



Proceedings of the 2024 Small Animal Conference

Together with the Veterinary Business Group

Novotel Sunshine Coast Resort, Qld
12-15 August 2024



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¹ Wernimont SM, Fritsch DA, Jackson M, et al. Specialized Dietary Fibers Alter Microbiome Composition & Promote Fermentative Metabolism in the Lower Gastrointestinal Tract of Healthy Adult Cats. *Current Developments in Nutrition*. 2019;3(S1):7.
² Fritsch DA, et al. Food with Novel Fiber Blend Improves Clinical Outcomes and Changes Gastrointestinal Microbiome Metabolism in Dogs (in a 2 Month Study). *J Vet Intern Med*. 2019;33(5):2513.
³ Wernimont SM, et al. Food with Specialized Dietary Fiber Sources Improves Clinical Outcomes in Adult Cats with Constipation or Diarrhea (in a 2 Month Study). *FASEB J*. 2020;34(1). Some cats may require multimodal management.



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Table of Contents

Small Animal

Brain, PH - Coughing, bronchomalacia and tracheal stenting	9
Brain, PH - Pulmonary hypertension	17
Brain, PH - The coughing dog with a heart murmur	27
Fry, C - Culture is key: How everyone can co-create more positive veterinary working environments	37
Fry, C - Managing strong emotions in veterinary workplaces	46
Hopper, B - Diagnosing canine heart failure in practice: Combining clinical examination, radiography, echo and TFAST	55
Hopper, B - Imaging and interpreting lungs better: Make the most of your radiographs and when do you really need CT?	63
Hopper, B - Aerodigestive disorders: How many of our respiratory sign patients actually have oesophageal disease?	73
Hopper, B - The mediastinum: Recognising and diagnosing disease in this space	75
Jackson, K - Pleural effusion clinical pathology: How to get the most out of your samples	83
Johnston, L - An update on pneumonia in dogs and cats	87
Johnston, L - Respiratory noise and character: How the sounds and patterns your patients make can lead you to a diagnosis	90
Johnston, L - Talking tubes: Understanding oesophageal dysfunction in dogs and cats	92
Lam, C - Diet-associated dilated cardiomyopathy (DCM): Update	95
Lam, C - Effect of systemic disorders on the heart: Feline	98
Lam, C - Feline aortic thromboembolism (FATE): Updates on management	103
Lam, C - Interventional treatment modalities for mitral valve disease	105
Lam, C - Overview on diagnosing feline cardiomyopathies	109
Lam, C - The effect of systemic disorders on the heart in the dog	112

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Coughing, bronchomalacia and tracheal stenting

Philip H Brain
BVSc FANZCVS (small animal medicine) FAVA
Registered specialist in small animal medicine

I'd like to begin by acknowledging that the presentation is being delivered from the lands of the Kabi Kabi and Jinibara peoples and pay my respects to Elders past, present and emerging.

These notes may contain opinions that are that of the author.

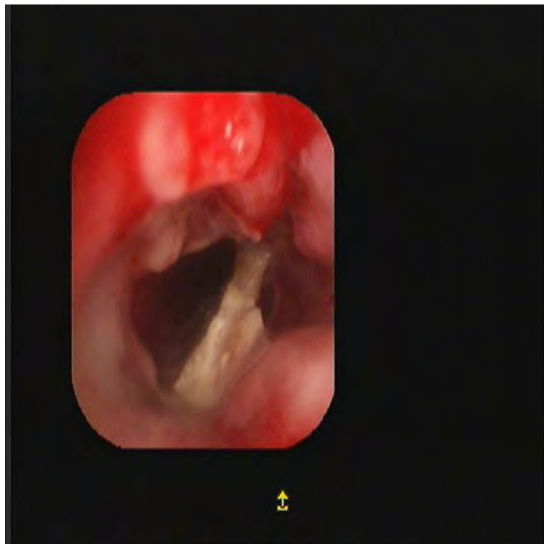
Coughing

- What is a cough?
- Video – not for you but to remind me...
- A cough is a sudden forced expiration against a closed glottis- sudden opening and turbulent airflow creates noise identified as a cough
- Cough pathway – cough receptors and sensory nerves in the airways, vagus nerve and central cough centre (brainstem and pons)
- Mechanical (proximal esp larynx/trachea) versus chemical irritation (bronchi and bronchioles (bronchial C fibres) and pulmonary (pulmonary C fibres))
- Chemical mediators include substance P, neurokinin and other tachykinins
- Less common causes of cough – disease involving pleura, pericardium, diaphragm, nose (nasal drip), sinuses, mediastinum
- Differentiate between coughing reflex and expiration reflex – no sharp intake of breath – laryngeal disease
- Acute coughing
- Rarer than chronic cough
- Cardiac- limited to acute onset CHF/pulmonary oedema - ruptured chordae tendinae but also I have recently seen in (recent traps)....arrhythmias, PE
- Respiratory-
- Upper airway- viral (canine infectious respiratory disease complex CIRDC- kennel cough), laryngeal or tracheal trauma, acute tracheitis, tracheobronchitis, foreign body,
- Lower airway – inhalation of FB
- Pulmonary- aspiration or FB pneumonia, infectious pneumonia, occasionally NCPO although more likely to be tachypnoeic rather cough

