is used in acute oliguric or anuric renal failure after adequate fluid loading of the patient. Occasionally a single dose may be administed for life threatening lung diseases when excessive intravenous fluids have been administered. Frusemide is of no benefit for aspiration pneumonia as it will not hasten removal of the aspirated liquid and by dehydrating the patient it will impair expectoration and clearance of the small airways.

Once a patient with congestive heart failure needs treatment with frusemide then they will need it for the rest of their life. The exception to this is if the patient has cardiac disease for which the underlying cause can be definitively treated such as thyrotoxic cardiomyopathy. Congestive heart failure can be suspected based on clinical signs but diagnosis should always be confirmed with thoracic radiographs. In patients with severe respiratory distress, cyanosis and a strong suspicion that left sided congestive heart failure is the cause then it is reasonable to start treatment with frusemide and a few hours later once the patient is more stable thoracic radiographs can be taken. However performing an outpatient treatment trial with frusemide is harder to justify unless there are severe financial constraints, even if a heart murmur is present. There are many older small breed dogs who have heart murmurs and audible crackles on lung auscultation but do not have left sided congestive heart failure, instead they may have bronchial disease or other lung pathology which is causing their clinical signs. When frusemide is administered to such patients they may have a temporary improvement in clinical signs due to drying of the airways and not due to resolution of the pulmonary oedema. In such patients ongoing administration of frusemide increases the risk of developing acute or chronic kidney failure.

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Clearance of dexamethasone sodium phosphate following nebulisation in horses

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Introduction

Equine asthma (inflammatory airway disease and recurrent airway obstruction) has a high prevalence in horses of all ages, breeds and disciplines. It is a proven cause of poor performance, resulting in financial losses and welfare concerns for the horse. Treatment for equine asthma is focussed on decreasing inflammation in the airway. Studies have found that reducing the exposure to airborne irritants, in particular dust particles, can result in an increase in performance. More severe cases, however, require medical intervention. Corticosteroids are potent anti-inflammatories and have been proven to quickly and effectively increase lung function and improve clinical symptoms. However, there are concerns of adverse side effects, such as suppression of hypothalamic-pituitary-adrenal (HPA) axis, with long term systemic use.[1] There is recent evidence that administration of dexamethasone sodium phosphate (DSP) by nebulisation has minimal systemic bioavailability and does not suppress HPA axis or induce lower airway inflammation.[2] Administration of dexamethasone sodium phosphate could, therefore, potentially provide a safe alternative for the long term administration of corticosteroids for the treatment of equine asthma.

In both Australian racing and Fédération Equestre Internationale (FEI) competition, medications that are considered performance enhancing are controlled. This ensures an even and level playing field, safeguards the public interest and assures the welfare of both the horse and rider. To maintain sport integrity, random plasma and urine swabbing are performed. As a guide for veterinarians treating competitive horses, withdrawal times (based on detection time studies) have been established for some controlled medications. Detection times have been established for the administration of dexamethasone sodium phosphate via intramuscular, intravenous and intra-articular routes. There have been no detection times published for the administration of dexamethasone sodium phosphate via nebulization.

The objective of this pilot study was to establish the clearance time of a single dose of dexamethasone sodium phosphate in plasma and urine in healthy adult horses.

Methods

Subjects: 6 Standardbred Mares ages 6-21 years (median 14 years), weight 428-505kg and no abnormalities on clinical examination.

These horses were acclimatized to the FlexinebE2® nebulizer mask one week leading up to the trial. Collection of samples occurred over two 5-day sample periods with three horses randomly allocated to each sampling period. A single dose of dexamesthasone sodium phosphate (Dexapent 5 mg/ml, Troy Laboratories) was diluted with sodium chloride based on total volume, as recommended by the FlexinebE2® nebulizer. Whole blood was collected into lithium heparin collection tubes through intravenous catheter or venipuncture at the following time points: 2, 4, 6, 8, 10, 12, 24, 32, 48, 72 and 96h post drug administration. Urine samples (50-100ml) were also collected using in-dwelling urinary catheters (Foley 28F 100cm), free catch or cystoscopy, at the following time points: 1, 4, 8, 24, 32, 48, 72 and 96h post drug administration. All samples were refrigerated at 4^{0} C until analysis.

An official Forensic Racing Laboratory analysed the samples within one week of collection using mass spectrometry to determine the plasma and urine dexamethasone concentrations. Calibrators and quality controls were established using blank samples. The limit of detection and quantification was also calculated by using serial dilutions of known amounts. The limit of detection was 0.04ng/ml for plasma and 0.07ng/ml for urine. The limit of quantification was 0.13ng/ml for plasma and 0.12ng/ml for urine.

A Waters Acquity UPLC system equipped with a Restek Raptor Biphenyl (50 X 2.1mm, 2.7um) analytical column and Restek EXP holder interfaced to an Applied Biosystems 4000 Q-Trap mass spectrometer was used to perform the analysis. The time taken to reach the maximum concentration of dexamethasone of plasma and urine post administration (t_{max}) and the maximum concentration of dexamethasone reached (C_{max}) were calculated. The time of elimination of dexamethasone in plasma and urine was also analysed.

Results

Urine

The point of t_{max} was the earliest sampling point of 1h for all horses with the C_{max} ranging from 3.2ng/ml to 23.8ng/ml of dexamethasone. One horse had no detectable levels in urine at 24h, another two horses has no detectable levels at 48h, two more had no detectable levels at 72h and one horse still had low detectable levels of 0.1ng/ml at the final measurement of 96h (Figure 1).

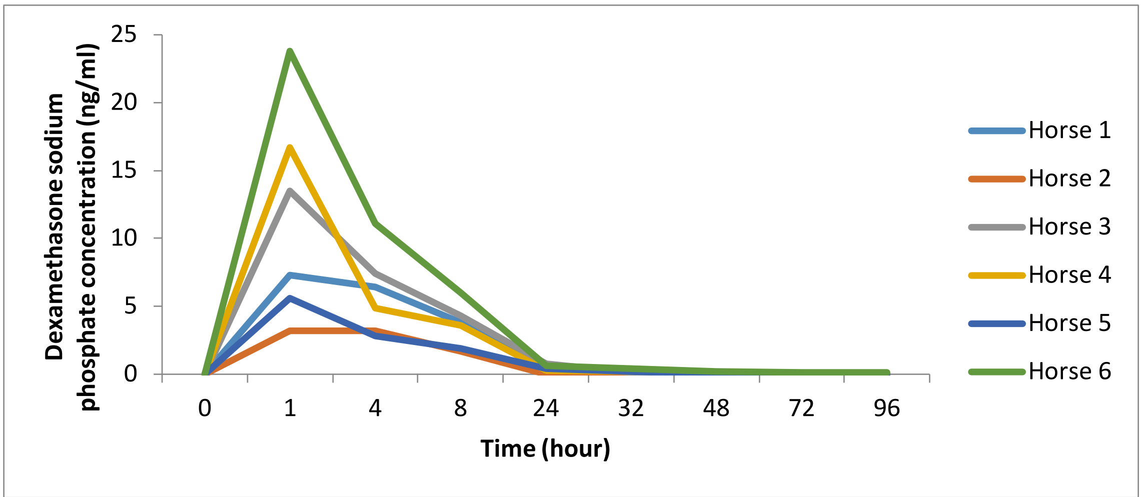

Figure 1: Equine Dexamethasone urine concentrations after a single nebulisation

Plasma

The point of t_{max} was the earliest sampling point of 2h for all horses with the C_{max} ranging from 0.6ng/ml to 1.8ng/ml of dexamethasone. Three horses had no detectable levels in plasma at 24h, another horse had no detectable levels at 32h and one horse at 48h. There was one horse that had not detectable levels at 32h, 48h and 72h but then had a low recording of 0.1ng/ml at the final testing point of 96h (Figure 2).

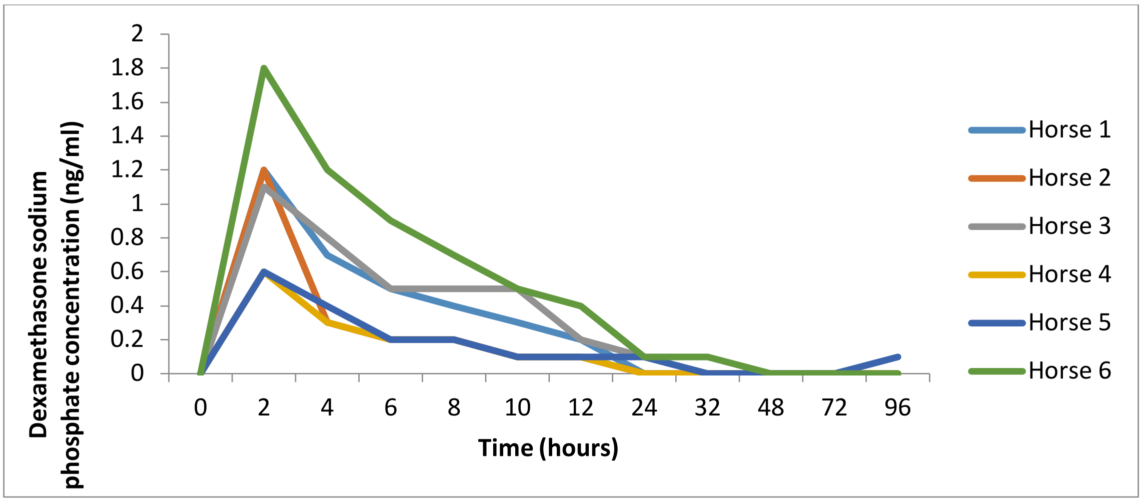

Figure 2: Equine Dexamethasone plasma concentrations after a single nebulisation

Discussion

The time taken to have low or no detectable levels of dexamethasone in urine and plasma of horses in this study is comparable to what has been previously published in studies looking at different routes of administration. Intravenous administration of a 100mg dose[3] and a 0.05mg/kg[4] have both been reported to have a clearance time of 48hours in plasma, while a single intramuscular administration has a reported clearance time of 48hours (0.05mg/kg)[4] to 72hours (20mg)[3] in plasma.

There was moderate variability identified between the final detection time of individuals, ranging from 24-96h+ hours for plasma and 24-96h+ for urine. Previous studies that have concluded that this is a due to the variability in the fraction of the drug inhaled and the fraction of the drug swallowed when using a nebulisation system such as a FlexinebE2® nebulizer.[2] Unfortunately this fraction will be different in each individual and at each nebulization. Additionally it is not possible to determine this, which means the overall bioavailability is difficult to assess accurately, and this should be taken in to consideration when accounting for clearance times.

The t_{max} and C_{max} occurred at the earliest sampling point for both plasma and urine. It should, therefore, be considered that the values in this study are not a true representation and that earlier sampling is required to determine the highest and earliest values reached. There was also one horse that had three consecutive readings of 0ng/ml in plasma from 32-72h and then returned a low reading of 0.01ng/ml at the last sampling point of 96h. This was an unexpected finding and may be associated with redistribution of the drug into fat or muscle tissue and then being released back into circulation.

There are several limitations to this pilot study, the biggest being that there was only a single administration of the dexamethasone sodium phosphate. This has been standard for detection time studies, however there are many variables in clinical practice including the dosage, concentration, length and frequency of treatment that could all impact on the time taken to clear the drug in plasma and urine. There is also limited data available on the recommended dosage of dexamethasone sodium phosphate when administered as an aerosol for the treatment of equine asthma.

Some other limitations to consider are the use of only healthy horses, with studies in humans finding a difference in systemic exposure to inhaled corticosteroids between healthy and asthma affected patients.[5] These horses were also rested for the duration of the trial and is have been found that mild exercise can prolong the clearance of dexamethasone when administered intravenously.[6]

Conclusions

A single dose of 0.04 mg/kg DSP administered through nebulisation was cleared in all 6 horses in plasma by 48 h, with one horse returning a detectable level (0.1 ng/ml) at 96 h, and five out of six horses in urine by 72 h. When determining withholding times for competition one must also take into consideration the dosage, frequency and chronicity of administration, disease status of the horse and activity levels.

Please note that dexamethasone sodium phosphate administered via nebulisation is currently off label usage of the drug.

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Improving detection, investigation and management of Emergency Animal Diseases

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Introduction

An EAD event such as an outbreak of foot-and-mouth disease affecting livestock in several states would cost more than $10 billion and have widespread impacts on animal health, welfare, productivity and social-economic impacts both associated with affected industries and downstream effects. Some EADs, including highly pathogenic avian influenza, rabies and Hendra are of major public health concern.

Veterinarians play an important role in detection, surveillance and management of Emergency Animal Diseases (EADs). Early detection and immediate reporting of an emergency pest or disease increases the chance of effective and efficient eradication. Veterinarians in the field are likely to be the first ones to detect suspicious animal diseases. Otherwise, they may be the first to be contacted by owners who have concerns about unusual clinical signs in their animals. Surveillance for the detection of EADs requires veterinarians in the field to be able to recognise the signs that point to a disease incursion.

An Emergency Animal Disease Training Package is being developed collaboratively by all Australian Veterinary Schools and funded by the Australian Government, Department of Agriculture and Water Resources (DAWR). The aim is for participating veterinarians from clinical practice to gain enough knowledge and information to consider an Emergency Animal Disease in their differential list and know how to proceed if an EAD is suspected.

Emergency Animal Disease Training Package

The project will develop a package of open access training materials that will inspire and educate practicing veterinarians and veterinary students on technical aspects of Emergency Animal Diseases (EADs) and their roles and responsibilities with regards to detection, investigation, management and reporting. The package will be structured as four approximately 20-minute online modules, including media from a range of modalities, with an introductory component and three engaging veterinary clinician-focussed case studies.

Each of the case studies will link to relevant Emergency Animal Diseases (EADs) resources such as the Field Guide for Veterinarians in Emergency Animal Disease Diagnosis and Investigation, existing content (case studies, activities and teaching materials) from the Universities' teaching programs and online information from Animal Health Australia. The course material will be peer reviewed by veterinarians from academia, government and private practice. Course materials and case studies from the training package will also be integrated into the curriculum of the veterinary science degree curricula around the country and could also be developed into workshops in collaboration with government veterinary agencies. Having similar material delivered to veterinary students as is made available to practicing veterinarians will increase knowledge retention and discussion amongst our profession. The modules will be engaging and interactive such that they are relatable and memorable for clinicians and veterinary students alike. A key theme that will be attractive to clinicians is diagnosing unusual differentials for common presentations.
The training package is expected to be freely available online from the 30th of June 2019.

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The challenge to improving lamb survival that won't go away

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Introduction

Improving lamb survival is a priority for the sheep industry from a production and welfare perspective. Approximately 30% of lambs born or almost 15 million lambs die each year. A recent review of national sheep reproduction rates and lamb survival undertaken for Sheep Producers Australia indicated that reducing these losses by half would result in an annual return of $750 million[1]. Furthermore, while recent and current R&D to improve lamb survival together with extension programs such as Lifetime Ewe Management and Bred Well Fed Well are making a difference, they are likely to achieve less than half of the targets set in the meat and sheep industry strategic plans for 2030. Extra investment and more effective RD&E is therefore needed to improve weaning rates and seriously tackle lamb survival, especially survival of twin lambs which are estimated to account for about 75% of all wastage. Improving survival will require adopting current best practice plus novel R&D to inform new and effective ways to reduce lamb wastage. This paper provides a snap shot of recent and current R&D undertaken largely on commercial farms by Murdoch University and its national collaborators to improve lamb survival.

Foundation research and extension to improve lamb survival

The Lifetimewool Project from 2001 to 2008 set the benchmark in terms of practical industry focused RD&E to improve lamb survival. Large-scale experiments across multiple years at 'Austral Park' in Victoria and 'Billandri' in Western Australia generated a wide range of ewe liveweight profiles during pregnancy and lactation that allowed us to generate equations that predicted the performance of both the Merino ewe and her progeny. This included ewe wool production and reproduction[2], lamb birth weight and survival[3], progeny growth to weaning and survival after weaning[4] and the production and quality of the progeny's wool during their lifetime[5]. These production responses were confirmed at a paddock scale in a range of environments[6,7] and economic modelling was used to determine the optimum liveweight profiles for different regions to improve whole-farm profit and animal welfare[8]. These guidelines were then developed into Lifetime Ewe Management (LTEM) that has been AWIs flag-ship extension program for the last decade[9].

LTEM has since been delivered to more than 4,000 wool producers managing more than 30% of the National ewe flock. Based on evaluation surveys completed during the 12-month period following graduation, the estimated number of lambs weaned by LTEM participants increased from 99% prior to LTEM to 106% after completion of the course (Thompson unpublished data). The impacts of LTEM on the number of lambs weaned is unprecedented by previous extension efforts in the Australian sheep industry. Participants of LTEM have also reduced ewe mortality from 4.0% to 3.0% whilst increasing stocking rates from 8.8 to 9.9 DSE/ha. The productivity gains achieved by LTEM participants can be attributed to: (i) changing attitudes to the importance of managing ewe condition score as a driver of farm profit; (ii) changing skills to manage ewes to achieve condition score (CS) targets; and (iii) adoption of management practices including pregnancy scanning for multiples and differentially managing twin-bearing ewes. To quantify the true legacy of LTEM on the National sheep industry it would be necessary to examine how these impacts of LTEM on participants have been sustained over the 5 to 10 years following participation in the course and to quantify the financial implications of adopting LTEM practices.

Recent research to improve lamb survival

Management guidelines to optimize the productivity of Maternal ewes

Almost 30% of ewes in Australia are non-Merinos and they produce about 45% of lamb supply. Whilst producers with non-Merino ewes achieved similar or even greater gains in productivity from participation in LTEM (Thompson, unpublished data), feedback from both deliverers and participants indicated a lack confidence in both the applicability of the Merino recommendations and or knowledge of how to adjust those CS targets to optimise productivity, profit and welfare outcomes including lamb and ewe survival. The overarching aim of the '*Lifetime Maternals*' project was therefore to use a combination of experimentation and economic modelling to develop ewe liveweight and CS profiles that optimise whole farm profit and welfare outcomes for maternal ewes.

Several large-scale grazing experiments involving 500 to 800 maternal ewes at each of four sites in WA, SA and Vic were conducted over two years. In 2014, ewes were managed to achieve a target CS at lambing varying from 2.5 to 3.6 and the treatments continued through until the end of lambing, so ewes in lower CS at lambing also lambed onto less feed on offer (FOO). As expected, improving ewe nutrition during pregnancy increased lamb birth weights[10,11] and the coefficients relating ewe liveweight profile to lamb birth weight were similar to those found in previous experiments for Merino and crossbred Border Leicester x Merino ewes. A 10 kg change in ewe liveweight during early-mid pregnancy changed lamb birth weight by 0.30 kg whereas a 10 kg change in ewe liveweight in late pregnancy changed lamb birth weight by 0.43 kg. Lamb birth weight was strongly correlated with survival to weaning, but single and twin-born lambs were equally likely to survive at the same birth weight unlike that observed in Merinos[3]. Changes in lamb birth weight had minimal effects on the survival of single lambs when ewes varied in CS from 2.7 to 3.3 at lambing, as single lambs still weighed 5.5 kg even on the lowest CS treatment. However on average the survival of single lambs was reduced by 7% in ewes fed to achieve CS 3.7 through to the end of lambing (89% compared to 82%), due to increased risk of birth injury and dystocia. Increasing ewe CS at lambing from 2.5 to 3.6 and especially up to CS 3.2 improved the survival of multiple born lambs by 17% and weaning rate from twin bearing ewes from 135% to 169%. In 2015, experiments at each site aimed to establish if higher FOO during very late gestation and lactation could mitigate the adverse effects of poor nutrition during pregnancy on birth weight, survival of twin-born lambs and weaning weights. While treatment effect on lamb birth weights and survival were less evident, it was clear that improving nutrition from the point of lambing until weaning does not fully counteract the adverse effects of poor nutrition during pregnancy on weaning weight of lambs from non-Merino ewes.

Overall, the results imply that CS targets at lambing of 2.7 for single-bearing ewes and at least 3.2 for multiple–bearing non-Merino ewes are likely to achieve near-maximum lamb survival and weaning rates. This clearly demonstrates the value of pregnancy scanning non-Merino ewes and differentially managing those with multiple foetuses. The carryover effects of pregnancy nutrition on lamb growth and weaning weights are also likely to have implications for optimising CS targets. Additional research over the last two years has refined the feeding standards for maternal ewes to enable the development of optimum CS targets. These CS targets will be included into existing extension programs.

Mob size targets at lambing

Management factors that enhance disturbance of ewes and lambs during the early post-natal period would logically increase the risk of separation and lamb mortality. A limited number of small-scale studies have suggested that higher mob sizes or stocking rates during lambing increases the risk of mis-mothering, ewe-lamb separations and hence lamb mortality. Lambing density is also expected to have a greater effect on the mis-mothering and survival of multiple born lambs because more lambs are born per day. In support of this, survey data collected from commercial sheep producers in south-east Victoria found that the survival of single and twin-born lambs increased by 1.4% and 3.5% from decreasing mob size by 100 ewes, and this applies for both Merino or non-Merino breeds[12]. The 'Lambing density' project aimed to quantify the effects of mob size and stocking rate during lambing on the survival of Merino and non-Merino lambs born on commercial farms across southern Australia.

Furthermore, it aimed to develop improved recommendations for sheep producers regarding the allocation of ewes to mobs and paddocks at lambing including the cost-benefit of investing in paddock subdivision to improve lamb survival.

The 'Lambing density' project involved three components which were completed across southern Australia; (i) on-farm research at 70 commercial farms to test a 2x2 factorial combination of mob size (high or low) and stocking rate (high or low) on the survival of twin-born lambs of Merino or non-Merino breed; (ii) on-farm research at 15 commercial farms to test the effect of mob size (high or low) on the survival of twin-born Merino lambs at low stocking rates; and (iii) a network of almost 200 sheep producers who contributed data for over 2000 lambing mobs from their own farms to investigate the impacts of mob size and stocking rate on the survival of single- and twin-born lambs of Merino and non-Merino breed across a broad range of management and environmental conditions. The experimental work found that lamb survival was poorer at higher mob sizes but not stocking rates[13]. A linear relationship between mob size and lamb survival was identified whereby the survival of twin-born lambs decreased by between 2% and 2.5% for each additional 100 ewes in the mob at lambing. This effect was consistent across Merino and non-Merino breeds and it was not influenced by stocking rate, ewe CS at lambing, FOO at lambing or the characteristics of the lambing paddock. More intensive research indicated a variable effect of mob size on lamb survival in two contrasting seasons. The survival of twin lambs decreased by 4% per additional 100 ewes in the mob when FOO was below 400 kg DM/ha and ewes were supplementary fed during lambing[14], whereas the survival of single- and twin-born lambs was not observed to differ between mob sizes of 50 and 130 ewes when FOO at lambing exceeded 2,400 kg DM/ha[15]. This may suggest that FOO and or supplementary feeding could influence the effect of mob size on lamb survival.

Economic modelling showed that reducing mob size at lambing is justified even if paddock subdivision is required. The economic return from reducing mob and paddock size at lambing is greater for twin- compared to single-bearing ewes, with the optimum mob size for twin-bearing ewes being 40% to 50% that for single-bearing ewes. For producers that do not pregnancy scan or only scan wet/dry, the optimum mob size is more like the mob size for twin- compared to single-bearing ewes. Integrating guidelines for reducing mob size at lambing with current guidelines for the optimisation of maternal nutrition and resource allocation will therefore contribute to improved+ marking rates within the sheep industry.

Grazing cereal crops and metabolic disorders

Grazing vegetative wheat, barley and oats (both dual purpose and traditional spring varieties) is becoming an important strategic and tactical grazing option on mixed farms. The higher winter growth rates of the crops offer an opportunity to fill winter-feed gaps and their high nutritive value makes them well suited to meet the requirements of reproducing ewes. However, their complex mineral composition (low sodium, magnesium and calcium and high potassium and DCAD) can increase the risk of metabolic disorders. Surveys of producers have indicated fewer ewe deaths on properties where pregnant ewes grazing wheat are kept in good condition and are fed mineral supplements. However, about 30% of producers that currently graze cereal crops have experienced metabolic issues and increased mortality with pregnant ewes, and an additional 20% no longer graze crops with pregnant ewes as current recommendations have failed to provide a practical solution to eliminate these risks (Masters and Thompson unpublished data). This project aimed to define the risks associated with grazing cereal crops and develop supplements that would minimise ewe mortality.

The project was conducted over two years. In the first year the mineral status of both forage and of reproducing ewes grazing wheat, oats or barley was monitored on farms in Western Australia (6 farms) southern New South Wales (7 farms) and central New South Wales (5 farms. In the second year, the effectiveness of two mineral supplements was assessed on six farms over three weeks. One of these was a standard industry supplement containing causmag, limestone, and salt (40:40:20), the other contained the same cations but as magnesium chloride, gypsum and salt (12.5:32.5:55). The second supplement was designed to reduce the dietary cation-anion difference in the ingested diet. Across both experimental years ewes gained CS (3 to 3.2) when FOO ranged from 200 to 3,400kg DM/ha during late pregnancy[16,17]. In year 1, a high proportion of farms had forage Ca (70%), Na (70%), Mg (18%) below published requirements and K (70%) above the published Maximum Tolerable Level. Analysis of samples collected from the ewes at the end of crop grazing indicated ewes on 94% of farms had alkaline urine and on 88% of farms Ca concentrations in the urine were in the marginal range. The forages had a complex mineral composition meaning grazing ewes had an increased risk of direct or induced Ca (hypocalcaemia) or Mg (hypomagnesaemia) deficiency, and the analysis suggested higher risks from grazing wheat and/or grazing crops grown on high K soils. In year 2, the supplemented ewes showed significant increases in Ca concentration in urine, plasma and Ca fractional excretion on all but one of the six farms, but there were no clear or consistent differences between the two supplemented groups of ewes. It was concluded that the Ca status of ewes grazing vegetative cereal crops in late pregnancy can be improved by providing supplements containing Ca, Mg and Na.

Despite increased knowledge of the impacts of supplements on metabolic status, little is known about the impacts on ewe or lamb survival after grazing cereal crops or when grazing cereal crops during lambing. Anecdotal evidence from producers is conflicting. Some report reduced lamb survival when grazing crops at lambing due to possible metabolic disease, whereas other producers report improved survival that is perceived to be the result of increased shelter.

Improving the reproductive performance of ewe lambs

Increasing the number of ewes mated to lamb at 12 to 14 months could be an effective avenue to rapidly build ewe numbers and increase lamb supply. However, the reproductive performance of ewe lambs is much lower and more variable than that achieved by adult ewes. A lack of information on the impacts of joining at 7 to 9 month on the mortality of the young ewe and her progeny, the longer-term impacts on the young ewe and her progeny or the financial ramifications of joining ewe lambs has also contributed to relatively poor adoption of the practice. The work is on-going but a series of small projects over the last 6-7 years have made significant progress towards development of management guidelines to improve the fertility and reproductive rate of ewe lambs. This includes understanding the importance of using teasers, liveweight and age at joining, growth rate during joining and sire of ewe lamb genetics on fertility and reproductive rate of Merino and or Maternal ewes lambs[18,19,20]. For example, for Maternal composite ewe lambs there appears to be little improvements in fertility or reproductive rate from joining beyond 7.5 to 8 months of age and 45 kg. Joining at 9 months of age does increase the survival of the progeny from ewe lambs by 4 to 5% per month (Thompson unpublished data), but obviously delaying joining reduces the recovery time to the hogget joining so is not a practical option. Achieving 45 kg at joining also maximises the survival of progeny from ewe lambs, however if 35 kg lambs do get pregnant the survival of their progeny is only 4 to 5% lower. Mortality of the ewe lamb also only varied from 2 to 4% across the range from 6.5 to 9 months of age and 30 to 50 kg at joining. We have also developed predictive models relating to management of ewe lambs post-weaning to optimise their hogget performance, but some knowledge gaps remain such as the effects of liveweight profile during pregnancy on the performance of the ewe lamb and her progeny.

The most recent project has used whole farm systems modelling to develop management guidelines to improve the reproductive performance of ewe lambs for different regions and times of lambing. The modelling indicated that joining maternal ewe lambs was profitable in all regions and times of lambing that were examined. The increased profitability from joining Maternal ewe lambs were greater than the benefits from mating Merino ewe lambs and greater for longer growing season environments regardless of ewe breed. In all analyses it was more profitable to join ewe lambs at 8 months of age rather than at 7 months of age and for the Maternal ewe lambs it was always more profitable to mate all ewe lambs. This was generally the case Merino ewe lambs as well, however the marginal return was often low when more than 50% of Merino ewe lambs were mated and therefore not mating a proportion of the Merino ewe lambs maybe a practical solution for farmers to reduce costs. The economic optimum liveweight at joining was generally about 75% of their mature weight and for all scenarios feeding to gain more weight during joining itself was more profitable. Selling the dry Maternal ewe lambs was always more profitable whereas it was generally more profitable to retain dry Merino ewe lambs. This analysis has also informed the development of a decision support tool for consultants and farmers to help them decide whether mating ewe lambs is a technology that they should be evaluating compared to other production alternatives. The DST also determines the optimal management system if mating Merino or Maternal ewe lambs together with the impacts on profitability of sub-optimal management. The DST will allow farmers to focus their decision-making and management on components of their production system that will provide the greatest financial returns.

Sensor technology to determine maternal pedigree

Greater knowledge of dam pedigree could have benefits to both ram breeders and commercial sheep producers regardless of breed. Ram breeders making the most rapid genetic gain across a range of traits more often record full pedigree, and this is especially the case for Merino breeders. More rapid genetic gains are possible because access to dam pedigree slightly improves the accuracy of breeding values plus enables adjustment for maternal effects such as dam age, pre-weaning environment such as birth type/rear type and the lambs own data such as date of birth. Most significantly, dam pedigree enables the generation of breeding values for reproduction traits, and potentially lamb survival in the near future. The current estimate is that less than 10% of Merino rams are sold with breeding values for number of lambs weaned. For a commercial producer, knowledge of dam pedigree and hence a dams rearing ability could also enable more accurate culling of non-performers and within generation improvements in weaning rates. This lack of dam pedigree is due to a large degree to the high cost and labour intensive nature of collecting maternal pedigree. This project tested the hypothesis that proximity sensors could accurately and rapidly determine maternal pedigree under commercial farming conditions.

The study measured the success of proximity sensors to match lambs to ewes with 40 flocks involving more than 15,000 ewes and lambs across southern Australia. The project aimed to compare dam-pedigree determined using proximity sensors to dam pedigree determined using other methods, including DNA profiling (n = 10), lambing rounds (n = 16), pedigree matchmaker (n = 4), yard mothering up (n =2). Ewes and lambs were typically fitted with proximity sensors for 2 to 4 days under normal farming conditions. This included flocks varying in ewe age (adults, hoggets and ewe lambs), lamb age (up to 125 days old), birth type (singles, twin or triple), paddock size (1 to 300 ha), mob size (30 to 400 lambs) and hence stocking rate (1 to 40/ha). Overall the average success of proximity sensors at matching a lamb to a ewe was 94.7% across the 40 flocks, and this increased to 96.4% after removal of three farms in which there where known problems with the protocol. The success rate was 97.3% across 20 farms that used lambing rounds or DNA to establish dam pedigree. Sensor success was 90% after 10 hours of wear time increasing to 95% after 22 hours, and the success rate of matching a lamb to a ewe were not influenced by ewe age, birth type, paddock size, mob size or stocking rate. Only one site had lambs above 100 days with sensor success of 89% for lambs between 80 and 125 days reducing to 48% for lambs older than 125 days presumably due to self-weaning. Sensor identification of dam matched farm pedigree information in 95.4% of lambs across 26 flocks (about 5000 lambs), and in comparison to each method this match was 97% for lambing rounds, 96% for DNA profiling, 91% for pedigree matchmaker and 84% for yard mothering up. The results show that proximity sensors can rapidly and accurately establish maternal pedigree and the method is robust across a range of conditions experienced on commercial farms. Private industry can now develop more cost effective sensor technologies with greater confidence that will enhance recording of dam pedigree and hence the rate of genetic gain across the sheep industry.

Current Research to improve lamb survival and reduce wastage

The role infectious disease in reproductive wastage of young ewes

There is substantial evidence to suggest reproductive wastage is greater in young ewes joined as either lambs or hoggets. The causes of foetal loss during pregnancy and the neonatal period in this age group are not well understood, however infectious disease (bacterial, viral, fungal) are often implicated. Furthermore, the specific timing, distribution and magnitude of these losses in young ewes across farms are poorly defined. In regards to infectious disease, the impacts on pregnancy are most severe when naïve exposure occurs during pregnancy, which may be a risk factor in maiden ewes. This project will utilise serology to determine antibody response at certain times throughout gestation. This will allow determination of exposure to particular infectious agents as well as timing of infection. The project seeks to determine the extent, timing and cause of foetal loss in young ewes, and its contribution to overall reproductive wastage between scanning and marking. A longitudinal study will be conducted on commercial farms located across WA, SA and VIC over consecutive years to account for variation across seasons and years. The project will focus on a combination of maternal ewe lambs (aged 7-8 months) and a mix of maternal and Merino maiden hoggets (aged 19-20 months), with a minimum of 5 mobs for each age group per state per year. Ewes will be weighed at joining. Body condition score will be measured, and blood samples collected on five occasions between joining and marking. Pregnancy diagnosis will be conducted at day 42-60 of gestation to determine foetal number and again at day 90-100 of gestation to determine the viability of the foetus(es). The number of live lambs will be recorded at lambing and again at marking. Where available, post-mortem examinations and tissue analysis for aborted and stillborn lambs will be used to determine likely cause of death. Initially, marking blood samples will be screened for evidence of exposure to the following infections: Toxoplasmosis (*Toxoplasma gondii*), Neosporosis (*Neospora caninum*), Campylobacteriosis (*Campylobacter fetus* ssp. *fetus, Campylobacter jejuni*) and Q fever (*Coxiella burnetti*). Previous blood samples (joining, day 42-60, day 90-100, day 140 of gestation) will be analysed only for ewes that have evidence of exposure (seroconversion) at marking to determine timing of infection and evidence of active infection relative to stage of gestation.

Boosting lamb survival by supplementing ewes with vitamins and minerals

Lambing in southern Australia ranges from autumn to spring and during this time ewes can experience deficiencies in Vitamin E (Vit E), selenium (Se) and/or Vitamin D (Vit D) depending on region and time of lambing. Vit E and Se prevent oxidative stress in the body, and Vit E also improves immune function and prevents disease. Changing to aerobic metabolism at birth can result in oxidative stress in the lamb and this can influence survival. Hence, low levels of Vit E and Se would confound the ability of the lamb to cope with oxidative stress at birth and compromise survival. Vit D is traditionally recognised for its roles in bone health but has more recently been linked to reproduction outcomes, foetal growth and development, immune function and disease in humans and animal models. This project follows on from the 'Enhancing immune competency to improve lamb survival' project funded by MLA which tested the effects of maternal supplementation with Vit E plus Se (Vit E/Se), Vit D or methionine during late pregnancy on immune responses and lamb survival. Whilst the effects of supplementation on the immune responses of lambs were small, supplementation with Vit E/Se and Vit D did appear to improve lamb survival to weaning (81% *vs*. 70%)[21,22]. The current project will establish on-farm research sites across southern Australia to validate these findings under commercial conditions.

The research involves two phases each involving a total of 30 farms. Phase 1 commenced in 2018 and surveyed farms assessing the levels of vitamins D and E along with selenium in soils, pastures and ewes during late pregnancy. Phase 1 confirmed that ewes lambing in autumn may be at risk of Vitamin E deficiencies as they graze pastures and stubbles with low levels of Vitamin E. Whilst supplementary feeds typically contained low levels of Vitamin E, the minimum requirements for Vitamin E appeared to be met through the variety of feeds consumed given ewes were of adequate Vitamin E status. Overall, Phase 1 has shown that ewes in late pregnancy that are consuming a variety of feeds, with or without vitamin or mineral supplementation, do not appear to be at significant risk of Vitamin D, Vitamin E or selenium deficiencies in southern Australia. However, the data collected is from a single time point and therefore it is unknown whether the status of the ewes along with the forages were inclining, stable or declining in concentration. The high metabolic demands of ewes during late pregnancy and lactation, in addition to the oxidative stress experienced by ewes and lambs at birth, means suboptimal vitamin and mineral status of ewes could contribute to poor ewe and lamb survival. However, supranutritional doses of the supplements could be required to achieve maximal benefits for ewe performance and lamb survival, rather than simply addressing deficiencies that correct clinical or subclinical disease in ewes. Phase 2 will commence in 2019 and over 3 years ewes will be supplemented with large doses of vitamin D, E and selenium during late pregnancy to assess the effect on lamb survival.

Managing fecund flocks to improve survival of triplet dams and their lambs

Many sheep producers have focused on increasing the reproductive performance of both Maternal and Merino ewes during the last decade through better ewe nutrition, management and selection. This has led to an increase in the percentage of triplet bearing ewes and these ewes and their lambs are at higher risk of mortality. Data on the mortality of triplet lambs is limited but it is likely to be in the order of 45 to 55%. The potential for higher rates of mortality in this cohort limits potential productivity gains and represents a downside risk if animal welfare practices do not align with consumer and community expectations. There has been little investment in developing strategies to reduce the mortality of triplet bearing ewes, and feedback from leading sheep meat producers across Australia is that the mortality of triplet bearing ewes and lambs is a growing issue that is not being adequately addressed by any R&D program. Whilst management guidelines based on ewe CS targets at lambing and feed on offer and mob size during lambing have been developed for single and twin bearing Merino and maternal ewes, these guidelines for triplet bearing ewes are less robust[23]. This review concluded that before such guidelines can be developed further research was needed to examine the impacts on both the triplet-bearing ewe and her lambs of varying feeding regimes in both pregnancy and lactation across a broad range of ewe condition scores and environments. In addition, they concluded that knowledge of the impacts of shelter and other paddock characteristics, stocking rate, mob size and human intervention are required.

This project commenced in 2018 and with a literature review and significant consultation with industry to better understand the needs of producers with fecund ewe flocks and their R&D priorities. The industry consultation included focus groups in Australia and New Zealand, in-depth interviews and almost 100 benchmarking surveys from farmers whom separate and differentially manage triplet-bearing ewes and those who do not normally manage triplet-bearing ewes separately. The five highest priorities identified from this process included (i) demonstration of the benefits from management of triplet ewes separately during late pregnancy and lambing compared to mixing triplet ewes with twins; (ii) CS targets at lambing; (iii) mob size targets for lambing; (iv) FOO targets for lambing and impacts of grain supplements; and (e) mineral supplementation. Up to 70 on-farm participatory research sites will be established in 2019 and 2020 to test the potential for these strategies to reduce the mortality of triplet bearing ewes and their lambs.

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Aphid associated photosensitisation

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Abstract
An extensive outbreak of photosensitisation in sheep, cattle and horses was observed across South Australia's Eyre Peninsula and Northern Adelaide Plains during the spring of 2017 and isolated cases in horses again in spring 2018. It was estimated that approximately 25,000 sheep developed photosensitisation following the ingestion of predominantly aphid-infested legumes during the episode. Stock owners reported lambs and adult sheep with swollen ears and faces, and visibly sunburnt facial skin.

The condition rapidly resolved when affected sheep were moved to fresh paddocks, although a small number of severely affected sheep died. Commonly observed heavy infestations of aphids on the grazed pastures concurred with reports in the literature of primary photosensitisation associated with the ingestion of aphids first recorded in horses and cattle in Pomerania, Germany in 1841.

Introduction
An extensive outbreak of photosensitisation in sheep was observed across South Australia's Eyre Peninsula and Northern Adelaide Plains from late September to November in 2017 (refer Figure 1). Stockowners commonly reported lambs and adult sheep with swollen ears and faces, and visibly sunburnt facial skin (refer Figure 2). These sheep had been grazing a variety of pasture types, but predominantly pastures with a high content of legumes such as medic (*Medicago spp*) and vetch (*Vicia sativa*). Some mobs were also affected while grazing green cereals.

Most producers sought advice from their animal health advisors and moved affected sheep from the pasture currently being grazed. The syndrome rapidly resolved although a small number of severely affected sheep died. Testing conducted on affected sheep indicated some with raised liver enzymes and hepatopathy was noted in two severely affected animals (refer Table 1).