Case report - Ace



- Signalment- 2 ½ yo male Maltese X
- History- 3 week Hx coughing- Presumptive Dx viral tracheitis. No response to antibiotics, progression to constant coughing. Thoracic rads NSA.
- But radiography including neck revealed....
- Tracheoscopy reveals.....
- Unremarkable recovery
- Take home message- atypical acute cough- take rads (that include the neck region)



Tracheal foreign bodies

- Not overly common
- True emergency
- Techniques to remove foreign bodies have been described and include endoscopic forceps, endoscopic guidance (beside scope), fluoroscopic guidance forceps **Tivers et al J Small Anim Pract 2006: 47(3); 155-9** and a Foley catheter (ET tube?) distal to FB, inflated and withdrawn **Goodnight et al JAVMA 2010; 237(6); 689-94**

Chronic coughing

- > 2mths
- Cardiac-.....let's get to that later
- Respiratory-.....go through quickly because all in text books
- Pharyngitis, tonsillitis, laryngitis, post nasal disease (nasal drip), gastro-oesophageal reflux (GERD)
- Upper airway- tracheobronchitis, bronchitis, bronchiectasis, asthma, tracheal collapse, bronchial collapse (bronchomalacia),
- Lower airway- feline allergic bronchitis (feline "asthma"), chronic bronchitis, hilar lymphadenomegaly
- Pulmonary parenchyma- pulmonary fibrosis, eosinophilic pulmonary disease, pneumonia (aspiration, bacterial, parasitic, fungal, protozoal), NCPO (electrocution, drowning, neurogenic, severe upper airway obstruction, fluid overload, ARDS)
- Neoplasia- mediastinal, laryngeal, tracheal, ribs (pleural) (absence pulmonary??)
- Pleural- chylothorax, pyothorax
- Miscellaneous- ACE inhibition (?) - one of first questions asked in humans, poorly defined in small animals

Tracheal collapse/Bronchomalacia

- Most common cause of coughing we see....
- Progressive degenerative disease of the tracheal and bronchial cartilage rings
- Hypocellularity, decreased GAGs, chondroitin and calcium content of cartilage but aetiology still poorly understood - multifactorial (genetics, inflammatory, nutritional)
- Usually dogs with TC have BM as well but can see TC or BM alone- use of term TBM (nearly 80% of dogs with TC have concurrent BM) **Madron ACVIM 2009**
- BM newly defined as *regional to diffuse dynamic airway collapse of segmental and subsegmental bronchi with associated clinical signs due to airflow limitation* **Reneiro and Masseau The Vet Journal 273: 2021**
- Usually middle age toy breeds but can be young dogs/large breeds (BM)
- Need to distinguish from other causes airway inflammation (idiopathic. CB; eosin bronchopneumonia etc) – often concurrent
- True prevalence not known but in those dogs having bronchoscopies half had some form of airway collapse (21% TC; 47% BM) **Johnson and Pollard. J Vet Intern Med 2010;24:298**
- Brachycephalic dogs (having airway examinations) 87.5% had fixed bronchial collapse **DeLorenzi et al J Am Vet Med Assoc 2009;235:835, Cote et al. JAAMA 2022; 260(13) 1**

Clinical signs

- Clinical signs well known- coughing, honking, respiratory distress –if ET- inspiratory dyspnoea, if IT- expiratory dyspnoea
- 2 differing clinical syndromes – coughing versus obstructive disease and dynamic versus fixed (or 'traditional' 71% versus 'malformation' 29%) **Weisse et al JAVMA 2019; 254:360; Bottero et al. J Vet Intern Med 2013;27:840**
- W shaped malformation recognised **Weisse et al JAVMA 2019, Weisse ECVIM 2023**
- Has implications for Rx options esp stenting
- Infections – 83% **Johnson LR, Pollard RE. J Vet Intern Med. 2010;24(2):298–305** 82% **Lesnikowski et al JVIM 2020;1** ..but....pathogenic 66% and progressive increase in % with age
- Association with laryngeal disease (30%) **Johnson LR, Pollard RE. J Vet Intern Med. 2010;24(2):298–305.**
- Association with liver disease (46% increased liver enzymes) **Bauer NB et al. J Vet Intern Med. 2006 Jul-Aug;20(4):845-9.** – likely chronic hypoxia-induced
- Association with myxomatous mitral valve disease – oh we will get to that in a separate lecture....

- Redundant dorsal membrane often contributes to pathogenesis



- Extent of RDM- bronchial involvement
- Cervical lung lobe herniation- another twist- 77% of dogs having fluoroscopy had cervical lung lobe herniation +/- extra thoracic tracheal kinking **Nafe LA ACVIM 2012, Can Vet J. October 2013;54(10):955-9.**

Grading (circa 1982)

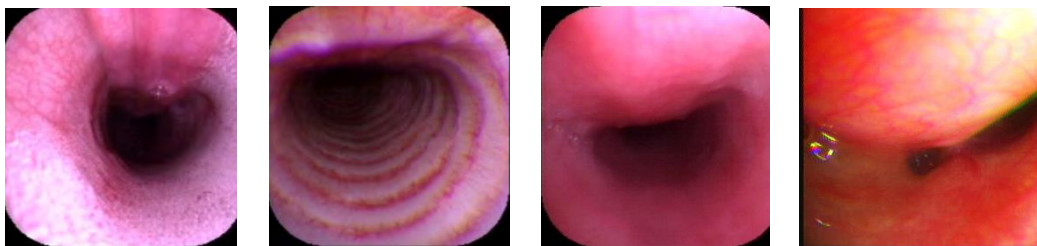
Grade I: Slight protrusion of the dorsal tracheal membrane into the airway lumen; reduction of tracheal lumen by 25 %

Grade II: Reduction of tracheal lumen by 50%.

Grade III: Reduction of tracheal lumen by 75 %

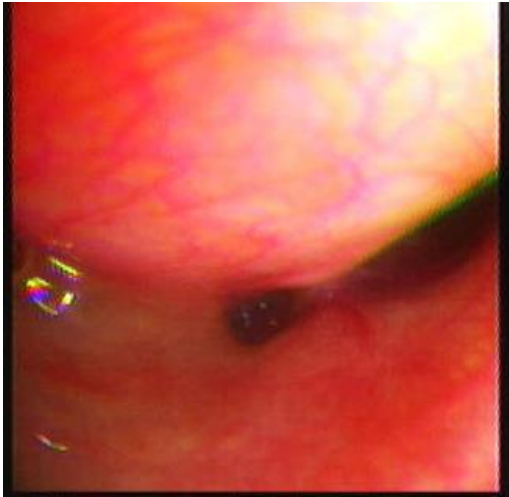
Grade IV: Tracheal rings flattened and less than 10 % of the tracheal lumen can be visualized.

- Likely superceded and only applies to dynamic/'traditional'



Diagnosis

- Signalment, history and clinical signs – usually but not always Toy breeds, clinical signs characteristic but beware concurrent disease
- Cervical and thoracic radiography- under (and over) estimates 76% sensitivity **Macready DM et al. J Am Vet Med Assoc. June 2007; 230 (12):1870-6.**
- Ultrasonography (?) **Eom et al. J Vet Sci. December 2008;9(4):401-5**
- Endoscopy - Bronchoscopy (+ laryngeal examination)
- CT – compare inhalation and expiration bronchial size
- Fluoroscopy
- Bronchoalveolar lavage



Medical treatment for tracheobronchomalacia

- Medical Mx effective approx 70% of time
- Doxycycline 10mg/kg q24h for 2-3 weeks
- Anti-inflammatories- corticosteroids (effective but...)- low dose (0.5mg/kg q12h)
- Cough suppressants- codeine, butorphanol, diphenoxylate (Lomotil), hydrocodone, dextromethorphan, tramadol, more novel Rx's such as gabapentin (!?) maropitant(?)
Grobman and Reinero J Vet Intern Med 2016 cannabis CBD versus THC Pers comm Leon Warne
- Sedatives and tranquillisers –buprenorphine, butorphanol
- Bronchodilators - theophylline, terbutaline – increasingly harder to get
- Inhalational – fluticasone (flixotide), +/- salbutamol (seretide), use of nebulisers
- Weight loss
- Use of harnesses rather than leads
- Removal allergens/smoke.
- Stanazolol **Adamama-Moraitou KK, et al. Int J Immunopathol Pharmacol. 2011;24(1):111–8**