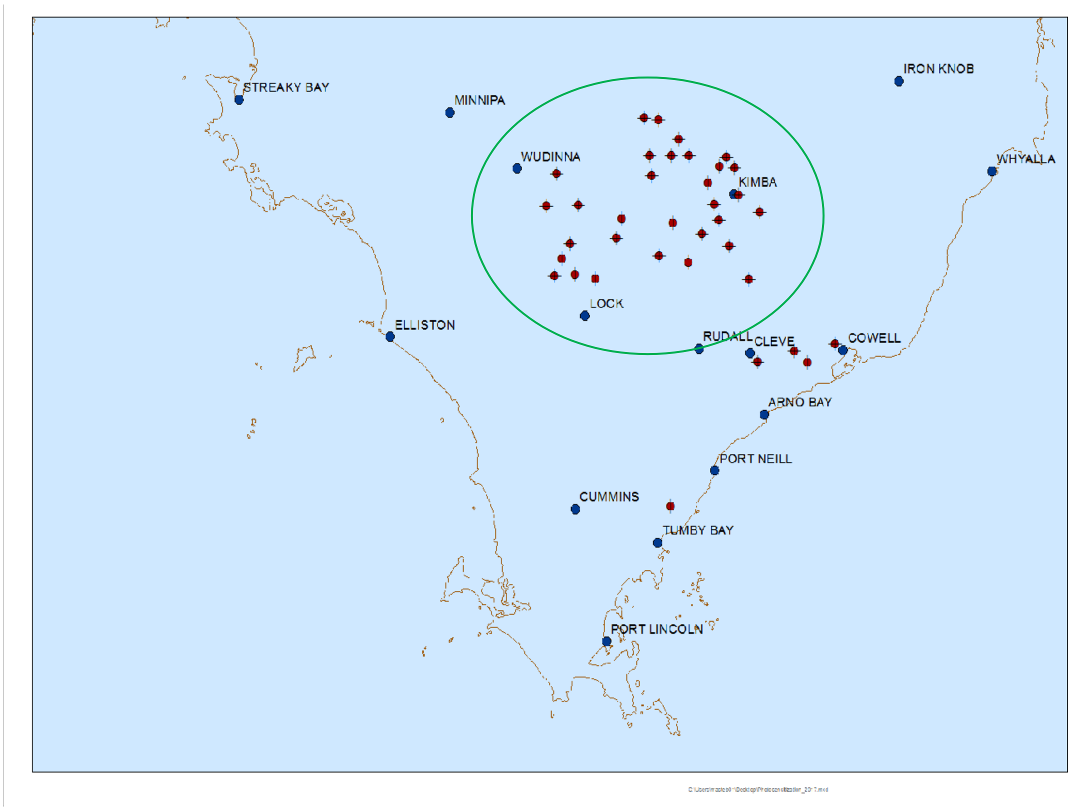
Producers commonly observed heavy infestations of aphids on the grazed pastures raising the spectre that aphid consumption was contributing to the condition (refer Figure 3). Other producers observed black smut like fungal growth near and on some plant species and a suspicion of mycotoxin induced hepatopathy was also speculated. However, some affected producers did not report the presence of either a heavy aphid infestation or fungal issues.

Primary photosensitisation in livestock is reported sporadically in association with consumption of photodynamic agents such as perloline in perennial ryegrass (*Lolium perenne*), hypericin in St. John's Wort (*Hypericum perforatum*) or lush forage such as lucerne (*Medicago sativa*), clover (*Trifolium spp*), vetch and oats (*Avena sativa*) with accumulated phylloerythrin.[1] Usually the condition occurs in small numbers of sheep on legume monocultures. On this occasion, an estimated 25 000 sheep on mostly legume pastures were affected over a short period. This condition has never been reported on this scale in South Australia. Disease investigations ruled out exotic diseases such as bluetongue and confirmed many cases as primary photosensitivity.

History

Cases of photosensitisation in sheep began to be reported to PIRSA (Primary Industries and Regions South Australia) in late September 2017. Reports and concern escalated through October as more sheep and properties were affected. PIRSA Animal Health collected data and blood and plant samples from 8 properties on the Eyre Peninsula. Another 17 affected properties were reported via Landmark agents while there were many more third party reports of affected properties.

Figure 1: A sample of Eyre Peninsula properties with reported cases of photosensitivity.



The syndrome was reported in ewes, lambs and wethers grazing annual ryegrass (*Lolium multiflorum*), vetch, vetch and medic, and medic pastures. Six of nine producers interviewed reported the presence of aphids, but with varying levels of infestation. One producer reported a black smut or mould growing in association with medic plants.

Skin lesions observed included swollen ears, swollen lips & face, scabby lips and ears (refer Figure 2), severe conjunctivitis, blindness and deaths in a small number of sheep. Most affected sheep recovered quickly when removed from the affected pasture. The recovery of some sheep was attributed to corticosteroid treatment and confinement in shearing sheds away from sunlight. More severe cases had crusty, thickened skin on the face, lips and ears and failed to grow well after being affected.

Figure 2: Examples of photosensitivity lesions in affected sheep on Eyre Peninsula

Some producers reported that they managed the problem by controlling aphids in pastures using insecticides such as LeMat 290 (Bayer) before reintroducing sheep. The aphids were identified as cow pea aphids, *Aphis craccivora* – see Figure 3.

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Figure 3: Photo of *Aphis craccivora* infestation on a legume stem

Test results

Four post mortem examinations were conducted by PIRSA, and a private veterinarian conducted one investigation. Eleven blood samples were collected from nine properties and the results are summarised in Table 1..

Table 1: Summary of sampling results from 9 properties affected by photosensitisation.

Collection Date	Animals affected	Samples submitted	Pasture type	Aphids observed	Liver pathology
29/09/17	lambs (on ewes)	blood	ryegrass	No	No
3/10/17	ewes & lambs	blood	vetch & medic	Yes	mild
3/10/17 11/10/17	ewes, lambs, wethers	blood blood, liver	vetch	Yes	yes
3/10/17	lambs only	blood	medic	No	mild
3/10/17	ewes & lambs	blood	medic	Yes	mild
12/10/17	ewes & lambs	blood liver	medic	Yes	mild
12/10/17	ewes & lambs	blood liver	medic	Yes	mild
12/10/17	ewes & lambs	blood liver	medic	Yes	moderate/ chronic
28/9/17	ewes & lambs	blood liver	?	?	yes

Diagnosis

Primary Photosensitivity

Most blood samples submitted showed only mildly elevated liver enzymes indicative of mild hepatic injury. On this basis primary photosensitisation is suspected.

Secondary Photosensitivity

Preserved liver samples in two cases of severely affected sheep did show evidence of hepatopathy with changes consistent with those observed with secondary photosensitisation. This suggests that in at least some of the severely affected sheep, liver damage was likely to have predisposed to the severity of the clinical signs.

Discussion

Sporadic primary photosensitisation in sheep is well documented and known to be associated with particular plants and crops at specific stages of development. The condition normally affects small numbers of animals within a group, some more severely than others, and is often associated with very good growing conditions.[2] Growing conditions across most of the Eyre Peninsula had generally been poor prior to Spring, with well below average biomass production across the region. Above average rainfall in August with a warm Spring produced good fodder growth on some of the affected properties, particularly in the Kimba area. Not all affected properties experienced this above average fodder growth. Sporadic, individual cases of primary photosensitization associated with fast growing, high protein pastures are reported in this area from time to time. The scale of this outbreak and the concurrent severe aphid infestations have not been reported previously in SA.

In this episode an estimated 25,000 sheep were affected across a wide area, with predominantly face and ear lesions observed in young and adult sheep. In contrast to the report by Salmon *et al* no feet lesions were observed.[2] Co-involvement of parapox virus infection (scabby mouth) was considered, but only confirmed in one case. In addition, exotic diseases that may involve face lesions such as vesicular diseases and bluetongue were excluded on six properties based on serological screening. Only sheep were reported to be involved in this episode, and mainly Merino or Merino cross. However, horses were observed with similar clinical signs and circumstances in October 2018 in the Ceduna area on Eyre Peninsula.

Primary and secondary photosensitisation have been described from a wide variety of green leafy plants, brassicas (*Brassica spp*), millets (*Panicum spp*), medics, lucerne and grasses.[3] Primary photosensitisation is due to the ingestion of exogenous photodynamic agents.[3] These substances are activated by sunlight leading to local dermatitis, cell death and the clinical signs decribed. Livestock are generally affected 4-5 days after going on to pastures and new cases cease when removed from the pasture affected. All species of grazing livestock may be affected but there can be variation in individual and species susceptibilities.

In some of these cases, sheep appeared to respond to injected corticosteroids and/or being confined out of sunlight in shearing sheds. Some lambs appeared to be severely affected (possibly due to secondary liver damage) and lost considerable condition and value (pers comm).

Secondary photosensitisation occurs when phylloerythrin the end-product of chlorophyll metabolism is unable to be excreted in the bile due to liver damage.[4] This may be a result of fungal toxins such as *Phomopsis leptostromiformis* in lupinosis or plant toxins such as pyrrolizidine alkaloid in Heliotrope (potato weed) poisoning. These toxins damage the liver causing phylloerythrin to remain in circulation and become activated by sunlight to produce clinical signs similar to primary photosensitivity.

Typically, severe cases of secondary photosensitivity do not respond well to treatment, are predisposed by liver damage detectable on blood tests (significant elevation of GGT and AST) and may be fatal. Livestock disease investigations on the Eyre Peninsula sometimes confirm cases of secondary photosensitisation, usually associated with Lupinosis or Heliotrope poisoning. The history of such cases occurring in the region and the occasional reports of jaundice or cirrhosis from abattoirs suggest that flocks sometimes include sheep with chronic liver damage. In cases of predominantly primary photosensitisation, individual sheep or even individual mobs may present with more severe signs of secondary photosensitivity, possibly due to underlying chronic liver pathology from their grazing history.

While there is no peer reviewed literature that proves causation of photosensitivity due to aphid consumption in sheep, there are several papers that associate the presence of aphids with the incidence of photosensitivity.[5,6,7] McCarthy and Tucker reported on cases in the literature from Pomerana, Germany in 1841 (and earlier in the case of cattle) where horses grazing vetch infested with aphids were severely photosensitised but no deaths occurred.[7] McClymont and Wynne proposed the possibility of aphids causing photosensitisation in sheep in NSW based on the work of Duewell *et al,* who demonstrated the presence of strong photodynamic pigments in some species of aphids, but they were unable to conduct their own research to prove this.[6,7]

Ferrer *et al* investigated whether photosensitivity in sheep grazing Lucerne was due to *Aphis craccivora* and/or seven-spot ladybirds (*Coccinella septempunctata*) larvae.[8] These authors concluded that the aphids were not implicated in the photosensitisation cases, while the ladybird larvae were.

The biochemical and other defence mechanisms that plants have evolved to protect themselves from insect and mammalian herbivores are well reported.[9,10] These anti-grazing attributes in plants either reduce their palatability, reduce their digestibility, or induce toxic effects when consumed. Some of these attributes are induced by particular seasonal conditions or by grazing pressure (including herbivorous insects), or by an interaction between these factors and growth stage. Launchbaugh *et al* describe how grazing animals have developed mechanisms to contend with the anti-grazing attributes of plants.[10] They discuss how grazing animals manage potentially harmful plant compounds by either: grazing selectively - diet selection skills involve cautious sampling, consuming a varied diet and consuming plants in a cyclic, intermittent or carefully regulated pattern; or possessing internal systems to detoxify or tolerate ingested plant toxins.

The ecological interactions which may have contributed to this animal health episode are not well understood. In contrast, the ability of sheep and other grazing animals to protect themselves from the harmful aspects of plants has been studied at length.[10] These protective grazing strategies tend to be less available to animals grazing pastures with less species diversity. The mix of plant species available to grazing livestock varies with seasonal conditions, agronomy, ecology, soil nutrition or interactions between any or all of these factors.

In cases where animals have chronic liver damage (due to longer term exposure to toxins) their ability to detoxify recently ingested material will be compromised, and signs of toxicity will be more pronounced and slower to resolve. Cereal hay without green matter is suggested as the safest feed for recovering photosensitivity affected animals.[4] To safeguard against photosensitivity-risk situations, one option may be to background livestock on cereal hay prior to grazing risky fodder, and to continue feeding palatable hay throughout the risk situation.

In the episode described affected producers contacted a range of industry sources for advice and assistance, although there were also many producers who did not. If this had been an exotic or new disease incursion, it is pleasing that many producers did seek advice from their animal health contacts. Stock agents were quick to contact PIRSA and funding was provided to producers to assist with investigation and testing. PIRSA Animal Health liaised with producers and collected samples for subsidised veterinary pathology testing and diagnosis. SA Sheep Connect (an industry / government partnership) also organised a webinar at short notice to inform South Australian livestock owners of the photosensitvity episode and advise how to resolve it.

Conclusion

The sporadic nature of the episode described limited the opportunity to test causality of the aphid association with photosensitivity. While there is signficant evidence of this association in the literature the question remains as to whether it will ever be conclusively proven.

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Australian wildlife – gaining experience in the field.

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Introduction

Gaining field experience with Australia's wildlife is not only a very rewarding experience, it can be beneficial for improving your knowledge of particular species and their habitats, as well as enhancing your wildlife handling skills and knowledge when treating wildlife in clinical practice. Field experience can also provide useful wider context for understanding the current threats facing Australia's threatened wildlife species. This seminar will provide insight into opportunities available to expand your field experience with Australian wildlife.

Opportunities to gain field experience with Australian wildlife will vary between states and territories. In this presentation, many of the opportunities and examples discussed are from a Western Australian perspective. For those based elsewhere across Australia who are seeking wildlife field experience opportunities, local research is encouraged to seek out local organisations and opportunities relevant to the wildlife species you are hoping to gain experience with.

Why and how

Your first-hand experience with Australian native wildlife may vary depending on your exposure to wildlife in clinical practice, the amount of wildlife related training included as part of your university studies, or your personal contact with wildlife through your life. The impetus to gain field experience with wildlife, may be to become familiar with handling specific species in clinical practice, or in emergency situations such as bushfires, or to build your skills and knowledge towards developing a career working with Australian wildlife in a dedicated wildlife hospital, rehabilitation facility, wildlife park, zoo or in wildlife conservation related research.

One of the most important points to make about seeking out field opportunities with Australian native wildlife – is to develop your contact network. Research and discover what vets and scientists are working with the wildlife species that you're interested in learning more about, and express your interest in any opportunities to assist them with their work. Remember that cultivating your contacts and any opportunities may take some time, so be persistent, determined and passionate about your goals.

Another thing to consider is to determine the amount of time you have available to devote to field opportunities, as the duration of activities can vary from days to weeks to months. It's also important to consider whether insurance is provided as part of the field work, or if you may need to organise your own. Depending on the type of wildlife you may be working with, be aware of any potential zoonotic risks (eg bat lyssavirus), and incorporate appropriate personal protective equipment into your field preparations.

Opportunities

The first area to explore for native wildlife experience is within the veterinary industry. Consider working or volunteering within a veterinary hospital that sees a large amount of wildlife – these practices may be located in outer-metropolitan areas or in regional areas. Some veterinary practices also service local wildlife parks, and some zoos and wildlife parks have their own dedicated veterinary hospitals where it may be possible to undertake work experience, internships, residencies and locum employment.

Wildlife rescue and rehabilitation centres offer a fantastic opportunity for exposure to Australian native wildlife. There may be opportunities to assist with rehabilitation or veterinary care, including accompanying more experienced vets for mentoring. Closely related to this are wildlife conservation opportunities offered through universities, such as residencies and post-graduate research projects. These programs may be more intensive and require a full-time commitment, however with the benefit of accumulating both theoretical and practical experience and potentially achieving a further qualification. Other scientific organisations such as museums, marine institutes and research organisations may offer opportunities to assist with field programs or research.

Each state and territory in Australia has a Government conservation or parks and wildlife department, with many of these offering volunteer field work opportunities. Similarly, you can also consider state fisheries departments for marine research opportunities, and Federal scientific organisations such as the Australian Antarctic Division for marine science opportunities. If you access your state Government's parks and wildlife department website, they also often have resources and links to community conservation organisations which focus on specific wildlife species or local habitat areas. This is another sector of the wildlife conservation space to explore for wildlife-related field opportunities, and includes 'Friends of' groups, local Landcare groups and natural resource management (NRM) groups.

There are several not-for-profit conservation organisations working across Australia such as the Australian Wildlife Conservancy and Bush Heritage Australia, which offer volunteer opportunities to participate in ecological field work involving Australian wildlife. The Australian Wildlife Conservancy also offers seasonal internships working in the field. Conservation Volunteers also coordinate projects where there may be opportunities to gain field experience with native wildlife. There may also be opportunities with ecological consultancies, assisting ecologists with undertaking fauna surveys and other research.

Professional development opportunities in wildlife conservation may also become available through continuing veterinary education providers and universities, and through courses run by Wildlife Health Australia, Wildlife Disease Association Australasia and other wildlife focused organisations. Wildlife Health Australia and Wildlife Disease Association Australasia are also great resources for hearing about opportunities with Australian native wildlife and connecting with vets with similar interests.

The experience

Once you have identified any opportunities of interest, participating in field work can be rewarding in terms of the exposure to native wildlife and improving your knowledge of particular species, together with expanding your contact network. The work may not be directly veterinary related, however the benefits of becoming familiar with handling native wildlife species, and learning from scientists working directly with species conservation, can provide useful learning and knowledge to take back to your clinical veterinary work. The native wildlife experience may also aid your career progression towards working with Australian wildlife in a veterinary capacity, opening doors to future opportunities.

The following resources are just a few examples of links which may be useful in researching opportunities to gain field experience with Australian wildlife:

Wildlife Health Australia (WHA) https://wildlifehealthaustralia.com.au/
Wildlife Disease Association – Australasia (WDA-A) https://www.wildlifedisease.org/wda/SECTIONS/Australasian.aspx
Zoo and Aquarium Association (ZAA) https://www.zooaquarium.org.au/
Perth Zoo https://perthzoo.wa.gov.au/
Taronga Zoo https://taronga.org.au/
Zoos South Australia https://www.zoossa.com.au/
Zoos Victoria https://www.zoo.org.au/
Australia Zoo https://www.australiazoo.com.au/
Native Animal Rescue https://nativeanimalrescue.org.au/
Native Arc https://www.nativearc.org.au/
Kanyana Wildlife Rehabilitation Centre https://kanyanawildlife.org.au/
Kaarakin Black Cockatoo Conservation Centre https://www.blackcockatoorecovery.com/get-involved/volunteer/
Department of Biodiversity, Conservation and Attractions (DBCA) https://www.dpaw.wa.gov.au/get-involved/volunteering-opportunities/506-current-volunteering-opportunities
Australian Wildlife Conservancy (AWC) https://www.australianwildlife.org/support-us/volunteer/
Bush Heritage Australia https://www.bushheritage.org.au/get-involved/volunteer
Conservation Volunteers https://bookings.conservationvolunteers.org/
Australian Antarctic Division http://www.antarctica.gov.au/
WA Museum http://museum.wa.gov.au/about/work-experience-volunteering
Project Numbat http://www.numbat.org.au/
Friends of the Western Ground Parrot http://www.western-ground-parrot.org.au/

Conclusion

If you are interested in pursuing a career in the wildlife conservation area, gaining field experience with native wildlife will be beneficial in terms of improving knowledge, practical skills and providing a broader understanding of the issues affecting Australian wildlife and their habitats. Field opportunities may be formal or informal, advertised or through word-of-mouth. By investing some time in researching opportunities and expanding your contact network through expressing your interest to those already working in this space, you will hopefully uncover rewarding field experiences with Australian wildlife to enrich your learning and your veterinary career.

Science communication to enhance community engagement in wildlife conservation.

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Introduction

We live in a busy world with lots of news, issues and events competing to capture everyone's attention. For those working in wildlife conservation, having an effective science communication strategy can make a significant difference when seeking to engage with the community about wildlife conservation issues and research.

Many aspects of wildlife conservation work can benefit from effective science communication. This might include raising the profile of threatened species or habitats, explaining the importance of research aims and outcomes, or building support, engagement and understanding within the community.

This seminar explains the options in the science communication toolbox, providing an overview of the different ways that those working in wildlife conservation can communicate effectively with a wide audience.

Developing a communications strategy will help you tell your story effectively in multiple media platforms. Science communication options include traditional print, radio and television media, social media, websites, citizen science projects, crowd-funding, publications, community presentations, face to face meetings and on-ground interpretation.

Using some or all of these methods to share the conversation about wildlife conservation, can help raise the profile of wildlife conservation issues, generate funding support, whilst providing opportunities for public education and community engagement.

The result of a good science communication strategy, is hopefully greater community awareness and support, contributing to enhanced conservation outcomes and highlighting the importance of wildlife to the community.

The importance of science communication

Explaining and communicating your science to non-scientists should be one of your most important jobs.[i] If you can capture the imaginations of non-scientists with your passion and enthusiasm for nature, then you are part way there in helping to bridge the gap to understanding scientific facts. It's important to explain your research in terms and ideas that your family or next-door neighbours can readily grasp.[ii] Use simple language, finding a balance between a scientific and conversational tone, whilst striving to be educational as well. Don't just rely on figures and statistics, bring numbers to life with examples and stories.

As wildlife veterinarians and conservation biologists, wildlife conservation work is often focused on researching or treating specific species or disease processes. Whilst your work may often be specialised or narrow in scope, it is important to maintain a wider picture, or landscape level view of how this research or work fits into the broader ecosystem. The reason this is important, is because research has shown that conservation biologists need to be proactive in increasing environmental understanding in the community through effective science communication and outreach - to help bring about the societal changes needed to reverse the trend of widespread ecological degradation and species extinction.[iii]

Place your wildlife conservation work in context. Consider the wider picture when looking at how to frame your conservation message or research – place it within an ecosystem, habitat or food chain, as focussing on a threatened species could be seen to be highlighting a symptom rather than the cause of an issue.[iv] It's worth remembering, that utilising scare tactics and saying an animal species is about to go extinct, can be counterproductive as the audience may switch off. It's important to get people thinking about ecological functions in a broader context for the long term.

According to research from Cornell University, researchers found that what scientists think statistics say about a species' risk of extinction, and how the public perceives that risk, can often be strikingly different. If you want people to care about endangered species, focus on how many animals are left, not on the chances of a species becoming extinct.[v] This point also highlights that if you have an important message you are planning to share through science communication, gain feedback on how the message is understood from a lay persons perspective before you unleash it to the general public – partners, parents, friends, can all help you when you're crafting your science communication content.

If you get the community on board with your wildlife conservation work, the benefits not only include increased awareness of the conservation issues, but community engagement also fosters two-way communication and knowledge sharing, where the community can become bonded to an ecosystem or animal through their experiences and participation in conservation activities.[vi] So it can be beneficial to find a balance between top-down outwards communications, and two-way participative communication.[vii] This also includes considering how you are portraying yourself and where you are located – engage with the local community and highlight your local connection, and be aware that if you are an 'outsider' working in an area, you'll need to look to strengthen your local community bonds to aid your engagement.[viii]

Science communication – where to start

The first thing to consider when looking to share your wildlife conservation story, is to develop a communications strategy or plan. This strategy should be designed to help you and your organisation communicate effectively to share scientific information, whilst meeting any core organisational objectives as well (such as stakeholder engagement, promotion, fundraising and business development).

A communications strategy can be useful whether you're working as part of an organisation, or if you're working individually on your own research and wildlife conservation activities. Having a strategy, will help you identify your key communication messages, which you can then build on when undertaking science communication activities.

The communications strategy should incorporate a brief overview of what you do, why you are doing it and where you are doing it. You can also consider reviewing what you are currently doing in terms of any science communication and how successful this has been.

- Perform a simple SWOT analysis, which looks at your communications strengths, weaknesses, opportunities and threats. Think about this in terms of your communications priorities, and whether you can turn threats into opportunities, and use your strengths to build effective communication.
- Look at what others working in the wildlife conservation space are doing, to see what science communication methods are working well, and also consider any potential opportunities for collaboration.

- If you are working in collaboration with other researchers or stakeholder groups, remember to try and coordinate communications where possible, and check with each other on crediting each stakeholder, and use of logos.
- Identify your audience and stakeholders – these can be both internal and external, including colleagues, local community, wider community and general public, industry stakeholders, media, Government, political, and funding bodies.
- When producing a particular piece of science communication, it's important to tailor it towards the particular segment/s of your audience that you are hoping to target. You will likely be communicating more frequently with particular sections of your audience.

Your story and your audience

Consider your science communication methods based on your target audience and what you are trying to achieve (short term gains or long term goals), your budget resources and timeframe. Try to ensure all communication is targeted and necessary, versus communicating just for the sake of communicating.

Think about the wildlife conservation story you want to tell:

- keep it simple – highlight particular aspects of an issue.
- keep it short – try not to pack in too much information, and consider the length of a video, social media post, media release or poster as part of your planning.
- consider what is the purpose of the story, ensure it is active – about doing something.
- incorporate a strong character if possible, whether this is a person, animal or location.
- consider the tone of your science communication – serious, or use of humour.
- use a call to action – something for the audience to do – such as asking for a donation, participation, awareness raising through sharing, or a change in values.
- relate your story to the community, why is it important locally or culturally.
- use images, videos, graphics and animations to explain complex scientific issues simply. Strong and compelling visual media, can help tell the story for you.

Develop your network to help you share your wildlife conservation story. This can include fostering media contacts, key stakeholders and members of the local community. It's important when developing your science communication campaign, that you firstly compile and prepare your aims, resources, promotional material, content and interview talent – so that you have everything ready to go before you 'release it'.

When considering how to get exposure in the media, utilise your contact network to target specific journalists or you can send a media release to the newsroom. It's also useful to think about the issue you are hoping to communicate, and whether it has local, state, national or international significance, and target media accordingly in print, radio, television and online outlets. Before you send out a media release, ensure you are prepared to be interviewed, and if it's not you, then have someone ready and available to speak to the media.

Methods in the science communication toolbox

- Online:
  - Website and blogs.
    - Having a website presence means you have a widely accessible platform to tell your story, plus you can easily include updates about your wildlife conservation work, and have a means of your audience being able to contact you.
    - Ensure your website is easy to navigate and load, and is mobile-responsive, and make use of compelling images and video to highlight the focus of your work.
    - Blogging provides an opportunity to share news and updates, and blog posts can be shared by an email distribution list. If you are blogging, remember to provide regular updates and always cross promote with your social media channels.
  - Social media.
    - Social media provides a virtual community to tap into, with the potential for free marketing, communication and awareness raising. With minimal cost social media offers a more informal style of communication compared to traditional media – allowing you to take your message directly to your audience without intermediaries.
      - Twitter – short text focused platform with images, video and links.
      - Instagram – image or video focused platform with text and tags, but no links.
      - Facebook – longer text-based platform with images, video and links, with more of a community development potential.
      - LinkedIn – professional contacts community supporting text, images and video, and building your contact network.
      - YouTube – video focused platform, which can link to other social media platforms and can also utilise on your website.
    - Social media platforms enable two-way communication – the audience can react, respond, engage and participate – whether positively or negatively. This feature can be ideal for promotional campaigns and fundraising, for asking for volunteer assistance in research projects, or for sharing crowdfunding requests to support wildlife conservation research activities.
    - You can directly target key people such as media, decision makers (politicians, Government), and other influencers in the conservation area or in the community more broadly.
    - Social media platforms vary slightly in terms of what content works best, but in general it's preferable to use simple strong messages accompanied by high quality images or video.
    - Offers the opportunity to bring together like-minded people – to engage and build community around a wildlife conservation issue or organisation.
    - Use social media to drive your audience to your website or blog or crowdfunding platform, and it is an easy platform to incorporate a call to action in your messaging.

        - Develop a social media plan, considering your budget and time available. Your plan can also incorporate the scheduling of content so that you don't physically have to be online when posting to social media. On Facebook, you can also consider a budget to pay to boost posts on Facebook to increase your reach and grow your audience.
        - Monitor your social media platform analytics to see what types of posts work and the optimal times to post to your audience.
    - E-Newsletters and email distribution lists.
        - There are various forms of online newsletters which you can create, which can feature news and updates about your wildlife conservation activities. E.g. Mailchimp, and many others.
        - E-newsletters are shared via an email distribution list which you can compile to target key contacts or supporters who are interested in your work.
        - It's important to keep the content fresh and relevant, accompanied by high quality images or video links. Ensure the platform contains mobile-responsive content so that it can be easily viewed on a mobile phone. Other considerations include, looking to write a strong subject line to catch attention, and consider an appropriate time of day to distribute them to your network.
- Media
    - Media release.
        - Writing a succinct and successful media release is an art in itself. Media releases can be distributed via email to newspaper, radio, television and online media outlets. You need to include contact details and be prepared to be available to be interviewed, and have images and video content ready to supply if requested.
    - Advertisement.
        - Depending on what you are trying to say, a cleverly constructed advert in a newspaper, or on radio, television or online, can still be an effective means of communication, however there is cost involved.
    - Write feature articles.
        - Outside of academic journals, pitching or writing an article for a print or online publication, can provide the opportunity to target a specific audience. Articles can then also be linked to your social media platforms and can lead to further engagement opportunities.
        - E.g. The Conversation website, Australian Geographic Magazine, nature interest publications and websites, etc.
- Publications
    - Print newsletters.
        - Printed newsletters can feature similar content and layout to an e-newsletter, and be used if you have an event, or a reception area where members of the community may visit. They can also be posted to supporters or key stakeholders.
    - Posters, flyers, brochures, books.
        - Depending on your audience, and budget resources, there are many forms of publications which can be used for science communication.
    - Interpretive signage.

        - If you are working on a wildlife conservation project that is associated with a local wilderness area, property or wildlife park, there may be the opportunity to develop and install interpretive signage which can help educate hikers and visitors about specific conservation issues.
- Interactions
    - Community presentations.
        - Presenting a seminar to your local community can be a powerful way of educating and communicating to build awareness.
        - There are a variety of opportunities to consider including schools, professional organisations, corporate groups, community groups, and other organisations such as Probus, U3A, Rotary groups etc.
    - Events – workshops, displays, conferences.
        - Depending on the focus of your wildlife conservation work, there may be engagement opportunities associated with professional development activities, scientific conferences, and community festivals and markets.
    - Citizen science projects.
        - If your wildlife conservation work offers opportunities for the community to participate in field work activities, then developing citizen science projects enables community involvement, enhancing awareness and understanding of wildlife species and wildlife conservation issues. E.g. Local Landcare groups, BirdLife Australia's Great Cocky Count etc.
    - Face to face.
        - Remember that one of the most effective forms of communication can be one on one with key people, which can help you to secure funding, cooperation, research guidance and to develop collaborative strategies associated with your wildlife conservation work.

Conclusion

To improve your science communication, remember to regularly review and evaluate your wildlife conservation science communication activities, to see what worked well and what you can improve on and do differently in the future. You can then incorporate any improvements into your science communication strategy. This evaluation can include analysis of website traffic, social media analytics, media coverage, resulting funding, community response and involvement – and what's been successful in helping to aid your wildlife conservation outcomes.

If science communication is not your strong point, then consider engaging a science communicator experienced with wildlife conservation to assist you to get your message out. Through taking the time to develop a science communication strategy, and thinking about what you are trying to communicate, to whom and how, you can ensure your science communication activities will be effective in terms of time and effort, and most importantly, that your science communication will have the best chance of contributing to improved community awareness and wildlife conservation outcomes.

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Never too old: Geriatric feline anaesthesia for general practice

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Introduction

Too often I hear clients say they have been told by their previous vet that their cat is "too old for an anaesthetic". Our geriatric feline patients shouldn't have to live with painful conditions such as dental disease due to a clinicians' avoidance of general anaesthesia. Treating dental disease has such immediate positive impacts on the cat's welfare, so it is our responsibility to learn how to safely anaesthetise them and treat this reversible source of pain and disease.

Brodbelt identified that cats have an increased risk of peri-anaesthetic mortality (0.11%) compared with that of the dog (0.05%).[1] Major factors that contribute to this increased risk include:

- Cats are often nervous, stressed or fractious in a vet clinic environment
    - Increased circulating catecholamines
    - Increased dose of anaesthetic drugs may be required
    - Assessment of underlying disease may be challenging in the conscious patient
- Cats have a relatively small body size compared to dogs
    - May be difficult to dose drugs accurately
    - Anaesthetic equipment may not suit their small size
    - Anaesthetic monitoring equipment may not work as well
    - Can become hypothermic more easily
- Cats have an increased risk of bronchospasm especially if intubated
- Cats are more sensitive to volume overload from the administration of intravenous fluids
- Cats have some specific drug metabolisms and responses
    - e.g. NSAIDS, opioids

Geriatric cats are defined as cats aged 15 years or more and they have additional anaesthetic risk factors that must be considered prior to anaesthesia.

- Higher risk of anaesthetic related death regardless of ASA status
- Higher incidence of underlying chronic diseases (cardiac, respiratory, liver, kidneys, endocrine)
- Central nervous system changes associated with age include lower cerebrovascular brain flow and reduced function. This means geriatrics are more susceptible to the effects of anaesthetic drugs, more prone to hypothermia, and can get easily stressed if they have geriatric cognitive dysfunction syndrome
- Occult cardiac disease is common, and it has been reported 42.6% of cats over 9 years of age with a heart murmur have clinically significant heart disease[2]
- Overall decrease in respiratory function with age, reduced tissue elasticity and weakened respiratory muscles mean decreased oxygenation
- Estimates of the prevalence of chronic kidney disease in geriatric cats have ranged from 35-81%. Treat the cat's kidneys like a temple, with respect![3]
- Higher risk of gastro-oesophageal regurgitation, higher risk of aspiration
- High incidence of osteoarthritis, underlying musculoskeletal pain

Pre-anaesthetic planning
Cats are masters at hiding disease, and it is imperative that geriatric cats are adequately screened prior to general anaesthesia. Initial assessment with a thorough physical exam including careful respiratory and cardiac assessment, fundic exam, and blood pressure measurement is essential and will help guide what further diagnostics are required.

Cats can become very stressed at veterinary clinics and this can mask clinical signs of underlying disease, interfere with the action of sedatives and anaesthetics, reduce wound healing and recovery times, and most importantly reduces patient welfare. Take proactive steps to reducing feline stress by educating owners on how to safely transport their cat to the vet and adapt their cat to the cat carrier. Include this information on your practice website, social media and have client handouts to distribute.[4]

The AAFP/ISFM Feline Friendly Handling Guidelines and AAFP/ISFM Feline Friendly Nursing Guidelines are excellent resources for your team to further their understanding of what it takes to be feline friendly. One of the key principles for creating a cat-friendly practice include minimising visual and olfactory cues that may lead to anxiety. This includes keeping dogs, cats and other patients away from the cat's line of vision and being proactive to manage odours. If possible, have a separate cat waiting area or move the owner through into a cat-only consult room upon arrival. Encourage owners to cover their cat carriers with a towel and have some clean towels available in reception to offer to owners that do not do this. Use a synthetic feline facial pheromone diffuser (Feliway™) in your waiting area and consulting rooms, and spray Feliway onto any towels used for handling.

Ensure all your veterinary staff are trained in feline friendly handling techniques including avoiding using scruffing for restraint. Approach cats slowly, quietly and gently, avoid direct eye contact and adapt your handling techniques based on their reactions. Many cats feel more secure being examined under a towel. Try to provide hospitalise cats with the ability to hide and perch as this increases their feeling of safety.[5] There is some evidence that classical music reduces stress in cats, and it additionally acts as an acoustic dampener for the potentially stressful background noise of humans, equipment and other animals.[6,7]

For cats that are known to be anxious or fractious, consider pre-appointment treatment with gabapentin. Use of pre-visit gabapentin in cats has been shown to reduce the fear response, decrease stress, and facilitates a more thorough physical examination. The author uses 50-100mg/cat PO (50mg for cats <4kg, 100mg for cats >4kg), and recommends giving one dose of 50-100mg the night before and repeat with another dose of 50-100mg two hours before leaving for the vet clinic. Clients are warned of side effects of sedation and possible salivation.[8]

A retrospective study of pre-anaesthetic blood screening of cats with mean age of 11.6 years reported that 16% had abnormal results that were concerning to the clinician. 1 in 100 cats that had abnormal results on their screening tests had no abnormalities detected on their clinical history or examination. In 9% of all cases tested, the anaesthetic protocols were changed, or procedures postponed because of the screening test results.[9] Thus, pre-anaesthetic screening for the geriatric cat is essential for detecting underling disease that could negatively affect the safety of anaesthesia.

A minimum data base for a geriatric cat prior to general anaesthesia should include a complete blood count, serum biochemistry, total T4, SDMA and urinalysis.[10] Further testing may be indicated based on the clinical examination findings or results of initial testing. Common co-morbidities include heart disease (most commonly HCM), hyperthyroidism, chronic renal disease, diabetes mellitus, lower airway disease (feline asthma), obesity and degenerative joint disease.

If chronic kidney disease is suspected or diagnosed, further staging is warranted with blood pressure measurement (if not already performed) and a urine protein:creatinine ratio, with consideration for abdominal ultrasound if there is renomegaly, irregular kidneys on palpation or there has been a recent significant deterioration in renal parameters.

If a heart murmur is detected, it is ideal to perform an echocardiogram to assess for underling heart disease as nearly half of cats if over the age of 9 years with a murmur will have clinically significant heart disease. If the owner has price constraints and the cat is asymptomatic, screening with cardiac biomarker tests (e.g. Idexx patient side NT-proBNP SNAP test) may be useful however has some limitations and results need to be interpreted in the light of the test's sensitivity and specificity.

- If the ProBNP SNAP test is positive in a cat >9 years of age, this individual cat has a 77.8% chance of having heart disease. This is clearly not a perfect test for heart disease, but it does mean that there is now more than twice the likelihood of heart disease being present in this cat (probability was 42.6%, now is 77.8%).[11]

- If the ProBNP SNAP test is negative in a cat >9 years of age, there is only <10% chance (9.4%) that the cat has clinically significant heart disease.[11]

Other pre-anaesthetic testing may include further imaging as indicated by the individual case details such as thoracic radiography or abdominal ultrasound, however the benefit of these procedures must be weighed up against the added risk for additional sedation and stress to the patient.

Pre-operative fasting is recommended to reduce the volume of stomach contents, and to prevent gastro-oesophageal reflux, regurgitation and aspiration. Traditional industry recommendations are to withhold food for 12 hours prior to the procedure i.e. “nothing after midnight” but this recommendation is not evidence based and there is currently no data in cats to establish the ideal fasting times. The evidence for duration of fasting in dogs is conflicting, with one study suggesting that feeding a small meal of wet food 3 hours prior to an anaesthetic reduces gastric acid reflux, and another study suggesting this is of no benefit.[12,13]

Longer fasting times do not guarantee that the cat's stomach is empty, and it is important to note that gastric emptying rate can be slowed by many factors including stress, meal size, food moisture (i.e. dry food) and comorbidities such as inflammatory bowel disease. There is some consideration for shorter fasting times in cats, and for most patients especially younger cats the author recommends a protocol of feeding the cat until midnight, fasting overnight and then providing a small feed of 1-2 teaspoons of wet food only around 4 hours prior to the anaesthetic. Older cats are more likely to have a slowed gastric emptying rate, so for geriatric cats this fasting recommendation is made on a case by case basis, and some geriatric cats may benefit from longer fasting times.

Geriatric cats are more prone to hypothermia due to their reduced central nervous system function, and lower muscle/fat insulation. Hypothermia has many deleterious effects including:

- Slowed anaesthetic recovery
- Impaired wound healing
- Increased oxygen demand (shivering)
- Decreased need for anaesthetic drugs (risk for drug overdose)
- Immune system depression
- Coagulation dysfunction, sludging of blood, risk of thromboembolism
- Decreased cardiac contractility

Prevention of hypothermia is always preferable than trying to treat it. Pre-warm all geriatric cats with a low-normal or low pre-anaesthetic body temperature from the time of premedication. Avoid administering the premedicant too early prior to the induction time. Once the cat has been induced under anaesthetic, it is important to note that their body temperature will immediately start to drop, so immediate use of active warming devices such as a forced warm air blanket is highly recommended. Ensure to keep the ambient temperature in the surgical room warm and minimise surgical time. If the cat requires any surgical procedures in addition to the dental procedure, use warmed fluids/scrub solutions and try to minimise clipping of the coat.

Premedicant choice

The choice of premedication drugs to use in a geriatric cat will depend greatly on the presence of co-morbidities, the cat's temperament, and the clinician's preference. The premedicant drug regime aims to decrease the patient's stress, reduce subsequent anaesthetic induction doses, provide pre-emptive analgesia and ensure a smooth recovery.

Minimising stress and fear will allow lower doses of drugs to be used and will improve the overall welfare and outcome for the patient. All staff should be trained in feline friendly handling techniques, and there should be a standardised protocol for managing fearful cats from the moment they arrive at the clinic. Providing in-patients with places to hide and perch in their kennels, and avoiding the sight, sound and smell of dogs (and other cats) can have a huge benefit. Try to minimise procedures and administration of drugs in the treatment room as this can often be a noisy and threatening place to a cat.

For calm, easy to handle cats, use of an opioid-only premedicant is often adequate. Methadone is the author's preferred opioid for premedication of geriatrics, as it is quick in onset and it is reversible. (Dose methadone 0.3-0.4mg/kg IM). Note that opioids can cause mild-moderate, self-limiting hyperthermia (<40.1C) and bradycardia in cats.[14] Avoid subcutaneous use of opioids, absorption in cats is slow and less predictable especially with buprenorphine.[15]

For more anxious or fractious geriatric cats, addition of a low dose of midazolam (0.1-0.2mg/kg IM) given with an opioid may better facilitate IV catheter placement. Benefits of midazolam are that it is also reversible. Avoid use of acepromazine in geriatrics due to its hypotensive effects and the risk for a prolonged recovery.

Use of NK-1 receptor antagonist maropitant as a premedicant has gained some popularity due to some evidence for efficacy of NK-1 antagonists against visceral pain. Niyom et al showed that intravenous administration of maropitant reduced the inhalant anaesthetic requirements in cats by 15% during ovarian ligament stimulation when dosed at 1mg/kg IV.[16] Additional benefits of pre-operative use of maropitant include reduction in the occurrence of opioid induced nausea and prevention of aspiration pneumonia. Not all nauseous cats will vomit, but the effects of nausea can lead to distress, food aversion, anorexia and weight loss. Maropitant should be given 1 hour before premedication to allow it to reach peak plasma concentration if given subcutaneously. Note maropitant may sting on subcutaneous injection. Administering maropitant intravenously avoids this pain however is considered off label use in Australia.