	Drug	Dose	Route	Frequency
Sedatives	Acepromazine	0.02–0.1 mg/kg (D) [*]	IV, IM, SC	q6–8h
		0.55–2.25 mg/kg (D)	PO	q6–8h
		0.02–0.1 mg/kg (C) [†]	IV, IM, SC	q6–12h
		1–2 mg/kg (C)	PO	q6–12h
	Butorphanol	0.2–0.4 mg/kg (D)	IV, IM, SC	q2–4h
		0.55–1.1 mg/kg (D)	PO	q6–12h
		0.2–0.8 mg/kg (C)	IV, SC	q2–6h
		1.5 mg/kg (C)	PO	q6–8h
	Midazolam	0.1–0.3 mg/kg (D)	IV, IM, SC	PRN
		0.05–0.5 mg/kg (D)	IV, IM, SC	PRN
Antitussives	Trazodone	5 mg/kg (D)	PO	q12h
	Butorphanol	0.05–0.1 mg/kg	IV, SC	PRN
		0.55–1.1 mg/kg	PO	q6–12h
	Hydrocodone [§]	0.22–0.25 mg/kg	PO	q6–8h
	Codaine	1–2 mg/kg	PO	q6–12h
	Morphine	0.1 mg/kg	SC, IM	q6–12h
	Co-phenotrope (diphenoxylate + atropine)	0.3–0.5 mg/kg	PO	q12h
	Dextromethorphan	0.5–1 mg/kg (D)	PO	q6–12h
		2–4 mg/kg (C)	PO	q6–12h
Glucocorticoids	Prednisone or prednisolone [*]	0.5–1 mg/kg (D)	PO	q12–24h
		1 mg/kg (C)	PO	q24h
	Fluticasone	110–220 mcg	Aerosol	q12h
Bronchodilators	Theophylline (extended release)	10–20 mg/kg (D)	PO	q12–24h
		25 mg/kg (C)	PO	q24–48h
	Aminophylline ^{††}	10 mg/kg (D)	PO	q6–8h
		5–6 mg/kg (C)	PO	q12h
		2–5 mg/kg	IV	q6–12h
		0.25 mg/cat	SC	q6–8h
	Fenbutaline	0.025 mg/cat	PO	q12h
		0.025–5 mg/dog	PO	q6–12h
		0.05 mg/kg (D)	PO	q6h
	Albuterol	90 mcg	Aerosol	PRN
Antihistamines	Chlorpheniramine	4–8 mg/dog ^{§§}	PO	q6–12h
		2 mg/cat	PO	q12–24h
	Diphenhydramine	2–4 mg/kg (D)	PO	q6h
Expectorant	Guafenesin	3–5 mg/kg	PO	q6h
Mucokinetic	Acetylcysteine	144 mg/kg (initial dose), followed by 70 mg/kg	IV	q6–12h
NK ₁ receptor antagonist	Maropitant ^{***}	1 mg/kg (D)	SC	q24h for up to 5 days or
		2 mg/kg (D)	PO	
		1 mg/kg (C)	IV, SC, PO	q48h

^{*} Dog

[†] Cat

[§] Caution in cats.

^{**} Taper to lowest effective dose.

^{††} Administer over 30–60 min.

^{§§} Maximum dose of 0.5 mg/kg (D).

^{***} Antiemetic doses. No studies have been performed to establish an antitussive dose.

TABLE 3. Drugs and dosages to manage respiratory disease (24, 47–53).

REVIEW article

Front. Vet. Sci., 21 January 2020

Sec. Comparative and Clinical Medicine

Volume 6 - 2019 | <https://doi.org/10.3389/fvets.2019.00513>

Tracheal collapse - case 1

- Puppacino- 10 yo MN Chihuahua
- Hx- 1-2 years worsening cough, acute onset dyspnoea and collapse. Non-cardiogenic pulmonary oedema (no heart murmur)

Bronchomalacia - case 2

- Rocky - 1yo ME Fox Terrier
- 9 month history coughing- no progression
- Bronchoscopy reveals

What about when medical treatment fails or is not enough?

- Rocky (!) 9 yo Yorkie, had ophthalmology procedure, now unable to be extubated, arrested twice the night before
- Tracheal stenting- what to tell the client- selection of patients
- Younger dogs may be better suited to extra-tracheal rings (life span of stents /long term follow up not yet done)

- Failure of aggressive medical management (not just owner compliance) 71% patients remained asymptomatic 12mths after Rx started **White RAS et al JSAP 1994 35:191**
- Predominantly intra-thoracic collapse (remember exp dyspnoea) but can be used in patients with both intra-thoracic and extra-thoracic collapse
- Other factors- small size and aged, those patients unable to be extubated*
*our experience
- Main factor may be obstructive/fixed or 'malformation' (honking, dyspnoea) versus dynamic or 'traditional' (coughing) **Weisse ECVIM 2023**

Tracheal stenting - what to tell the client

- Concurrent main-stem bronchial collapse (remember nearly 80% of dogs with TC have concurrent BM **Madron ACVIM 2009** - but not necessarily a contra-indication to tracheal stenting **but** is a poor prognostic sign and likely will result in persistent cough that must be treated with medical management despite stenting (50% clinical improvement versus 70-90%)
- Stenting is essentially palliative and will not cure their dog and cough is likely to persist (at least for 'traditional' TBM)
- Following stenting
 - 89% had improvement in goose honking and noisy breathing **Weisse et al 2019**
 - 84% had improvement in dyspnoea **Weisse et al 2019**
 - but (only) 43% in coughing **WSAVA 2019**

Tracheal stenting - outcomes and what can go wrong

- Clinical improvement rates in 75–90% of animals treated with self-expanding, intra-luminal stainless steel stents have been reported **Weisse C. WSAVA/FECAVA /BSAVA World Congress 2012; Weisse et al 2019; Weisse ECVIM 2023**
- Immediate complications were mostly minor, peri-operative mortality rate of approximately 10%.
- Stent likely not associated with infection **Lesnikowski et al JVIM 2020:1**
- Stent migration (occurred if poor measuring or stent selection) or poor placement (technique), probably overstated **Weisse ECVIM 2023**
- Late complications included stent shortening; excessive 'granulation*' tissue; progressive tracheal collapse; stent fracture (persistent coughing, or stent not maximally expanded)* likely inflammatory and steroid responsive
- 3 monthly radiography after stent placement

Tracheal stenting - when I don't want to do it

- When the client comes in and says "I don't want to give my dog medication" (anymore)
- When the client comes in and says "I don't want my dog to cough anymore"
- When the bronchoscopy show severe bronchomalacia with mild tracheomalacia and tracheal collapse (if both may help)
- On a Friday night or weekend- not always possible but better as an expedient elective
- When we can't extubate a patient – first one needed to be BUT stress, decisions, cost, equipment

Tracheal stenting - procedure

- Nitinol - (titanium, nickel) 2 critical properties- super-elasticity and shape memory
- Pre-stent placement and measurement
- Stent deployed through the endotracheal tube through a bronchoscope adaptor so that the patient can remain on the anaesthesia circuit



Take home messages

- Likely need to differentiate between dynamic/traditional and obstructive/malformation to provide prognosis associated with treatment
- Clinical signs useful – coughing versus dyspnoea/honking
- Stenting is great but medical treatment 70% effective (in dynamic disease) and does have limitations and risks

Pulmonary hypertension

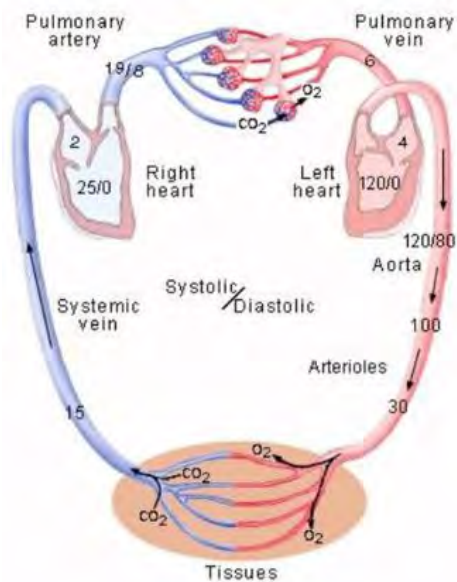
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Pulmonary hypertension

- Pulmonary hypertension – one of my favourite conditions
- Combination of internal medicine and cardiology
- Normal pulmonary pressures 25/10mmHg (systolic/diastolic)



- Pulmonary Hypertension > 30/20mmHg
- Old classification of pulmonary hypertension

Pulmonary arterial hypertension	Congenital left-to-right shunt	Atrial septal defect Ventricular septal defect Patent ductus arteriosus Arteriovenous malformation
	Pulmonary disease	Chronic obstructive pulmonary disease Interstitial pulmonary fibrosis Neoplasia Reactive pulmonary artery vasoconstriction
	Thrombotic or embolic disease	Neoplasia Immune-mediated hemolytic anemia Protein-losing disease Hyperadrenocorticism Disseminated intravascular coagulation Sepsis Trauma Cardiac disease Recent surgery Heartworm disease
	High altitude disease	
	Parasitic disease	<i>Dirofilaria immitis</i> <i>Angiostrongylus vasorum</i>
	Necrotizing vasculitis/arteritis	
	Idiopathic	
Pulmonary venous hypertension	Left heart disease	Mitral valve disease Left ventricular failure Pulmonary venous stenosis Myocardial disease

- The 2020 ACVIM Consensus Statement on PH in dogs proposed an alternative classification method. Usefulness in cats with PH is unclear.