Preparation of individualised emergency drug dose charts where all drug doses are calculated for the patient prior to induction are of huge benefit. Drugs to consider including on this chart include atropine, methadone, naloxone, fluids rates, positive inotropes (e.g. dobutamine, ephedrine), adrenaline, lignocaine, and flumazenil. Record all pre-operative parameters (body weight, resting respiratory rate and heart rate, temperature) on the anaesthetic chart for quick reference.

Induction

Choice of induction agent will depend on the patients' co-morbidities, temperament, and clinician's preference. All patients must have placement of an intravenous catheter which can be inserted after the premedicant to minimise stress. All geriatrics regardless of cardiorespiratory function should be pre-oxygenated for 5-10 minutes as geriatrics are likely to have poorer oxygenation due to decreased respiratory function associated with ageing.

Intravenous fluid therapy has been identified as a risk factor for cats undergoing anaesthetic.[1] Potential reasons for this include:

- Potential of occult heart disease
- Small blood volume in cats = 50-60ml/kg e.g. 180ml for a 3 kg cat
- Challenge to accurately deliver smaller volumes of fluids

The recommended intraoperative fluid maintenance rate is 3ml/kg/hr however this may need to be reduced in patients at risk of fluid overload such as with cardiac or severe renal dysfunction. To ensure accurate delivery, use of a fluid or syringe pump is highly recommended.

Ideal induction agents for geriatrics include alfaxalone and propofol. Both these drugs are short acting and titratable, meaning they can be given slowly and to effect to ensure more accurate dosing. Both drugs have similar effects on the cardiovascular and respiratory system. Taboada et al showed that there were no significant differences in cardiorespiratory parameters (mean pulse rate, Doppler arterial blood pressure, respiratory rate, end-tidal carbon dioxide partial pressure) and recovery times in cats when comparing alfaxalone and propofol anaesthesia.[17] Alfaxalone has the additional benefit that it is generally well absorbed intramuscularly. Therefore, alfaxalone can be used at a low dose (1mg/kg IM) for fractious cats to facilitate sedation for catheter placement however its absorption can be variable in some individuals and it is important to take care with cumulative dose rates especially with geriatrics.

Induction of very fractious geriatric cats can be a challenge. The anaesthetic plan for these cats should always begin with avoiding the triggers that get the cat in a fractious state in the first place. Pre-appointment treatment with gabapentin and minimising excessive noise and stimulation for the cat is essential. Many geriatric cats have degenerative joint disease so be gentle with handling if able to avoid causing pain.

Avoid chamber inductions with gaseous anaesthetics, this is a stressful procedure in a compromised patient where one cannot control the dosage. Ensure the veterinary nurse is wearing appropriate PPE if the cat is known to be highly aggressive. The author's preferred technique for handling fractious cats is to leave the cat contained in their carry box, carefully and quickly lift the lid off and cover the cat gently with towel. This will facilitate restraint for injection of and intramuscular premedication followed 10-15 minutes later by a second injection of a low dose of intramuscular alfaxalone. This generally provides adequate sedation in the geriatric patient for intravenous catheter placement and subsequent induction with intravenous alfaxalone. Ensure to reduce the total induction dose and dose slowly to effect.

Intubation of cats for anaesthetics was identified as a risk factor in the Brodbelt study however intubation is unavoidable for dental procedures to maintain a patent airway, prevent aspiration and deliver oxygen, inhalant anaesthetics and supportive ventilation.[1] Most anaesthetic deaths occur within 3 hours after the anaesthetic ends and can commonly be due to airway obstruction. The cat has a small airway and their larynx is highly prone to spasm when manually stimulated. To place an endotracheal tube, use a suitably sized laryngoscope with clear lighting to clearly visualise the arytenoids to help reduce risk of spasm. Lubricate the ET tube with water soluble lubricant and apply local anaesthetic to the arytenoids. Note that commercial products that use a nozzle for delivery of local anaesthetic can damage the delicate laryngeal mucosa and are a risk for spread of infectious diseases such as feline calicivirus. It is preferable to apply 0.2ml total dose of 2% lignocaine (without adrenaline) directly to the arytenoids using a single use sterile 1ml syringe (without a needle or catheter as this can dislodge and become a projectile airway foreign body!).

Dentistry increases risk of tracheal damage and has been reported to be associated with up to 70% of iatrogenic tracheal injuries.[18] Use of a cuff manometer device such as the AG Cuffill cuff inflator can help to ensure high cuff pressures are avoided and are relatively inexpensive. Always disconnect the patient from the anaesthetic before moving the patient and ensure to always firmly tie in the ET tube and check mid-procedure.

Maintenance

Careful monitoring during the duration of the anaesthetic and recovery has been shown to be associated with lower mortality and should be routine for all patients. A dedicated and experienced anaesthetist is essential for ensuring early detection and intervention of abnormalities. For smaller sized practices, this means booking in geriatric procedures when you know you are adequately staffed to do this. In addition to this, frequent and repeated monitoring and documentation of trends is critical.

Anaesthetic monitoring should include:

- Physical observation
  - Heart rate and rhythm, pulse strength and character
  - Respiratory rate, depth and rhythm, look for evidence of fluid overload
  - Mucous membrane colour and capillary refill time
  - Jaw tone, eye position, patient movement, presence of reflexes
  - Response to surgical stimulation
- Essential monitoring equipment
  - Blood pressure – oscillometric systems can take repeated readings at times intervals but can be unreliable and inaccurate in small patients. Check any abnormal results using a Doppler machine
  - Pulse oximetry (with wave form)
  - Oesophageal stethoscope
  - Temperature probe
  - Fluid pump
  - It is optimal to also have capnography (with waveform), ECG
  - If indicated by patient status, consider blood gases +/- blood glucose if low on induction or long procedure

Many geriatric patients will have underlying musculoskeletal disease so ensure all patients are on soft, padding bedding and try to avoid positioning in unnatural postures.

Common problems

Use of rigid metal mouth gags commonly available from veterinary dental suppliers have been identified as potential risk factor for cerebral ischaemia and blindness in cats. The cat's maxillary arteries are main source of blood supply to retinae and brain and there is significant circulatory compromise during maximal mouth opening. It is critical that the surgeon does not force the cat's mouth to remain fully open. Stiles et al reviewed the medical records of 20 cats with reported post-anaesthetic cortical blindness. It was found that a mouth gag was used in 16/20 of the cats. 4/20 (20%) remained permanently blind whereas 14/20 cats (70%) had documented recovery of vision.[19,20]

Bradycardia is commonly encountered with geriatric anaesthesia and can be classified as a heart rate of <100bpm with normal intracranial pressure. Relative bradycardia is also important and can be described as a >30% reduction from the patient's pre-induction heart rate. To address bradycardia, first check the heart rhythm/ECG, if it is normal, lightening the plane of anaesthesia may resolve it. If the patient is already under a light plane of anaesthesia, consider dosing with atropine (0.01-0.02mg/kg IV) in addition to improving active warming if the cat is hypothermic. If the ECG is abnormal, this warrants further investigation and may require aborting the procedure.

Hypotension is another common problem encountered during geriatric anaesthesia and is defined as MAP <60mmHg or SAP <90mmHg (Doppler). Firstly, establish that the blood pressure reading is correct, ensuring the that the cuff is placed correctly on the patient and that it is the right size (approximately 40% of the circumference of the limb or tail at the site of cuff placement). Oscillometric blood pressure machines can be inaccurate in cats, and thus if possible confirm the accuracy of the blood pressure reading with a Doppler machine. Once it has been established that the cat is hypotensive, assess the anaesthetic depth and lighten if possible and address bradycardia if present. If the blood pressure does not improve, consider giving a crystalloid bolus of IV fluids over 10-15 mins of 5-10ml/kg. Take great care to avoid fluid overload and monitor carefully.

If blood pressure remains low and the anaesthesia depth becomes too light, a balanced anaesthesia approach can be very beneficial. Balanced anaesthesia involves using a combination of anaesthetics or analgesics to allow further reduction of the isoflurane % and thus improve the blood pressure. Examples of approaches include:

- Use of local nerve blocks - remember the maximum dose for lignocaine in cats = 6 mg/kg (includes initial dose for intubation)
- Use of other analgesics
    - Methadone 0.1 mg/kg IV can repeat once more later if required
- Use of incremental doses of induction agent to increase anaesthetic depth allowing isoflurane % reduction
    - Alfaxalone/propofol give 1/10th to ¼ of the induction dose as needed – usually given every 5-10 minutes. May be able to maintain anaesthesia without isoflurane if needed.

If the patient remains hypotensive, then consider use of a positive inotrope (dobutamine CRI 1-5 ug/kg/min IV, or ephedrine 0.1-0.2mg/kg IV). For reference, the recently published 2018 AAFP Feline Anaesthesia Guidelines has excellent trouble shooting guides that can be printed out and laminated for use in practice.[8]

Recovery

Most anaesthetic deaths will occur within the first 3 hours in recovery after turning off the inhalant anaesthetic.[1] Death can occur due to decreased monitoring and support in the recovery phase, or due to unrecognised/untreated complications during the maintenance phase of anaesthesia.

Upon completion of the dental procedure, clear/suction the oral cavity and pharynx prior to extubation and ensure to remove cotton pharyngeal packing. Tying a string around the gauze swab packing and attaching to the end of the ET tube at the beginning of the procedure can help to avoid overlooking this step on recovery. Patient warming should continue and maintain mask oxygenation until sternally recumbent.

Ensure that the patient is monitored and supported until safely awake and don't forget to continue to minimise stress by ensuring the recovery area is quiet and calm and the patient is comfortable. Continue to check their vitals in the post-op period to detect abnormalities early (i.e. heart rate, respiratory rate, spO2, blood pressure, body temperature, pain scale).

Geriatric dental procedures commonly include multiple extractions due to the high likelihood of advanced periodontal disease and tooth resorptions. Creating a post-op analgesia plan is essential using a multimodal approach. The author prefers repeating a post-op opioid injection +/- tramadol injection (e.g. methadone 0.2-0.4mg/kg IM +/- tramadol 1-2mg/kg SC) however more extensive dental procedures such as a full mouth extraction may require stronger analgesia such as a fentanyl CRI for the initial post-op period. Repeated assessment of pain scores can help to titrate and adjust the plan for the patient.

Home care analgesia is also critical and is individualised for each cat depending on their underlying co-morbidities and the ability of the owner to medicate their cat. Oral transmucosal (OTM) administration of buprenorphine has been shown to provide similar analgesia to IV administration in some cats however absorption can be quite variable between individuals.[21] To maximise the likelihood of optimal absorption via the OTM route, educate owners on how to administer the medication (i.e. not to be given orally, to be given in the cheek pocket or under the tongue where it will absorb through the mucous membranes).

For additional home care analgesia, oral use of gabapentin (6.5-10mg/kg bid PO) may also be of benefit as an adjunct analgesic for acute pain although its mechanism of action for pain management has not fully elucidated. Oral tramadol is extremely bitter tasting, and its use orally in cats should be avoided as it can result in severe hypersalivation, anxiety and food aversion.

It may be prudent to avoid non-steroidal anti-inflammatory drugs (NSAIDS) such as meloxicam in the immediate post op period for geriatrics and only use if indicated once the cat is eating and hydrating well. If NSAIDS are used in geriatrics, it is recommended to use a lower dose rate (meloxicam 0.01-0.02mg/kg PO sid) and take caution if there is renal/hepatic insufficiency,

Conclusion

Feline geriatric anaesthesia requires careful planning, adequate staff training and an excellent standard of anaesthetic and patient monitoring and care, however it is not out of the capabilities of the general practitioner. Age is not a disease, and letting our senior patients suffer with painful dental disease is not an ethical option for many cats. Learning how to confidently perform anaesthesia in this cohort of patients will give excellent client and patient satisfaction and will often add significant quality time for owners with their loved elderly cats.

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Assessing and optimising the quality of life of zoo animals

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Introduction - What do we mean by a "modern zoo"?

The World Association of Zoos and Aquariums (WAZA) has played a key role in defining the role of zoos in terms of their contribution to collaborative conservation, with the aim of achieving "viable populations of ... species thriving in healthy ecosystems".[1] However, this goal must occur within a framework of excellent animal welfare for the animals in zoo collections. Conservation of wildlife is considered to be the core purpose of the modern zoo, while the core activity of the modern zoo is promoting excellence in animal welfare.[2] Both animal welfare and conservation outcomes therefore have equal emphasis in the modern zoo.

In 2015 WAZA released its Animal Welfare Strategy which defines the best practice of zoo animal welfare using the Five Domains model of welfare assessment and management.[3] This model has also been adopted for the welfare framework of the Zoo Aquarium Association (ZAA), the peak body representing zoos in the Australasian region.

Zoo accreditation and animal welfare

In recent years, the ZAA's accreditation process has been revised to align accreditation outcomes with the ability to demonstrate positive animal welfare against specific criteria, based on the Five Domains model. Through this model, the welfare of zoo animals is assessed as positive, neutral or negative in relation to four physical domains (nutrition, physical health, environment and behaviour) and one mental domain ("affective state", the positive or negative subjective experiences of an animal), using criteria which are specific to that species.

This approach to the assessment of animal welfare enables member zoos to clearly identify positive, neutral and negative experiences and welfare outcomes for animals. Zoo animal management is geared towards achieving positive or neutral welfare outcomes across all domains for all species, a goal which is a requirement for successful accreditation as well as a desirable welfare target.

The quality of life of an animal in a modern zoo

The assessment of zoo animal welfare is an ongoing and iterative process for the modern zoo which involves staff at all levels of operation and management. This enables rapid recognition and response to changes in animal welfare state and promotes optimisation of all aspects of husbandry, housing, animal enrichment and medical care. A consequence of this high level of care is that animals in a modern zoo will often exceed the life expectancy of their wild counterparts.

In the past, extreme longevity was interpreted as evidence of good animal welfare and was considered to be a desirable outcome for zoo animals.[4] However, with development of more sophisticated understanding of animal welfare, zoos are increasingly shifting towards practices and evaluations which emphasise quality of life over quantity of life.

Ongoing quantitative assessment of welfare feeds naturally into considerations of quality of life. Mellor (2016) describes a scale for defining the relative balance of positive and negative experiences an animal may have, which in turn allows an assessment of whether the animal is experiencing "a life worth living" (Figure 1).[5] A commitment to excellent zoo animal welfare must therefore also include the capacity to assess quality of life, and to respond in a timely and humane manner when quality of life is unacceptably compromised.

Table 2. A Quality of Life (QoL) scale where the different categories are defined in terms of the relative balance of positive and negative experiences animals may have (adapted from [32]).

Category	Description
A good life	The balance of salient positive and negative experiences is strongly positive. Achieved by full compliance with best practice advice well above the minimum requirements of codes of practice or welfare
A life worth living	The balance of salient positive and negative experiences is favourable, but less so. Achieved by full compliance with the minimum requirements of code of practice or welfare that include elements which promote some positive experiences
Point of balance	The neutral point where salient positive and negative experiences are equally balanced
A life worth avoiding	The balance of salient positive and negative experiences is unfavourable, but can be remedied rapidly by veterinary treatment or a change in husbandry practices
A life not worth living	The balance of salient positive and negative experiences is strongly negative and cannot be remedied rapidly so that euthanasia is the only humane alternative

Figure 1 (Mellor 2016)

Perth Zoo's approach to evaluating quality of life

Most modern zoos have a documented process for making decisions on quality of life (QOL) which enable them to identify at-risk animals, assess QOL against relevant criteria and make decisions based on the findings. These processes tend to be semi-quantitative, tailored to the species and/or individual concerned, and are subject to ongoing review and reassessment. [6,7,8]

At Perth Zoo, the need to develop QOL documentation for an individual is usually raised by keeping staff, veterinarians or curators as animals become aged, develop chronic illness or otherwise become debilitated. While some zoos have established species-specific age thresholds which automatically raise the need for development of documentation,[8] this approach is not currently being used at Perth Zoo.

QOL documents are developed by keeping staff from a template, with input from veterinarians and animal management staff. Figure 2 is a hypothetical example of a finalised QOL document. The document begins with an agreed summary of the animal's current health and welfare status, and then identifies a series of "triggers": specific welfare criteria or thresholds which flag a deterioration in the animal's well-being in some way. Triggers can address any of the Five Domains. They frequently include assessment of physical parameters such as body weight, abnormal medical findings, body condition and mobility. Another important aspect for developing triggers is consideration of the animal's "agency". Animals demonstrate agency when their behaviours are voluntary, self-generated and goal directed.[9] Behavioural agency in turn is associated with positive affective experiences that the animal finds rewarding. The demonstration of agency is an important aspect of defining QOL as animals age or develop chronic illness. The emergence of negative affective criteria may also be identified as triggers, such as aggression towards an enclosure mate or loss of interest in a particular activity.

The need to create or review a QOL document can be raised by any zoo staff member at any level. However, the decisions arising from a QOL assessment are made by a group of key stakeholders, which includes the section keepers, section supervisor, curator and a veterinarian.

Animals identified as requiring QOL surveillance have a formal review of their QOL by the identified stakeholder group on at least an annual basis, but more frequent review may also be mandated as part of the document. In addition to these routine reviews, the occurrence of any of the criteria or thresholds in the triggers list will "trigger" an immediate QOL review. Meeting a trigger is not grounds for euthanasia; it merely indicates that it is necessary to review all aspects of the animal's situation and ensure the balance of well being is still being met. The outcome of such a review may be one of five scenarios:

- QOL is acceptable and no additional changes to management are required;
- QOL is acceptable, but changes to treatments or management are actioned;
- Further information is required to establish QOL – additional observations, additional diagnostics or investigations are actioned;
- QOL is not acceptable but changes can be actioned to bring about a return to acceptable QOL;
- QOL is not acceptable and euthanasia is indicated.

Figure 2: Hypothetical QOL document for African lion “X”

Background/Context

“X” is a 19 year old lioness, and is in good general health. Expected longevity for captive African lions is 20-21 years of age. She is housed with an aged male conspecific.

X’s mobility is still good, although given her advanced years she is likely to have some age-related degenerative joint disease. Examination under anaesthesia in December 2016 did not detect any significant joint problems and her dentition was good at that time. X was started on Cosequin four times a week in March 2017 and keepers feel that this has resulted in improved mobility.

In August 2018 it was noted by keepers that X was exhibiting an increase in pacing behaviour along the air lock fence. Possible causes of behaviour included food anticipation/hunger, seeking keeper interaction, or disruption due to nearby construction (new lion exhibit). By December 2018, after a diet increase, this pacing was noted to have decreased based on behavioural ethograms. Her quality of life was assessed as acceptable in December 2018, however the pacing behaviour must be monitored and reviewed over time to ensure it is not affecting her overall demeanour and quality of life.

Triggers

Any or all of the following occurrences will trigger a meeting of key staff to review quality of life:

- **Change in appearance** – body weight, muscle tone, overall shape (e.g. abdominal distension). X’s ideal body weight range is 115-120kg.
- **Change in demeanour** – aggression, loss of enthusiasm for enrichment, negative interactions with keepers or with other animals in the enclosure
- **Change in response to normal routine** – decline in appetite or unwillingness to eat preferred types of food that persists for more than 3 consecutive feed days; inability to feed normally; inability or disinclination to follow normal husbandry movements or routines that persists for more than 3 consecutive days
- **Change in health status** – X develops a medical condition that warrants reassessment in the opinion of PZ veterinarians

Decision making

Any of the key staff (duty keepers, supervisors, curator, duty veterinarian, director) are entitled to report triggers and raise the need for a meeting. Concerns should be directed through the section/duty Supervisor, who organises the meeting. The group will discuss the case and decide on the appropriate course of action.

Next quality of life review

If X’s condition remains stable and no meeting of key staff is triggered in the meantime, a meeting of key staff to review quality of life will be scheduled for: *November 2019.*

Challenges

The unapproachable and wild nature of many zoo animals can create challenges for assessment of QOL. The normal arsenal of veterinary investigative techniques, such as radiography, blood testing and CT scan, are often logistically challenging, if not unachievable, in the zoo context. For example, many large ungulates such as rhino and giraffes are prone to dental disease as they age, yet it is impossible to fully evaluate their dentition without full anaesthesia, a process which carries much more inherent risk in these animals than in domestic ungulates. Likewise, the large size of elephants restricts the use of conventional radiography as a diagnostic tool. In addition to diagnostic constraints, many of the palliative treatments which might be effective in domestic animals are often not feasible in zoo animals due to lower compliance or impractical dose rates. Where such constraints exist, QOL documentation plays a valuable role in ensuring all stakeholders understand and accept the limitations of investigation and treatment.

The capacity of zoo animals to mask pain, discomfort and medical conditions can be an obstacle to effective assessment of QOL, particularly given the diagnostic constraints which may arise. Post mortem examination of aged zoo animals has demonstrated that in the absence of careful QOL evaluation, advanced degenerative disease (musculoskeletal and dental) and neoplasia may go undiagnosed until necropsy.[8] In many cases, signs of discomfort and debilitation may emerge very gradually, and without structured scrutiny may be considered to be "just the way that animal is". This puts the animal at risk of long-standing negative welfare, particularly if it is a long-lived species such as a great ape, giant tortoise or giraffe. Ongoing discussion and consultation between veterinarians, keepers and supervisory staff is essential for developing specific triggers for each species or individual, to best articulate what pain and suffering might look like in the case at hand, and develop understanding of how that might correlate with disease and QOL. At Perth Zoo, specific scoring systems are developed to assist with objective longitudinal assessment of subtle clinical signs. This process often provides valuable insight into fluctuations in key triggers over time.

Decisions regarding QOL in zoo animals are often made under substantial public scrutiny. It may be a challenge to bring people to understand the need to euthanase a beloved animal which appears to them to be in good health. Additionally, some may struggle to understand why a condition which is readily treatable in humans, such as arthritis, may be associated with an unacceptable QOL in a zoo animal. QOL decisions may also attract a negative backlash from activists who are ethically opposed to zoos. Under such scrutiny it is essential that all zoo animal custodians understand the basis for any decisions relating to QOL, particularly euthanasia. Perth Zoo's QOL documents are shared with the zoo's Ethics Committee and Board, so they have a good understanding of the QOL process for high profile individuals well in advance of any decision, and feel competent to explain those decisions to the broader community. We have found QOL documentation to be a highly valuable tool for bringing lay staff to an understanding of the complexities of QOL decisions; conversely, the questions posed by lay staff such as our media and communications team, the board and the ethics committee, challenges us to document our QOL decisions in accessible ways.

The timely and thorough documentation of QOL, coupled with a regular review process, gives zoo staff the confidence to make timely euthanasia decisions for chronically ill and geriatric animals, which might otherwise have a slowly progressive slide into poor welfare. It also ensures that such decisions are made in a highly consultative manner, with the consensus of keepers, animal managers and veterinarians. Furthermore, the process provides the framework and language to articulate the decision pathway to managers, the media and the general public.

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Foot and mouth disease: are we prepared for an outbreak?

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Introduction

Several transboundary animal diseases threaten the Australian livestock industries. Of these, FMD would have a significant veterinary public health impact with direct and indirect costs amounting to billions of dollars and potentially years of lost trade. To ensure we are prepared should such an incursion occur, a team of scientists and other stakeholders have been working on developing tools that will assist in early recognition of disease and accurate diagnosis. In addition, computer applications are being developed that will assist decision makers during an outbreak to help trace the disease and also to cost the control decisions and their impact.

An integrated approach

Complex problems that require foresight and novel thinking are best addressed by multidisciplinary teams. The FMD Ready Project aims to improve preparedness by focusing on four different areas that could improve our response times and effectiveness. Not only is it important to ensure access to fast and accurate diagnostic tools, but it is essential that on-farm surveillance is sensitive to ensure diseases are detected in a timely manner. The social factors that impact on surveillance need to be understood and novel approaches for collaboration between stakeholders trialled. Several pilot groups have been established across various industries that involve role players across the production chain.

Once an outbreak is recognised, the most efficient and ideally cost-effective control strategies need to be implemented. Australia has a FMD vaccine bank, and the decision to vaccinate may have a major effect on the duration of an outbreak and the subsequent economic impact. Animal trials to test the vaccines in the bank have shown that these vaccines are efficacious despite the high amount of variation in field viruses, whilst also providing data on virus excretion that could be useful in disease models.

The Australian national model of livestock disease spread and control (AADIS), that helps predict the potential success of outbreak control, is now being updated to cost the decisions made. During an outbreak it is also important to trace the spread of the disease and a novel application that uses short distance wind dispersion and genomic information will assist in this complex task of understanding the routes of disease spread.

With these new tools and novel approaches to surveillance, Australia is better prepared not only for an incursion of FMD, but many other transboundary animal diseases as well.

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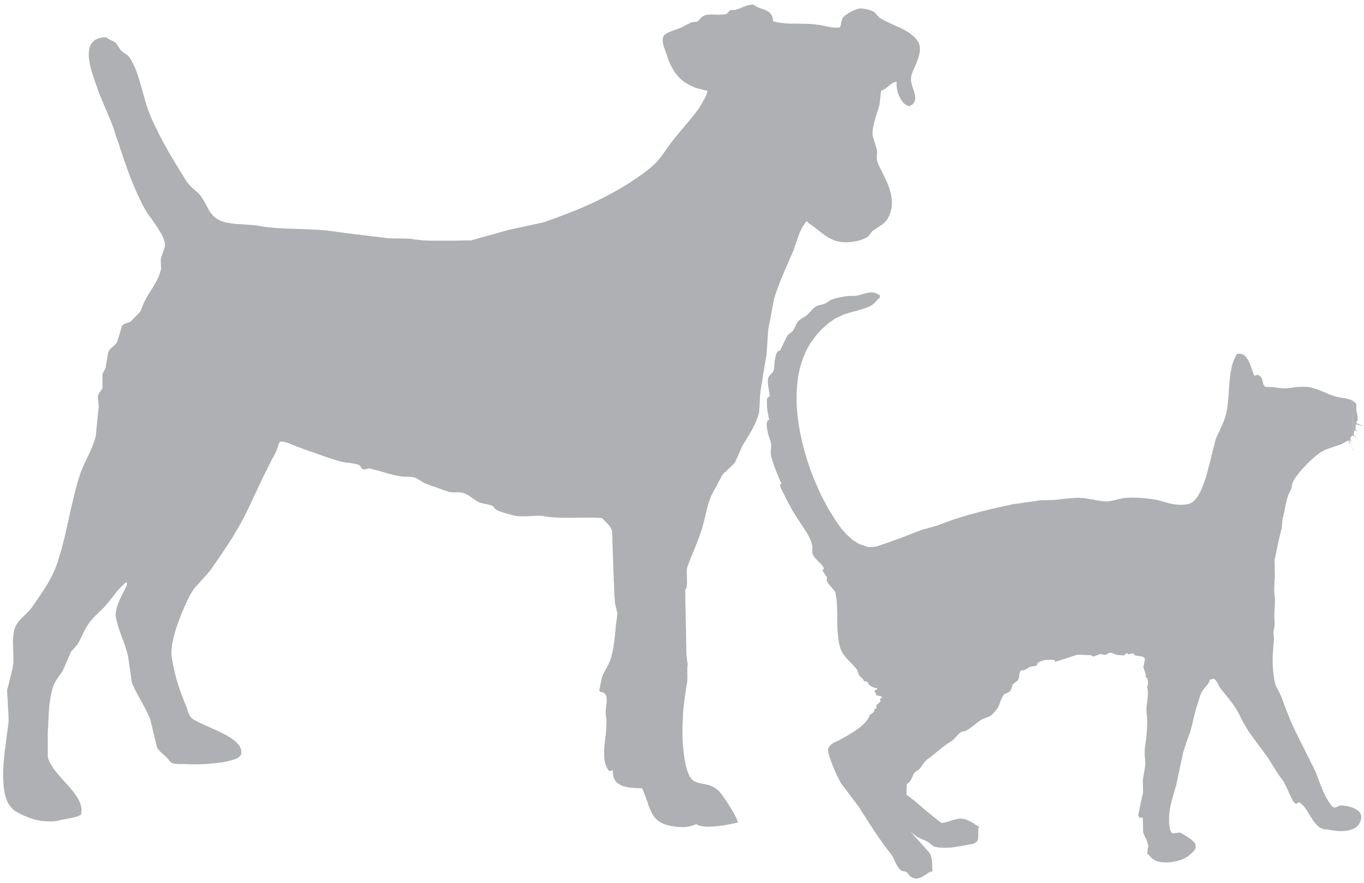
Standards of Care

Anaesthesia Guidelines for Dogs and Cats



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Warne, L – Standards of care, Anaesthesia Guidelines for Dogs and Cats

Standards of Care

Anaesthesia Guidelines for Dogs and Cats

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INTRODUCTION

The Australian Veterinary Association has prepared these standards to support veterinarians in offering the highest standards of care to their patients. The standards set out in this document detail the ideal standards of anaesthetic care for dogs and cats within a General Practice setting. The Australian Veterinary Association believes that owners should be advised of the best available care as set out in these standards. These standards have been based on the latest peer-reviewed scientific research, surveys of Australian veterinarians and broad consultation within the veterinary profession. Anaesthesia is a continually evolving discipline, with frequent advances in pharmacology and technology. It is mandatory for all members of the anaesthesia provision team to periodically undergo training to refresh their knowledge. Referral to a board-certified veterinary anaesthesiologist should be considered for cases that are beyond the practitioner's level of expertise or comfort.

These standards will be reviewed and updated based on feedback from veterinarians and the publication of relevant new research.

PREANAESTHETIC EVALUATION

Signalment

Information pertaining to species, age, breed, neutering status and demeanour should be noted for each patient. An understanding of age and breed characteristics may provide information about additional anaesthetic concerns and prompt further diagnostic tests. Geriatric animals may pose higher anaesthetic risk due to reduced cardiac, hepatic and renal function, which may not be apparent on routine physical examination. Similarly, certain breed differences can lead to greater risks for airway obstruction, increased responsiveness to anaesthetic drugs, and delayed recovery, all of which can result in increased anaesthesia-related morbidity and mortality. Individual genetic variability can trigger unexpected and adverse responses to anaesthetic drugs, which need to be identified by good recordkeeping and consistent patient monitoring. Although genetic differences are typically held responsible for prolonged recoveries and increased drug responsiveness, true genetic sensitivity has been demonstrated in only a handful of breeds, including the greyhound and the collie (Table 1). Demeanour should be taken into consideration when planning individual anaesthetic drug protocols, reducing patient perioperative stress and ensuring safety for veterinary personnel.

TABLE 1: Selected Breed-related Anaesthesia Concerns

Category	Concern	Precaution
BRACHYCEPHALIC BREEDS (e.g. Pug, Bulldog, Boxer, Pekingese, Persian cats, etc.)	Brachycephalic airway syndrome; increased risk of upper airway obstruction. Consideration should be given to delaying the elective procedure until corrective airway surgery has been performed.	• Preoxygenate • Caution with excessive sedation • Diligent monitoring from admission to discharge • Prepare for difficult intubation • Provide supplemental oxygen until extubation • Extubate ONLY after patient is bright and alert with a gag-reflex present • Monitor breathing pattern and oxygenation status post extubation • Be prepared to re-anaesthetise and re-intubate the patient in recovery if required
SIGHTHOUNDS (e.g. Greyhound, Whippet, Borzoi, Saluki, Afghan hound, Irish Wolfhound)	Delayed drug metabolism, possible delayed recovery from drugs such as barbiturates, propofol and acepromazine; lower body fat percentage; hypothermia; stress-induced hyperthermia; myopathy	• Use low-dose acepromazine (0.005 – 0.02 mg/kg IM) • Avoid barbiturates • Administer propofol or alfaxalone SLOWLY to effect • Monitor patient temperature and actively warm as required • Treat stress/pain-induced hyperthermia with prompt anxiolytics, analgesics, cooling • Provide adequate padding during long procedures
HERDING BREEDS (e.g. Collie, Border Collie, Australian Shepherd, Shetland sheepdog)	*ABCB1* (MDR1) mutation causes defect in P-glycoprotein pump, resulting in accumulation of certain drugs into the central nervous system, causing excessive prolonged sedation	• Diligent monitoring following sedation • Reduce premedication/sedation dose by at least 25% • Consider using drug which can be reversed/antagonised • Consider genetic screening prior to anaesthesia for elective procedures

TOY BREEDS (e.g. Chihuahua, Pomeranian, Shih Tzu, Brussels Griffon)	Hypothermia due to large body surface area to volume ratio; difficulty monitoring; hypoglycaemia	• Active patient warming and diligent monitoring of body temperature • Utilize Doppler blood pressure monitoring device, along with ECG, pulse oximetry, temperature • Monitor blood glucose concentrations prior to and during anaesthesia as well as recovery; supplement as required
GIANT BREEDS (e.g. Saint Bernard, Newfoundland)	Increased response to sedatives	• Dose drugs based on lean body mass or allometric scaling (e.g. α_2-adrenergic agonists)
DOBERMAN PINSCHER	Predilection for dilated cardiomyopathy, von Willebrand disease	• Evaluate coagulation status • If von Willebrand disease suspected, administer desmopressin prior to surgery • If NSAID are required, administer COX-2-selective drug
Boxer	Acepromazine-induced vagal response, marked hypotension, and bradycardia has been reported in Boxer dogs of UK lineage; Boxer cardiomyopathy	• Avoid acepromazine in Boxer dogs if possible and reduce acepromazine dose (0.005 – 0.01 mg/Kg IM) if alternative sedation agents are not available • Thorough patient history and physical examination including ECG analysis prior to anaesthesia

History and Reason for Anaesthesia

A detailed, accurate history should be considered an essential part of a preanaesthetic evaluation. Previous conditions take on significance when their residual effects compromise the patient while under anaesthesia or become exacerbated as a result of the stress of anaesthesia and recovery. For example, a patient with cardiac disease may be more intolerant of fluid therapy or a brachycephalic breed may have a higher risk of recovery complications due to brachycephalic airway syndrome. The history should include pertinent information regarding prior and concurrent drug therapy and whether the patient has had any adverse reactions or sensitivities to medications or anaesthetic agents. When possible, the anaesthetic record of a patient that has been anaesthetised previously should be retrieved and reviewed thoroughly for any adverse responses during induction, maintenance, or recovery from anaesthesia.

Physical Examination

A thorough physical examination of all body systems should be performed prior to general anaesthesia and sedation. For further information please refer to STARDARDS OF CARE – REGULAR HEALTH CHECK STANDARDS FOR DOGS AND CATS available online:
www.ava.com.au/sites/default/.../ASAVA_StandardsofCare_Web.pdf

Clinical Diagnostics

Preanaesthetic diagnostics are a consideration to allow detection of underlying disorders that may influence the management of the patient or influence the prognosis associated with any given disorder. The decision regarding when to perform preanaesthetic diagnostics and which tests to include is a decision that needs to be based on the patient's history as well as physical examination and addressed individually by each practice on a case-by-case basis. Evaluation of patients at least one day prior to an elective procedure can help avoid time conflict between the surgery schedule and the benefit of additional clinical diagnostics. Recommended diagnostic testing for specific conditions are discussed later.

PREANAESTHETIC LABORATORY EVALUATION

Preanaesthetic diagnostic testing is an adjunct to a detailed patient history and thorough physical examination to aid in the detection of disease. While there can be no doubt that preanaesthetic biochemical and haematological analysis are valuable for certain patient groups (e.g. geriatrics) and any patient that is clinically unwell, questions have been raised as to whether they are justified for every patient.[1] The use of extensive laboratory screening of patients has not been shown to significantly improve patient outcomes or prompt changes in anaesthetic technique.[2] Despite this conclusion, preanaesthetic blood analysis was found to have resulted in a reclassification to a higher ASA status (defined below – Table 2) in 8% of dogs and additional preoperative treatments in 1.5% of dogs which would have otherwise not been evident from the patient history or physical examination and should therefore be considered for all patients undergoing anaesthesia.[2] Given increased understanding of the limitation of population based reference intervals, there is value in comparing results of pre-anaesthetic biochemical parameters to previous results for that individual, to see if there has been significant changes, even if the values remain within the population based reference intervals.[3] Recent publications have shown that significant changes can be detected by diagnostic tests in patients which would have otherwise been considered to be in good health based on history and physical examination alone (6.2% of dogs and 19.2% of cats).[4] The type and timing of diagnostics should be determined by the veterinarian based on previously mentioned factors, as well as any changes in patient status or the presence of concurrent disease. There is no evidence to indicate the minimum timeframe prior to anaesthesia within which laboratory analysis should be performed. However the timing should be such as to best reflect current changes that may impact anaesthetic risk.

When faced with financial or technical limitations which prevent extensive preanaesthetic biochemical and haematological analysis the authors recommend the following minimal preanaesthetic screening be mandatory: packed cell volume (PCV), total solids (TS), blood glucose, blood urea nitrogen (BUN) and urine specific gravity (USG). Prior to major surgical procedures the authors also recommend performing a peripheral blood film evaluation to enable prompt identification and characterization of conditions such as anaemia and thrombocytopaenia.

ADVANCED LABORATORY EVALUATION

Von Willebrand disease (vWF) – Breeds that have a high incidence such as Doberman Pinschers should have a buccal mucosal bleeding time (BMBT) performed prior to anaesthesia for any surgical procedure. A finding of prolonged BMBT indicates further testing for vWF is required.

Platelet counts and coagulation panels – Indicated for patients exhibiting signs of unexplained/easy bruising, petechiae or ecchymosis and for procedures where significant haemorrhage is possible.

Arterial blood gas, acid base and electrolyte analysis – Indicated for patients with suspected pathophysiologic abnormalities that can alter gas exchange or acid-base disturbances. Interpretation of a blood gas profile should be accompanied with consideration of electrolyte concentrations. This should focus on recognition of acid-base and electrolyte patterns; and while this rarely leads to a specific diagnosis, it allows for tailoring of fluid therapy and specific intervention which can be lifesaving (e.g. addition of potassium in hypokalaemic patients).

DIAGNOSTIC IMAGING

Suspected or known conditions requiring diagnostic imaging for the purpose of evaluating anaesthetic risk include the following:

Cardiac or respiratory disease – known or clinical suspicion of cardiac or respiratory disease should be investigated by means of thoracic radiographs, and if indicated echocardiography or electrocardiography prior to anaesthesia.

Anaesthetic Risk and Physical Status

Following completion of a thorough patient history, clinical examination and interpretation of findings from ancillary diagnostics, the American Society of Anesthesiologists (ASA) Physical Status Classification System can be assigned (Table 2). The practice of assigning an ASA Status to a patient provides a framework for the clinician to summarise their preanaesthetic evaluation and encourages the clinician to think about the physical status of the patient rather than the solely focusing on the procedure itself. It is also a useful standardised method of documenting patient risk from a medico-legal standpoint as high ASA scores have been shown to be predictive of anaesthetic morbidity and mortality in veterinary patients.[5]

TABLE 2: ASA Physical Status Classification System

1 - A normal healthy patient

2 - A patient with mild systemic disease

3 - A patient with severe systemic disease

4 - A patient with severe systemic disease that is a constant threat to life

5 - A moribund patient who is not expected to survive without the operation

If the procedure is an emergency, the physical status classification is followed by "E" (for emergency) for example "3E" would be for a patient with severe systemic disease undergoing emergency anaesthesia.

OWNER COMPREHENSION AND INFORMED CONSENT

Prior to anaesthesia clients should be fully informed of all relevant known risks associated with the planned procedure including the anaesthesia and associated loco-regional anaesthesia techniques, and signed consent should be obtained. In addition, this discussion should include consented instruction as to what degree of intervention should be performed in the event of a life-threatening emergency such as a cardiac arrest. Utilization of a colour coded system has been recommended for clarity within the practice setting (e.g. CODE Red - No resuscitation should be attempted; CODE Amber -

Resuscitation should be attempted but should be limited to basic life support techniques such as closed chest cardiac compressions – unless the abdomen is open and trans-diaphragmatic direct cardiac massage is feasible; CODE Green - All available means of resuscitation should be employed including open-chest direct cardiac massage, when trained personnel capable of performing such techniques are present). Should more patient information be obtained (e.g. from additional diagnostic tests), the risk assessment associated with anaesthesia may change. The owners should be made fully aware of such changes.

PREPARATION FOR ANAESTHESIA

Individual Patient Plan

An individualized anaesthetic and analgesic plan should be constructed for the management of each patient based on risks identified in the preanaesthetic evaluation and incorporating the staffing, equipment and drug resources available. Contingencies should be made for potential adverse events, and emergency drugs should be available, and doses calculated.

STABILIZATION

Where possible concurrent disease or conditions which may contribute to an elevated anaesthetic risk should be addressed prior to anaesthesia. Elective procedures should not proceed whilst concomitant and untreated risk factors exist. Patients requiring emergency procedures should receive optimal stabilization prior to anaesthesia.

FASTING

It is recommended that healthy dogs and cats are fasted for at least 6 hours prior to being anaesthetized, whenever possible, to reduce the risk of regurgitation and aspiration pneumonia.[6] Water should not be restricted until just prior to anaesthesia at the time of premedication.[6] Exceptions to this would include patients undergoing gastric surgery and those with megaoesophagus. Dogs and cats aged less than 8 weeks or weighing less than 2 kg are at greater risk of hypoglycaemia and should not be fasted for greater than 1 – 2 hours.[6]

PREMEDICATION & ANALGESIC PLAN

The importance of a well-planned premedication and analgesic plan cannot be understated. The choice of premedication will be influenced by signalment, temperament, concurrent disease, the procedure to be performed, drug availability, personal familiarity and preference. The appropriate choice of premedication drugs should aim to achieve the characteristics outlined below. No individual drug possesses all of the following properties, and as such various combinations of agents are used to best achieve these characteristics:

Sedation and stress reduction – Calm or immobilize a patient to enable minor procedures (e.g. catheterisation, clipping etc.), reduce stress and decrease anaesthetic drug requirement during induction of anaesthesia and reduce adverse arrhythmogenic autonomic activity.[6]

Safe handling – Facilitate safe patient handling and preanaesthetic preparation (e.g. catheterisation, clipping etc.) for both the animal and personnel.

Analgesia – Analgesia is a key component to well-balanced anaesthesia for all surgical and potentially painful procedures. It is also important to note that appropriate preanaesthetic stabilization of a patient should involve the treatment of any pre-existing pain. Experimental studies have shown a reduced requirement for post-operative analgesia when analgesics are administered "pre-emptively".[7] Typically an opioid forms the analgesic component of the premedication for surgical procedures. The selection of the particular opioid and initial dose rate should be made on the basis of:

- the expected intensity of the pain
- the duration of action required
- the desired speed of onset
- relevant side effects of the particular drug.

For a detailed and practical resource designed to assist practitioners in recognizing, assessing, and treating pain please refer to The WSAVA Global Pain Council Guidelines available online: http://www.wsava.org/guidelines/global-pain-council-guidelines

Balanced anaesthesia – By providing analgesia and sedation the premedication should also facilitate a dose reduction of other potentially more physiologically compromising drugs (e.g. inhalational agents) used to produce anaesthesia.

Calm recovery – Both the analgesic and sedative components of the premedication drug combination should be present at the time of emergence from anaesthesia to promote a calm recovery. If the duration of therapeutic effect of the individual agents is not sufficient to achieve this, redosing of one or both components may be required prior to the conclusion of the anaesthetic procedure.

Premedications are typically administered intramuscularly (IM) or subcutaneously (SC) 15-45 minutes prior induction of anaesthesia. Whenever permissible by drug labelling guidelines premedicants should be administered intramuscularly, as this route affords more reliable drug absorption, compared with subcutaneous administration.[8]

VENOUS CATHETERIZATION

An intravenous (IV) catheter is the patient's lifeline while under the effects of general anaesthesia and sedation. Intravenous access allows for direct administration and rapid uptake of anaesthetic, analgesic and emergency drugs as required perioperatively. Various indwelling catheters placed using an 'over-the-needle' technique are appropriate for peripheral veins and the catheter choice depends on animal size and personal preference. To reduce resistance-to-flow and clot formation, the largest possible gauge catheter appropriate for the vessel to be catheterised should be selected. The catheter type and size, along with the time, date and site of insertion should be noted in the anaesthetic/medical record.

The cephalic vein of the thoracic limbs or the lateral and medial saphenous veins of the pelvic limbs are the most common peripheral veins catheterised. With placement of catheters, strict asepsis is important. Appropriate clipping of the area and an aseptic preparation of the skin (with a 1-2% iodine tincture, iodophors, chlorhexidine or 70% alcohol solution) is necessary. Appropriate hand hygiene must be applied, and ideally sterile gloves worn, particularly for long-stay catheters.

Topical anaesthetic agents (e.g. Emla® cream) may be applied 45 minutes to 1 hour prior to cannulation to reduce stress and facilitate catheter placement in anxious or hyperaesthetic patients. Cover the topical anaesthetic with an occlusive dressing after application to ensure it is not ingested. Constant supervision is advised.

To decrease risk of catheter-related complications, sterile placement, daily inspection and rewrapping of the catheter site is advised. When possible, administration of irritating or hypertonic solutions into a peripheral vein should be avoided. Regular inspection enables early identification of complications including, but not limited to:

- phlebitis, or inflammation of the vessel
- thrombosis or formation of a thrombus on the catheter or vessel wall
- embolism
- catheter breakage
- subcutaneous fluid infiltration.

Tight taping or bandaging, leading to swelling distal to the catheter, may contribute to resistance to the flow of fluids and/or drugs being given via the IV catheter. There is little evidence from human patients that the type of dressing significantly reduces the incidence of catheter infection compared with catheters left exposed and kept clean and dry.[9] Furthermore, transparent "breathable" dressings have not been shown to offer significant advantage for human patients over gauze dressings unless impregnated with chlorhexidine (e.g., 3M™ Tegaderm™ CHG Chlorhexidine Gluconate I.V. Securement Dressing), and do not adhere effectively to animal skin.[10, 11]

Although peripheral IV catheters are typically removed after 72 hours in human patients, there is no evidence in veterinary medicine that this is necessary if there are no identified complications.[12] Venous catheters should be flushed and inspected every six hours and rewrapped every 12 hours. If the catheter site becomes wet or soiled, the catheter should be removed, and another placed aseptically. While monitoring an indwelling catheter, record the date, catheter site and type, and comments daily. Catheters used for continuous fluid therapy do not need to be flushed routinely. Unused IV catheters should either be removed or flushed every six hours to maintain patency. This should be done using 0.9 per cent sodium chloride with or without heparin. There is little evidence to support the use of heparinised saline over normal saline to maintain patency of intravenous or arterial catheters.[13, 14]

Equipment Preparation

In order to reduce the risks of anaesthesia, foreseeable problems must be prevented. Patient related problems will be identified by the preanaesthetic evaluation. Checking equipment will help to identify and prevent problems due to technical faults or errors (see Table 3). It is critical that all members of the anaesthetic team (both veterinarians and nursing personnel) are familiar with the anaesthetic machine, monitoring systems and related equipment. All members of the team should be able to troubleshoot common equipment dysfunctions.

Table 3: <u>Equipment Checklist</u>

The checks outlined below should be carried out prior to each anaesthetic procedure. Servicing of anaesthesia delivery systems should be performed regularly, at specified intervals in accordance with the manufacturer's documented service requirements. Servicing requirements should be noted on a label and displayed on the equipment in a prominent position. The label should list the date of the most recent service and the due date for the next service.

<u>Anaesthetic Machine</u>

- For piped gases: Connect the oxygen, nitrous oxide or medical air to the correct gas pipelines and perform a 'tug test' (pull to check the integrity of the connection to the anaesthetic machine). Check and ensure that there is a second supply of oxygen for emergencies (a full oxygen cylinder safely secured near the anaesthetic machine).
- For oxygen cylinders: Check there are two oxygen cylinders accessible, one 'in use' cylinder and one 'full' reserve cylinder.
- Turn the oxygen flowmeter on to maximum and off again, check the ball/bobbin is rising/rotating freely respectively. Repeat this step for nitrous oxide and medical air if required.

- Check the machine for leaks: Turn on the oxygen to 4 L/min. Occlude the fresh gas outlet and check that the oxygen flowmeter bobbin/ball drops. This should be performed with the vaporizer you wish to use in place on the back bar and repeated if a different vaporizer is placed on the anaesthetic machine. *Nb. Please refer to anaesthesia machine's documentation for specific leak-checking procedures applicable to each machine.*
- Operate the emergency oxygen bypass control (flush). Ensure flow occurs and ceases when control is released.
- Switch the oxygen off.

Vaporizer

- Check that the vaporizer(s) for the required volatile agent(s) is sitting correctly on the back bar of the anaesthetic machine and locked in place.
- Check that the dial turns fully through the full range. Turn dial off.
- Check that the vaporizer(s) are adequately, but not over, filled and that the filling port is tightly closed.

Scavenging

- For active scavenging systems: Check that the scavenging pipe is connected to the scavenging system outlet. Ensure that the active scavenging system is turned on.
- Ensure that the end of the scavenging system is correctly attached to the breathing system.
- For passive scavenging systems: Ensure that the scavenging hose is connected to a charcoal absorber. The charcoal absorber should be weighed weekly to assess the degree of absorption and replaced when the maximum weight is reached (as recommended by the manufacturer).

Table 3: Equipment Checklist (*cont.*)

Breathing system

- Select the appropriate breathing system (see Selecting Breathing Systems and Fresh Gas Flow Rates) and check that the reservoir bag is an appropriate volume for the patient (tidal volume [10 to 20 mL per kg] x 5).
- Connect the breathing circuit to the fresh gas outlet, ensuring all connections are secured tightly.
- Connect the scavenging tube to the circuit.
- Perform a leak test by closing the APL (Adjustable Pressure Limiting/“pop-off”) scavenging valve, and turning on the oxygen flowmeter.
- Occlude the “to the patient” end of the circuit with your hand. Let the reservoir bag fill completely with oxygen.
- Turn off the flowmeter when the pressure gauge registers 30 cm H_2O or when the reservoir bag is fully inflated and slightly distended if the anaesthetic machine does not have a pressure gauge.
- If the pressure holds steady the system is leak-free, but if the pressure drops (or the bag deflates when squeezed) the system will need to be checked for leaks.
- **Open the APL valve** to depressurise the system prior to removing your hand from the patient end of the breathing system.
- When using a rebreathing circuit, the soda lime should be checked for signs of exhaustion (according to manufacturer’s recommendations) and the unidirectional valves checked to ensure they are moving freely.
- When using a non-rebreathing circuit (e.g. Bain) check the integrity of the inner tube by performing an occlusion test of the inner tube. Occlusion of the inner tube while oxygen is flowing should cause the flowmeter bobbin/ball to drop temporarily.
- All circuits with adjustable scavenging valves should be checked to ensure the valves open and close then **leave the valve open** prior to use.

Ventilator

- Check that the ventilator is configured correctly for its intended use.
- Ensure that the ventilator tubing is securely attached.
- Set the controls for use and ensure that adequate pressure is generated during the inspiratory phase.
- Check that alarms are working and correctly configured.
- Check that the pressure relief valve functions correctly at the set pressure.
- Two-bag test: A two-bag test should be performed after the breathing system, vaporizers and ventilator have been checked individually.
    1. Attach the patient-end of the breathing system (including angle piece and filter) to a test lung or rebreathing bag.
    2. Set the fresh gas flow to 5 L/min and ventilate manually. Check the whole breathing system is patent and the unidirectional valves are moving (if present).
    3. Check the function of the APL valve by squeezing both bags.

Table 3: Equipment Checklist (*cont.*)

4. Turn on the ventilator to ventilate the test lung. Turn off the fresh gas flow or reduce to a minimum. Open and close each vaporizer in turn. There should be no loss of volume in the system

Breathing systems should be protected with a test lung or rebreathing bag when not in use to prevent intrusion of foreign bodies. Conduct a check before every procedure.

What if a leak occurs?

- **Reservoir bag** – If leak occurs, replace reservoir bag.
- **Breathing Circuit** – Install new breathing circuit or obstruct inhalation/exhalation openings to determine if leak originates from the breathing circuit.
- **Vaporizer Fittings** – Verify fittings and tubing are securely attached.
- **Absorber Canister Gaskets** – Check for loose absorbent grains between canister housing gaskets and verify that the canister is seated properly.
- **APL Valve** – Remove valve and obstruct opening to determine if leak originates from the APL valve.

Ancillary equipment

Endotracheal tubes

- Select the appropriate size (diameter and length) and type of tube. Prepare three tubes of different sizes for each patient.
- Check the cuffs for leaks. Inflate the cuff and leave it inflated for a few minutes to detect leaks. Deflate cuffs.
- Ensure that the lumen of the tube is clean and free of debris.

Laryngoscope

- Attach the appropriate length and type of blade to the handle. Typically, a Miller style straight blade is most appropriate for small animal patients.
- Open/engage the blade to ensure that the light source is working properly.
- Laryngoscope blades should be washed between patients.

Monitoring equipment

- Check that the monitor is switched on and in the correct “work” mode rather than standby.
- Check the required monitoring cables are correctly connected.

- Check that the available monitoring probe types and sizes are suitable for your patient.

Cleaning and reuse of anaesthetic equipment

- Currently, there are no official guidelines for veterinary anaesthesia related to the cleaning and reusing of anaesthesia breathing circuits, endotracheal tubes and ancillary equipment between patients, with the cost–benefit ratio of using sterilized breathing systems or filters

Table 3: Equipment Checklist (*cont.*)

remaining questionable. We recommend that all anaesthesia equipment that has direct patient contact (e.g. endotracheal tubes, laryngoscope blades, pulse oximeter clips, oesophageal stethoscope, thermometer etc.) should be thoroughly cleaned in mild soap and water, rinsed thoroughly, dried and disinfected between patients.

- Anaesthetic circuits and endotracheal tubes be routinely changed ideally between individual patients but at a minimum on a daily basis. If visibly contaminated, circuits, endotracheal tubes and ancillary equipment should be changed and thoroughly cleaned between patients. Circuits and endotracheal tubes used for highly infectious cases should be safely discarded.
- Regular culturing of breathing circuits, absorbent canisters, and rebreathing bags should be performed for both bacterial and fungal contaminants.

NOTE: Vaporizer In Circuit (VIC) Systems

Most modern vaporizers are agent-specific, concentration-calibrated, out of the circuit, and high-resistance vaporizers that are compensated for temperature, flow, and back-pressure. Non-precision, in the circuit vaporizers are still occasionally found in veterinary practice, however without proper specific training and inhalant anaesthetic agent monitoring these vaporizers pose unnecessary risks during anaesthesia and should not be used.[15] All vaporizers mentioned within this document will unless otherwise stated refer to vaporizer agent-specific, concentration-calibrated, temperature-compensated, out of the circuit (VOC) vaporizer systems.

Selecting Breathing Systems and Fresh Gas Flow Rates

Anaesthetic gas exits the anaesthesia machine (via the common gas outlet) and then enters a breathing circuit. The function of the circuit is to deliver oxygen and anaesthetic gases to the patient and to eliminate carbon dioxide. The carbon dioxide may be eliminated by gas inflow (**non-rebreathing systems**) or by soda lime absorption (**rebreathing systems**).

Breathing systems have been classified using various schemes without consensus and uniformity (i.e., open, semi-open, and semi-closed). This inconsistency in nomenclature found within the literature and teaching institutions is a source of confusion and inconsistency. For this reason, it has been suggested that these terms be abandoned. For clarity it is easiest to classify the breathing system into one of two groups: those designed for rebreathing of exhaled gases (**rebreathing systems**) and those designed to be used under circumstances of minimal to no rebreathing (**non-rebreathing systems**).[16] It has also been suggested that in addition to describing the design of the breathing system, the fresh gas flow (in mL/kg/min) needed to prevent or enable rebreathing to occur should be stated to fully describe how the system is being used.[17]

The amount of rebreathing that occurs with any particular anaesthetic breathing system depends on four factors: the design of the individual breathing circuit, the mode of ventilation (spontaneous or controlled), the fresh gas flow rate and the patient's respiratory pattern. Circuits may eliminate rebreathing either by ensuring an adequate flow of fresh gas which flushes the circuit clear of expired alveolar gas, or additionally, in the case of a circle system, by the use of soda lime, which absorbs the carbon dioxide so that lower fresh gas flows may be used.

REBREATHING SYSTEMS

A rebreathing system allows for the rebreathing of the exhaled gases. The expired carbon dioxide is removed and the fresh gas mixture along with inhalant anaesthetic vapour is continually added. The components of a rebreathing system include: the fresh gas inlet; absorber circuit; manometer; rebreathing bag; hoses; Y-piece; unidirectional valves (inspiration & expiration); APL valve; and a scavenger system. These components increase the resistance to the movement of the gas mixture in the system as well as the total volume of the system comparative to a non-rebreathing system. Human adult size rebreathing circuits are typically used for patients weighing greater than 10 kg. It is advisable that patients in the weight range 2.5 to 10 kg be placed on paediatric size rebreathing systems with a reduced circuit diameter. There is no minimum patient size for using a rebreathing system accepted among anaesthesiologists; however, the minimum patient size is generally suggested as not less than 2.5 kg, when using a modern anaesthesia machine with light weight unidirectional valves. Recommendations for selecting fresh gas flow rates in rebreathing systems are outlined in Table 4.

Table 4: <u>Recommendations for Selecting Fresh/Carrier Gas Flow Rates in Rebreathing Systems</u>

<u>Following Induction (with an injectable agent)</u>

- For patients induced with an injectable anaesthetic agent and subsequently intubated and connected to an anaesthetic machine with a rebreathing system, the initial flow rates should be relatively high (**50-100 mL/kg/min**) to facilitate rapid inspired concentration increases in anaesthetic agent within the system and to replace the anaesthetic vapour that is dissolving into patient tissues during the initial uptake period of anaesthetic delivery (e.g., the first 10 -20 min following induction).[15]

<u>During Maintenance</u>

- Once the patient has reached a satisfactory depth of anaesthesia, the flow rate may be reduced to a maintenance level. Rebreathing systems require relatively lower flow rates compared with non-rebreathing systems during the anaesthetic maintenance period because carbon dioxide is removed from the expired gases, which are then returned to the patient. Provided that there are no leaks in the system and the carbon dioxide absorber is functional, the carrier gas and anaesthetic can be recycled continuously, with only a small amount of additional fresh gas requirement. Following the initial uptake period of anaesthetic delivery (e.g., the first 10 -20 min following induction) flow rates are reduced.

Table 4: <u>Recommendations for Selecting Fresh/Carrier Gas Flow Rates in Rebreathing Systems *(cont.)*</u>

Lower flow rates are beneficial in conserving moisture and heat. During this period flow rates of **20-50 mL/kg/min** are recommended for small animal patients.[15]

- <u>Flow Rates When Making Changes in Anaesthetic Depth</u>: Higher fresh gas flow rates (**50-100 mL/kg/min**) are also recommended to facilitate more rapid equilibration of anaesthetic concentration within the breathing circuit, and ultimately the patient's depth if a patient is judged as being too "deep" or too "light", and the vaporizer dial setting has been adjusted accordingly.

- <u>Low-Flow Anaesthesia</u>: The minimum safe fresh gas flow during maintenance supplies enough oxygen to satisfy the patient's metabolic oxygen consumption. To economise on gas use and waste during the maintenance period, the oxygen flow rate can be reduced to equal the metabolic oxygen requirement of the patient (**5-10 mL/kg/min**), plus enough additional gas flow to replace gases lost because of leaks in the circuit and/or via a side-stream gas analyser (See APPENDIX 1: Recommendations for performing low-flow anaesthesia). The anaesthetist should be aware that at flow rates less than 250 mL/min, some precision vaporizers and flowmeters may not accurately deliver the dialled vaporizer concentration and oxygen flow rate.

***NOTE: Specific technical training in this technique and/or the use of gas analysers to monitor inspired and expired oxygen and anaesthetic partial pressures is recommended.

<u>During Anaesthetic Recovery/Emergence</u>

- Immediately after the vaporizer has been turned off, increase the fresh gas flow rate to **200-300 mL/kg/min** (or as close to non-rebreathing flow rates as possible). Then with the APL valve open, apply gentle pressure to evacuate the reservoir bag, repeating this process when it refills. Where such high flow rates are applied and the fresh gas flow rate matches or exceeds the patient's minute ventilation (tidal volume x respiratory rate), a rebreathing system can be made to function as a non/minimal-rebreathing system. This strategy will wash out waste anaesthetic gas, increase oxygen concentration in the breathing circuit, and hasten the patient's emergence from anaesthesia. It is recommended that these high flow rates be maintained until the patient is extubated.

NOTE: See APPENDIX 2 - Quick Reference Chart: Oxygen Flow Rate Guidelines for Rebreathing Systems (L/min)

- *<u>IMPORTANTLY</u>* the APL valve should be fully open at all times when the patient is spontaneously breathing and only closed temporally during manually (bag) assisted ventilation by the anaesthetist.

NON-REBREATHING SYSTEMS

The distinguishing feature of non-rebreathing circuits is that elimination of carbon dioxide is accomplished by removing all expired gases from the system and venting them to the atmosphere. This is normally achieved by using the fresh gas flow from the anaesthetic machine to direct the expired gases out of the circuit via a valve or other arrangement. In general, non-rebreathing systems provide good control of the inspired gas concentrations, since the anaesthesia vaporiser concentration setting closely matches the inspired isoflurane concentration at all times and fresh gas delivered from the anaesthetic machine is inspired in each breath. They are, however, less economical in use than rebreathing systems because the minute volume of ventilation (or more) must be supplied to the patient to prevent rebreathing, and they contribute more to the problem of atmospheric pollution with anaesthetic agents. Appropriate scavenging of anaesthetic waste gases is mandatory.

Non-rebreathing systems have traditionally been recommended for patients <5 kg, due to lower resistance during spontaneous breathing, less equipment deadspace, and smaller total circuit volume compared to adult rebreathing circuits.[15] However, by using newer paediatric and neonatal rebreathing systems, many of the aforementioned advantages of non-rebreathing systems are negated and it is possible to maintain patients <5 kg safely using rebreathing systems providing that the patients tidal volume is sufficient to operate the unidirectional valves.[15]

There is no patient size recommendation for selecting a non-rebreathing, paediatric/neonatal rebreathing or adult rebreathing system that has been universally accepted among anaesthesiologists. It is however generally accepted that non-rebreathing systems are advisable for patients <2.5 kg. Patients ranging from 2.5 to 10 kg are well suited to paediatric/neonatal rebreathing systems; and patients >10 kg adult rebreathing systems. Recommendations for selecting fresh gas flow rates in non-rebreathing systems are outlined in Table 5.

Table 5: Recommendations for Selecting Fresh/Carrier Gas Flow Rates in Non-Rebreathing Systems

The precise fresh gas flow rate to minimise the rebreathing of carbon dioxide in non-rebreathing systems differs with each individual patient and type of system. Ranges have been reported from 100-600 mL/kg/min for patients <7 kg. In general, an approximate range of 100-300 mL/kg/min with a minimum of 500 mL/min and a maximum of 3 L/min is recommended for patients <6.8 kg.[18] Continuous monitoring of expired carbon dioxide values (by capnography) is the ideal modality to determine an individual patient's fresh gas flow requirement.

NOTE: *IMPORTANTLY* the APL valve should be fully open at all times when the patient is spontaneously breathing and only closed temporally during manually (bag) assisted ventilation by the anaesthetist.

Anaesthetic Waste Gas Scavenging

It is advised that practitioners familiarise themselves with relevant Australian state/territory-based legislation pertaining to occupational health and safety procedures or requirements, concerning control of waste anaesthetic gases. Veterinary Surgeons Boards within each individual state/territory can provide information regarding complying with all relevant standards.

For a detailed resource outlining recommendations for the control of waste anaesthetic gases in the workplace please refer to ***Commentary and recommendations on control of waste anesthetic gases in the workplace*** produced by The American College of Veterinary Anesthesia and Analgesia available online: http://www.acvaa.org/docs/2013_ACVAA_Waste_Anesthetic_Gas_Recommendations.pdf

INDUCTION AND MAINTENANCE OF ANAESTHESIA

Team approach

All personnel involved in the delivery of anaesthesia should be fully informed of the details and plan related to the patient's scheduled procedure, relevant clinical history, and aware of their individual role and responsibilities. It is also essential for all members of the anaesthesia and procedural team to be familiar and practiced in the delivery of any potential emergency contingencies. All team members should be familiar with emergency protocols and supplies (including emergency drug doses calculated) for each individual patient prior to anaesthetic induction. A full description of the most current clinical cardiopulmonary resuscitation (CPR) guidelines, including new algorithms and drug dosing charts are available online: https://www.veccs.org/recover-cpr/

A diligent anaesthetist who is ready for varying contingencies can intercept developing problems before they reach the "crisis" stage. Diligent monitoring and timely, well thought out responses to changes in patient status are crucial in avoiding adverse events.

The maintenance period is made easier by prior planning. For those practices which may not have a dedicated anaesthetist, making sure that everything required for the procedure is organised prior to induction (e.g. surgical kit, suture material etc.) This will enable personnel responsible for anaesthesia maintenance to be able to spend more time dedicated to monitoring of the patient and less time spent performing other tasks during this period.

Surgical safety checklist

The management of risk and patient safety are major drivers in the implementation of surgical safety checklists. Checklists designed to ensure effective communication as well as improve collaboration and the delivery of patient care have been implemented in both human and veterinary healthcare sectors.

The World Health Organization (WHO) surgical safety checklist has been shown to decrease mortality and complications and has been adopted worldwide.[19] The WHO surgical safety checklist has been modified to achieve veterinary relevance and successfully implemented by many practices and institutions. An example of one such checklist can be found in Appendix 3. Development of a surgical safety checklist is strongly recommended and should be tailored to individual needs and work practices. The development of such a surgical safety checklist should involve all members of the perioperative team in order to ensure engagement and compliance.

Patient preparation

Preparation of a patient for anaesthesia should include the following:

- IV catheter placement (mandatory).
- Connect monitoring equipment and record the patient's pre-anaesthesia baseline blood pressure, heart rate, haemoglobin oxygen saturation (pulse-oximetry) and evaluate the ECG for any anomalies.
- Manage/optimise any detected cardiovascular or respiratory problems. ***Anaesthesia for elective procedures should be postponed until a patient is appropriately stabilised***. Cardiovascular stabilization and ongoing maintenance of patients, may include, but is not limited to:
    - Administration of IV fluids. Hypovolemic patients may require isotonic crystalloids, colloids, and/or hypertonic saline to optimise intravascular hydrostatic pressure, improve venous return, cardiac output, and improve tissue perfusion. Dehydration should be corrected in animals that are moderately to severely dehydrated, prior to anaesthesia if time permits. Only 75% to 80% of the dehydration deficit should be replaced during the 24 hours before anaesthesia to avoid fluid overload. The remaining fluid deficit due to dehydration can gradually be replaced over 24 to 48 hours after surgery.[20] In addition preoperative volume loading of normovolemic patients is not recommended, and, in humans, blood volume was unchanged after overnight fasting.[21-25]
    - *Managing cardiac arrhythmias (when feasible based on the patient's condition and the urgency of the procedure to be performed).*
    - *Administration of blood products (when indicated and available).* Anaemia and coagulation disorders can decrease the delivery of oxygen to tissues, and hypoalbuminaemia can alter drug transport and binding and effect fluid balance.
- Preoxygenate patient with 100% oxygen via a firm fitting face mask (demeanour permitting) for a minimum of 3 minutes prior to induction of anaesthesia. Preoxygenation for 3 minutes prior to induction of anaesthesia increases the time to desaturation of haemoglobin by a factor of 3 to 4 compared to non-preoxygenated patients and is particularly beneficial if a prolonged or difficult intubation is expected.[26] Removal of the rubber diaphragm from the facemask may increase patient tolerance.
- When the patient is as deemed to be as stable as possible, proceed according to the individual patient plan.

Anaesthetic Induction Phase

Anaesthesia is best achieved via IV administered induction agents carefully titrated slowly to effect. The equipment and steps involved are detailed in Table 6. Mask or chamber inductions increase patient anxiety, are stressful on the cardiovascular and respiratory systems, delay airway control, and increase the risk of environmental and personnel exposure to gaseous anaesthetics.[27] It is strongly advised to reserve these techniques as options of "last resort" where other alternatives are not suitable.

Table 6: Endotracheal Intubation

Equipment:

- 3 – 10 mL syringe for cuff inflation. It is recommended not to use greater than a 3 mL syringe for cuff inflation in cats.
- Endotracheal tubes in varying sizes - the correct ET tube should be measured from a point immediately rostral to the upper incisors to the thoracic inlet. The safest endotracheal tubes are the high-volume, low-pressure cuffs because they generate less pressure per given volume of inflation with less risk of tracheal trauma.
- Gauze squares (to grasp the tongue).
- Laryngoscope – While it is possible to intubate a cat without laryngoscopy, the visualization is poor and therefore the risk of trauma is much greater. If there is not a laryngoscope in your practice, you may choose to purchase your own as the time and stress it will save is invaluable. There are various models available from most veterinary equipment suppliers. Alternatively, a good light source and a tongue depressor could be used (head torches can be useful)
- Facemask and oxygen supply
- Lubricant (preferably sterile)
- Supplies to secure the endotracheal tube (i.e., rolled gauze to tie the tube to the upper jaw, lower jaw, or behind the head).
- Stylet - recommended for use with small, delicate or flexible silicon tubes. Use a stylet with a flexible tip to avoid tracheal trauma. Once the stylet has been inserted slightly beyond the vocal chords, the tube should be advanced over the stylet rather than advancing a protruding stylet into the trachea, which may result in tracheal trauma, especially if the patient is lightly sedated and coughs or the neck is bent down during intubation.

Table 6: Endotracheal Intubation (*cont.*)

Steps:

1. Ideally, patients should have been pre-oxygenated with an oxygen mask for a minimum of 3 minutes before intubation. Oxygen flow-by should always be available. **Two team members should be involved in the procedure.**
2. Induction agent is administered until a suitable plane of anaesthesia is reached – manifested by a loss of righting reflex. Patients should typically be placed in sternal recumbency; however, team members should also prepare for and be able to intubate patients in lateral and dorsal recumbency. When intubating a patient in sternal recumbency, an assistant should place one hand behind the base of the head (occipital bone), while the opposing hand holds the maxilla (upper lips) and extends it in a dorsal direction. This should fully extend the head and neck as close to a horizontal position as possible.
3. When the patient has received an adequate amount of anaesthetic to sufficiently relax the muscles of the jaw and decrease the gag reflex; the anaesthetist should gently pull the tongue forward, and down between the lower incisors, thus pulling the lower jaw down and the epiglottis forward. This should be done in a gentle manner as the hypoglossal/glossopharyngeal nerves can be stretched leading to neuropraxia and problems with the tongue post-anaesthesia.
4. The blade of the laryngoscope should be placed just in front of the epiglottis and downward pressure applied to depress the tongue, so that the epiglottis is drawn forward and the soft-

palate disengaged, allowing visualisation of the larynx. Assess the glottal diameter to determine an appropriate ET tube size. If possible, avoid directly contacting the epiglottis as this can cause damage leading to swelling and laryngeal spasm.

5. Spray the larynx with a single spray of lignocaine (ALL cats, AND dogs with sensitive reactive/inflamed larynx). Test the spray before use to ensure the pump mechanism is loaded.
6. Allow at least 60-90 seconds for the local anaesthetic to take effect. Initially the larynx may spasm due to irritation from the spray but once the local anaesthetic has taken effect the larynx should relax. During the waiting period the tongue should be relaxed and the laryngoscope removed from the oral cavity.
7. Flow-by oxygen should be provided, and additional induction agent administered if further relaxation of the jaw or larynx is required.
8. Lightly lubricate the balloon of the selected ET tube avoiding the lumen and side-port (Murphy-Eye) of the endotracheal tube to prevent obstruction. Cuff lubrication has been shown to reduce pulmonary aspiration.[28]
9. Reposition the patient, with their head and neck extended, (to allow better visualisation and personal comfort this can be at more of an angle to the table than before). Use the laryngoscope as before to depress the tongue.
10. With the laryngoscope in place, there should be a clear view of the arytenoids. Guide the ET tube over the epiglottis, aiming the bevel at the most ventral point of the laryngeal opening. Gently advance the tip of the ET tube through the laryngeal opening during inspiration (the laryngeal folds should abduct), a gentle twisting motion can be employed to aid passage of the tube. It is very important to visualize the tube passing between the arytenoids and into the glottal opening. It is not sufficient simply to pass the tube over the

Table 6: <u>Endotracheal Intubation (*cont.*)</u>

epiglottis as this will most likely result in the tube passing into the oesophagus. Other methods to assure proper placement include observing condensation in clear tubes as the patient breathes, auscultation of lung sounds and observation of a capnography trace.

11. Once correctly positioned, the ET tube should be secured by tying rolled gauze first around the tube then in a bow behind the canines and around upper or lower jaw; or behind the base of the head in cats and brachycephalic breeds.
12. Connect the ET tube to the breathing circuit. Remember that the cuff can cause shear damage to the wall of the trachea if twisted so during patient repositioning the ET tube should be disconnected from the breathing system.
13. Inflate the ET tube cuff to achieve an appropriate tracheal seal.
    ET tube cuff inflation techniques: An inflatable cuff has a pilot tube with a port for a syringe. It is ***<u>important not to over-inflate</u>*** the cuff as tracheal ischaemic necrosis or rupture can result.[29] To ensure correct inflation pressure, attach a syringe full of air to the pilot tube; inflate the lungs by forcing air down the endotracheal tube (by squeezing the reservoir bag) and listen for air escaping around the tube; slowly inject air into pilot tubing, inflating the cuff until you no longer hear air escaping. Once the cuff has sealed the trachea, the leak will cease, and an optimal airway pressure of 20–30 cmH_2O should be maintained (if an airway manometer is present).[30] Some pilot tubes have a valve, while others have to be clamped to keep the cuff inflated. A reliable method for ensuring that cuff pressures are within the recommended range is to use a cuff monitor to inflate cuffs. A cuff monitor is essentially a low-pressure manometer similar to those used for Doppler blood pressure measurement that is attached to the pilot balloon of the cuff and provides a measure of intra-cuff pressure.
14. Remember to assess your patient's vital parameters immediately after intubation.

Anaesthetic Maintenance Phase

POSITIONING

During anaesthesia patients should, if possible be positioned in a normal physiological position. The head should be extended to promote a 'free' airway and reduce the likelihood of endotracheal tube kinking. The eyes should be protected from contact with abrasive surfaces and corneal lubricant should be applied following induction to protect the eyes from corneal ulceration.

TEMPERATURE REGULATION

Anaesthesia invariably effects temperature regulation. This will typically manifest as hypothermia however during warmer weather large hairy dogs may overheat. Small animals with large surface area to volume ratios lose heat rapidly under anaesthesia and sedation but all animals may develop clinically relevant hypothermia. Hypothermia has many physiological effects and will delay greatly a patient's recovery from anaesthesia.[31] Care should be taken to monitor body temperature, and when required provide supplemental heating to maintain a patient's temperature to as close to normal during anaesthesia. Thermal support may include warm IV fluids, use of a fluid line warming device, insulation on the patient's extremities (e.g., bubble wrapping of the feet), heated conductive warming blankets, circulating warm-water blankets, and warm air circulation systems. Do not use supplemental heat sources that are not designed specifically for anaesthetized patients as they can cause severe thermal injury.[32]

INTRAVENOUS FLUIDS

The provision of fluids during anaesthesia, as well as the type and volume used, should be based on the individual patient's signalment, physical condition, and the length and type of procedure. The dogma of administering crystalloid fluids at 10 mL/kg/hr perioperatively, with higher volumes for anaesthesia induced hypotension is not evidence-based and should be re-evaluated. Such high fluid rates are likely contributors to adverse patient outcomes, including increased body weight and lung water; abnormal pulmonary function; coagulation deficits; reduced gut motility; reduced tissue oxygenation; increased infection rate; decreased wound healing; decreases in packed cell volume and total protein concentration.[33, 34]

In the absence of evidence-based anaesthesia fluid rates for veterinary patients and taking into consideration the growing weight of evidence relating to the adverse effects of hypervolemia, the authors suggests that rarely should there be a need to exceed 3 mL/kg/hr of balanced crystalloids in dogs and cats undertaking surgical procedures. Fluids volumes administered above this rate should be for the purpose of resuscitation (i.e. replacing blood volume as it is lost from the intravascular space).

Experimental data in isoflurane anaesthetised dogs and clinical studies in people suggest that crystalloid fluid replacement ratios from 4 to 5 mL or greater for each millilitre of lost blood (4:1 or 5:1) are required.[20, 35-37]

Monitoring

Clinical observation and assessment by a vigilant anaesthetist is essential for safe patient care during the peri-anaesthetic period. During the maintenance period ***good monitoring is critical***. The person monitoring the patient should be suitably trained and skilled and ***should not*** be the practitioner performing the procedure. Clinical monitoring should be supplemented with monitoring devices as necessary, to assist the practitioner responsible for the anaesthesia. The anaesthetist whose sole responsibility is the provision of anaesthesia care for that patient must be constantly present from induction of anaesthesia until the patient has recovered. In exceptional circumstances brief absences of the anaesthetist primarily responsible for managing anaesthesia may be unavoidable. In such circumstances observation, including recording of observations of the patient and a plan for responding to significant changes in monitored physiological variables must be temporarily delegated to a suitably trained and skilled colleague who is judged to be competent for the task.

MINIMUM MONITORING STANDARDS

The monitoring of a patient undergoing any type of anaesthesia should include regular assessment and accurate recording keeping of physiological parameters including circulation, oxygenation, ventilation and temperature, as well as diligent post-anaesthetic care in the recovery period:

The following guidelines for anaesthesia monitoring are adapted from existing standards developed by The American College of Veterinary Anesthesia and Analgesia (ACVAA).

- **Circulation**

  Objective: to ensure adequate circulatory function.

  Methods:

  - Palpation of peripheral pulse to determine rate, rhythm and quality, and evaluation of mucous membrane colour and capillary refill time (CRT).
  - Auscultation of heart beat (stethoscope; oesophageal stethoscope or other audible heart monitor). Continuous (audible heart or pulse monitor) or intermittent monitoring of the heart rate and rhythm.
  - Pulse oximetry to determine the % haemoglobin saturation.
  - Electrocardiogram (ECG) continuous display for detection of arrhythmias.
  - Blood pressure:
    a. Non-invasive (indirect): oscillometric method: Doppler ultrasonic flow detector
    b. Invasive (direct): arterial catheter connected to an aneroid manometer or to a transducer and oscilloscope.

  Recommendations: Continuous awareness of heart rate and rhythm during anaesthesia, along with gross assessment of peripheral perfusion (pulse quality, mucous membrane colour and CRT) are mandatory. Blood pressure and ECG should also be monitored.

- **Oxygenation**

  Objective: to ensure adequate oxygenation of the patient's arterial blood.

  Methods:

  - Pulse oximetry (non-invasive estimation of haemoglobin saturation).
  - Arterial blood gas analysis for oxygen partial pressure (PaO_2).

  Recommendations: Assessment of oxygenation should be done whenever possible by pulse oximetry, with blood gas analysis being employed when necessary for more critically ill patients.

- **Ventilation**

  Objective: to ensure that the patient's ventilation is adequately maintained.

  Methods:

  - Observation of thoracic wall movement or observation of breathing bag movement when thoracic wall movement cannot be assessed.
  - Auscultation of breath sounds with an external stethoscope, an oesophageal stethoscope, or an audible respiratory monitor.
  - Capnography (end-expired CO_2 measurement).
  - Arterial blood gas analysis for carbon dioxide partial pressure ($PaCO_2$).
  - Respirometry (tidal volume measurement).

  Recommendations: Qualitative assessment of ventilation is essential as outlined previously, and capnography is required when using continuous mechanical/mandatory ventilation, with blood gas analysis as necessary.

- **Temperature**

  Objective: to ensure that patients do not encounter serious deviations from normal body temperature.

  Methods:

  - Rectal thermometer for intermittent measurement.
  - Rectal or oesophageal temperature probe for continuous measurement.

  Recommendations: Temperature should be measured periodically (ideally at least every 15 minutes) during anaesthesia and recovery; and if possible checked within a few hours after return to the wards (more frequently if temperature abnormalities are present).

- **Record keeping**

  Objectives:

  - To maintain a legal record of significant events related to the anaesthetic period.
  - To enhance recognition of significant trends or unusual values for physiologic parameters and allow assessment of the response to intervention.

  Recommendations:

  - Record all drugs administered to each patient in the perianaesthetic period and in early recovery, noting the dose, time, and route of administration, as well as any adverse reaction to a drug or drug combination.
  - Record monitored variables on a regular basis (minimum every 5 minutes) during anaesthesia. The minimum variables that should be recorded are heart rate and respiratory rate, as well as oxygenation status and blood pressure.
  - Record heart rate, respiratory rate, and temperature in the early recovery phase.

- Any untoward events or unusual circumstances should be recorded for legal reasons, and for reference should the patient require anesthesia in the future.

RECOVERY PERIOD

Half of all anaesthesia related deaths occur during the recovery period (47 % in dogs, 60% in cats), most frequently during the first 3 hours.[38] ***It is critical that patients are diligently monitored during this period*** to ensure a safe and comfortable recovery from anaesthesia. Extubation should ONLY be performed once the patient has regained a gag-reflex and no patient should be left intubated and unattended during the recovery period.

Patient care during recovery from anaesthesia

The frequency with which patients are monitored following anaesthesia will vary depending on the individual patients' procedure, signalment, disease process, pain profile, and on-going medical and/or surgical management requirements. The following recommendations are considered mandatory for the provision of care during the period following extubation or recovery from anaesthesia:

- Observation of respiratory pattern.
- Observation of mucous membrane colour and CRT.
- Palpation of pulse rate and quality.
- Measurement of body temperature, with appropriate warming or cooling methods applied if indicated. Do not use supplemental heat sources that are not designed specifically for anaesthetized patients as they can cause severe thermal injury.
- Re-apply eye lubrication during the recovery period until an appropriate blink reflex is present, particularly if an anticholinergic was administered.
- Manually express a patient's urinary bladder if distended to minimise any associated discomfort.
- Observation of any behaviour that indicates pain, emergence delirium or dysphoria, with appropriate pharmaceutical intervention as necessary. Re-assess the patient's pain level and, if necessary, adjust the pain management plan.
- Other measurements as indicated by patient's medical status, e.g. blood glucose, pulse oximetry, PCV, TP, blood gases, etc.