CONSENSUS STATEMENT | [Open Access](#) |

ACVIM consensus statement guidelines for the diagnosis, classification, treatment, and monitoring of pulmonary hypertension in dogs

Carol Reinero , Lance C. Visser, Heidi B. Kellihan, Isabelle Masseau, Elizabeth Rozanski, Cécile Clercx, Kurt Williams, Jonathan Abbott, Michele Borgarelli, Brian A. Scansen

First published: 17 February 2020 | <https://doi.org/10.1111/jvim.15725> | Citations: 109

- Group 1:** Pulmonary arterial hypertension
- Group 2:** Left-sided heart disease
- Group 3:** Respiratory disease/hypoxia
- Group 4:** Pulmonary emboli/pulmonary thrombi/pulmonary thromboemboli
- Group 5:** Parasitic disease, e.g. *Dirofilaria*, *Angiostrongylus* spp.
- Group 6:** Multifactorial disorders or unclear mechanisms

Pulmonary hypertension - signalment, history and PE

- Signalment – any breed or age and no sex predilection
- History and clinical signs - can present with a variety of clinical signs, depending on the primary aetiology of PH or directly from the PH. These signs include cough, respiratory distress, lethargy, syncope, and exercise intolerance.

- Physical examination – once again depends on the underlying cause- PH patients may have mitral murmurs, tricuspid murmurs, mitral and tricuspid murmurs, a split-second heart sounds, pulmonary crackles, cyanosis, and ascites.

Pulmonary hypertension - diagnosis

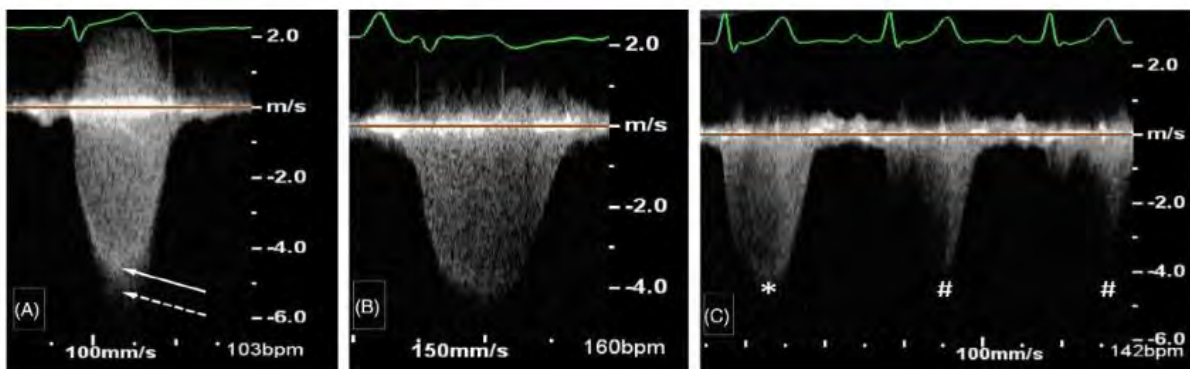
- How do we diagnose
- Gold standard invasive haemodynamic assessment with right heart catheterisation which helps determine right atrial, right ventricular, pulmonary artery and pulmonary capillary wedge pressures.
- Rarely do this except during catheter interventional procedures
- Clinical pathology may support underlying causes - polycythaemia if R → L shunting, prothrombotic if PTE
- Thoracic radiography +/- CT



- Echocardiography

TABLE 2 Echocardiographic probability of PH in dogs

Peak tricuspid regurgitation velocity (m/s)	Number of different anatomic sites of echo signs of PH ^a	Probability of PH
≤3.0 or not measurable	0 or 1	Low
≤3.0 or not measurable	2	Intermediate
3.0 to 3.4	0 or 1	Intermediate
>3.4	0	Intermediate
≤3.0 or not measurable	3	High
3.0 to 3.4	≥2	High
>3.4	≥1	High



- Cardiac bio-markers - proBNP

- Thoracic radiography may not be diagnostic of PH but can help determine the underlying aetiology of PH (for example left-heart disease, interstitial pulmonary fibrosis, neoplasia, PDA)
- Secondly, thoracic radiography help support a diagnosis of PH (i.e., right heart enlargement, main pulmonary artery enlargement, branch PA enlargement, tortuous and blunted PAs).
- Non-cardiogenic pulmonary oedema can also form as a result of PH. The distribution of oedema, in these cases, is typically patchy and often associated with dyspnoea and syncope.
- Echocardiography can estimate the systolic, diastolic, and mean pulmonary arterial pressure.
- The peak systolic TR velocity and gradient reflects the estimated peak systolic right ventricular pressure and is used to classify PH as mild (≥ 2.8 to < 3.5 m/s, ≥ 31.4 to < 50 mm Hg), moderate (3.5 – 4.3 m/s, 50 – 75 mm Hg), or severe (> 4.3 m/s, > 75 mm Hg).
- There can be problems with the estimation of the PAP based on TR gradients, and this measurement should be used in conjunction with other echo parameters of PH. Other parameters that echo can evaluate include: RV morphology, septal flattening, pulmonary artery systolic flow profiles, PA systolic time intervals, Tei index of myocardial performance, systolic time intervals, RV tissue Doppler, tricuspid annular plane systolic excursion and the right PA relative area change/distensibility index.