Patient discharge

Discharge of patients having undergone anaesthesia should only occur after the patient is awake, aware, warm, comfortable and ambulatory (if the patients conditions allows). Evaluate the animal for its responses and its ability to interact safely with owners and maintain physiologic homeostasis. Provide written instructions for owners, outlining the dose and potential side effects of analgesics and other medications to be given to the patient at home.

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APPENDIX 1

RECOMMENDATIONS FOR PERFORMING LOW-FLOW ANAESTHESIA

During Induction

- Use a total fresh gas flow that approximates minute ventilation and increase the fresh gas flow only if the measured (inspired) concentrations of oxygen and anaesthetic vapour are less than the set concentrations on the vaporizer.
- Decrease vaporizer setting as the difference between exhaled and inspired anaesthetic concentrations diminish. Monitor the exhaled concentration of anaesthetic vapour to ensure adequate anaesthetic depth.

During Maintenance

(When expired anaesthetic concentration approaches the inspired concentration)

- Estimate patient oxygen consumption to be 5 mL/kg/min.
- Set total oxygen flow (oxygen + 21% of air flow if used) to equal estimated oxygen consumption.
- Add 200 mL/min to total oxygen flow if using a sidestream gas analyser that does not return sampled gas to the circuit.
- Add the volume of any estimated/quantifiable leaks in the system to the total oxygen flow.
- Oxygen flow can be reduced in 50 mL/min increments until the inspired oxygen concentration begins to decrease.
- Monitor inspired oxygen concentration and set low oxygen concentration alarm at desired level.
- Monitor exhaled anaesthetic vapour concentration to ensure adequate MAC. Set alarm for desired level of expired vapour concentration.

During Recovery/Emergence

(When expired anaesthetic concentration approaches the inspired concentration)

- Increase the total fresh gas flow to greater than 200 mL/kg/min (effectively converting the system to a non/minimal-rebreathing circle system). This will promote the scavenging of exhaled anaesthetic agents from the system.

***NOTE: Specific technical training in this technique and/or the use of gas analysers to monitor inspired and expired oxygen and anaesthetic partial pressures is recommended.

APPENDIX 2

Quick Reference Chart: Oxygen Flow Rate Guidelines for Rebreathing Systems (L/min)				
Weight (kg)	During Induction, and Changes in Depth (50-100 mL/kg/min)	During Maintenance (20-50 mL/kg/min)	Low-Flow Anaesthesia During Maintenance (5-10 mL/kg/min)	During Emergence – Minimal rebreathing system† (200-300 mL/kg/min)
2.5	0.25 – 0.3	0.25	0.25*	0.5 – 0.8
5	0.3 – 0.5	0.25	0.25*	1.1 – 1.5
10	0.5 – 1	0.25 – 0.5	0.25*	2 – 3
15	0.8 – 1.5	0.3 – 0.75	0.25*	3 – 4.5
20	1.0 – 2	0.4 – 1	0.25*	4 – 5
25	1.3 – 2.5	0.5 – 1.25	0.25*	5
30	1.5 – 3	0.6 – 1.5	0.25 – 0.3*	5
40	2 – 4	0.8 – 2	0.25 – 0.4*	5
50	2.5 – 5	1.0 – 2.5	0.25 – 0.5	5
60	3 – 5	1.2 – 3	0.3 – 0.6	5
70	3.5 – 5	1.4 – 3.5	0.35 – 0.7	5
80	4 – 5	1.6 – 4	0.4 – 0.8	5
90	4.5 – 5	1.8 – 4.5	0.45 – 0.9	5
100	5	2.0 – 5	0.5 – 1.0	5
150	5	3.0 – 5	0.75 – 1.5	5
>150 kg Switch to large animal anaesthesia machine				

* At flow rates less than 250 mL/min, some precision vaporizer and flowmeters may not accurately deliver the dialled vaporizer concentration and oxygen flow.
† Minimal rebreathing occurs only when the oxygen flow matches or exceeds the patient's minute ventilation.

APPENDIX 3

The University of Melbourne U-Vet Animal Hospital Surgical Safety Checklist

BEFORE INDUCTION

Surgeon: _____ **Anaesthetist:** _____ **Nurse:** _____

- SURGEON AND ANAESTHETIST +/- NURSE CONFIRM **PATIENT DETAILS**
  - ☐ Identity
  - ☐ Procedure
  - ☐ **CPR CODE** confirmed
  - ☐ **TPR** AND **BLOOD WORK** performed and reviewed
  - ☐ CONFIRM **SURGERY SITE** L / R, front/hind limb
  - ☐ Are there second surgery sites?
  - ☐ Is a scope or other imaging planned?
- ANAESTHESIA **SAFETY CHECK** COMPLETE
  - ☐ Anaesthesia machine checked
  - ☐ Monitoring equipment checked
  - ☐ Ventilator checked
- **PRE-EXISTING CONDITIONS** OR SURGICAL/ANAESTHESIA **RELATED RISKS** IDENTIFIED AND REVIEWED
  - ☐ Yes
  - ☐ No
- ARE **ANTIBIOTICS** NEEDED?
  - ☐ Yes, and ready to administer
  - ☐ No
- IS **PRE-OP OR INTRA-OP IMAGING** REQUIRED?
  - ☐ Yes, Request form submitted Yes No
  - ☐ No
- NURSE AND SURGEON
  - ☐ CONFIRM PATIENT **POSTITIONING**, SPECIAL **EQUIPMENT, IMPLANTS** and **OR SETUP**

Patient Identification Label

BEFORE SKIN INCISION

Surgeon: _____ **Anaesthetist:** _____ **Nurse:** _____

☐ CONFIRM ALL **TEAM MEMBERS** HAVE INTRODUCED THEMSELVES BY **NAME** AND **ROLE**

- SURGEON AND NURSE VERBALLY CONFIRM
  - ☐ Patient Identity
  - ☐ Procedure
  - ☐ Surgery site
- Ask ANAESTHETIST has **ANTIBIOTIC PROPHYLAXIS** been given at least 30 minutes previously?
  - ☐ Yes
  - ☐ No
- SURGEON AND ANAESTHETIST VERBALLY CONFIRM **ANTICIPATED CRITICAL EVENTS**
  - ☐ Blood test results reviewed
  - ☐ What are the critical steps?
  - ☐ What is the anticipated surgical time?
  - ☐ What is the anticipated blood loss?
  - ☐ Are blood products available if blood loss anticipated?
  - ☐ Are there any anaesthetic concerns?
- NURSE REVIEW
  - ☐ Has sterility (indicators) been confirmed?
  - ☐ Confirm implants / equipment in the OR
  - ☐ Surgical sponges counted and reported
- IS **IMAGING DISPLAYED IN OR?** SURGEON AND NURSE
  - ☐ Yes
  - ☐ No or Not applicable
- SURGEON ASKS ANAESTHETIST
  - ☐ Is anaesthetic depth adequate for surgery?
  - ☐ Is additional induction agent available?

BEFORE PATIENT LEAVES SURGERY

Surgeon: _____ **Anaesthetist:** _____ **Nurse:** _____

- NURSE CONFIRMS
  - ☐ Name of procedure has been recorded
  - ☐ Record if sterility was compromised, how?
  - ☐ Sponge count reported and correct
  - ☐ Any equipment concerns
- IS **POST-OP IMAGING** IS REQUIRED?
  - ☐ Yes
  - ☐ No
- CONFIRM **LOCATION OF RECOVERY**
  - ☐ Wards or ICU
- SURGEON AND ANAESTHETIST **REVIEW KEY CONCERNS FOR RECOVERY AND POSTOP MANAGEMENT PLAN**
  - ☐ Were there any anaesthetic concerns?
  - ☐ Were there any surgical concerns?
  - ☐ Analgesia
  - ☐ Antibiotics
  - ☐ Intravenous fluid rate
  - ☐ Particular medication
- SURGEON AND NURSE
  - ☐ Confirm responsibilities for submission of samples collected
  - ☐ CONFIRM **WHO WILL CALL THE OWNER** and **WHEN**

Prior to the patient progressing to the next stage, a minimum number of signatures must be obtained to ensure successful completion of each stage of the checklist. A signature from the surgeon and anaesthetist are mandatory at each stage.

DATE: _______________

Before it's too late – Conservation Medicine Research at Murdoch University

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Introduction

Research in the fields of wildlife conservation medicine involves studying the health of endangered wildlife species within ecological contexts in order to assist recovery efforts to conserve these species. The research focuses on disease as a threatening agent for endangered species, and the role that it plays in driving population declines and extinctions. Disease is increasingly being recognised as a major threatening factor to wildlife conservation, and we know that the emergence of new diseases is often associated with disease inter-relationships between human, animal and ecosystem health and changes in these inter-relationships associated with anthropogenic ecological change.

This paper provides an overview of research projects being undertaken at Murdoch University in the fields of wildlife and conservation medicine, with a particular focus on research on the health and ecology of Western Australia's threatened black cockatoo species.

Staff and students in the Conservation Medicine Program are involved in research on threatened wildlife species, throughout Australia and around the world. There is usually a paucity of baseline data available for the endangered wildlife species studied by staff and students in the Conservation Medicine Program; so our research often requires initial establishment of blood reference intervals for species investigated. This provides a critical clinical tool and knowledge base for studying wildlife population health and investigating future disease outbreaks. There is also continuing strong interest from the international scientific community in developing effective and ethical methods of tracking wildlife, given the importance of determining post-release survivorship of captive-bred and translocated individuals.

Black cockatoo health and ecology research

The Carnaby's cockatoo is an endangered black cockatoo species, endemic to Western Australia[1,2]. Long term research on the health and ecology of black cockatoos has been conducted by the Murdoch University research team since 2010, in collaboration with the Department of Biodiversity, Conservation and Attractions and Perth Zoo. The research has involved health sampling of wild Carnaby's cockatoo (*Calyptorhynchus latirostris*) nestlings, and GPS and satellite tracking of adults to better understand flock movements and habitat use at breeding sites.

The black cockatoo health research investigated the prevalence of beak and feather disease virus (BFDV), avian polyomavirus (APV), Adenovirus and *C. psittaci* infection in Carnaby's cockatoo nestlings at key breeding sites across this species' distribution range.

A total of 278 nestlings were sampled between 2010-2014. Blood, feather and swabs (conjuctival, choanal and cloacal) were collected to enable establishment of baseline health parameters and test the birds for BFDV, APV, Adenovirus and *C. psittaci.* PCR was used to screen for these diseases, and haemagglutination and haemagglutination inhibition testing was also used to screen for BFDV.

BFDV was detected in five (2%) nestlings based on PCR of blood samples. Eleven (4%) nestlings tested positive to APV, and three chicks were concurrently infected with both BFDV and APV. Five nestlings (2%) tested positive for adenovirus and 38 (14%) nestlings tested positive for *C. psittaci*. The clinical significance of these infections in Carnaby's cockatoo populations remains unknown.

The GPS and satellite tracking of Carnaby's cockatoos at breeding sites has enabled identification of critical habitat resources in the landscape, which will assist with conservation efforts to conserve this endangered species.

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Investigating Sudden Death in Cattle and Sheep

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Introduction

Sudden death is commonly reported in Australian livestock in part because our stock are often infrequently observed. This paper will discuss the diagnosis of the most common and/or important causes of sudden death in cattle and sheep on the Central Tablelands of NSW. These include bloat, hypomagnesaemia, lightning strike, enterotoxaemia, phalaris, nitrate, lead and cyanide poisoning. Anthrax which can occur on the western fringe of the Central Tablelands will also be discussed.

Causes of sudden death in cattle and sheep on the Central Tablelands

Bloat

While bloat classically occurs when cattle (and especially young weaned cattle) graze on fresh legume pastures, it can occasionally occur on other feeds and in sheep. Bloat can be diagnosed based on history and at necropsy with the hallmarks being conjunctival and tracheal haemorrhage, anterior congestion and posterior blanching and a ‘bloat line’ on the oesophagus at the thoracic inlet.

Nitrate poisoning.

Nitrate poisoning is most commonly encountered in hungry livestock (usually cattle) being fed millet hay, canola silage or oaten hay that is high in nitrates as a result of using nitrogenous fertilisers. Losses can be substantial but are unpredictable with the same fodder fed to cattle on several properties but losses only occurring on some. Measuring the nitrate and nitrite levels of the fodder and aqueous humor can confirm the diagnosis.

Hypomagnesaemia

Hypomagnesaemia is the major cause of mortality in adult cows on the Central Tablelands. It most commonly affects mature cows in the first 2 months of lactation. While hypomagnesaemia typically occurs in the late winter and early spring (when most cows on the Central Tablelands are lactating) it can occur after rain in the autumn, presumably due to lactating cows grazing pastures that are high in potassium and low in magnesium and sodium. It is difficult to definitely diagnose hypomagnesaemia at necropsy because, as a metabolic disease there are no specific gross or microscopic lesions (Parkinson et al). Hypomagnesaemia can be confirmed by measuring magnesium levels in the aqueous humor but in my experience this may not be conclusive, perhaps because aqueous levels may lag behind serum and cerebrospinal magnesium levels (Paynter D, pers. comma). Parkinson et al (2010) mentions measuring cerebrospinal and urine magnesium levels in addition to aqueous levels.

Lightning strike

Lightning strike occasionally causes sudden death in livestock on the Central Tablelands. It is suspected when multiple animals are found dead under trees, along fence lines and around self-feeders although single animals are no doubt also struck in paddocks. Observation and meteorological records should show electrical activity at the time of death.

Struck cattle may have characteristic (pathognomic) singe or burn makes that can be observed even several days after death.

Enterotoxaemia

Enterotoxaemia caused by *Clostridium perfringens* type-D is a common disease of sheep and is routinely diagnosed by history and necropsy findings. It can be confirmed by brain histology, epsilon toxin assay of small intestinal contents, glycosuria and glucose in the aqueous humor. While enterotoxaemia is reported in cattle and often suspected, the author has never conclusively diagnosed it. However as Jones et al (2015) noted, for a definitive diagnosis of *C. perfringens* type-D to be made in cattle, brain histology is essential and the presence of intestinal epsilon toxin, while supportive, is insufficiently sensitive or specific to be diagnostic.

Acute phalaris intoxication

Phalaris (*Phalaris aquatica*) is one of the most important pasture species in southern Australia and is well known to cause two apparently unrelated disorders; a chronic 'staggers' form and an acute 'sudden death' form. In turn two forms of 'sudden death' are described, the more common nervous form and the rarer cardiac form. The nervous form has been shown to be a peracute ammonia toxicity caused by an increased nitrogen load from sudden access to young, rapidly growing phalaris that is both high in protein and has a urea cycle enzyme inhibitor (Bourke et al 2005). High levels of ammonia in the aqueous humor can confirm the diagnosis.

Lead poisoning

Lead is the most common and important cause of poisoning in cattle worldwide (Seawright 1982) but is rare in sheep. While cattle intoxicated with lead probably always show neurological signs before death, these may not be observed. In the absence of clinical signs, lead poisoning is suspected when cattle are given access to new or disturbed paddocks, rubbish tips and house paddocks. Lead affected cattle may walk aimlessly until they become entangled in fences and other structures where they die. Lead poisoning can be confirmed even several days after death by measuring liver and kidney lead levels.

Cyanide poisoning

Cyanide poisoning is rarely diagnosed on the Central Tablelands in part because the most potent sources of cyanogenetic glycosides, grasses such as *Sorghum spp,* are not grown widely in the region. A case of cyanide poisoning poisoning in sheep, caused by the ingestion of the garden shrub African daisy (*Osteospermum spp*) will be presented. A diagnosis of cyanide poisoning is made on a history of access to plants containing cyanogenetic glycosides, consistent necropsy findings and analysis of the suspect plant. Proving that the animal ate the plant in sufficient quantities to kill it remains problematic because cyanide is highly labile making animal tissue (including rumen content) testing unreliable.

Urea poisoning.

Urea kills cattle and sheep remarkably quickly and the carcases may be found close to the source. History, rumen pH and blood or aqueous ammonia levels enable post mortem diagnosis.

Anthrax

Anthrax typically causes livestock to die suddenly in the 'anthrax belt' on properties with a history of anthrax and in the warmer months of the year. A feature of anthrax is that is causes deaths in a range of livestock classes (for example ewes, rams and lambs) and species. While blood exuding from orifices is regarded as a feature of anthrax it is not always observed and may occur with other diseases. The immunochromatographic test (ICT) enables an on-site preliminary diagnosis of anthrax while awaiting laboratory confirmation.

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Tips on sunshinevirus infection in pythons

Clinically important principles for diagnosis and management of sunshinevirus infection from an experimental infection study in Australian pythons

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Introduction

Sunshinevirus was first found in pythons from a Sunshine Coast collection that experienced an outbreak of neuro-respiratory disease in 2008.[1,2] A polymerase chain reaction (PCR) test for the virus was developed and testing has been available at Murdoch University to veterinarians since 2011. Sunshinevirus has since been found in collections across Australia and in most Australian python species.[3]

Although many cases of sunshinevirus infection have been found in association with disease, many pythons that test positive for the virus do not show clinical signs of disease. Some of these have been serially sampled and been found to shed virus for months, and in some cases, even years.[3] At least some of these animals have gone on to develop neurological signs. Because the date of first exposure to the virus in these naturally-acquired infections cannot be known, it has not been possible to determine the virus incubation period. Without this knowledge, it has been difficult to make informed recommendations about quarantine periods for newly acquired snakes, how best to diagnose infection, and how to manage infected animals. To address these knowledge deficits, a study at Murdoch University was recently performed where carpet pythons were experimentally infected with sunshinevirus under controlled conditions.[4]

Experimental Infection Studies

A number of infection studies were performed. In the first study, virus was inoculated directly into the body cavity (intracoelomic) or trachea of four coastal carpet pythons (*Morelia spilota mcdowelli*). Two different doses of virus were inoculated into the snakes, and all four pythons became infected. In the second study, two healthy south-western carpet pythons (*M. s. imbricata*) were co-housed with infected coastal carpet pythons from the first study. This was to explore the natural transmission of sunshinevirus. In this second study, two other south-western carpet pythons were inoculated with virus. In both studies, control animals were sham-inoculated with virus-free medium.

Oral and cloacal swabs, and blood, were collected from all the snakes in all the studies once or twice a week. All pythons were monitored for 4-12 months, before they were euthanased for necropsy. In addition, environmental swabs were collected from the pythons' enclosures, both while the infected snakes occupied them, and after the snakes had been removed following euthanasia. Other surfaces in the snake room were also sampled with swabs.

Key Results

- Five out of six virus-inoculated snakes became infected. One of the two snakes put in contact with an infected animal became infected. Control (sham-inoculated) animals did not become infected.

- Infection was first detected by PCR within 14 days of direct viral inoculation. Oral-cloacal swabs and blood samples were both PCR-positive at that time. PCR testing does not discriminate between viable and inactivated virus.
- Viable (replicating) virus capable of infecting another snake could be cultured from swabs (from 17 to 238 days after inoculation) and blood. The first swab sample tested for infectivity was from day 17 and the last sample tested was from day 238.
- Once a positive PCR result was obtained, each subsequent sample that was tested was also positive. This applied to both oral-cloacal swabs and blood samples.
- One of the two pythons co-housed with an infected python became infected after four months of continuous cohabitation. This was interpreted as a natural, horizontal transmission of virus. The second python that was co-housed with an infected python did not become infected, despite sharing an enclosure for six months.
- Four of the six infected pythons developed obvious clinical signs of disease, from two-and-a-half to seven months after infection was first detected. Non-specific signs of disease included dysecdysis (two pythons), anorexia (two pythons), and dyspnoea (one python). Neurological signs were recorded in three out of the six infected pythons and ranged from disorientation and incoordination (three pythons), to neck twisting and abnormal mouth gaping, which was worse with stimulus (one python). Occasional kinking or flaccidity of the caudal tail (three pythons) and intermittent muscle fasciculations (two pythons) were also observed. Two infected pythons showed only equivocal signs of disease during four months of daily monitoring.
- All six infected pythons had histological evidence of sunshinevirus infection. Changes in the brain (vacuolation and gliosis) were the most consistent finding, but pathology was found in a wide range of tissues, e.g. cytoplasmic inclusions in epithelial cells in the respiratory tract, renal ducts, and gall bladder. The severity of histopathology was not closely correlated with the length of infection or the observed clinical signs of disease. However, pythons that had been exposed to the virus for longer had pathology in a greater range of tissues.
- Estimated total white blood cell counts (eWCCs) increased following virus exposure, peaking two to five months after inoculation. The eWCCs then gradually declined. Estimated total white cell counts increased the most (five-fold) in the pythons that were inoculated with virus into the body cavity. Two out of three pythons inoculated with virus intratracheally, as well as the python that became infected through exposure to an infected snake, had similar, but more modest (two-fold), white cell increases. The greatest increases were in lymphocyte and monocyte counts, while heterophil counts typically decreased. Statistical analyses of these changes are pending.
- The two control pythons, one inoculated python and one python that was in contact with an infected python, were always PCR-negative and that result is assumed to mean they remained uninfected. They showed no obvious clinical signs of disease over four, nine, five or eight months of observation, respectively, and no histological lesions consistent with sunshinevirus infection were detected in their tissues.
- Areas in direct and indirect contact with infected pythons (e.g. perches, water bowls, sampling areas) yielded small amounts of virus that could be detected by PCR, but infectious virus (viable virus that could be isolated into cell culture) was not detected.

Virus detected by PCR was found in excreted materials (faeces, urine and proteinaceous [hemipenal] plugs).

Limitations of experimental infection studies

A limitation of experimental infection studies is that it is not known how closely the conditions, especially the routes of administration and doses of virus used, correlate with those in naturally-acquired infections. However, there are several factors that suggest that extrapolation from experimental to naturally-acquired infection was valid for these studies. First, the second study included an example of a naturally-acquired infection. Second, the patterns of infection and disease observed in all the experimentally infected animals were comparable, regardless of the route and dose of virus administered. Finally, the clinical and pathological results were comparable with observations of snakes from Australian collections that naturally acquired the infection.

Detecting sunshinevirus infection

Species susceptibility

The experimental infection studies confirmed the susceptibility of two sub-species of carpet python to sunshinevirus infection. Previous research[3] has shown that most species of Australian pythons are susceptible. Sunshinevirus has also been found in a few exotic species of pythons and boas from collections in Germany and Thailand,[5,6] suggesting that these related species should also be susceptible. The susceptibility of other snake and reptile species is unknown. Only limited numbers of venomous snakes and other reptile species have been tested.[3]

Polymerase chain reaction (PCR)

Of all the methods employed to determine the infection status of the pythons in the experimental infection studies, PCR was the most specific, sensitive and versatile. It was used on a variety of samples from live snakes and consistently detected virus in samples from animals that had been inoculated with virus, once they commenced viral shedding. This is probably the most useful test clinically. Because it is very sensitive, it can accommodate considerable variations in sample quality. However, it does not differentiate between viable and inactivated virus, and because of its sensitivity, care must be taken to avoid contamination of samples with even small amounts of virus from other animals or the environment. Since infected animals seem to shed virus persistently, a negative or positive PCR result is likely to be a reliable indicator of an animal's infection status. However, if there have been recent opportunities for introduction of virus to a collection (e.g. new snakes brought in, or a snake loaned out for breeding has returned to the collection), then two samples, spaced at least six weeks apart, while an animal is in quarantine, may be required. In all situations, if an equivocal result is obtained, a second test at least six weeks after the first, is suggested to clarify the snake's infection status.

Estimated white cell counts

Estimated white cell counts are of limited use in detecting sunshinevirus. The limitations of this method include the inherent inaccuracies in the technique itself, the time required for changes to manifest (up to five months to reach peak levels in the experimental infection studies), the inconsistency in changes observed and the lack of specificity of such changes for sunshinevirus. Detection of indicative changes in absolute and relative white cell ratios may be used to suggest a viral infection in the absence of clinical signs. However, the absence of an elevated eWCC cannot be relied on to rule out an infection.

Clinical signs

Clinical signs were not a sensitive or specific indicator of sunshinevirus infection. When obvious clinical signs were noted in infected pythons, they commenced nearly three months after inoculation, whereas virus shedding (including shedding of infectious virus), had been detected more than two months earlier. Some infected pythons never showed obvious clinical signs of illness and only half (three out of six) showed neurological signs during 3-12 months of infection. Nevertheless, clinical signs may be used to suggest disease and prompt further testing.

Although only six pythons in the study became infected, two manifestations of clinical disease were observed.

1. Obvious signs of disease following months of asymptomatic infection (one python)

In this one south-western carpet python, no signs of disease were seen for 112 days after inoculation. On day 113, the python refused a feed for the first time, and a day later it started to display neurological signs. These signs included abnormal mouth gaping, neck twisting and incoordination. The mouth gaping and neck twisting could be triggered by external stimulus, such as a hand moving towards the animal.

2. Slow progression of mild signs of disease (five pythons)

The pythons in this group initially displayed signs of disease that were not always obvious, such as a possible head tremor. These signs were only displayed intermittently. Of the five pythons in this disease manifestation, those that were euthanased later developed more obvious signs of disease (see table below), and displayed them more frequently, indicating that the disease was progressive.

Table. Clinical signs recorded in pythons with slow disease progression

Python sub-species	CCP[a]	CCP	SWCP[b]	CCP	CCP
Length of observation period (days)	111	117	148	359	360
Signs of disease detected (total days)	22	2	42	183	20
First and last days disease detected	23 87	2 82	49 148	79 359	6 360
Clinical sign					
Possible head tremor or twitch	X	X	X		
Muscle fasciculations				X	X
Reluctance to move		X	X	X	X
Possible caudal flaccidity	X			X	
Tail kinking			X		X
Incoordination			X	X	
Twisted coils in repose					X
Raspy breathing					X
Clear fluid in mouth				X	
Dysecdysis	X		X		
Reduced mental activity			X		
Reduced muscle tone			X		
Anorexia				X	X

[a] coastal carpet python; [b] south-western carpet python

Virus isolation

Isolation of viable virus in cell culture was useful for indicating virus infection. It was used on samples from live snakes and consistently detected virus in samples from animals that had been inoculated with virus, once they commenced shedding virus. However, for clinicians, it is less useful than PCR. It is time-consuming and requires ready access to a specialised research laboratory, since sample quality is important, and reptile cell cultures are required for the virus isolation. The method is also not specific for detection of sunshinevirus. Cultures that become infected need to be tested by some other method, usually PCR, to determine the identity of the infecting virus.

Histology

In the experimental infection study cases, histology was a reliable method for indicating sunshinevirus infection when interpretation was performed by a pathologist experienced in reptile histopathology. Its biggest limitation is that it can only be performed on dead animals since the brain is the preferred specimen. It is also only suggestive and not specific.

Sunshinevirus contamination in the environment

During the experimental infection studies, surfaces outside the python enclosures were disinfected with 70% ethanol in water, or F10SC®, a veterinary disinfectant. As an enveloped virus, sunshinevirus is expected to have limited survival time outside a host and should be highly susceptible to commonly used disinfection solutions. However, these solutions do not destroy the viral genetic material which can be detected by PCR.

Since sunshinevirus was detected in cloacal swabs and products excreted by the pythons, it is not surprising that swabs of enclosure surfaces and cage furniture contained virus detectable by PCR. However, no infectious virus (viable virus that could be isolated into cell culture) was recovered from the swabs. This suggests that it was either inactivated virus, or that the quantity present was not sufficient to produce an infection. Sunshinevirus was also detected by PCR from swabs of areas immediately surrounding the infected snake enclosures and from areas that had been in contact with infected snakes, as well as the room's air vent, indicating contamination of the wider environment with viral genetic material.

Environmental contamination has the potential to cause a misleading positive PCR result if animals or samples are contaminated with residual viral genetic material from the environment. In order to address this problem, experiments investigating the destruction of viral genetic material were carried out on enclosures after routine cleaning. The disinfection treatment that was most effective at reducing viral RNA levels was 10% bleach (equivalent to 0.4% hypochlorite), freshly reconstituted, and left on for 30 minutes. More heavily contaminated enclosures required two treatments. Surfaces were rinsed afterwards to render them safe for animal contact.

Virus transmission

During the experimental infection studies, infectious virus was recovered from oral-cloacal swabs and blood, so the sampled fluids may be potential agents of viral transmission. Infection was produced by either tracheal or intracoelomic inoculation, suggesting that sunshinevirus could gain entry via the respiratory or circulatory systems. Therefore, potential routes of horizontal transmission include cloacal-oral and oral-oral, as well as inoculation directly into the circulatory system (perhaps by haematophagous ectoparasites). Mechanisms for cloacal-cloacal transmission (e.g. by mating) were not investigated by these studies.

Virus was not detected by PCR in samples from the control pythons or from within their enclosures. These animals were housed near infected animals (~5 cm between enclosures) in shared air space for more than a year. This suggests that aerosol transmission of virus is unlikely.

Transfer of infection from one python to another while co-housed together took approximately four months to occur. A second co-housed python did not become infected after six months of cohabitation. These findings suggest that even direct contact and shared facilities are not a reliable method of transmission, but it is unclear if other factors, such as the length of time that a python has been infected, influence how readily infection is transmitted.

In summary, results from the experimental infection studies support the possible routes of horizontal transmission as including cloacal-oral, oral-oral and via the circulatory system. Further investigation is needed on the possibility of fomites being a means of virus transmission.

Conclusion

The experimental infection studies described in this document have provided valuable information that can assist in providing guidance for detecting and managing sunshinevirus infection in pythons.

Testing for sunshinevirus by PCR plays an important role in the biosecurity of Australian python collections. It provides a rapid, highly specific and highly sensitive method for identifying the virus and can be performed on live animals. When an animal infected with sunshinevirus has been removed from a collection, special attention should be given to procedures for cleaning and disinfection, in order to avoid future misleading positive PCR results.

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Bites, scratches, scrubs - infection control in companion animal practices

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Introduction

Personal and environmental infection control practices (IC) are essential tools that prevent nosocomial (hospital-acquired) and zoonotic infections as well as reduce the drivers for the development of antimicrobial resistance (AMR). In recent years, companion animal veterinary clinics have reported an increase in the incidence and severity of nosocomial infections such as virulent feline calicivirus.[1-3] In addition, the emergence of zoonotic diseases such as brucellosis and Q fever in companion animal practice highlights the need for a paradigm shift in attitudes towards infection control to protect the health and safety of veterinary staff, their patients and the community. This is important because veterinary staff are required to ensure they promote safe practices within their workplace to reduce risks to clients and colleagues.[4, 5]

The development of AMR is associated with poor IC practices.[6] Effective hand hygiene is the mainstay of effective IC programs in human healthcare settings because it reduces the incidence of nosocomial transmission and thus reduces the need for antimicrobial use.[7, 8] Antimicrobial resistance is a global issue that requires cooperation between human and veterinary practitioners because it affects both human and animal health.[9] The Australian National AMR Strategy identifies the need to improve IC in veterinary practice (Strategic objective 4).[10]

Whilst there are similarities between human and veterinary health care contexts that allow veterinary adoption of current IC practices there are some important differences that require modification of protocols and approaches. For example, the unpredictability of working with animals, the requirement to perform some procedures in contaminated areas such as on the floor, and the lack of direct or indirect government funding. In fact, veterinary clinics may be more similar in process to human dental clinics than to medical facilities. Veterinary and dental clinics are predominantly owner operated and perform surgery in-house, mostly as day only procedures. Patients with a greater acuity are often managed in or transferred to 24-hour facilities.
This presentation will present the results of a survey of companion animal veterinary staff with regards their IC practices and high-risk behaviours.

Methods

A digital questionnaire was distributed via email, professional networks and social media to practicing companion animal veterinary staff over four months in 2016-2017. The Australian Veterinary Association Guidelines for Veterinary Personal Biosecurity [11] and Hand Hygiene Australia [12] were used for recommended IC practices. Focus groups of veterinarians and veterinary nurses were conducted to further expand on issues identified.

A total of 228 companion animal veterinarians and veterinary nurses responded to the digital questionnaire. Respondents were primarily from general practice clinics (70%) with the remaining comprising specialist and emergency centres (28%) and shelter or mobile practitioners (2%).

The questionnaire included questions on self-reported compliance rates of a range of high-risk behaviours, (such as recapping of needles, neonatal resuscitation) and glove use. The attitudes of respondents to a range of recommended personal infection control practices such as hand hygiene, the use of personal protective equipment and personal vaccination were also explored.

High risk behaviours

On average, 80% of veterinary staff had been bitten or scratched in the preceding six months. Most wounds were managed by the individual within the workplace. Sharps injuries were also a common occurrence with 60% of veterinarians and almost 50% of nurses sustaining a sharps injury in the preceding six months. Only 22% of veterinary staff completed an incident report after sustaining a bite, scratch or sharps injury.
A total of 8% of veterinarians and 14% of veterinary nurses performed mouth to mouth between one and five times in the preceding six months with one veterinarian reporting that they had performed unprotected mouth to mouth more than five times in the six-month period.

Hand hygiene

Respondents were asked to identify when they were more likely to wash their hands in several scenarios. Veterinarians reported that they washed their hands “always” or “mostly” 60% of the time before entering a consultation or treatment room. Nurses reported that they were “always” or “mostly” compliant 51% of the time. Washing hands before a procedure (“always” or “mostly”) increased to 92% for veterinarians and 78% for veterinary nurses. Ninety seven percent of veterinarians and veterinary nurses reported that they washed their hands “always” or “mostly” after performing a procedure. Comparing recent graduate veterinarians (5 years or less) with veterinarians that have graduated 20 or more years did not find any differences in hand hygiene compliance before entering a treatment room, before a procedure or after performing a procedure.

Discussion

The results of this survey confirmed that companion animal veterinary staff participate in a number of high-risk events during the course of their day-to-day work. The proportion of veterinary staff sustaining bites and scratches is similar to previous reports,[13, 14] including the observations that staff often do not seek medical care and self-prescribe antibiotic treatment [14]. Correct management of injuries is important because even minor injuries can cause discomfort and injuries that are more serious, can cause significant pain, loss of function, scarring or absence from work.[15, 16] In addition, blood loss exposes other staff to potential human infectious diseases.

The frequency of sharps injuries reported by respondents is similar to the level observed in other studies. A Canadian study found that 74% of veterinary staff had experienced a needle stick injury in the preceding 12 months [17] while an American study reported that 77% of staff had experienced a sharps injury [18]. Sharps injuries can transmit blood or bodily fluids as well as pharmaceutical products which can have a range of effects.[13, 18]
Furthermore, the low level of compliance with reporting requirements for workplace injuries is a concern because it obscures the scale of the problem and breaches current workplace health and safety laws. Understanding the true number and severity of injuries within the workplace will enable an evidence-based approach to managing and communicating risks to veterinary staff. This may lead to changes in the practice of veterinary science to reduce risks, such as greater caution with animal handling and the use of muzzles, animal restraint devices or sedation.
Unprotected mouth-to-mouth resuscitation is a risky behaviour and exposes the individual to zoonotic diseases such as Q fever and brucellosis.[11, 19, 20]The recent clusters of Q fever

associated with feline caesareans [21-23] highlight the risks from atypical reservoirs of *Coxiella burnetti*. Ensuring that veterinary practices have appropriate resuscitation devices available for use can easily reduce the incidence of this behaviour to zero.

It is interesting to note that the majority of veterinary staff believe that they are practicing good hand hygiene because they wash their hands after procedures. However, hand hygiene is a more complicated concept that requires a paradigm shift in veterinary settings. The WHO initiated the “5 Moments” program to simplify the process to those moments critical for pathogen transmission to protect the patient or everyone else. [12, 24] An attempt to introduce the 5 moments into veterinary practice was unsuccessful [25]. This is not surprising given the challenges and gaps in compliance experienced by human healthcare providers.[26-28] Compelling veterinary staff to perform regular and frequent hand hygiene to reduce the risk of nosocomial infection is a challenge worth undertaking. This study demonstrates the need for the development of a simple, cost effective and adaptable protocol that is suitable for a wide range of veterinary contexts. The universal adoption of this protocol will require a significant shift in the culture of the veterinary profession.

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How To: Overview of diagnostic approaches for small ruminant disease investigations

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Summary
A systematic approach to investigating diseases of small ruminants should aim to identify the epidemiological, clinical, pathological and ancillary information required for a definitive diagnosis. It determines what body systems are involved and which of the 5 pathological processes should be considered: inflammation; degeneration or malformation; vascular disorder; growth disorder; and pigmentary deposits. This leads to construction of a morphological diagnosis, estimating the duration, severity and distribution of sign(s) and lesions(s) to inform the nature of the pathological process(es) and informs the differential diagnoses. This differential diagnosis then directs the laboratory investigations, including a 'step-wise' choice of tests that can affordably provide the evidence, that when considered in relation to lesions and field observations, provides the definitive diagnoses and advises potential control and therapy options. Systematic livestock epidemiological, clinicopathological investigations with rigorous necropsy procedures and laboratory support, improves diagnoses and enhances surveillance for endemic, emerging and transboundary disease entities. A training course in NSW entitled "Production Animal Pathology for Field Veterinarians' has been developed and trained 6 cohorts of veterinarians in these skills between 2009-2011 and 2015-2017. That field veterinarians possess these skills is increasingly important as it supports the superior health status of Australian livestock in the increasingly challenged and often under-resourced global food security system.

Introduction
Future agriculture is a complex challenge, requiring the balance of dwindling land, water and non-renewable energy supplies including fertilisers, with increasing demand and trade in livestock and their products. This is occurring in an environment of rapid urbanisation with a disconnect from food production, plus increasing risks from global financial turmoil, climate change, political fragility and worryingly, the emergence of a robust dialogue on social media that largely rejects 'evidence-based solutions' for meetings these challenges. Managing emerging animal health issues that may potentially compromise our fragile food security system is a most significant challenge, one often misunderstood by political and agricultural policy makers. Animal health management systems rely on efficient surveillance and skilled diagnostics to enable rapid reporting and response. However, what are the skill levels of the field veterinary and laboratory service personnel that are required to deliver this service?

Pathology is a scientific 'art' requiring continuous learning and professional development, particularly as diagnostic challenges are continually emerging. Older veterinary scientists mostly find it remarkable that so many new disorders have emerged during their careers as veterinarians or veterinary public health scientists. In recent decades there have been many new infectious pathogens (e.g. neosporosis) and non-infectious disorders (e.g. new toxic plant entities) emerging, with older (e.g. Ovine Johne's disease; Bluetongue virus) and newer diseases transmitting across boundaries (e.g. Schmallenberg virus), plus older disorders finding the means of masquerading as something new or finally being discovered (e.g. atypical scrapie). Field veterinarians are the frontline for early recognition of new or unusual disorders, requiring they have confidence in their abilities to conduct through disease investigations, including necropsies of livestock and use of veterinary laboratory diagnostics. As public laboratories are now centralised and often struggle to maintain their services, fewer trained pathologists are performing necropsies. Further, the costs of animal transport, lab necropsies and carcass disposal has become increasingly prohibitive. This increases the reliance on field veterinarians to provide superior quality livestock disease investigations, that include systematic necropsies. This is an increasingly serious problem for the veterinary profession globally. There is an emerging expectation that veterinary curricula on production animals provide 'day one competencies' for veterinarians that are capable of contributing to improved livestock health surveillance services for the livestock industry-based global food security system. This is very challenging for most veterinary schools and there is an urgent need for the global food security community to recognise this deficiency and promote postgraduate training for livestock veterinarians to improve their disease investigation and necropsy skills. This paper discusses the 'how to' when conducting systematic disease investigations. It also provides an example of a successful initiative to enhance pathology training of field veterinarians in NSW in the 'Certificate in Production Animal Pathology for NSW Field Veterinarians', a collaboration of the veterinary schools at The University of Sydney and Charles Sturt University, with the NSW Local Lands Services (LLS) and NSW Department of Primary Industries (DPI). A similar course is operating in Victoria and such initiatives that deliver training in pathology for rural veterinarians to improve disease diagnostic investigations and quality surveillance information is suggested for other states and countries.

In conducting a systematic disease investigation, the key information required to direct the diagnostic process in small ruminants involves the gathering of the relevant evidence from: the epidemiological data; the clinical signposts; the detailed necropsy information; and consideration of ancillary diagnostic information in relation to the findings in the field.

The epidemiological investigation

This aims to determine the relevance of potential risk factors. Were the animals located in areas where infectious agents, teratogenic plants or nutritional aberrations may occur? Diligent examination of the environmental conditions including plants, feeds, soil, water, housing, plus animal, crop and forage treatments that occurred prior to and during the onset of the disease is advised. Considered information on epidemiological metrics, includes:

- signalment i.e. species, age, breed and sex of animals involved,
- the numbers of animals in the 'at risk' population,
- the numbers of animals in the 'morbidity' population,
- the numbers of animals in the 'mortality' population,
- the relevant spatial information: where the disease appeared to arise and is the current location relevant e.g. are arboviruses relevant, nutritional deficiencies or intoxications likely?
- the relevant temporal information on when the disease first occurred i.e. did the 'index' case occur recently, so the disease is considered per-acute, acute or subacute, or is it a chronic problem?
- the relevant management information: vaccination and therapeutic history
- for reproductive losses, the numbers of animals offered for service (joining rate), successfully mated (pregnancy tested), neonatal animals delivered, numbers of animals marked (marking rate) and weaned (weaning rate) is calculated as these measures enable attribution of the stage(s) in the reproductive cycle that the losses occurred. Knowledge of the most likely differential diagnostic list for diseases that cause known losses at each stage of the cycle then informs a more cost-effective disease investigation.[1]
- for plant and chemical intoxications, knowledge of the potential access of the animals to known toxic plants (e.g. *Phalaris* spp. etc) or feeds (e.g. cottonseed containing gossypol; silage or water with *Listeria* sp.) and history of chemicals previously used and possibly

stored (e.g. lead) requires detailed paddock examination and relevant regional knowledge, in addition to access to the published literature of the numerous resources available that document plants associated with livestock losses in Australia.[2]

- for inherited disorder investigations, examination of breeding information is required and as most newly recognised inherited abnormalities are transmitted in an autosomal recessive manner, both dam(s) and sire(s) will be heterozygous for the suspected defect. For flocks with single sire matings and have accurate records, a simple pedigree analysis may determine if familial relationships exist between the affected animals and their parents plus antecedents if several cases occurred. DNA parentage confirmation determine whether more than one sire is involved. Inspection of the database of animal disorders, 'Online Mendelian Inheritance in Animals' is advised as OMIA regularly updates the documented genotypes and phenotypes associated with inherited disorders (OMIA: https://omia.org/home).

The clinical examination

This aims to identify the most probable anatomical location of the lesions, using a similar approach to that commonly applied to companion animals. The clinician aims to identify dysfunctional areas, then use diagnostic aids, including necropsy where necessary, to provide appropriate therapeutic and disease control measures. The signs may help define and localise lesions to one or more functional regions and suggest whether the disease is either focal, multifocal or diffuse, in one or more of the 10 specific functional systems, including:

(1) cardiovascular;
(2) respiratory;
(3) alimentary;
(4) renal;
(5) neurological and ocular;
(6) muscular;
(7) skeletal;
(8) lympho-haemopoetic;
(9) skin and appendages;
(10) reproductive systems.

The examination commences with a clinical history to ascertain the rapidity of onset, the duration of illness, and whether the signs are intermittent, continuous, progressive or non-progressive. Prior to restraining the animal, one is usually able to identify abnormalities of:

- condition: condition score 1-5, and weakness ie 'tail of the mob'
- cardinal signs e.g. tachycardia, dyspnea
- mentation: depression, hypo- or hyperaesthesia
- posture: recumbency, ambulatory, circling
- gait: paresis or paralysis; dysmetria, and
- head position e.g. head tilt

Following restraint, heart and respiration rate and body temperature of one or more animals and a closer assessment of the mental state should be recorded. If neurological disease is suspected, cranial nerve examination aims to identify if: vestibular disease is present (VII), if ocular responses are normal (II and III, involving assessment of the menace response, palpebral and papillary light reflexes, and the presence of Horner's syndrome), an auditory (cranial nerve VIII) responses is normal, and if nasal irritation (I) or abnormal facial and pharyngeal responses (V, VIII, XII) are present. In most small ruminants, assessment of muscle tone (hypo- or hypertonic), quality of patellar and withdrawal reflexes and the presence of any placement deficit or absence of proprioceptive responses, is amenable and should be recorded. Diagnostic imaging of skeletal structures is increasingly available and often very useful, enhanced by the increasing availability of CAT and MRI imaging equipment, particularly at university clinics, supplementing more traditional X Ray studies that have been reported in small ruminant studies for many decades.

An important question is whether the clinical signs suggest one or more body systems are involved, if a focal or a more widely disseminated lesion is likely, and if the disease history suggest the disease may be progressive? Generally, inflammatory diseases produce multifocal lesions and the clinical signs displayed may be generalised and progress more rapidly than with inherited degenerative disorders. Signalment (age, breed and sex) is important, particularly as inherited degenerative conditions such as storage diseases, abiotrophies and movement disorders are often confined to specific breeds or their derivative breeds and crossbreeds. Examining all the body systems to locate possible lesions facilitates the differential diagnoses, enabling more rational investigations and use of diagnostic aids, laboratories, therapies, control measures, and importantly, helps decisions on euthanasia and necropsy. The usual procedures used in companion animals are mostly appropriate for small ruminants, including the detailed systematic neurological examination as recently described[1].

Another important consideration is whether the dominant signs are secondary to disease in another organ system (e.g. liver or kidney) or a generalised deficiency or toxic disorder (e.g. hypocalcaemia, urea poisoning) resulting in systemic or neuromuscular signs. Ruminants with severe liver disease may develop hepatic encephalopathy that is likely due to endogenous toxic substances (especially ammonia) produced in the gut that are not detoxified by the liver because of extensive hepatocellular injury or the presence of portosystemic shunts, resulting in progressively severe spongiform change in the CNS white matter. Renal failure may also produce neurological signs (e.g. uraemic encephalopathy). A common cause of metabolic encephalopathy is hypoxia where insufficient oxygen to the brain causes neural injury.

Finally, at this stage it may be appropriate to consider therapy as a diagnostic aid. The classical approach for the diagnosis of thiamine deficiency Polioencephalomalacia (PEM) and Hypocalcaemia includes a positive clinical response to IV thiamine (10mg/kg) and calcium borogluconate therapy, respectively.[2]

The necropsy investigation

This aims to ensure appropriate diagnostic lesions are identified, described and then selected for microscopic examination, if required. If an intoxication is suspected, a search of the rumen contents for leaves of toxic plants occurring in the environment can ensure a diagnosis (e.g. *Trema aspera*). A rigorous process enables consideration of all abnormalities as attributable to one or more of the 5 pathological processes:

1. inflammation (e.g. viral, bacterial, protozoal and fungal diseases, plus other agents);
2. degeneration or malformation (e.g. metabolic disorders, intoxications, genetic diseases);
3. vascular disorder (e.g. ischaemia, trauma);
4. growth disorder (e.g. neoplasia, metaplasia) and
5. pigmentary deposit (e.g. melanosis, anthracosis).

Categorising the signs and lesions in this way will aid in developing the differential diagnosis for each disease mechanism, following deductive reasoning that:

- leads to construction of a morphological diagnosis, estimating the duration, severity and distribution of sign(s) and lesions(s) to inform the nature of the pathological process(es).

- informs the differential diagnoses, directing further investigations, including choice of ancillary diagnostics to arrive at the definitive diagnoses, and
- indicates the potential aetiological diagnoses, considering from the list of:
    1. trauma,
    2. neoplasia,
    3. metabolic,
    4. intoxication
    5. nutritional deficiencies,
    6. infectious disorders
    7. congenital and acquired genetic disorders.
- this suggests the most appropriate samples to be collected for ancillary laboratory examination,
- then directs the possible interim control and therapy options for the 'most likely' of the differential diagnoses of disorders until the definitive diagnosis is achieved.

An important consideration is that these examinations facilitate more rapid diagnosis of endemic and emerging disorders, enhancing our surveillance for incursions of transboundary diseases. Important examples included Foot and Mouth Disease (FMD) and Transmissable Spongiform Encephalopathies (TSEs), as disorders exist in this country presenting with symptoms and lesions initially considered suggestive of such diagnoses (e.g. Orf virus; segmental axonopathy, respectively). Further, the early detection, reporting, therapy and management of diseases of small ruminants reduces the welfare burden of unmanaged disease by reducing the potential for transmission of disease to other flocks and to our wildlife and human populations (eg Listeriosis).

Ancillary laboratory diagnostic examinations

These are very useful in achieving a definitive diagnosis and the best advice for veterinary clinicians is to establish a trusting relationship with an experienced pathologist who can help guide the investigation. This is essential to reduce the cost of the investigation, selecting tests in a 'step-wise' manner commencing with the most likely on the differential diagnostic list and discussing whether a surveillance program is available to support the investigation (e.g. TSE Surveillance Scheme). Haemogram and urine studies, plus biochemical profiling of serum, plasma, CSF, anterior ocular chamber and other body fluids may help support diagnostic evaluations, particularly if inflammatory (e.g. meningoencephalitis) or metabolic deficiencies (eg Ca, Mg) are present. The most common laboratory examinations for livestock species are for infectious pathogens, combining direct (e.g. body fluid microcopy, smears) and indirect (e.g. serology, pathogen isolation) tests with microscopic tissue examinations to confirm the presence of lesions attributable to the pathogen(s) demonstrated.

A critical issue in diagnostic pathology is awareness that demonstration of the presence of a pathogen is not necessarily diagnostic of the disease and that disease diagnosed at the laboratory may not necessarily be diagnostic of the main problem in the field. Pathogen detection information needs interpretation in relation to pathological lesion information, with both requiring interpretation in relation to the field epidemiological information.

Pathology for Field Vets Course, NSW

A course aimed at upskilling the disease investigative skills of District Veterinarians and Veterinary officers in NSW, was developed in 2008-2009 by a joint committee, led by Professor Peter Windsor and Dr Michelle Dennis of The University of Sydney, with A/Prof John Glastonbury from Charles Sturt University (CSU) veterinary school, the then Livestock Health and Pest Authorities (LHPA), and the NSW Department of Primary Industry (DPI). The course was run successfully in 2009, 2010 and 2011, comprising four 3-day workshops, held in Camden (sheep, cattle, poultry), Yanco (cattle feedlot), Dubbo (sheep abattoir, zoo), and Wagga (pigs, sheep, cattle). The course was revived in 2015, 2016 and 2017 following a period of consolidation of the LHPA into their new structure as the Local Land Services (LLS) and consolidated to three 5-day sessions (Wagga/Yanco, Dubbo, Camden). The course covers necropsy technique for production animals, detailed pathology of endemic and emergency diseases of production animals, including sheep, cattle, pigs, poultry, camelids, goats, horses, using clinical features of disease to localize abnormalities of a tissue, recognition and description of gross pathological features of disease, developing differential diagnoses and selection of samples to be submitted to a laboratory, use of diagnostic laboratories and quality of laboratory submissions, communication with the laboratory, and interpretation of laboratory findings, and communication of pathologic findings with the profession and scientific communities.

Coursework and activities were designed to address the following learning objectives, where by the end of the course, participants will can:

- Perform field-based necropsies in a systematic and thorough manner in making a diagnosis of the cause of sickness in and/or death of livestock.
- Report necropsy and other relevant disease investigation findings to veterinary pathologists and industry stakeholders including agencies responsible for primary industry, public health, animal welfare and law enforcement.
- Communicate learning in the disease investigation with colleagues & in 'Flock & Herd'.

The emphasis of the practical classes was on descriptive pathology, and how tissue changes would influence formation of differential diagnosis and direct laboratory investigations. The classroom-based components of each session focused on system-specific diseases, with each session preceded by distribution of gross pathology cases ("diagnostic exercises") to examine prior to the didactic session. Classroom discussions were based on these exercises, focused mainly on cattle and sheep, with inclusion of goats, camelids, pigs, birds, horses, fish and some wildlife. Each of the participants were asked to prepare to:

- Describe the lesion
- Provide morphologic diagnoses
- Provide differential diagnoses for suggested aetiologies
- Discuss potential ancillary investigations, including laboratory tests (if applicable)
- Provide a case study communication task for publication in 'Flock & Herd'

Following review of these cases, the instructors presented a variety of related lesions and disorders including those of similar lesions with a different aetiology and pathogenesis. Then followed case presentations from participants, involving their own disease investigations that exemplified lesions relevant to designated system/topics. Case presentations were 10-15 minutes, followed by approximately 5-10 minutes of classroom discussion. Each didactic topic covered via assigned exercises integrated the following: clinical examination of an animal/herd, collecting and recording of historical information, clinical terminology and description of clinical findings, localisation of lesions based on clinical findings, necropsy technique, recognition of macroscopic lesions, description of pathological findings, imaging of lesions, use of clinical and pathological differential diagnoses to direct diagnostic investigation, selection and submission of laboratory samples, interpretation of laboratory findings, the use of information resources, verbal and written communication of diagnostic investigation for clients and in particular, the scientific community, plus disease treatment and control, regulatory implications etc. Images from investigations were shared during discussions and where images were unavailable, a search for pictures of a similar lesion archived in various online resources was suggested. Useful resources used included various selected published papers[3,4,5,6,7,8,9] and the following:

- *Pathologic Basis of Veterinary Disease*, by M. Donald McGavin and James F. Zachary – reading suggestions which complement classroom topics will be given from this text
- O.L.I.V.E.R. (http://oliver.vetsci.usyd.edu.au/) – a USYD resource not in the public domain; contact Peter Windsor for login details.
- John King's Necropsy 'Show and Tell' (http://w3.vet.cornell.edu/nst/nst.asp)
- 'Flock and Herd' (http://www.flockandherd.net.au/)

This project fostered the partnership of the two universities with veterinarians of the LLS and DPI and enhanced numerous collaborations, with DV's contributing many articles to the LLS website 'Flock and Herd', many images to the USYD database O.L.I.V.E.R and case materials for the training BVSc and now DVM students. The trained DV's and VO's are more confident when training students on extramural rotations, particularly instructing students in performing necropsies and engaging in diagnostic investigation with greater confidence and higher levels of skill. These initiatives have led to several applied projects resulting in published papers[7,8] with some in preparation.

Conclusion

Encouraging a systematic approach to investigating diseases of small ruminants is important, utilising epidemiological, clinical, pathological and ancillary information identify which of the 5 pathological processes should be considered in developing a morphological and then the differential diagnosis. This process directs further investigations aimed at demonstrating additional information from pathogen detection or other diagnostic tests, to be evaluated with lesion description and definition, then considered with field-derived information to arrive at a definitive diagnosis and provision of control and therapy options. The systematic livestock disease investigation improves diagnoses and enhances surveillance for endemic, emerging and transboundary disease entities. A training course in NSW entitled "Production Animal Pathology for Field Veterinarians' was developed and trained 6 cohorts of veterinarians in these skills between 2009-2011 and 2015-2017. That field veterinarians possess these skills is increasingly important as it supports the superior health status of Australian livestock in the increasingly challenged and often under-resourced global food security system. With new disease entities continuing to emerge and the growing importance of food security and animal welfare being recognised as a global challenge, more highly skilled livestock disease surveillance veterinarians are required. They can ensure that future food security issues associated with increasing prosperity and demand for livestock protein in countries with rapidly developing economies and changing dietary preferences, can be met more efficiently with greater safety.

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Investigating Congenital Malformations in Cattle

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Summary

As there are many pathological processes capable of damaging the development of the embryo or foetus during gestation, what are the best approaches to resolve these problems? Firstly, investigators need to consider the epidemiology of potential risk factors and particularly, were the animals located in areas at vulnerable periods where infectious agents, teratogenic plants or nutritional aberrations may occur? Secondly, investigations are assisted by information on the metrics of breeding, including: the occurrence of successful matings; pregnancy status; neonatal animals delivered; then marked and weaned. These measures provide information on the success of each of the steps in the breeding cycle that produced normal and viable offspring versus abnormal animals. Identification of which stage(s) in the reproductive cycle that the losses occurred, offers a 'most likely' differential diagnostic list and informs a more cost-effective disease investigation. Thirdly, detailed laboratory examinations may be required, particularly where the presence of infectious agents is suspected; collection of a foetus, neonate and/or placenta and body fluids & tissues is advantageous as such specimens may allow demonstration of pathogens and more importantly, confirmation of attributable lesions in tissues. Fourthly, where arbovirus infections may occur, viral serological and insect vector distribution maps can be consulted, particularly as arthropod vector populations are dynamic and the risks of movements of insects from known endemic areas, appears to be increasing. Finally, addressing the question of whether a genetic disorder is causing abnormalities or losses, regular examination of the database of inherited animal disorders 'Online Mendelian Inheritance in Animals' (OMIA; http://omia.angis.org.au/) is advised. Veterinarians have an important role in both educating the public on the inevitability of congenital malformations and contributing to often important investigations of disorders of development in a timely manner that can bring benefits to improved welfare of livestock and potentially, humans and other animals.

Introduction

Understanding the causes of abnormalities occurring during development in pregnancy, particularly those presenting as embryonic, foetal or congenital malformations in livestock, is an increasingly complex and important area of global animal and human health science. There are concerns that the risks of teratogenic agents capable of altering foetal morphology and function appear to be increasing, with known teratogens including chemical toxicities, nutritional deficiencies and physical conditions such as heat. Alarming for a world undergoing climate change, is that *in-utero* infections due to arboviruses will be increasingly important in both animal and human populations as shown by emergence of Schmallenberg (Agerholm et al, 2015) and Zika viruses respectively (Kim & Shresta, 2016). Further, as inbreeding is common in domestic animal production systems, there is likely to be increasing risk of congenital abnormalities occurring from inherited factors, particularly where artificial reproductive techniques have enabled vast numbers of progeny to be descended from individual elite sires (Windsor & Agerholm, 2009). Finally, there remains a long list of abnormalities that are yet to be fully characterised and where the pathogenesis remains uncertain (Windsor, 2018).

In Australia, there are many known congenital disorders of Australian livestock and new disease entities continue to emerge. To ensure that our livestock health surveillance system continues to aim towards the highest of standards, we need vigilance in our search for and description of new and emerging disease entities, particularly those present at birth as these often cause considerable anxiety to producers and on occasions, severe economic losses (Jago et al, 1993). Efficient disease surveillance is required to identify and estimate the impact of those where we may need to develop evidence-based interventions in a timely manner, in order to manage risks to livestock production. The pathogenesis and methods for investigating congenital disorders and introduces a range of inherited disorders that appear to be emerging here or are recognised overseas and may well be here but are largely or completely unrecognised.

Aetiologies of disorders of development

Mammalian embryonic development is complex and involves synchronisation of numerous events occurring through gametogenesis, fertilisation, blastogenesis, embryogenesis, then foetal development and growth. Identification of the cause of an abnormality that presents prior to or at birth, is made more difficult by the fact that the clinical presentation observed likely reflects lesions resulting from events that occurred much earlier *in-utero.* Aberrations occurring between gametogenesis and embryogenesis that result in mortalities, are usually only recognised as reproduction failure or 'return to service'. Further, lethal insults of foetal development later in gestation may present as abortions, foetal resorption or mummification, stillbirth or neonatal mortality. Non-lethal insults and abnormalities may lead to lowered viability at or shortly after birth, reflecting that with some disorders there is protection of an abnormal foetus provided within the uterine environment, as occurs in the inborn errors of amino acid metabolism (Windsor et al., 2011).

Infection of a pregnant ruminant with a teratogenic arbovirus may induce various lesions of the developing nervous system of the foetus, particularly after the foetus has commenced to develop an immunological capacity, usually at the end of the first trimester of gestation; the lesions induced reflect the stage of development of the nervous system of the foetus. An excellent example is in calves born to susceptible cows infected during gestation with Akabane virus (AKAV) in Australia, with lesions of hydranencephaly induced by infection at the end of the first trimester, versus lesions of arthrogryposis induced by infection in mid-gestation (Kirkland et al., 1988). In both instances, such cases are usually readily distinguished from those due to infection in mid-gestation by Bovine Virus Diarrhoea Virus (BVDV) when lesions of cerebellar dysplasia and occasionally ocular dysgenesis (micropthalmia) may be induced.

However, if the agent only induces harm in the developing embryo when aligned with deprivation of maternal nutrition or excess, or other maternal physiological disturbance such as an intoxication, then identifying the aetiology of the loss or congenital disorder may become complicated. An example of deficiency includes the suspected association of nutritional deficiency induced chondrodysplasia in calves from cows in certain areas grazing drought affected pastures, erroneously attributed overseas to the grazing of Acorns and now referred to as congenital chondrodysplasia of undetermined origin (CCUO; White and Windsor, 2012). An example of a toxin is the plant *Dysphania glomerulifera* now shown to be the cause in sheep and cattle of neonates affected by biliary atresia following grazing by their dams adjacent to a depleted water reserved during drought (Harper et al., 1990). Understanding that the cause of this disorder was due to exposure of the foetus during gestation to the unique and newly identified toxin biliatresone that induced inhibition of post-hepatic biliary development in-utero (Lorent et al, 2016). This required several decades from the initial observations before biochemical research in laboratory animals led to evidence that this suspected plant was the source of the disorder (Koo et al., 2015).

Potentially, the simultaneous ingestion or administration of certain trace elements and a wide variety of chemical substances in food additives, fertilisers, fungicides, herbicides, and other chemicals used in agriculture, can create a challenging investigation. Whilst chemicals and therapeutics may be shown not to be teratogenic in laboratory animal studies and when used separately in livestock, enabling their registration by regulatory agencies, the concurrent, inappropriate and unintended exposure to some agents considered 'safe' then only later shown to have induced potentiating or synergistic effects with non-chemical environmental factors, makes identification of the primary aetiology more difficult. Further, a broad range of physical agents may be involved in the induction of deleterious effects, including maternal infections and non-infectious maternal disorders, hypoxia, hypothermia, hyperthermia, maternal immobilsation, plus seasonal variations in the occurrence of infections and available dietary components. This broad range of risk factors for congenital disorders in farm animals, requires that investigators possess considerable knowledge of the nutritional and dietary, physical, chemical, hormonal, viral and other agents plus genetic pathways capable of inducing lesions during gestation of the embryo and foetus (Windsor, 2018).

Surveillance for congenital disorders is challenging

Although surveillance to enable the early recognition of new and emerging inherited diseases is important, it is often challenging as the clinical signs of congenital diseases do not necessarily provide signposts to the aetiology of the disorder. When examining a ruminant animal with congenital disease, the clinician commonly asks the question, what are the non-genetic disorders that can produce these signs, plus, what are the genetic disorders that occur in this age of animal and in this species, breed and body system? For this reason, known inherited disorders are usually tabulated according to species, breed and age to aid the diagnostic process. When a genetic disease or malformation is diagnosed, it is worth remembering that this case probably represents only the 'tip of the iceberg' (Jolly and Windsor, 2010). Further, there is often an expectation that inherited disorders will only occur in pure-bred animals. However, in-breeding in the cattle industries with sire-daughter mating or similar close-breeding is still commonly practised, with some mutations first found in mixed-breed animals.

The diagnosis of a suspected inherited disorder usually requires the exclusion of other aetiologies and particularly infections, adding to the cost of disease investigation. This is particularly important in Australia where toxic plants are widespread, bovine pestivirus (BPV) is broadly endemic, and the Simbu group of viruses and Akabane virus (AKV) are present in certain areas. The regional endemicity of arboviral diseases can change at the whim of climatic events that influence the distribution of vectors; midges in the case of AKV. Numerous toxic plants cause dysfunction of enzymic cellular mechanisms that have also been described as compromised by mutations, as in Darling Pea poisoning and alpha-Mannosidosis (Windsor et al, 2011a). Both BPV and AKV cause a range of congenital disorders in ruminants that are readily confused with inherited disorders, as recognised in Arthrogryposis Multiplex of Angus that was initially confused with AKV disease (Windsor et al, 2011a,b).

Currently the cost of investigating congenital disorders is largely imposed on producers. This is a major impediment to the comprehensive studies that are required for detailed investigation of new inherited defects. What is required is systematic clinical and pathological and epidemiological investigation and documentation, elimination of ‘phenocopies’ from the differential diagnosis, parentage verification, pedigree analysis and collection of samples from homozygous affected, heterozygous and homozygous normal populations for molecular studies that can lead to development of heterozygote detection tests. Although the recognition of a new recessively inherited disorders can be difficult during the emergence of early cases, the cost of controlling the disease when it is well established is considerably greater and more complex when anxiety levels and financial damage through loss of reputation of breeders is heightened. Pro-active surveillance to identify and limit the dissemination of genetic defects at an early stage in the course of the emerging disease, through industry funded surveillance programs has been suggested as being more cost effective than efforts to eliminate a new genetic defect several generations after becoming established (Windsor and Agerholm, 2009).

Investigating disorders of development

Most livestock owners are keen to know the cause(s) of reproductive losses and congenital abnormalities that occur in their animals, although may not necessarily be keen to report such occurrences to authorities, particularly when a genetic defect is suspected. Livestock owners may jump to such conclusions and approach investigators with the question 'is this animal affected by an inherited disease previously recognised in this breed?' However, it may be more appropriate to first consider the question 'did this animal become infected or was it exposed to something that caused this aberration during gestation'? To provide an answer to the question on whether an abnormal animal may have been affected by an infectious agent, nutritional deficiency or excess, or a toxin that may act as a teratogen from a plant or other chemical source that damaged the development of the embryo or foetus during gestation, the investigator is advised to follow the following pathway:

(1) Consider the epidemiology of potential risk factors. Were the animals located in areas where infectious agents, teratogenic plants or nutritional aberrations may occur? Diligent examination of the environmental conditions including plants, feeds, soil, water, housing, plus animal, crop and forage treatments that occurred during the gestation period is advised.

(2) Consider information on breeding metrics, including:

- the numbers of females successfully mated,
- pregnancy status: numbers of females successfully maintaining pregnancy until the time of pregnancy testing,
- neonatal animals delivered and marked i.e. mustered for initial interventions, including application of identification devices and health products including vaccines, and finally
- the weaning rate.

The calculation of these measures provides a 'rate' on the outcome of each of the steps involved in producing normal and viable offspring in the population, enabling attribution of the stage(s) in the reproductive cycle that the losses occurred. Knowledge of the most likely differential diagnostic list for diseases that cause known losses at each stage of the cycle then informs a more cost-effective disease investigation.

(3) Consider the laboratory examinations required, particularly if infectious agents are suspected. The collection of a foetus, neonate and/or placenta, or if available, several of these most valuable of specimens, is advantageous in demonstrating the presence of pathogens and the important step of confirmation of the diagnostic lesions. Potential pathogens may be demonstrable in smears and lesions, plus successful culture and/or detection of antigens or PCR-products assists. Collection of pre-suckle serum or body cavity fluid from the foetus or affected neonates and serum from the dam enables laboratory demonstration of evidence that a pathogen capable of causing abnormalities was formerly present, producing a pathogen-specific antibody response. If there is uncertainty on whether a pathogen may have been involved, demonstration of an elevation in non-specific immunoglobulin (e.g. IgG) that is suggestive that an infectious process occurred prior to birth, is advised. Extending this examination by conducting pathogen-specific herd or flock sero-profiles is also be useful. This enables comparison of

the serological status of the two cohorts of females in the population; those 'affected' (i.e. lost a foetus) versus those 'unaffected'. Identification of a higher rate of seropositivity for a particular pathogen in the population of dams producing the 'affected' progeny, enables definition of the 'at risk' population and hopefully evidence for 'inclusion' or 'exclusion' of a pathogen.

(4) Consider spatial and temporal information for assessing the risk of arbovirus infections:

- where arbovirises may occur, viral serological and insect vector distribution maps should be consulted, particularly as arthropod vector populations are dynamic and the risks of movements of insects from known endemic areas, appears to be increasing. In eastern Australia, the distribution risk of exposure of the livestock population to an arbovirus infection such as AKAV is well documented in the NAMP (National Arbovirus Monitoring Program). Similarly, in parts of Europe, Bluetongue (BTV) and Schmallenberg (SBV) viruses may cause a range of congenital malformations in the foetus and neonate, depending on the stage of foetal neural development when the dam is infected (Agerholm et al, 2015).

- a complication of these investigations of arboviral-induced abnormalities is that in most cattle populations, BVDV remains endemic and capable of causing a broad range of congenital malformations in the foetus and neonatal calf over a longer period of the gestation. In cattle and sheep, consideration of any likelihood of exposure of the foetus in mid-gestation to an animal that is persistently infected (PI) with pestivirus is advised; in cattle BVDV and in sheep Ovine Border Disease (OVBDV) virus (Kessell et al, 2011).

- the timelines for occurrence of *in utero* vulnerability of the foetal neuromuscular system to a virus with teratogenic potential is important in diagnosing congenital abnormalities, as the pathology that occurs following infection is determined by the stage of gestation when foetal infection occurs. For example, in cattle, intrauterine infection of the foetus by BVDV, AKAV, SBV and BTV during the susceptible periods of development (Days 60-180 of gestation) may cause malformations in the central nervous system. Lesions in the brain of AKAV/AV occur from infection at Days 85-105 of gestation when typically, hydranencephaly (HE) occurs, although porencephaly and rarely, secondary hydrocephalus may eventuate, with or without the Arnold-Chiari malformation that occurs from progression of the cerebellum into the foramen magnum. Calves with cerebellar dysplasia from BVDV have typically been infected at around 150 days of gestation and such cases need to be investigated for pestivirus infection. BDVD can induce numerous abnormalities, including micropathalmia, and sometimes extension of the cerebellar dystrophy lesions to occipital lobe cerebral porencephaly that may progress to resemble HE. Infection with the Simbu viruses (most commonly AKAV) between about 120-160 days in gestation, may result in lesions in the spinal cord with depletion of lower motor neurones that typically cause arthrogryposis, presenting as malformations of the axial and appendicular skeleton.

- note that the endemic presence of the teratogenic viruses may delay the initial diagnosis of emerging inherited 'phenocopies' of these disorders. Arthrogryposis multiplex (AM; OMIA 001465) in Angus calves is an inherited disorder that emerged globally in the early millenium. However, in eastern Australia, the earliest cases of AM presenting with arthrogryposis were initially considered attributable to AKAV infection.

(5) Consideration of involvement of plant toxins as a cause of the disorder requires knowledge of the epidemiology of and potential access of the pregnant dam to known potential teratogenic plants during the first trimester of gestation (e.g. *Verratrum*

californicum, *Conium maculatum, Nicotiana glauca* or grazing lupins or sorghum). In addition to the published literature of congenital abnormalities associated with specific toxic plants (Keeper and Stuart, 1987; Harper et al., 1990), there are numerous other resources available that document plants associated with livestock losses *in-utero,* including the comprehensive book of Australian toxic plants (Mackenzie, 2012).

(6) Finally, in answering the question on whether an inherited disorder is the cause of an abnormality or losses, regular examination of the database of animal disorders, 'Online Mendelian Inheritance in Animals' (OMIA;) is advised (Nicholas and Harper, 1996). OMIA documents the known genotypes and phenotypes associated with inherited disorders, enabling examination of the published literature of disorders described in a refereed publication. The increased availability of molecular tools for characterising mutations has greatly facilitated the recognition of disease-causing mutations in animals. Investigations of suspected inherited disorders are facilitated by examination of pedigrees and use of parentage verification by blood tying or molecular methods, assists in confirmation of the veracity of this information. Consideration of the pedigree information as a predictor of the potential mode of transmission is also advisable. As the majority of newly recognised inherited abnormalities are transmitted in an autosomal recessive manner, both dam(s) and sire(s) will be heterozygous for the suspected defect. For flocks and herds that have consistently used registered sires, single sire matings and have accurate records, a simple pedigree analysis may determine if familial relationships exist between the affected animals and their parents plus antecedents if several cases occurred. If it is deduced that all the affected progeny derive from the same or closely related sire(s) and the dams of affected progeny are also related to the sire(s), then an inherited pathogenesis is more likely, enabling a more targeted approach to laboratory investigations of non-genetic causes. DNA parentage analysis can also be used to determine whether more than one sire is involved. The important question to consider is whether the affected phenotype is present in the progeny of a parent animal at a prevalence exceeding that likely by chance, after consideration of other factors. Confusion can occur, for example, where one single-sire mating group is bred in the only paddock with a teratogenic plant, or at the time of year when insect vectors capable of transmitting teratogenic viruses were active, or in cattle and sheep when an animal persistently infected with BVDV or OBDV was in the vicinity.

Conclusion

As the range of pathogenic modalities invoking embryonic and foetal losses or congenital disorders in livestock is complex, such investigations can be challenging for veterinarians. However, rather than dismissing such occurrences as inevitable costs of production, investigations of congenital disorders in animals are worthy as new entities appear constantly and may the 'tip of the iceberg', plus may provide important scientific contributions to the mechanisms of disease pathophysiology and animal models for studies of human disorders. Congenital abnormalities the 'canary in the coalmine' that environmental or breeding practice change has led to deleterious reproduction. Droughts in Australia are likely to become more severe. Drought led to the discovery of biliatresone from *Dysphania spp*. plants in Australian sheep, providing a model of toxic biliary atresia in infants, the most common cause of infantile liver transplantation. Diligent surveillance for congenital disorders has become increasingly important with growth of the international elite animal germplasm industry and artificial breeding. Within the dominant global breeds (e.g. Holstein-Friesian, Angus and Brahman) the efficient sharing of elite sires, each carrying known or unrecognized mutations that may transmit lethal disorders, results in regular appearance of congenital abnormalities. This global business in elite semen and embryos increases inbreeding and inevitability, more genetic defects will occur from the drive for more efficient livestock production, requiring increased rigor in identification and management of inherited disorders (Windsor and Agerholm, 2009). Recent developments in genotyping of newly recognised disorders indicates that this is increasingly understood. However, scrutiny by consumers and welfare advocates of farm production is increasing. Veterinarians have an important role in both educating the public on the inevitability of congenital malformations and contributing to more systematic investigations of disorders of development, providing benefits to improved efficiency of livestock production and potentially, welfare of humans and other animals.

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Diagnosis and management of respiratory diseases in small ruminants

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Summary
Globally, the most common respiratory disorders of sheep are rhinitis/sinusitis and pneumonia due to a range of host-pathogen-environment interactions that reflect the various sheep husbandry systems used in different countries. In Australia, where extensive husbandry systems predominate, verminous rhinitis (*Oestrous ovis*) and pneumonia (Lungworm), lesions due to inappropriate anthelmintic administration causing aspiration pneumonia, plus Caseous Lymphadenitis (CLA) bronchial granulomas, are considered the most frequent but usually sporadic causes of ovine clinical respiratory disease. However, abattoir surveys indicate that pneumonic diseases may contribute more to lowered sheep productivity than is generally recognized. Further, as pneumonic lesions are frequently seen at necropsy in sheep of all ages and clinical signs such as dyspnea and/or coughing are often evident when flocks of sheep are driven and housed, it is an area deserving of more detailed research. With increasing pressure on global food security to meet the burgeoning demand for red meat and milk protein driving more intensive small ruminant husbandry systems, infectious respiratory pathogens cause the most important disease issues in many European countries and beyond. Although in Australia the abundance of our grazing lands contributes to our relative freedom from many of the respiratory diseases occurring elsewhere, consumer demands for high quality meat and dairy products from sheep and goats is creating marketing opportunities that will likely result in more intensive small ruminant husbandry systems here. Inevitably, this will lead to more small ruminant respiratory disorders, including the likelihood of an emerging, mainly bacterial 'respiratory disease complex' not unlike that commonly diagnosed in beef feedlots. Ruminant veterinarians need awareness of the 'cost of respiratory disease from intensification' and are encouraged to visit intensive small ruminant systems in other countries where respiratory disorders are the dominant disease issue and there is frequently, very high dependence on antimicrobials and astonishing use of vaccination (often autogenous) to manage these diseases, plus emerging concerns about these practices.

Introduction
In many countries, managing respiratory disorders is the most challenging issue for intensive livestock systems, including small ruminants. This is not the case currently in Australia, where extensive husbandry systems predominate and verminous rhinitis (*Oestrous ovis*) and pneumonia *(Mullerius and Dictyocaulus spp*), aspiration pneumonia lesions due to inappropriate anthelmintic administration, plus CLA bronchial granulomas, are considered the most frequent causes of mainly sporadic ovine respiratory disease[1]. However, CLA remains of great importance to the sheep meat processing sector and is generally considered the most serious cause of economic loss in abattoirs[2]. Unlike our goat industry where CAE incursion occurred, we do expect our national biosecurity system will protect our sheep industry from the lentivirus infection scenarios common in Europe. However, the management of small ruminant respiratory disease is very likely to become a more challenging task than it has traditionally been in Australia. This reflects the increasing pressure on the global food security system to meet the burgeoning demand for red meat and milk protein, especially from rapidly developing nations, with extremely large human populations altering their dietary preferences (eg China) and driving the development of intensive small ruminant husbandry systems.

In Australia, our abundance of grazing lands has contributed to our relative freedom from many of the infectious respiratory pathogens that cause important disease issues in other countries. However, consumer demands for high quality meat and dairy products from sheep and goats is creating marketing opportunities that will result in more intensive small ruminant husbandry systems. Inevitably, this will lead to more small ruminant respiratory disorders, including the possibility of an emerging, mainly bacterial 'respiratory disease complex' not unlike that commonly diagnosed in beef feedlots. As an understanding of small ruminant respiratory diseases is likely to be more important, it is herein briefly reviewed.

Upper Respiratory Tract Disorders

Diseases of the upper respiratory tract of sheep and goats in Australia include rhinitis/sinusitis and pharyngeal disorders. Sinusitis may be caused by the larvae of *Oestrus ovis*, nasal foreign bodies including grass seeds, and less commonly, nasal mycotic infections and tumours. Clinical signs associated with sinusitis may include the following signs: unilateral or bilateral, serous to mucopurulent nasal discharge; decreased or lack of airflow through one or both nostrils; coughing; sneezing; and mild to severe respiratory distress. The various nasal neoplasms reported include adenopapillomas (nasal polyps), adenomas, adenocarcinomas, lymphosarcomas (goats), and squamous cell carcinomas (sheep). Pharyngeal disorders include trauma and abscessation, particularly in association with overly aggressive use of equipment used to administer boluses that cause injuries resulting in discrete abscesses or extensive and diffuse cellulitis that can interfere with swallowing and occasionally lead to respiratory distress. Bacteria commonly isolated after an incident of pharyngeal trauma include *Trueperella, Pasteurella multocida, Mannheimia haemolytica,* and *Fusobacterium.* Laryngeal chondritis is an obstructive upper respiratory tract disease characterised by severe dyspnea most commonly encountered in meat-breed rams 18–24 months old. Acute onset of severe respiratory distress with marked inspiratory effort is caused by oedema of the arytenoid cartilages of the larynx, resulting in narrowing of the lumen and affected animals stand with the neck extended, head held lowered with flared nostrils, and mouth open.

Enzootic Nasal Tumour (ENT)

In European countries, ENT is caused by an exogenous retrovirus (ENTV) that can be transmitted experimentally by tumour homogenates, explaining the widespread occurrence of this condition within some flocks. ENT generally affects mature animals (2–4 yr old), although has been reported in animals as young as 4 months of age. The lesion may be unilateral or bilateral, resulting in either unilateral or bilateral serous, mucoid, or mucopurulent nasal discharge, with deviation of the nasal septum in advanced unilateral tumours. Affected animals show progressive signs of inspiratory dyspnea with open-mouth breathing, decreased airflow at the nares, dullness on percussion over the turbinates, sneezing, and head-shaking. Stridor may also be caused by compression of the larynx by enlarged retropharyngeal lymph nodes associated with abscessation of the head. A major differential diagnosis of ENT in Spain, is a granulomatous rhinitis causing tumour-like projections of chronic inflammatory tissue from one or both nares, associated with infection with a *Salmonella sp*. Laryngeal chondritis also results in inspiratory dyspnea of varying severity. With advancing tumour growth, exophthalmos and facial deformity may occur, although metastasis is uncommon. Outcome depends on the tumour type, condition of the animal, and extent of the lesion, but affected animals are usually culled for animal welfare and commercial reasons (surgical removal of a noninvasive tumour is rarely undertaken).

Acute Lower Respiratory Tract Disorders

In Australia, lesions due to inappropriate anthelmintic administration causing aspiration pneumonia may well be the most frequent causes of ovine respiratory disease. However, abattoir surveys suggest that pneumonic diseases may contribute more to lowered sheep productivity than is generally recognized. Further, as pneumonic lesions are frequently seen at necropsy in sheep of all ages and clinical signs such as dyspnea and/or coughing are often evident when flocks of sheep are driven and housed, ovine respiratory disorders is deserving of more detailed research. With increasing pressure on the global food security system to meet the burgeoning demand for red meat and milk protein, especially from rapidly developing nations where altering dietary preferences are driving more intensive small ruminant husbandry systems. This is likely to intensify production systems in Australia where our abundance of grazing lands contributes to our relative freedom from many of the infectious respiratory pathogens that cause important disease issues in Europe and other countries.

Acute ovine viral and bacterial pneumonias
Acute sporadic cases of pneumonia in sheep may be associated with infection with parainfluenza type 3 (PI-3), adenovirus, and respiratory syncytial virus, most often affecting lambs (and kids). PI-3 is an enveloped RNA virus (family Paramyxoviridae) that induces a generally mild interstitial pneumonia with signs that may include coughing, a serous nasal and/or ocular discharge, fever (40°–41°C), and hyperpnoea/dyspnoea. The single PI-3 serotype for sheep that has been identified is distinct from the bovine PI-3 serotype and the diagnosis is confirmed by its isolation from nasal swabs from affected animals, or by comparison of acute and convalescent serology. Treatment is usually only warranted in severely affected animals where secondary pathogens including *Pasteurella multocida, Mannhaemia haemolytica*, and *Mycoplasma spp* may have become established (note that PI-3 vaccines have not been specifically developed for use in small ruminants).