TABLE 3 Anatomic sites of echocardiographic signs of PH used to help assess the probability of PH in dogs

Anatomic site 1: Ventricles	Anatomic site 2: Pulmonary artery	Anatomic site 3: Right atrium and caudal vena cava
Flatting of the interventricular septum (especially systolic flattening)	Pulmonary artery enlargement (PA/Ao >1.0 ^{17,18})	Right atrial enlargement ^{19,20}
Underfilling or decreased size of the left ventricle ^a	Peak early diastolic PR velocity >2.5 m/s	Enlargement of the caudal vena cava ¹⁹
Right ventricular hypertrophy (wall thickening, chamber dilation, or both) ^{19,21}	RPAD index <30% ^{17,22}	
Right ventricular systolic dysfunction ^{19,23-27}	RV outflow Doppler acceleration time (<52-58 ms) or acceleration time to ejection time ratio (<0.30) ^{17,18,28} Systolic notching of the Doppler RV outflow profile (caution: false positives are possible)	

Abbreviations: PR, pulmonary regurgitation; RPAD, right pulmonary artery distensibility; RV, right ventricular.

^aNot applicable for dogs with group 2 PH due to the confounding effects of LV remodeling secondary to LHD.

Echocardiography – newer parameters

- A recent study suggests that PV/PA can be useful as an additional, non-invasive, and indirect variable to identify precapillary PH in dogs.
- PV/PA (2D) was identified as the strongest predictor moderate PH (TRPG > 50 mmHg) using a cutoff value of < 0.70 (sensitivity = 96%, specificity = 82%).
- Study documenting PH in WHWT showed a high prevalence of PH at diagnosis in WHWTs affected with CIPF and also highlighted the utility of PV/PA as a non-invasive surrogate for assessment of PH in this population.
- Echocardiography – newer parameters (cont)
- Right pulmonic artery distensibility index
- Left ventricular eccentricity index.
- Cardiac biomarkers
- NT-proBNP has been shown to be increased in dogs in the presence of PAH and PVH.
- NT-proBNP can be helpful in supporting a diagnosis of PH in a dog with precapillary PH, especially when an estimated PAP is difficult to interpret.

Pulmonary Hypertension - treatment

- The goals of treatment are to:
 1. treat the underlying disease.
 2. promote PA vasodilation.
 3. suppress cellular proliferation.
 4. improve cardiac output by decreasing PVR and
 5. enhance RV inotropy.
- The ideal PH therapy would decrease PVR, spare the systemic circulation, and increase RV inotropy.

PDE- PDE-5 inhibitors

- Sildenafil (Viagra, Revatio), tadalafil (Cialis), vardenafil, udenafil, avanafil are orally active, highly selective PDE-5 inhibitors.
- Multiple studies have demonstrated the benefits of sildenafil in people and dogs with PH.
- Sildenafil appears to produce beneficial effects in PH by multiple mechanisms (i.e., improvement of RV and LV function, ventricular interdependence, pulmonary lymphatic drainage) but the primary mechanism operative in PH patients appears to be direct pulmonary artery vasodilation.

- Sildenafil responsiveness is variable in dogs with respiratory-associated PH, but improved QOL was demonstrated in dogs surviving >1 month, and long-term survival was noted in some cases.
- The primary mechanism operative in PH patients appears to be direct pulmonary artery vasodilation.
- Sildenafil should be used cautiously in cases of left-heart disease. If the PA pressure falls below the left atrial pressure, with sildenafil, forward blood flow from the right heart to the left heart can become severely impaired or the pulmonary capillaries can flood because of higher pulmonary venous pressures, resulting in pulmonary oedema.
- Tadalafil has also been described in clinical and experimental settings.
- Pimobendan and levosimendan are calcium-sensitizing agents and PDE-3 inhibitors.
- PDE-3 has activity at the level of large and small (resistance) PAs, whereas PDE-5 exerts its activity in primarily large pulmonary arteries. PDE-3 inhibitors promote PA vasodilation via enhancement of cAMP-dependent/adrenergic relaxation.
- The dual action of PDE-3 inhibition and the positive inotropic effects of calcium sensitization may provide some attenuation of PH, especially in PH secondary to left-sided heart disease.
- Ultimately, the RV needs to be supported if it fails and pimobendan may be beneficial as a RV-positive inotrope.