Ovine Respiratory Disease Complex (ORD)
The pathogens *M haemolytica* (Mh), *P multocida* (Pm), *Mycoplasma spp, Chlamydia pneumoniae,* and *Salmonella spp* may also be associated with primary acute to subacute bronchopneumonia in sheep and goats, although Mh and Pm can be cultured from the upper respiratory tract of normal sheep and goats. *Mycoplasma ovipneumoniae* can cause a mild bronchopneumonia alone, although is more often isolated along with Mh from sheep and goats with severe pneumonia, suggesting that the Mycoplasma (potentially with some, or all of the above viral infections) may predispose the lung to invasion by this organism, with peracute cases manifest as septicaemia also occurring. The Host Pathogen Environment (HPE) 'stress' factors involved in acute bronchopneumonia of small ruminants include: introduction of new animals; high stocking density; poor ventilation; and sudden alterations in nutrition (eg acute to subacute acidosis); although other factors may also predispose to development of pneumonia in this Ovine Respiratory Disease Complex, a major cause of mortality and morbidity in lambs in intensive husbandry in many parts of the world.

Chronic Lower Respiratory Tract disorders
In Australia where extensive husbandry systems predominate, verminous pneumonia (Lungworm), chronic iatrogenic lesions from sublethal inappropriate anthelmintic administration and CLA bronchial pyogranulomas, are considered the most frequent causes of chronic lower ovine respiratory disease.

Verminous pneumonia
The three species of lungworm that occur in sheep or goats in Australia include the large lungworm *Dictyocaulus filaria* and the small lungworms, *Muellerius capillaris* and *Protostrongylus rufescens*. These parasites prefer cool conditions as the developmental stage in slugs or snails restricts *Muellerius* and *Protostrongylus* to mainly cooler regions. These parasites can be readily identified by:

- *Dictyocaulus filaria* is white, several cm’s long and commonly found in broncho-tracheal exudates and sputum.
- *Muellerius capillaris* is a thin worm that is tightly coiled in small granulomas in sub-pleural lung observed as grey foci 2–4 mm in diameter.
- *Protostrongylus rufescens* is a red coloured worm 16–35 mm long located smaller bronchioles.

Signs of lungworm include lethargy, weight loss, nodular lesions (lungs), pneumonia and as they irritate bronchiolar and tracheal epithelium, the main symptom is coughing. At necropsy the white, thread-like worms are clearly visible either in the airways or in nodular lesions under the pleural surface, although these nodules may also contain inflammatory exudates. Diagnosis is by consideration of clinical signs (coughing) and examination of sputum, the larvae in fresh faeces may be observed and recovered using a Baermann apparatus, and lesions are readily identified at necropsy. Although all broad spectrum drenches have activity against adult lungworm, control can occasionally be difficult to achieve.

Iatrogenic aspiration pneumonia from anthelmintic administration
Both episodic and epidemic outbreaks of morbidity and mortality presenting with dyspnoea and death of various degrees occur relatively commonly in Australia, a country highly dependent on the use of anthelmintics to control helminths and trematodes. Lesions of moderate to severe acute to chronic active suppurative to gangrenous bronchopneumonia are usually distributed in the anteroventral regions of the lungs and often progressing to fibrinous pleurisy. Invariably, such cases are due to inappropriate drench technique with administration of foreign material into the trachea, although detective work may be required to identify how such episodes occurred (eg the grandpa with the bad back syndrome). Advice on correct drenching technique needs attention, particularly with new workers such advice includes taking care not to lift the sheep's head above horizontal when drenching; and avoiding drenching in the cradle, and not plunge dipping tired or thirsty sheep.[3]

Ovine caseous lymphadenitis (CLA)
CLA is a serious disease of the global sheep industry, mainly causing losses from abattoir condemnations and in some countries, compromised productivity and clinical cases of suppurative dermatitis. However, in Australia CLA is now generally considered a disease of minimal clinical concern on-farm with cases reported rarely, although the disease remains an important food quality issue for abattoirs.[2] There is evidence from longitudinal abattoir surveillance that CLA prevalence has been declining, from estimates of 26% in 1995–5.2% in 2009.[4] Changed management factors relevant to reduced prevalence and producer concerns for CLA in Australia have been examined, including drivers and motivation for change, resistance to change, knowledge management, farming systems dimensions and leadership.[5] Although extension programs addressing disease risk factors, such associations of CLA with dipping for external parasite control and frequency of close confinement, may be of relevance to improved disease risk management by producers, improved CLA control on many Australian sheep farms is considered largely attributable to the introduction of vaccination programs for CLA in 1983, with inclusion of CLA antigen within clostridial vaccines (eg '6 in 1' and 3 in 1 vaccines). This innovation enabled routine annual CLA vaccination to occur in an increasing proportion of the national flock, despite several surveys that have shown that producer knowledge of CLA is low.[6]

Promoting the persistent use of 'combination' vaccination to continue suppression of CLA infection and improved biosecurity to reduce reintroduction of disease, plus targeting properties that are not using vaccination, remains a challenge for animal health authorities. As CLA in Australia is now primarily an issue for the sheep meat processing sector, efforts to improve feedback to producers suffering carcass condemnations at slaughter should been encouraged Persistent vaccination for CLA improves the health and welfare of sheep and this strategy deserves wider recognition in all sheep and goat production countries where vaccine usage is sporadic.[6]

In Australia in recent years, the differential gross diagnosis of subcutaneous CLA has become more difficult with the advent of Gudair® vaccination against Ovine Johne's Disease due to *Mycobacterium avium* subspecies *paratuberculosis.* Gudair® contains paraffin oil adjuvant that in muscle is highly necrotising, with lesions progressing to granuloma formation, at and beyond injection sites including regional lymph nodes.[1,5] Alternatively, in some countries the differential diagnosis of CLA can be complicated by Morel's disease (MD), caused by *Staphylococcus aureus* subspecies *anaerobius.*[8] Although CLA is an issue almost wherever sheep and goats occur, MD occurs in some southern European countries (eg Spain), African, Asian and Middle East countries (eg Saudi Arabia). MD is readily misdiagnosed as CLA due to the chronic and generally subclinical nature of the subcutaneous abscesses and local lymphadenitis. The poor response to antimicrobial therapy, plus use of vaccines that only target only CLA, make control of MD difficult and contribute to the high prevalence in some countries. Where MD is common, it is considered of similar importance to CLA and development of a bivalent vaccine has been suggested. Ovine or caprine MD has not yet been diagnosed in Australia.

Chronic Viral Pneumonia (Maedi-Visna; Ovine Progressive Adenomatosis)
With the exceptions of Australia and NZ, chronic, progressive viral pneumonia is relatively common in adult small ruminants throughout the world. These entities involve lesions of progressive interstitial pneumonia and are due to lentiviruses (family Retroviridae). They include ovine progressive pneumonia (Marshes progressive pneumonia in North America or Maedi in European countries) and ovine pulmonary adenocarcinoma (Jaagsiekte or the contagious lung tumor of sheep and infrequently, of goats). Note that the retrovirus that causes pneumonia in goats, CAE virus (Caprine Arthritis and Encephalitis) has been present in Australia since the 1970's. Chronic, progressive, interstitial proliferative pneumonia is usually associated with the lentiviruses (so-called slow-virus infections). In both progressive pneumonia and pulmonary adenocarcinoma, the entire lung can be affected in a gradual proliferative process of pulmonary tissue resulting in progressive dyspnoea, anorexia, and weight loss.

Maedi-Visna: Maedi-visna is a chronic, progressive viral infection characterised by a prolonged incubation period and predominantly two clinical manifestations; pneumonia and encephalomyelitis (Maedi means dyspnoea and Visna means wasting in Icelandic language). The virus also infects the udder causing a chronic mastitis and reduced milk yield. Generally, only one form of the disease occurs in one animal and often one form predominates in any one flock. The disease does occur in goats but transmission between sheep and goats does not usually occur in field cases. Vertical transmission, by the excretion of virus-infected leucocytes in colostrum and milk, is the main form of spread of Maedi-visna in flocks. Transmission to the foetus *in utero* occurs but is rare and it is believed that transmission via ova or sperm does not occur, with horizontal transmission occurring mainly by the inhalation of infected respiratory secretions from infected sheep. Infection is introduced into clean flocks by horizontal transmission from an introduced, infected sheep. Although the virus does not survive for more than two weeks outside the host, the incubation period may exceed several years. Following infection, viral replication is restricted for a prolonged period, during which the viral genetic material resides in infected cells as proviral DNA until the infection progresses and clinical signs are associated with the chronic progressive proliferation of lymphoid tissue in lungs, brain, udder and joints. The pulmonary lesions in Maedi are an interstitial pneumonia that restricts the alveolar space, leading to anoxia, although many sheep remain in a sub-clinical phase of the disease. The variation in expression of the disease as primarily respiratory, nervous or mastitic, is presumed due to different tissue tropism of the strains of the virus, differences between breeds and flocks of sheep in their susceptibility and the effects of different management factors.

Animals with Maedi are listless, emaciated and dyspnoeic. There is coughing and nasal discharge, but most affected sheep retain their appetite. Udder induration, hind-limb paralysis and, in some cases, swollen joints with or without lameness, may also be present in the flock. Clinical signs last for 3 to 12 months but the disease is inevitably fatal. Sheep pulmonary adenomatosis (Jaagsiekte) can produce similar clinical signs with similar flock history and, in some countries, can be simultaneously present in the same flock and the same sheep, although is usually characterised by a profuse nasal discharge and a shorter clinical course (the two diseases can be readily differentiated on histopathology). Parasitic pneumonia and melioidosis also have signs of chronic respiratory disease.

Diagnosis of Maedi is confirmed either by one of several techniques for virus identification or by serology used as a flock diagnosis. Note that negative serology in individual cases is not reliable evidence of freedom from infection, as the time between infection and seroconversion is variable and may be prolonged, for as long as one or more years, with some infected animals remaining seronegative. There is no treatment for Maedi-Visna. National control programs in endemically infected countries vary in their approach but are based largely on the identification of clean flocks by serological testing; separation of lambs from ewes and artificial rearing of lambs in flocks with low prevalence of infection; or complete destocking and replacement in flocks with moderate or higher levels of infection.

Ovine Pulmonary Adenomatosis (OPA, Jaagsiekte): OPA is a chronic progressive pneumonia of sheep characterised by histological lesions of adenomatous proliferations in alveolar walls. Clinically affected sheep produce profuse watery mucous in the lungs leading to excessive discharge from the nose, aided at examination by 'wheel-barrowing' the suspected affected animal. It is also caused by a retrovirus (and is one of the three so-called 'slow virus' infections of sheep with Maedi-Visna and Scrapie, although the latter is now well recognised as a prion disorder or TSE). The disease occurs in Britain, continental Europe, South Africa, Israel, Asia and Iceland. Only sheep are infected in natural cases and transmission is presumed to occur by inhalation of infected droplets and by vertical transmission to the foetus. Close housing in winter, as occurs routinely in Iceland, promotes transmission but is not essential for flock outbreaks. The incubation period is 1 to 3 years and as the adenomatous proliferations encroach on the alveolar space and lead to anoxia, coughing occurs, although it is not a prominent sign. Emaciation, dyspnoea and panting after exercise, plus profuse watery discharge from the nose, are characteristic signs, with moist rales on auscultation over affected areas of lung. The disease is inevitably fatal and at necropsy, the lungs are enlarged, heavy from consolidation and there is frothy fluid in the bronchi. Histopathology is characteristic. There is no treatment. A vaccine has been used successfully in Kenya.

Discussion

In Australia, the abundance of grazing lands has contributed to our relative freedom from many of the infectious respiratory pathogens that cause the important disease issues in other countries. As extensive grazing dominates our sheep production systems, verminous rhinitis (*Oestrous ovis*) and pneumonia (Lungworm), lesions due to inappropriate anthelmintic administration causing aspiration pneumonia, plus Caseous Lymphadenitis (CLA) bronchial lymphadenitis pyogranulomas, are considered the most common causes of mainly sporadic ovine clinical respiratory disease. Abattoir experiences indicate that CLA remains of concern and as lesions of pneumonia and pleurisy also occur regularly, there is a likely a greater contribution of respiratory disease to lowered sheep productivity than is generally recognized. This conclusion is supported by the common occurrence of clinical signs of dyspnea and/or coughing evident when flocks of sheep are driven and housed, and respiratory system lesions observed at necropsy in sheep of all ages.

Attention to CLA control through encouraging persistent vaccination plus a renewed focus on improved drenching management are clearly priorities for Australian sheep health extension programs. CLA programs in Australia have been reviewed recently.[6] Extension advice for the control of pneumonia and pleurisy in Australian sheep is to encourage close and rigorous attention to the risk factors that predispose to infection of the respiratory tracts.[3] These risk factors include: (1) yarding & mustering; (2) optimising stock health and minimising stress; and (3) appropriate drenching & dipping technique. An audit of the following practices on farms where respiratory disease is a problem is advisable, noting that sheep are: driven slowly; overcrowding in yards and prolonged or unnecessary yarding is avoided; yarding stock when conditions are extremely cold or hot, dry and/or dusty is avoided or if necessary when dusty, consider hosing yards; able to walk slowly back to paddocks at their own pace after yarding; able to access to clean water and sudden diet changes are avoided or minimised; not mixed with other mobs; receiving good worm control and appropriate vaccinations; well fed and any nutritional or mineral deficiencies are addressed promptly; provided with appropriate shelter from extremes of temperature and dusty feed is avoided; drenched correctly, with care not to lift the sheep's head above horizontal when drenching plus train new workers to drench properly; and drenching in the cradle plus plunge dipping of tired or thirsty sheep is avoided.

In Europe where ORD and the chronic viral pneumonia entities are endemic, there is often extreme dependence on antimicrobials, with regular vaccination emerging against many pathogens recovered from small ruminant respiratory lesions. With increasing antimicrobial resistance (AMR) in livestock systems, there is an emphasis on alternative disease management approaches in Europe. This led to emergence of a disorder in sheep following repeated vaccinations, first observed following the Bluetongue epidemic in 2008, and considered a form of the autoimmune/autoinflammatory syndrome (ASIA syndrome) hypothesised to have been induced by aluminum adjuvants. This syndrome occurred in commercial sheep and was linked to the repetitive inoculation of aluminum-containing adjuvants through vaccination.[9] The syndrome involves an acute phase affects < 0.5% of animals in a flock, appearing 2-6 days after an adjuvant-containing inoculation; presenting as an acute neurological episode with dullness and acute meningoencephalitis with most animals recovering. A chronic phase was observed in a higher proportion of flocks, often following the acute phase and triggered by external stimuli; mostly low temperatures. Animals in the chronic phase display excitation followed by weakness, extreme cachexia, tetraplegia and death. Gross lesions are related to a cachectic process with muscular atrophy, and microscopic lesions are mostly linked to a neurodegenerative process in both dorsal and ventral column of the gray matter of the spinal cord. Experimental reproduction of ovine ASIA in a small group of repeatedly vaccinated animals was successful, with detection of Al(III) in tissues indicating aluminum was present in the nervous tissue of experimental animals. Multiple, repetitive vaccinations as occurred in the compulsory European vaccination program against Bluetongue in 2008 have been suggested having negative consequences, with ovine ASIA syndrome a potential model of similar autoimmune diseases in humans and other animals, although a major research effort is required to understand the complex pathogenesis of the disorder.

Preparing for the 'cost of respiratory diseases from intensification' requires greater awareness of both the endemic and transboundary respiratory disease risks that can compromise the health and productivity of our small ruminant production systems, plus the welfare and public health management issues that are emerging in other countries and may potentially arise in Australia from small ruminant intensification.

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Pain relief for cattle husbandry procedures: update

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Summary
Pain in farmed cattle usually results from injury, disease or most commonly, occurs during aversive husbandry procedures used routinely on farms to assist livestock management and improve welfare. For ethical, economic and marketing assurance concerns, consumers, producers and other livestock industry stakeholders are increasingly demanding that routine pain management for livestock be provided for such procedures. Research to establish the efficacy of products to achieve this, particularly on-farms by producers, has become increasingly important. This research supported the registration of new products aimed at the Australian extensive livestock systems, delivering topical anaesthesia and/or non-steroidal anti-inflammatory pain relief by producers. These products are demonstrating pain relief efficacy for an increasing range of applications, including disbudding, dehorning, castration, calving and lameness. Other applications of these products, new forms of delivery, and even new products, are envisaged. International producers and consumers of livestock products need increased awareness of these improved welfare attitudes and practices in Australian livestock agriculture. This 'pain management revolution' is empowering producers and their advisors on farms to reduce suffering in animals, enhance animal welfare and importantly, address concerns raised in activist-led campaigns advocating that livestock production as inherently cruel. Pain relief in livestock is now an important risk management intervention for the global animal industries and the research developments for the cattle industry in Australia are briefly reviewed.

Introduction
Pain is a cause of poor welfare in production animals. There is increasing demand for options that can provide provide pain management for cattle herds, driven by ethical, economic and market assurance concerns of consumers, producers and other livestock industry stakeholders. Research to alleviate both peri-operative and post-operative pain in cattle from the common aversive livestock husbandry procedures in cattle has made significant progress in recent years. The initial studies on pain relief in Australian livestock were initiated following an animal welfare activist-led campaign against the Australian sheep industry acceptance of the mulesing operation in lambs, to reduce life-time risk of sheep blowfly strike. This led to introduction of a farmer-applied spray-on topical anaesthesia (TA) wound dressing formulation (Tri-Solfen®, containing a combination of the local anaesthetics lignocaine and bupivacaine with adrenaline and cetrimide in a gel) in 2005 and eventual registration of this product in 2012 to manage the pain of mulesing and hasten healing of wounds (Lomax et al, 2008).

An important finding in these studies was confirmation of extended positive welfare outcomes for periods well beyond the duration of action of the TA's (Lomax et al, 2013), plus evidence of efficacy of the TA wound dressing for castration and tail docking of lambs (Lomax et al, 2011). TA for mulesing has now been widely adopted with currently in excess of 100 million lambs treated, plus use for castration and tail docking promoted. These developments have enabled improved welfare of sheep susceptible to flystrike in the extended period required until genetic alterations of Australian Merino phenotypes can progress sufficiently to successfully address the risk of myiasis (Windsor and Lomax, 2013). This success led to broader applications with demonstrations of efficacy of TA in other livestock husbandry procedures involving surgical interventions, particularly cattle. Research with castration of calves (Lomax and Windsor, 2012), disbudding and dehorning of young calves (Espinoza et al, 2013), plus general on-farm wound and disease management treatments, particularly shearing cuts and lameness in sheep and cattle. Efficacy for beef cattle dehorning by amputation achieved conflicting results (Van der Saag, 2008b,c) and speying by the Willis dropped ovary technique has proved initially unsuccessful (unpublished).

Studies have continued in developing 'best practice' multimodal approaches to pain management, aimed at capturing the effectiveness of two or more individual agents in optimal dosages, offering theoretical synergies in acute pain prevention and relief and potentially minimising the side effects from each drug (Young and Buvanendran, 2012). Amelioration of wound sensitisation by use of various nonsteroidal anti-inflammatory drugs (NSAID's) has been extensively studied, although when used for wounds has less clinical impact than blockage of nociception by TA wound dressing, as occurs in dehorning (Espinoza et al, 2015). Delivery of meloxicam by oral administration was developed in Australia, enabling administration of NSAID's by producers following registration of Buccalgesic® (Small et al, 2014). Meloxicam has now been shown to be efficacious by oral or injected administration when combined with Tri-Solfen® for castration (Van der Saag et al, 2018a), and for castration and dehorning when both procedures are performed concurrently (Van der Saag et al, 2018c). Research on options to modify cognition (e.g. by centrally-acting drugs including low dose xylazine) have been attempted but the concerns of food safety and occupational health are likely to limit their application in Australia. Research to prolong the availability of NSAID's is continuing, as is examination of the best 'age' for animals undergoing these procedures, particularly as the assumption they should be done in neonates or very young animals is impractical in extensive Australian beef systems.

Progress in these developments and the increasing uptake of routine pain management applications during aversive husbandry procedures, is consistent with the new paradigm that emerged in Australian agriculture, termed the 'livestock pain welfare revolution' (Windsor et al, 2012; 2015). It is increasingly important and possible to address the welfare concerns of pain during husbandry procedures that have been used routinely in livestock management for centuries, with no pain relief applied. This paradigm has achieved some international recognition (Grandin, 2014) and is now offering research support for wound pain management studies in humans (unpublished). Efforts are required to ensure these developments continue to be adopted and that pain relief for surgical husbandry procedures and other painful disorders becomes routine in livestock agriculture. As these advances address the ethical, economic and market assurance concerns of consumers and producers of dairy and beef products globally, they are reviewed in this paper.

Pain and pain management for cattle

Pain is intended to elicit awareness of a visceral or environmental insult. Wound pain involves the three stages of nociception (with modulation), sensitization, and central cognition. Following a noxious stimulus, peripheral nociceptors, with enhancement by release of pain-sensitising chemical mediators, produce peripheral to central activation of sensory neurones via the peripheral nerves, then the dorsal horn of the spinal cord, stimulating sensory areas of the brain resulting in central cognition of pain. Clinical assessment of pain is challenging, particularly as 'prey' species tend to limit their display of pain incurred by injury. Further, assumptions that animals at an early age suffer less pain than those that are older is now considered an erroneous assumption that an animal with a less developed central nervous system (CNS) may experience less pain than an older animal where the CNS is more mature. This may be of relevance in altricial species but is inappropriate in precocial species including ruminants, where the CNS is sufficiently advanced at birth (i.e. 50% myelinated in ruminants) to enable neonates to be ambulatory and respond to noxious stimuli in the immediate neonatal period. Perhaps the acceptance that aversive husbandry procedures should routinely be conducted at an early age, derives from the ease of restraint that can be applied to younger animals?

Pain management of ruminants as performed routinely by veterinarians in the field in Australia, is also driven by the need for restraint. Many vets use sedatives to restrain animals, most commonly with compounds with analgesic properties (e.g. xylazine), plus infiltrate a local anaesthetic(s) into the surgical site or nearby peripheral nerve to 'block' nociception. More recently, NSAIDs have been recruited to ameliorate sensitization by inhibiting prostaglandin production through blockade of two cyclo-oxygenases (COX-1 and COX-2) and generally producing anti-inflammatory, analgesic and antipyretic effects. The effects of NSAIDs for pain alleviation after disbudding/dehorning (Stilwell et al., 2012), castration (Stilwell et al., 2008), left-displaced abomasum surgery and calving (Stilwell et al., 2014) and recently, lameness management (Stilwell, unpublished data), with veterinarians now commonly using NSAID's for pain management in ruminants in Australia and other countries. However, as pain management provided by veterinarians is impractical in most extensive ruminant systems, particularly in Australia, there was an urgent need to provide safe and practical agents for use by non-veterinarians for pain management in livestock. As the positive welfare effects of inhibiting wound nociception by spray-on TA by farmers has shown to be safe and practical, the inclusion of a farmer-applied NSAID to ameliorate wound sensitisation is considered a logical additional strategy. Although the addition of NSAID's to a regimen using TA has shown marginal or even nil improvements in welfare outcomes in some trials (Espinoza et al, 2015), the development of oral NSAIDs offers a convenient approach for farmer-applied use in improving pain management in cattle (Van der Saag et al., 2018a,b). The multimodal use of TA and NSAID is increasingly recognized by producers and their advisors as a preferred pain management strategy on-farm, as it reduces the need for the presence of a veterinarian in extensive industries where the veterinary provision of pain management is impractical.

As the administration by farmers of centrally-acting drugs (e.g. xylazine) to modify cognition would require greater veterinary supervision than is likely to be achievable in the Australian extensive livestock industries, it is an approach that is unlikely to receive support, particularly as heavy sedation with muscle relaxation may lead to poorer pain management because farmers, or even vets, may confuse immobility with anaesthesia. Work with disbudding has shown that sedated calves will have very high levels of cortisol when handled even if no stress behaviour is apparent (Stilwell et al., 2012). Anticipatory control of pain or 'pre-emptive analgesia' has been shown to be extremely beneficial (Stilwell et al., 2008; Stilwell et al., 2012; Van der Saag et al., 2018a,b,c) because it limits the subsequent pain experience by preventing central sensitization and peripheral hyperalgesia and there is evidence that the analgesic effects of longer-acting NSAIDs given preoperatively, may persist for several days after hot-iron disbudding or castration (Stilwell et al., 2008).

Research on more important sources of pain in cattle

Disbudding and dehorning

Disbudding is a common painful husbandry procedure on dairy farms performed on millions of young calves each year. Disbudding destroys horn-producing epithelial cells and usually involves either use of thermocautery (hot-iron disbudding) or caustic paste. Dehorning involves physical removal of the horn buds, preferably prior to their attachment to the skull, although on extensive beef properties, this is usually not possible and the horns and germinal epithelial tissue are physically removed, using knives, scoop dehorners or other equipment. Numerous studies have been conducted on pain management treatments for these procedures, mostly comparing either tissue sensory testing responses by algometry, pain related behaviours, including head rubbing and shaking, ear flicking, time spent lying and in locomotion etc.), and/or plasma cortisol levels.

Findings from these studies indicate that caustic paste disbudding, even in very young calves, causes pain that is not easily controlled (Stilwell et al., 2008) and that amputation or scoop dehorning causes severe and prolonged pain and should not be used in dairy calves (Stilwell, 2008) unless TA wound treatment is applied (Espinoza et al, 2009). Achieving satisfactory pain relief for amputation dehorning in beef cattle with both TA and NSAIDs is also difficult to achieve, although field trials can be difficult to interpret if compromised by flystrike (Van der Saag, 2018b). Cornual nerve blocks with lidocaine efficiently eliminates pain during hot-iron disbudding but is less effective in the case of caustic paste or amputation (Stilwell et al., 2008) and that signs of pain increase after the nerve block diminishes by 2-3 hours. However, pain is still inhibited if an NSAID is provided (Stilwell et al., 2008) and pre-emptive analgesia with an NSAID (e.g. flunixin-meglumine or carprofen) reduces sensitization to subsequent stimuli. Of interest, xylazine alone does not effectively control pain from hot-iron disbudding (Stilwell et al., 2010) with higher cortisol levels in sedated calves even when no painful procedure is applied. This research concludes that hot-iron disbudding after cornual nerve block and parenteral NSAID, is an efficient and practical pain management protocol that should be used by all intending to disbud young dairy calves.

Castration

Castration aims to reduce aggressiveness, sexual activity and modify carcass characteristics (e.g. increased marbling and reduced of dark-cutting meat) and usually involves either: surgical removal of the testicles; application of a constricting elastic band (rubber ring) at the base of the scrotum; and bloodless castration by external 'burdizzo' clamping; and potentially chemical castration, although this remains controversial. Castration produces physiologic, neuroendocrine and behavioural changes indicative of pain and stress, especially if done without analgesia as has been common practice. A study compared the pain and rate of healing after one (longitudinal over the scrotum midline) or two incisions (longitudinal over each testicle) concluded that, although one incision causes less initial pain, the inflammation, probably due to reduced drainage, leads to more intense and prolonged pain than when performing two incisions (Stilwell et al., 2008). Numerous studies have now been published demonstrating reduced sensory responses, pain-related behaviours, hair or serum cortisol and weight loss when anaesthetics/analgesics are applied at castration (Stilwell et al., 2008; Lomax and Windsor, 2012). Surgical castration is the most reliable method for cattle castration and most amenable to pain management if TA wound dressing is applied to the scrotum during the procedure (Lomax and Windsor, 2012). A study comparing surgical and band castration in feedlot calves indicated that surgically castrated calves spent less time lying and meloxicam mitigated the more pronounced inflammation and improved overall weight gain for both methods (Roberts et al, 2018). In Australia, there is a strong trend for surgically castrated calves treated with TA wound dressing and buccal meloxicam to have reduced pain following castration and dehorning, with improved weight gain, increased lying activity and reduced scrotum inflammation in the first few days following the procedures has now been published (Van der Saag et al., 2016; 2018a,c).

Lameness

It is well established that lame cows have reduced nociceptive thresholds consistent with hyperalgesia and that treating cows with some hoof lesions trimming is important. However, trimming may cause or exacerbate pain and cows may react violently, creating a safety issue for trimmers. As pain management be perform when trimming, a study in Portugal to test the efficiency of Tri-Solfen® on pain during trimming (Stilwell, pers. comm.). Sixty-two lame dairy cows were scored for lameness before entering the crush for treatment. After confirming the presence of a hoof lesion animals were randomly distributed to two groups: C – usual trimming with no pain control; T – trimming with local anaesthetics being applied immediately after live corium was exposed. During curative trimming, behaviour observation was conducted by two observers blind to treatment. Algometry measurements were performed before and after the procedure, to assess animal reaction to pressure and lameness scoring was again performed on completion. Results showed that the use of these topical anaesthetics significantly reduces reaction to trimming and lameness score after trimming, when compared with non-treated animals. Algometry values showed increased pressure threshold after application of topical anaesthetics. This study demonstrated that the use of topical local anaesthetics will improve animal welfare and trimmer safety, and that Tri-Solfen® is well-suited for farmers' use because of its low cost, practicality and easy application (Stilwell, unpublished).

Conclusions

Farmer-applied spray-on TA (Tri-Solfen®) for mulesing in sheep rapidly achieved widespread adoption with over 100 million lambs now treated. This indicates that suitable product(s) can rapidly change the attitudes of farmers to livestock welfare as there is now widespread recognition in Australia that if aversive husbandry procedures used commonly to facilitate management of farmed livestock populations are to continue, then the pain inflicted on animals during these procedures should be ameliorated. Farmer-applied TA is a new paradigm for addressing the welfare concerns of aversive procedures used routinely in cattle and is very likely to continue to become firmly established in the Australian livestock industries and beyond. Despite the many technical difficulties and cost, safety and public health and food safety concerns, particularly with the use of some drugs of potential efficacy that are required to be excluded from food producing animals (e.g. xylazine and opioids, respectively), there is now a rapidly emerging interest by numerous Australia livestock producers and many of their veterinary advisors, in the use of TA and NSAID product(s) for improved animal welfare during husbandry procedures in cattle. Progression of research on TA and NSAID and registration of these products for use in cattle and other farmed species for routine husbandry procedures has advanced animal welfare in Australia and potentially beyond, empowering farmers to take control of managing pain by reducing the suffering experienced by their animals undergoing aversive husbandry procedures. Importantly, it addresses concerns raised in activist-led campaigns promoting improved welfare of farmed animals and is an important market-risk management intervention that could be routinely adopted in livestock husbandry globally.

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Syndromic surveillance – a biosecurity frontline for northern Australia

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Introduction

Australia is free from many agricultural pests and diseases that may affect the quality and quantity of food and fibre sources, providing a strong basis for production and a widely recognised reputation for exporting high quality 'clean' produce to many economically important international markets. Although Australia is geographically isolated, and with an effective national biosecurity system, the remote and vast northern coastline is vulnerable to the introduction and establishment of exotic agricultural pests and diseases from neighbouring countries. In the absence of a robust and effective surveillance system, many of these diseases could go undetected for years. This unique northern Australia biosecurity risk profile resulted in the formation of the Northern Australia Quarantine Strategy (NAQS) in 1989.

The NAQS program was established, within the Australian Government's Department of Agriculture and Water Resources, to address the risks associated with the vulnerability of the coastline between Broome and Cairns, and north through the Torres Strait. Established seaports and airports are controlled in accordance with relevant risk profiles; however, the northern coastline is exposed to wind and ocean currents, potentially unregulated or illegal vessels, busy shipping channels, marine debris and migratory animals. The NAQS program is a multidisciplinary group that comprises of veterinarians, entomologists, botanists, plant pathologists and scientific support staff tasked with managing the biosecurity risk of these unregulated pathways.

Each of the disciplines within NAQS maintain a list of target diseases and pests that have a potential pathway into northern Australia. These lists and pathways are reviewed annually through a variety of in-house, and external, science and evidence-based processes. The current target disease list for the animal team includes surra (*Trypanosoma evansi*), foot and mouth disease, rabies, classical swine fever, avian influenza as well as many others. Recently, the list has been updated to include African swine fever due to the recent outbreaks in China (2018) and Vietnam (2019).

Indigenous Ranger Groups

While the arrival of unchecked biosecurity risk material is outside of regulatory control, early detection of exotic diseases and pests can limit the impact of such pathogens to production, trade, the environment and human health. As such, in addition to the structured remote area targeted surveillance strategy, the department engages the services of indigenous ranger groups across northern Australia to establish a network that endeavours for timely reporting on unusual animal health events.

To date, 69 indigenous ranger groups monitor over more than 10 000 km of northern Australian coastline, islands and inland regions, extending from Broome in Western Australia to Cairns in Queensland. This area encompasses approximately 1.4 million km^2, an area that would otherwise be very difficult or impossible to access for any meaningful disease surveillance. NAQS engages with each of these groups on a fee for service basis for a range of biosecurity surveillance activities that provide an early warning system for plant and animal exotic pests and diseases. Such activities include assistance with NAQS survey preparation and logistics, assisting NAQS field officers with avian influenza surveys, marine debris monitoring along the coastline and community animal health reports.

Community Animal Health Reports

The Community Animal Health Report (CAHR) is passive syndromic surveillance system that collects observations collated by northern Australian ranger groups. Sources of observations may include community health clinics, council animal workers and members of the community. Historically, the CAHR data was generated from a paper-based questionnaire that was collated onto a spreadsheet. This has since been superseded through the use of a "Ranger app" that has been loaded onto tablets for use in the field. The objective of the CAHR is to monitor for the incursion of an exotic/emerging animal disease in remote north Australia communities, provide qualitative evidence that complements Australia's freedom of certain animal diseases, and establishes a baseline of normal animal health at the community level.

The questions in the CAHR are intentionally biased in design with a focus on NAQS target animal diseases. An unreported benefit of this reporting system is that it serves as a prompt for the ranger groups to engage and maintain relationships with NAQS, and feel comfortable with reporting anything unusual. Given that ranger groups have responsibilities outside of biosecurity, the Ranger App and CAHR serve as simple prompts to facilitate the collection of data without double handling of data and paperwork.

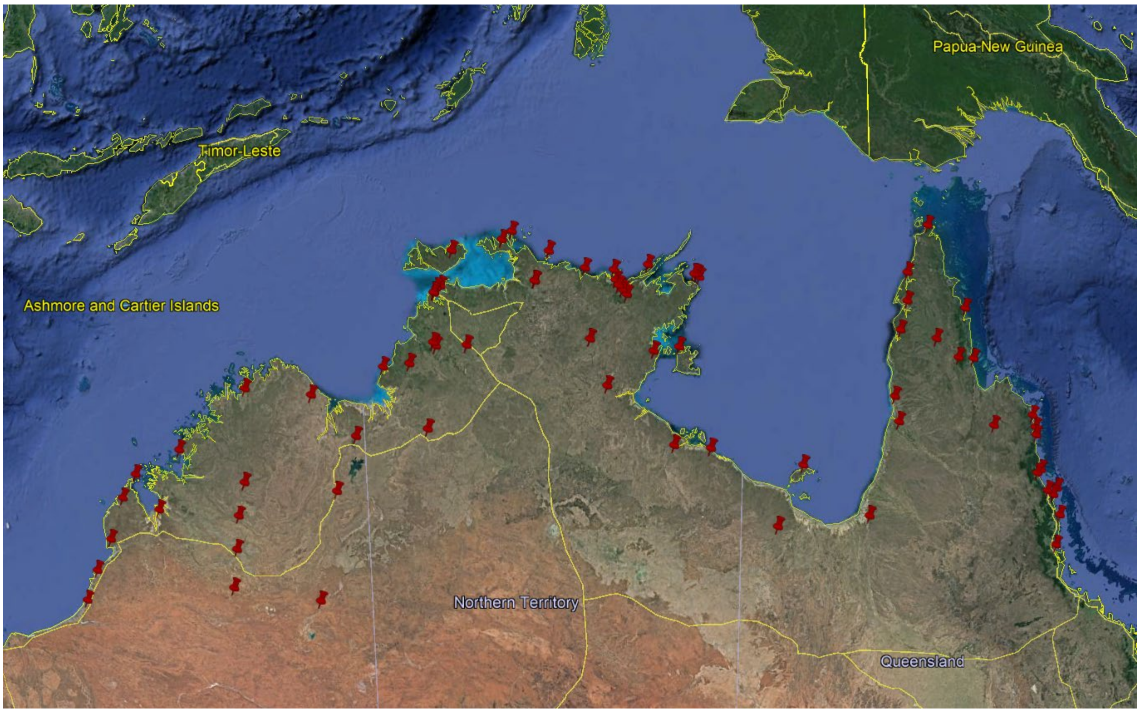

Fig 1. Indigenous ranger groups reporting for NAQS across northern Australia

Antimicrobial Resistance in Companion animal practice: A tragedy of the Commons

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Disclaimer:
The views and opinions expressed within this presentation and abstract are my own and may not be representative of The Department of Agriculture and Water Resources.

Introduction

The tragedy of the commons concept was first reported in 1833 by an economist by the name of William Forster Lloyd. He observed that communal grazing land was often in a poorer condition than privately owned land. A little over a hundred years later, ecologist Garrett Hardin revived the concept to issues such as overpopulation, clean air and fishing locations (Hardin, 1968, Hardin, 1994). Hardin argued that short term self-interests are often prioritised over the common good – with the individual receiving the benefits while the negative effects are dispersed and shared by the rest of the population.

The concept has applications to antimicrobial resistance, especially in companion animal practice.

The complex web of antimicrobial resistance

Antimicrobial resistance is complicated and there are a range of pathways and factors that have been proposed to have facilitated the development of resistant genes in bacteria at a global level, such as:

- Imprudent use of antimicrobials in humans and animals, especially for the treatment of viral diseases
- The over-the-counter availability of antimicrobials in a number of countries
- Poor health literacy amongst patients and clients
- The over-reliance of antimicrobials in certain sectors of intensive animal production
- Global movement of people, animals and goods that may harbour bacteria with resistance genes.
- The effect of the environment as a mixing pot and reservoir of resistant genes.

The weight of contributions from the above-mentioned drivers (as well as others) is poorly defined and it is best to describe the AMR issue as multifactorial and complicated (Woolhouse et al., 2015).

Yet, an easy response from any group within a silo is to blame other groups and professions for poor stewardship practices (Kahn, 2017).

The tragedy of the commons in companion animal practice

The structure of practice in companion animal medicine is at an individual, case-by-case level. Our goal is to utilise the best available science-based medicine for the benefit of our patient. Furthermore, there are a number of drivers that may influence our antimicrobial prescribing behaviour:

- Client expectation for the provision of antibiotics
- Experience and comfort with certain antimicrobials.
- Concerns regarding the "what if?" scenario – ie. the patient deteriorating
- Client dissatisfaction resulting in loss of business to a local competitor
- The use of antimicrobials to accelerate patient recovery
- Financial constraints of the client – limiting the number of review consults
- Patient/client compliance around dosing

These factors may be part of the risk-benefit equation that clinicians consider with each case prior to prescribing antimicrobials. Yet, due to business pressures and competition between clinics, especially in metropolitan regions with multiple clinics, we tend to lose sight of our prescribing practices or longitudinal trends of resistance at the regional level.

The zoonotic edge

While our patients, for the most part, are not interacting with one another like a herd, they do interact with other species, namely humans. This close interaction, clustered within a shared household, can result in the sharing of microbes – especially bacteria. While not every bacterial species encountered in veterinary practice is zoonotic, there are a few that are capable of jumping between species (Dotto et al., 2018, Rijks et al., 2016).

Antimicrobial stewardship

This concept is not new and has been discussed in previous proceedings, conferences and journals at great length. Through a series of actions and heightened awareness, veterinarians can preserve the effectiveness and availability of antimicrobials for the benefit of our patients, the wider public and the environment. Yet this requires a degree of oversight and responsible, evidence-based utilisation of the antimicrobials we have available.

Furthermore, barriers at the clinic level need to be acknowledged and addressed (Hardefeldt et al., 2018). Parallels with the medical profession may help identify some of these barriers such as the influence of senior clinicians on junior physicians or even the intra-team dynamics (Lewis and Tully, 2009, Papoutsi et al., 2017).

This talk will discuss the application of the antimicrobial stewardship principles at various levels as follows:

- Individual practitioner level: reduce the use of antimicrobials in conditions and procedures that don't require their use (Hardefeldt et al., 2017, Knights et al., 2012).
- Client population level: Proactively engage with the local community through media (traditional and digital) on why antimicrobial use requires stewardship through a One Health prism (Stallwood et al., 2019).
- Clinic level: Utilise guidelines and establish clinic agreement on empiric treatments. Furthermore, establish a review process on a 6 monthly or annual basis to identify trends and areas for improvement (Lloyd and Page, 2018). Finally, at a bare minimum, seek to improve clinic-level hand hygiene (Anderson and Weese, 2016).
- Professional/regional level: Explore applications of peer feedback (Hallsworth et al., 2016).

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Abnormal faeces in the adult rabbit

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Diarrhoea is uncommon in adult rabbits. True diarrhoea, which is defined as loose, watery unformed faeces needs to be differentiated from uneaten caecotrophes, which are the product of "first pass" digestion in rabbits. In young rabbits (<1 yr of age) diarrhoea is a more common finding. Coccidiosis is the most frequently reported underlying cause, but mucoid enteritis and other bacterial infections are also causes of diarrhoea in young rabbits – and left untreated it can be fatal (Varga 2014).

The causes of adult onset diarrhoea in rabbits are not always obvious and can be difficult to investigate. Megacolon, inflammatory bowel disease and bacterial or yeast infections have been anecdotally noted as a cause of altered or loose faeces in rabbits (Hersey-Benner, 2008), or discussed as individual case reports, but larger studies into the underlying causes and treatment options of diarrhoea for rabbits have not been undertaken. The usual methods of diagnosing bowel disease in other species, including biopsy and endoscopy, are difficult and potentially dangerous to perform in rabbits.

More work on the causes of diarrhoea in adults has been undertaken in other species. Anatomically, horses are similar to rabbits in that they are hind gut fermenters, have a large and complex gastrointestinal system and cannot vomit. In horses, a type of inflammatory bowel disease (ISBD) has been described and investigated. ISBD is characterized by poor body condition, weight loss, failure to gain, decreased appetite, increased gastrointestinal motility, mild recurrent colic and diarrhoea (van der Kolk, 2012). In some of these cases gluten sensitivity has been noted, and high levels of gluten dependent antibodies have been found. In one of these cases a change to a gluten-free diet, including the use of lucerne hay has provided symptomatic relief (van der Kolk, 2012). There have been no such investigations or diet trials in rabbits with diarrhoea or other symptoms similar to ISBD. Rabbits may also experience a gluten sensitivity, similar to horses, which could account for their diarrhoea. A change in diet away from gluten rich materials may therefore improve the symptoms.

Aim

The aim of this investigation was to determine what effect a change of hay to lucerne might have on diarrhoea in adult rabbits.

Study Criteria

Ten cases of adult onset diarrhoea that were not responsive to a normal diet exclusion trial were included in the study.

Methods

History

A full history was taken for each rabbit, getting details of current and past diet, any possible inciting cause or other changes the owner may have noticed. Owners were asked to bring in samples or photographs of the "diarrhoea". All rabbits that were producing uneaten caecotrophes with no evidence of true diarrhoea were excluded from the study.

Owners were also asked about any perceived weight loss, bloating or gut stasis episodes that may have occurred. In contact rabbits were also examined. Owners were asked about any previous or current treatments and what, if any effect these had on the diarrhoea.

Clinical exam

All rabbits had a full clinical exam performed, including abdominal palpation, weight and assessment of body condition score. Any other existing medical problems were noted.

Faecal analysis

All rabbits had a faecal analysis by microscopy performed in house before any treatment plan or diet trial was started.

Blood testing

Full haematology and biochemistry were performed in four of the cases. In the other rabbits the owners declined blood testing for various reasons.

Imaging

Abdominal ultrasound was undertaken in one case, the only rabbit who did not respond to the lucerne only diet trial.

Diet trial

All of the rabbits in the study were originally on pellets (varying brands), different types of vegetables and fruits and all were on oaten hay. The rabbits were put on an exclusion diet trial. Pellets were removed first, then fruit, and then every few days one type of vegetable was removed from the diet until the rabbit was solely on oaten hay. The owners were asked to keep a "poo" diary and report amount and frequency of any diarrhoea noted.

Although there was some / temporary improvement in some of the rabbits, all were still producing at least some watery loose faeces. They were then asked to switch hay type to lucerne – and to keep on the lucerne only diet for at least two weeks.

Results

Six males and 4 females were entered into the study. All were desexed, indoor rabbits. The average age of the rabbits was 2.2yrs (range 1.5-9yrs). Average length of onset of symptoms before referral to the clinic was (range 3-52wks).

Of the ten rabbits, three owners reported the rabbits had always had diarrhoea, two reported signs appearing shortly after desexing, and two reported signs after change in hay. One rabbit developed symptoms shortly after being rehomed, and one was reported to always be sensitive to some types of vegetables.

Most (9/10) rabbits were underweight, all had weight loss reported and 9/10 were in poor general body condition.

9/10 rabbits had a negative faecal parasite test prior to the diet trial. One rabbit, a 2yo male had coccidia present in his faeces, and was treated with toltrazuril. His diarrhoea did not improve considerably and was therefore entered in to the diet trial phase of the study.

Of the ten rabbits in the trial the diarrhoea cleared up completely in nine cases, within one week, as self-reported by the owner and confirmed by the vet.

One rabbits' diarrhoea improved but did not clear up completely, and on haematology a lymphocytosis was noted with an overall increase in white cell count. Abdominal ultrasound was performed and enlarged abdominal lymph nodes were noted. The lymph nodes were not biopsied due to the increased anaesthetic risk to this rabbit. Tentative differential diagnoses of inflammatory bowel disease or intestinal lymphoma were suggested and a course of prednisolone 2mg bid was started. After 3 months the diarrhoea had resolved completely, and the lymphocyte and white cell counts had returned to normal. The prednisolone was then tapered down and stopped. The total course of prednisolone was 5 months, and no side effects except increased appetite were noted. The rabbit has remained clinically normal and diarrhea free for 5 months post stopping all medication.

Conclusion

In cases of non-parasitic diarrhoea in adult rabbits, changing the hay in the diet from a gluten based source, to lucerne, can improve the overall quality of faeces.

Discussion

The results and apparent 90% success rate of this study is compelling. However, there are several problems with this study. Firstly, and most importantly we do not have a definite clinical diagnosis for any of the cases in this trial. It has been assumed by association and the finding of similar symptoms in the horse that the diarrhoea in these rabbits may also be caused by gluten intolerance. Future work could look at testing adult rabbits with diarrhoea for increased gluten dependent antibodies compared to normal rabbits.

The use of lucerne hay as part of an adult rabbits' diet is somewhat controversial. Lucerne is higher in calories and calcium and is usually only recommended for young rabbits or nursing does. However, as the rabbits in this study were almost all underweight, weight gain in this instance would be a positive side effect.

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Further data to support the repellency and efficacy of the Seresto® Collar for Cats against *Ixodes holocyclus* for 8 months

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Aims

Few products are registered to control the Australian paralysis tick (*Ixodes holocyclus*) on cats, and no registered products have the ability to repel ticks, or to be acaricidal on contact so that the tick does not need to bite the host to be killed. These features, offered by Seresto® for Dogs, would also benefit cats by minimising the discomfort of tick bites and reducing the potential risk of transmission of tick-borne diseases. There is little known about tick-borne diseases in Australian cats, but putative pathogens have recently been isolated from paralysis ticks collected from cats (Gofton, 2015; Greay, 2018). A novel study design was adopted to evaluate the repellency and efficacy of the Seresto® Collar for Cats (Seresto) against *I. holocyclus,* and data from this study was previously reported (Lyons, 2018). A second study was conducted, to confirm the results of the first, and to fulfil the requirements for product registration.

Methods

An un-blinded, randomised, controlled study was conducted, from March to November, to evaluate the repellency and efficacy of a 10% imidacloprid and 4.5% flumethrin collar (Seresto® Collar for Cats) on cats, against induced infestations of *I. holocyclus*. Twenty four domestic cats, immunised against holocyclotoxin, were included in the study. On Day 0, placebo or Seresto collars were fitted to the cats in Group 1 (n = 12) and Group 2 (n = 12), respectively. Assessments were conducted from Day 7 and then at monthly intervals over 8 months. At each assessment, tick infestations were conducted by releasing 20 ticks alongside sedated cats in an infestation chamber and leaving them to attach naturally over 1 h (± 5 min). Repellency was determined at 6 and 24 h post-infestation using the total number of attached ticks (ToA) on cats (ToA = Live Attached + Dead Attached + Moribund). Efficacy was determined at 48 and 72 h post-infestation using the total number of live (ToL) ticks on cats (ToL = Live Free + Live Attached + Moribund).

Results

At 6 h post-infestation, there was a repellency effect ranging from 97 to 100% on Seresto-treated cats over the 8 month study. A single live tick was found on one cat 48 h following the final infestation on Day 246, but subsequently died within 72 h of infestation. Consequently the efficacy of Seresto at 48 h post-infestation was 100% from Day 7 to Day 219, but reduced to 99% on Day 246. The efficacy of Seresto at 72 h post-infestation was 100% throughout the 8 month study. The difference in ToA and ToL tick counts between Group 1 and 2 was significant ($p<0.01$) at every tick challenge.

Conclusions

Seresto was highly effective at repelling (>97%) and killing (≥99%) *Ixodes holocyclus* on cats within 6 h and 48 h of infestation, respectively, for 8 months, from 7 days after collar application.

Clinical significance of the results

This study confirms the very high repellent and acaricidal efficacy of the Seresto Collar for Cats against *Ixodes holocyclus* for eight months, as previously reported.

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Conflict of interest statement
Employment of the authors by Bayer as indicated in institution or practice details above.

Investigating the magnitude and causes of foetal loss in young ewes

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Aim: To determine the magnitude and timing of reproductive wastage in young ewes.

Methods:
Fieldwork methodology for a national project investigating magnitude and causes of reproductive wastage in young ewes was piloted on four farms in WA. At each farm, approximately 200 maiden ewes (aged 7-20 months) were monitored throughout pregnancy. Pregnancy status was determined using transabdominal ultrasonography at 76-80 (scan 1) and 116-119 (scan 2) days from initial exposure to rams, with a 35-46 day joining period. Number of lambs born and present at marking were recorded by the farmer. Aborted tissues and lambs that died within 48 hours of birth were collected, and necropsies were performed to determine cause of death.

Results:
Overall marking rates and lamb survival appear similar across all farms, except for farm 3 where greater loss of potential lambs was observed between scan 1 and lambing and contributed to greater overall reproductive wastage (Table 1). Wastage between scan two and lambing includes abortion, as well as stillborn, dead or predated lambs not located in the paddock.

Table 1: Magnitude and timing of foetal loss

	Farm 1	Farm 2	Farm 3	Farm 4
Age at joining (months)	18-20	18-20	7-9	7-9
Scanning rate (%)	116	120	96	102
Marking rate (%)	94	85	51	80
Wastage between scan 1 and scan 2 (%)	0	0	7	0.5
Wastage between scan 2 and lambing (%)	2	4	12	7
Perinatal loss (birth-marking) (%)	18	26	35	16
Overall wastage (%)	19	29	47	22

Post mortem exams were performed on 135 lambs. Cause of perinatal death were unable to be determined in 30% cases. Starvation-mismothering-exposure (27%), dystocia (23%) and stillbirth (19%) were the three most common causes of death.

Conclusions:
Perinatal death was the most significant source of reproductive wastage in maiden ewes, consistent with previous observations for mature ewes.[1] However, mid and late gestation losses were evident on one farm. This is consistent with a previous small study that suggested in utero losses are a significant source of reproductive wastage in ewe lambs.[2] However, the degree and causes of wastage during mid and late gestation in maiden ewes is not well understood. The proportion of deaths due to starvation-mismothering-exposure and dystocia were similar to previous studies in adult ewes, suggesting that whilst lamb survival may be lower for maiden ewes, causes of perinatal death follow similar patterns as for adult ewes.[1] Stillborn lambs represented an important component of wastage, and the potential role of abortifacient agents as a source of overall wastage in maiden ewes warrants further investigation.

Clinical significance of the results:
Preliminary results suggest that mid and late gestational foetal loss may be an important contributor to overall wastage for ewes bred at 7-9 months of age on Australian farms. This project is being expanded in 2019-2020 to better determine the timing and extent of reproductive wastage for young ewes, and determine if infectious disease has an involvement.

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Conflict of interest statement
None of the authors listed have any conflict of interest to declare. The project was funded by Meat and Livestock Australia. Meat and Livestock Australia approved the manuscript for publication, but were not involved in the collection, analysis or interpretation of data. All sample collection methods used were approved by the Murdoch University Animal Ethics Committee (Approval no. R3004/17).

Safety and availability of tetrahydrocannabinol (THC) and cannabidiol (CBD) after oral dosage in the dog: Influence on pain and inflammatory pathways associated with the endocannabinoid system (ECS).

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Abstract:
Introduction: The most prevalent cannabinoid (CB) in cannabis is THC, a partial agonist of CB receptors. The next most abundant is CBD, an indirect antagonist of CB receptors which interacts with several other receptors systems. These receptors and endogenous CBs form the endocannabinoid system (ECS), which influences cognitive, physiological and metabolic functions in animals, including pain and inflammation control. The ECS is targeted therapeutically by medical cannabis.

Materials and Methods: A randomised three group parallel dose-evaluation study was conducted using 11 healthy male Beagle dogs. Single oral doses were administered to each dog 2.5 hours after feeding. Group 1 (n=4) received THC (0.24 mg/kg) and CBD (0.12 mg/kg), Group 2 (n=4) received THC (0.12 mg/kg) and CBD (0.24 mg/kg), Group 3 (n=3) received a placebo containing no CBs. Dogs were monitored for clinical effects for 3 days. Samples for plasma and whole blood were drawn at 15 timepoints in 72 hours after dosing. Plasma samples were analysed for THC and CBD concentrations and a range of other biomarkers. The effect of treatment on pain and inflammatory pathway associated gene expression was evaluated using qPCR on whole blood.

Results: No adverse events or psychotropic changes were observed in any dog. The time of peak concentration (T_{max}) for both CBD and THC across treatment groups occurred from 40 to 90 min post treatment. Mean THC peak concentration (C_{max}) in Groups 1 and 2 were 46.6 ng/mL and 38.8 ng/mL and mean CBD-C_{max} were 13.1 ng/mL and 37.8 ng/mL, respectively. The terminal elimination half-lives were found to be slow for both CBD (12.6 h) and THC (18.5 h). Gene expression was compared at 1.5 hour and 72 hour time points for each group, accounting for pre-treatment values. A significant ($p<0.05$) change in fold regulation was seen in expression of Chemokine ligand 5, CB2 and Interleukin 8 in Group 1 compared with placebo; and Chemokine ligand 5, Cerebellar degeneration-related protein 2, CB2 and Interleukin 8 in Group 2 compared with placebo. The effects of THC and CBD on inflammatory cytokines and neurotransmitters were tested at 7 time points from 0-24 hours in all groups. Significant differences ($p<0.05$) accounting for time, pre-treatment values and group x time interaction were observed in some biomarkers known to be associated with modulation of anti-inflammatory processes in treatment groups compared to placebo.

Conclusion: THC and CBD are bioavailable, reaching plasma after oral administration. These data suggest that THC and CBD have the potential to control pain and inflammation in dogs and are slowly eliminated from blood circulation. These effects are likely to be a combination of direct effects and supporting the ECS allowing animals to maximise endogenous pain control processes.

Facilitating effective, responsible and sustainable antimicrobial mastitis treatment in the Australian Dairy Industry: Susceptibility of Local Pathogens to Penicillin and Cloxacillin

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Aims:

More than $150 million is lost to Australian dairy farmers each year through poor udder health, and the major cause is mastitis. Antimicrobial therapy remains an essential tool in the management of mastitis, however it raises the issue of antimicrobial resistance (AMR). Demonstration of evidence-based usage and commitment to ethical product stewardship is necessary to maintain safe and effective veterinary use of antimicrobials.

In a recent large survey of dairy cattle in SE Australia, the most common bacterial pathogens cultured from clinical mastitis cases were: *Streptococcus uberis* (54%), *Staphylococcus aureus* (15%), *Escherichia coli* (12%) and *Streptococcus dysgalactiae* (9%) – i.e. predominantly gram-positive bacteria (Charman et.al. 2012).

The aim of these two studies was to determine the current susceptibility of the most common gram-positive mastitis pathogens to two common antimicrobials used in the dairy industry: penicillin and cloxacillin. In addition, the development of a farm-specific anti-microbial monitoring program is discussed.

Methods:

Between 2016 - 2018 up to 30 bacterial isolates of *Streptococcus uberis*, *Staphylococcus aureus, S. agalctiae* and *S. dysgalactiae* were sampled from mastitis cases on commercial dairy farms in SE Australia. The Minimum Inhibitory Concentrations (MICs) of penicillin and cloxacillin were determined as the lowest concentration of the antibiotic that was found to inhibit bacterial growth.

Results:

Table 1. MICs for Cloxacillin

Bacterial pathogen	MIC50	MIC90	MIC range	Susceptibility*
Streptococcus uberis	0.5	4	0.125 - 4	100%
Staphylococcus aureus	0.25	0.25	<0.06 - 0.25	100%
Streptococcus agalactiae	2	2	0.125 - 2	100%
Streptococcus dysgalactiae	0.06	0.5	<0.06 - 2	100%

Table 2. MICs for Penicillin

Bacterial pathogen	MIC50	MIC90	MIC range	Susceptibility *
Streptococcus uberis	0.0312	0.125	0.0156 - 2	97%
Staphylococcus aureus	0.0312	4	0.0156 - 4	87%
Streptococcus agalactiae	0.0625	0.0625	0.0625 - 0.0625	100%
Streptococcus dysgalactiae	0.0078	0.0156	0.0078 - 0.125	100%

**Susceptibility was determined for each antimicrobial based on clinical breakpoints defined in the guidelines of the Clinical and Laboratory Standards Institute (CLSI).*

Conclusions

Common gram-positive bacterial pathogens cultured from mastitis cases of Australian dairy cattle showed 100% susceptibility to cloxacillin and high susceptibility to penicillin. The lowest susceptibility for penicillin was found in isolates of *S. aureus*. Resistance of *S. uberis* to penicillin in mastitis cases has been shown to be increasing globally (77% intermediate susceptibility or resistant, Canada, Cameron et. al. 2016, and 35.6% intermediate susceptibility, Europe, de. Jong et. al. 2018).

Clinical significance of the results

MIC testing of local mastitis pathogens gives a snap-shot of arising resistance. One mechanism for the accelerated development of antibiotic resistance is exposing bacteria to repeated, inappropriate antibiotic treatments causing them to become more resilient and harder to kill. Antibiotics remain a valuable tool in the dairy industry, and when used responsibly, are vital for maintaining animal health and welfare.

The use of narrow-spectrum, targeted antimicrobials and using antimicrobials wherever possible that are not on the 'restricted' list for human health are practices recommended by the recently published Veterinary Antimicrobial Prescribing Guidelines (prepared through collaboration between University of Melbourne's Asia Pacific Centre for Animal Health (APCAH) and the National Centre for Antimicrobial Stewardship).

Currently in Australia most mastitis prescriptions are made without local knowledge of the true quantitative antimicrobial susceptibility on that particular farm (MIC). The only commercial testing available previously for antimicrobial susceptibility was the agar disc diffusion assay, which had significant limitations and problems with interpretation.

Bayer is promoting the effective, sustainable, and responsible use of antimicrobials on Australian dairy farms by offering a new test, the DAIRY ANTIBIOGRAM. This test offers an easily accessible MIC assay of common mastitis pathogens tailored to a specific farm, based on a bulk milk tank sample.

The DAIRY ANTIBIOGRAM gives Australian dairy veterinarians a unique opportunity to build reliable susceptibility testing into their mastitis consultancy, and to use this data to guide responsible antibiotic use.

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Conflict of interest statement

Employment of the authors by Bayer as indicated in institution details above.

Stopping Canine Parvovirus – a multi-faceted research-and-intervention approach

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Aims:

Canine Parvovirus (CPV) disease occurs across Australia and worldwide, causing severe and often fatal gastroenteritis in dogs, especially puppies. The disease emerged in the late 1970s and early 1980s with a worldwide pandemic and remains endemic in many areas despite highly effective vaccines being available. This paper will describe a multifaceted approach to establish (a) the number of CPV cases across Australia and where/when they are occurring (b) social, climatic, seasonal, wildlife and geographic factors associated with cases/outbreaks (c) effective veterinary intervention strategies (d) whether discounted vaccination programs can improve herd immunity and (e) the best design for a CPV-specific disease surveillance-and-alerting program. The ultimate goal of this approach is to reduce or stop CPV cases in specific high-risk areas, and to then scale up to a successful program nationally.

Methods:

A national Australian veterinary profession survey was conducted in 2017 to determine CPV case numbers, regions affected, veterinary opinions on disease surveillance and CPV intervention strategies. Analysis on survey data examined risk factors relating to climate (from Bureau of Meteorology (BOM) data), socioeconomics (from Australian Bureau of Statistics (ABS) data), and remoteness (from ABS remoteness area (RA) data). Local veterinary clinics experiencing outbreaks supplied historical CPV data for analysis of seasonality patterns. Wild dogs tissue samples (n=171) from NSW and QLD at the urban fringe are being examined using quantitative PCR (qPCR) to determine if wild dogs are CPV carriers.

Results:

Australian companion animal veterinary clinics (n=2,260) were contacted and 569 veterinarians from 532 unique clinics (24%) responded to the survey. 4,219 CPV cases that occurred in 2016 were reported; this was extrapolated to an estimated national caseload of 20,110. Geospatial analysis revealed large CPV case numbers in rural and remote areas.[1] CPV was identified by qPCR in 9/69 (13%) wild dog samples analysed to date, full testing is yet to be completed. Examination of local CPV outbreak patterns in Rockhampton QLD and Wagga Wagga NSW reflected some seasonality yet inconsistency of timing and severity. An online CPV-monitoring and alerting system (Parvo ALERT, parvoalert.com) was designed and launched via a staged version release, with feedback from veterinary participants being sought to further improve the program. Discounted vaccination programs have started for disadvantaged pet-owners, in collaboration with local vets, councils, pet-lover-volunteers, shelters and rehoming groups. Over 4 months, 376 dogs (mostly puppies) were vaccinated in the Rockhampton region. Beneficiaries of the vaccination program are surveyed to gauge feedback, improve the system and update the model.

Conclusions:

CPV outbreaks are associated with socioeconomic disadvantage, remoteness and rural areas. Wild dogs do appear to carry CPV and whether they play a role in transmission requires further research. Extensive planning, stakeholder engagement, the formation of a charity and social-enterprise have all assisted in the success of the project to date.

Clinical Significance of the results:

Early results suggest that a discounted vaccination and communication strategy for socially disadvantaged people/pets, monitored by a disease-alerting system, could be used to prevent CPV cases in rural and remote areas.

Ethics Committee Approval:

Ethics approval for the veterinary survey (2016/802) and client survey (2017/358) was granted by the Sydney University Human Research Ethics Committee.

Conflict of Interest Statement:

The lead author, Mark Kelman is a co-founder and director of the charity, Paws for A Purpose. The charity is the owner of the Parvo ALERT disease-surveillance-and-alerting system. This study also represents part of a PhD at University of Sydney by the lead author.

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Luteinizing hormone receptor expression and function in canine lymphoma

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Aims

Luteinizing hormone (LH) is mainly considered a reproductive hormone, but LH receptors (LHR) have been identified in normal and neoplastic non-reproductive tissues. The objectives of this study were to: 1) determine LHR expression in canine B-cell and T-cell lymphomas and; 2) determine if LHR expression in canine lymphoma cells was functional in vitro.

Methods

Objective 1: Tumor samples removed during the routine clinical management of canine lymphoma (n=47 dogs) were formalin-fixed, paraffin embedded, sectioned onto charged slides and then subjected to routine immunohistochemical techniques using heat-induced epitope retrieval. Rabbit polyclonal anti-human LHR antibody (1:100 dilution) or rabbit negative control was applied to slides.
Objective 2: Immortalized T-cell lines from three dogs with T-cell lymphoma were plated in 96-well plates in RPMI 1640 (phenol and protein free) media at 37°C with 5% CO_2. For each cell line, standard curves from 10,000 to 500,000 cells/well were plated in triplicate. Increasing concentrations of human chorionic gonadotropin (hCG; 4 U/mL to 40,000 U/mL) or canine LH (0.002 ng/mL to 20 ng/mL) were added to wells containing 100,000 cells plated in triplicate. Plates were incubated for 24, 48, and 72 hours for hCG or 48, 72, 96 and 120 hours for cLH before cells were counted using a commercial MTT cell proliferation assay.
Data analysis: The percentage of cells positive for LHR and the staining intensity (scored 0-3) were compared using a Students t test or a one-way analysis of variance where appropriate. Mean ± SD cell number was compared between hormone concentrations using a one-way analysis of variance. Significance was defined as $p<0.05$.

Results

Objective 1: All lymphoma tissue samples contained cells positive for LHR but percentage of cellular expression and staining intensity varied between individual dogs. There were no significant differences in the percentage of LHR positive cells or staining intensity when compared by body weight, breed, sex, and lymphoma phenotype (B-cell vs. T-cell).
Objective 2: Activation of LHR in isolated canine T-lymphoma cells induced significant cell proliferation in all three cell lines with both hCG and cLH, but varied between concentrations and incubation times. The highest amount of proliferation from hCG occurred at the 40,000 U/mL concentration following 72 hours of incubation in all three cell lines. The highest amount of proliferation from cLH occurred at the 2 ng/mL concentration following 96 hours of incubation in all three cell lines. However, there was a significant decrease in cell counts following administration of cLH at the highest concentration (20 ng/mL) at all incubations times.

Conclusions

This is the first study to provide evidence that canine B-cell and T-cell lymphomas express LHR and that these LHR are functional (e.g. activation induces cells proliferation).

Clinical significance of the results

The current research provides an explanation for why desexed dogs are 3-4 times more likely to develop lymphoma. Clinical trials are planned to include deslorelin-induced LH down-regulation with conventional chemotherapy to prolong survival times in dogs.

Conflict of interest statement

Virbac provided funding for the travel and accommodations of the abstract presenter.

Comparison of the efficacy of sodium pentosan polysulfate alone with the combination of sodium pentosan polysulfate and N'acetyl glucosamine for the treatment of osteoarthritis in dogs

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Aims

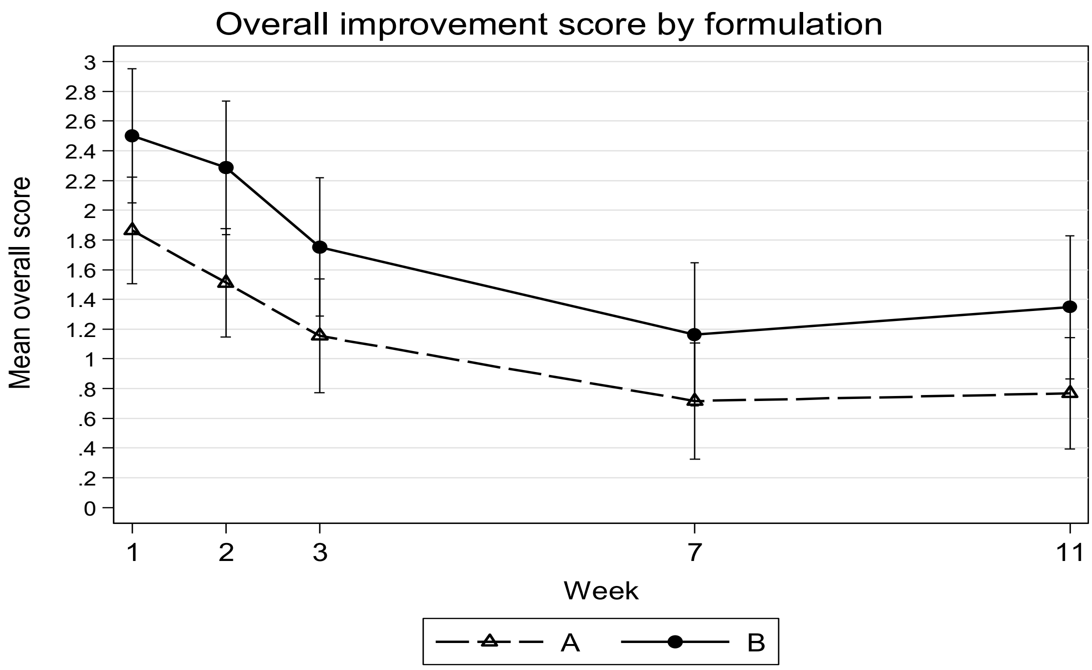
The efficacy of sodium pentosan polysulfate (PPS) and N'acetyl glucosamine (PPS-NAG), was compared in a double-blinded randomised, positive controlled clinical trial in dogs with confirmed radiographic evidence of osteoarthritis (OA).

Methods

Forty eight dogs with lameness and radiographically confirmed OA and no other diseases or recent OA treatment were randomly assigned to treatment with a 4 day course of carprofen or meloxicam and then 4 weekly SC injections of 3mg/kg PPS (Cartrophen Vet Injection, Biopharm Australia) or the combination PPS-NAG (Synovan® Injection, Ceva Animal Health). The dogs were assessed by veterinary investigators at baseline (week 0) than weeks 1, 2, 3, 7 and 11 for lameness, range of motion, pain on palpation and overall response to treatment. The dog owners completed a Canine Brief Pain Inventory (CBPI) immediately before (baseline) and then weekly for 11 weeks after treatment. CBPI scores provided measures of pain severity (assessed as worst, least, average and now), and of pain interference (assessed as general activity, enjoyment of life, ability to rise, ability to walk, ability to run and ability to climb up) as described by Brown et al.[1] Statistical analysis was conducted on 36 dogs, 22 treated with PPS-NAG and 14 treated with PPS alone.

Results

Improvement over 11 weeks was seen in both treatment groups with significant reductions in lameness score, overall improvement and CBPI scores over time. The scores for veterinary assessment of lameness score, overall improvement, CBPI pain severity and interference for PPS-NAG treated dogs were consistently lower numerically than those for PPS treated dogs. CBPI pain severity scores for PPS-NAG treated dogs were significantly lower in weeks 5-9. Overall improvement scores for PPS-NAG treated dogs (A) were significantly lower than PPS treated dogs in weeks 1 and 2 (Figure). There was a more rapid response to PPS-NAG with lameness scores in PPS-NAG treated dogs showing a significant decrease in weeks 1-2 and 2-11 compared with dogs treated with PPS, which showed a decrease later, from weeks 3-7.



Conclusions

This study demonstrated that PPS treatment of OA in dogs both alone and in combination with NAG, is effective in reducing the clinical signs of OA. Although this study lacks a negative control it supports earlier studies[2] which reported the benefits of the use of PPS. The response following PPS-NAG treatment was significantly greater and more rapid than the response following PPS treatment. This is supported by in vitro trials which have demonstrated a greater anti-inflammatory effect for the combination of PPS and NAG compared with PPS alone,[3] it is recognised that combinations of drugs can assist in the management of OA in humans.[4]

Clinical significance of the results

Enhanced efficacy and a more rapid response to treatment will not only benefit patients immediately but also may lead to better long term outcomes as this will be noticeable to clients and encourage them to return for future veterinary treatments. The more rapid reduction in pain may be an important factor in establishing patient loyalty and having those clients return for booster injections.

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Finola McConaghy is an employee of Ceva Animal Health, manufacturer of Synovan injection

Innovative telepathology solutions

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Increasing client and owner demand for faster turnaround of specialist reporting of pathology, combined with a shortage of pathologists in Australia and globally, has driven demand for digitization of pathology specimens which enables workload distribution. While current architectural evaluation of histopathology still requires formalin fixed tissues to be cut, paraffin embedded and thin sections prepared and stained, the digital scanner now can take the place of the microscope, with pathologists viewing slides as a virtual image through web-based platforms.

Some of the benefits of this approach include the ease and rapidity with which cases can be distributed, shared, discussed and collaboratively reported, but bonuses include the ease of use for training of residents and for education of the submitting veterinary client, and owner of the animals, through pictorial reporting alongside traditional text-based reporting. Cytology digitization and pictorial reporting in particular allows practitioners to become more confident of their own skills and to learn directly from pathologist reports.

Removing the microscope from the equation also has some striking benefits for workplace comfort and occupational health and safety. Pathology is still largely a sedentary occupation for those not on the autopsy room floor, but it no longer requires a fixed neck position and the same degree of eye strain.

Idexx introduced and validated technology now employed for the majority of our histopathology cases around the world, and now extends this digital work to include cytopathology, haematology slide review, and even gross specimen trimming through webcams and live oversight of tissue preparation between the scientist and pathologist. The veterinary world has for once overtaken the human medical world in this field.

Pathologists are often geographically and therefore socially and academically isolated. Digital pathology enables flexible and remote working with benefits to family and work-life balance, but also helps to reduce isolation for those working from home, through our weekly consensus rounds. This collaborative event provides a group experience of more than 100 years in pathology all contributing opinions, along with team building and support. The teams use real time slide review, markup, and discussion to progress a diagnosis or discuss next steps. We can also reach out to a worldwide network of pathologists and other specialists with knowledge in specialist areas. For example, we recently had a case of psittacine beak and feather disease in a non-psittacine bird, which we were able to confirm on the same day by collaborating with an expert in that field remotely. This means that the trend of sub-specialist pathology knowledge and expectations that are already evident in human pathology will gradually increase in the veterinary field, to the benefit of our animal patients.

Discovery and Early Development of a New Antigiardial Drug Class

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Aim
Giardia duodenalis is a ubiquitous protozoan pathogen that causes significant morbidity and mortality in humans worldwide[1]. Furthermore, globally, *Giardia* is the most frequently reported intestinal parasite of dogs[2], who are commonly infected with clades of *Giardia* that are zoonotic[3]. Failure of drug therapy is increasingly encountered, potentially due to poor patient compliance as a result of the need for repeated drug administration, off target adverse drug effects and, importantly, parasite drug resistance[4]. In recognition of the current limitations of existing treatments of giardosis and the dearth of new treatments under development, the objective of this discovery project was to study the *in vitro* efficacy and selectivity of a library of aminoguanidine compounds against *Giardia* and identify lead compounds that met the target product profile.

Methods
Standard *in vitro* methods included bacterial microbiological inhibition concentration determination, the resazurin reduction assay of cell metabolism, a modified adherence assay, a treatment recovery assay, and cytotoxicity assessments in human cell lines. A mouse model of *Giardia* infection was developed specifically for this project[5].

Results
From an initial library of 150 compounds, 2 novel entities displayed low micromolar giardicidal activity, no antibacterial activity, were highly effective in the *in vivo* mouse infection model, and were acutely safe in mice given 2,000mg/kg *per os*.

Conclusions
Arising from a comprehensive *in vitro* and *in vivo* characterisation project, a novel class of microbiologically selective antigiardial compounds has been identified with a large margin of safety[6]. Studies of pharmacology, safety and effectiveness in dogs are currently scheduled for later in 2019.

Clinical significance of the results
A novel class of safe and effective drugs to treat *Giardia* infections in humans and animals is a major unmet need due to the emergence of resistance to all currently approved agents. The availability of a new treatment has the potential to improve animal health and welfare, reduce zoonotic risks, and significantly reduce the adverse effects of infection in humans, particularly children, in the developing world.

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Conflict of Interest Statement

SWP is a Director of Neoculi; SWP, AMC and AJS are co-inventors of the novel chemical library.

Paraoxonase-1 activity as marker for diagnosis of equine metabolic syndrome

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Aims

Equine metabolic syndrome (EMS) is a clinical syndrome recognized in horses and ponies characterized by obesity, dyslipidemia, adipokine dysregulation and insulin dysregulation (ID).1,2 Current diagnostic strategies are centred around demonstration of ID, and some of these tests have questionable sensitivity and repeatability.3,4 Human metabolic syndrome shares similar pathophysiological features to EMS and a new serum marker, paraoxonase-1 (PON1) activity, has been proposed to improve the diagnosis.5 PON1 is a serum enzyme that binds to lipoprotein, which has an active role in modulation of the inflammatory response and is a noteworthy antioxidant given its ability to protect lipid oxidation.6 Systemic inflammation and increased lipid oxidation are important in the pathogenesis of HMS and a consistent decrease in PON1 activity has been demonstrated.5 The aims of this study are: 1) to compare PON1 activity, cholesterol, triglycerides, high density lipoprotein (HDL) and basal insulin in horses with EMS and clinically normal horses; 2) to compare PON1 activity with cholesterol, triglycerides, HDL and basal insulin..

Methods

Thirty-four clinically normal equids and 34 with EMS (confirmed ID) were enrolled in the study (Permit R2887/16). PON1 activity, cholesterol, triglycerides and HDL were measured using an automated spectrophotometer.

Results PON1 activity was not significantly different between horses with or without EMS (p=.278). Triglycerides and base line insulin were significantly higher (p=.000) in EMS horses than in healthy control group. PON1 activity is positively correlated with cholesterol (r=.526, p=.000), and HDL (r=.600, p=.000) and weakly with basal insulin (r=.385, p=.006).

Conclusions

PON1 activity does not appear to be a useful diagnostic marker in horses with EMS, however a larger sample size is required to account for type II error. However, the PON1 activity changes are positively correlated with lipid changes supporting the link between lipid metabolism and PON1 activity. More investigation on PON1 activity's role on lipid oxidation in horses is warranted.

Clinical significance of the results

Despite human and EMS sharing some similarities, PON1 activity does not appear to a useful diagnostic test in horses with this condition.

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Conflict of interest statement

This project has been funded by Morris Animal Foundation

Numnuts – humane tail docking and castration

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Aims

'Marking' i.e. tail docking and castration of lambs is coming under increasing scrutiny by animal welfare groups together with growing consumer focus on humane treatment of all animals, suggest improvements to traditional animal husbandry procedures.
Normal practice is to apply latex castration rings to the testes and tails of both male and female lambs. The constriction forces generated by the ring develop into acute ischemic pain. This is at its most severe from 15 to 45mins. Over 70 million procedures are carried out in Australia annually.
Well established research (Mellor and Stafford 2000) highlighted this pain infliction which can cause restrictions to their healthy development. Additional evidence has shown that local anaesthetics (LA) can alleviate this pain (Kent et al 1997). Until now, the risk of needle stick injury to the farmer, time required to administer LA using a needle and syringe and uncertainty about how and where to administer creates a barrier to the adoption of LA as part of normal 'marking' practice.

Methods

In 2013 a partnership was formed between 4c Design (Scottish design engineering company) and Moredun Research Institute (Scotland) to develop a solution. With funding from MLA and AWI, CSIRO was engaged to perform the field research.

Results

Numnuts® is the unique, practical approach which has resulted. The Numnuts® device combines the application of tail docking and castration rings with a simultaneous injection of a fixed, controlled dose of NumOcaine® LA, targeting the point of pain infliction on the animal.
Numnuts® comprises three major components combined in one easily operated device:-

- Vastly improved, ergonomically designed, ring applicator
- Unique injector system providing a safe discharge mechanism
- Quick Change Cartridge (QCC) locking-in the S4 LA, 'NumOcaine™' in a safe, easy to broach, tamper-proof package.

Numnuts® works when all the component parts are combined as a complete system.

Conclusions

Operator safety, ease of application and productivity all at a (reasonably) low cost have been the key factors in the development of the product.
Numnuts®, launching at this conference, addresses a need, long identified by the veterinary profession, which until now could not be met, neither practically nor economically, on-farm.

Clinical significance of the results

Numnuts® is a collaboration of veterinary science combining with engineering design and manufacture to produce a practical, economic and safe solution to a necessary animal husbandry procedure which addresses contemporary societal demands.

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Conflicts of interest statement: I am not aware of any conflicts of interest

A comparison between juvenile pubic symphysiodesis and juvenile pubic symphysectomy: a one year follow-up

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Hip dysplasia (HD) is one of the most common orthopaedic diseases of large-breed dogs[7]. In affected dogs, joint laxity, joint incongruence, and secondary osteoarthritis can lead to crippling pain[2, 5, 7]. Current topics on hip surgery involve the technique of juvenile symphysiodesis (JPS), a technique to improve the congruence of growing coxofemoral joints in young dogs[2,9,11,12,13]. Although other procedures do exist, the primary researcher proposes a modified technique: juvenile pubic symphysectomy (JPSec).

Radiographic and computed tomography (CT) measurements were made in the study group of 21immature puppies aged 16 weeks. The following measurements were used for the determination of HD: Subluxation index (SI) with the Flückiger method[6], Norberg angles[5], lateral center-edge angles[4,5], dorsal acetabular rim angles[4,5] and acetabular ventro version angles (VV)[4]. Joint laxity was determined subjectively by using the Barden's lift technique and the Ortolani sign. The HD positive puppies were randomly selected for the surgical techniques used in this study. The study consisted of three groups: Group 1 = HD free (non-surgical) control group (N=7), Group 2 HD at risk positive dogs treated with the JPS technique using electro cautery (N=7) and Group 3 the JPSec technique was performed on the pre-determined hip dysplastic dogs (N=7).
The pelvic ventroversion angles were assessed pre-and immediately post-op, at 16 weeks, and with follow-up assessments at 20, 24 and 54 weeks of age in all the groups.

The JPSec technique resulted in immediate change in the acetabular ventro version angles by ±5° post surgically (24-48h) due to rotation of both acetabulae (ventroversion). The JPSec appeared to provide greater and more dorsal acetabular covering (DARA) after 24 and 54 weeks than JPS. The lateral center edge angles also improved on follow-up assessments of both the techniques. There was a significant change in ventroversion angle improvement, more so with the JPSec than with JPS procedure and as a result, also better dorsal coxofemoral coverage by the dorsal acetabular rim with decreased hip joint laxity. JPSec and JPS did not have any significant effect on the sacral width or sacral conformation.

The pubic symphysectomy procedure is a simple surgical technique that yields good results without the need for specialized equipment. The age of the dogs is an important factor. It is believed that the benefits of removing the *pubic symphysis* growth plate at 16 weeks of age would be optimal. The difference between the two techniques lies in the severity of the HD of the dog. It was found that the JPS yielded the best results with a Norberg angle between 95° and 102°, whereas JPSec heralded the best results between 85° and 95° degrees.
The JPSec technique requires fixation of the pubic ramii and symphysis with orthopedic wire, these implants will serve as an indicator of a previous corrective surgical procedure when such an animal is later presented for HD evaluation.

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