Endothelin antagonists

- Bosentan, sitaxsentan, and ambrisentan are endothelin antagonists that are given orally, yet have historically been cost prohibitive (i.e., > \$40,000/year).
- Prostacyclin Analogs
- Epoprostenol, treprostinil, and iloprost are prostacyclin analogs that are administered as continuous-rate infusions, frequent SQ injections, or frequent inhalations (i.e., 6–12 x/day).

L-Arginine

- L-arginine is an amino acid that results in NO and L-citrulline production. There is the potential for L-arginine supplementation to improve PH by increasing NO bioavailability and potentiating the effects of PDE-5 inhibition.

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Pulmonary hypertension - let's look a little closer at the 2020 consensus statement

- Changed focus so that parameters broadened to multiple anatomical sites.
- Continued to use TVR velocity but recognized the limitations of using solely.
- Beyond my remit- but we use as many of the echocardiographic markers at multiple sites
- Also provides useful; algorithms that are beyond the scope of lecture to go into detail about.

Pulmonary hypertension - case 1

- Honey
- Signalment -15 yo FS Chihuahua
- Hx - Recently seen by SASH IM and Surgery for chronic enteropathy and bilateral adrenal masses identified at RV. Hypocobalaminaemia identified. HyperA diagnosed 2 years prior and treated with trilostane
- Rapid breathing started today, owner very aware of this due to other dog passing away with pulmonary hypertension last year.
- Abdominal distension, Dxed at ref vet as modified transudate
- Given 5mg Trilostane this morning.
- No vomiting or diarrhoea in the past week
- No excessive drinking in the past week
- Recent weight gain - usually 3.2kg, now 3.6kg
- PE - HR/PR 112, auscultation grade 4 murmur, loudest on the right, pulse quality normal, rhythm regular. ECG NSR. Jugular pulses.
Respiratory: RR 36 (arrived in O2 box from RV), RE very mild increase in expiratory effort, auscultation normal. SpO2 95-100% in O2, 89-93% in room air.
Abdomen: Soft, non-painful, no appreciable masses on palpation. Moderate abdominal distension, 'pot-bellied' appearance.
- Echocardiographic findings
 - Right atrial enlargement with interatrial bowing
 - TC regurgitation > 3.4m/s
- Echocardiographic findings
 - Pulmonic insufficiency > 2.2m/s
 - Pulmonary artery enlargement

1. Myxomatous Mitral and Tricuspid Valvular Degeneration equivocal B1
 2. Echocardiographic criteria for pimobendan benefit met based on right-sided changes and pulmonary hypertension
 3. Echocardiographic parameters not suggestive of increased left atrial pressure consistent with congestive heart failure (based on PV:PA, Mitral E max and E:A ratio)
 4. Echocardiographic parameters were suggestive of severe pulmonary hypertension (based on TC, PI regurgitant velocity and RPA distensibility index) based on the estimated modal TR velocity as per the ACVIM guidelines on Pulmonary hypertension.
- Diagnosis
 - Myxomatous Mitral and Tricuspid Valve Disease equivocal stage B2
 - sMINE score 5 Moderate
 - Pulmonary hypertension severe
 - DDx PTE (especially given co-morbidities), other causes pulmonary hypertension
 - CT findings
 - Extraaxial space-occupying lesion right occipital lobe, consider meningioma, others less likely
 - Otitis media bilateral
 - PTE left pulmonary artery
 - Bilateral enlarged adrenals and enlarged pituitary gland, consider macroadenoma with Cushings disease. Right adrenal with suspected adhesions to the cd vena cava.
 - Splenic nodules, consider lipoma. Soft tissue attenuating nodule, consider EMH, primary or metastatic neoplastic disease possible.
 - Bilateral chronic renal disease
 - Treatment
 - Pimobendan 0.625mg PO q8hr
 - Sildenafil 3.125mg (0.79mg/kg) PO q8hr
 - Rivaroxaban 5mg (1.26mg/kg) PO q24hr
 - Butorphanol 0.2mg/kg IV PRN
 - Trazodone 25mg PO PRN for anxiolysis
 - Outcome
 - mth later, echocardiography revealed persisting moderate pulmonary hypertension; but decreasing size of her right heart
 - Clinically doing well

Pulmonary hypertension - case 2

- Pablo
- Signalment - Age 14 years 2 months Sex Male Neutered Cross Breed
- Hx – Previously diagnosed with MMTVD stage B2 and bronchomalacia.
- Has been going OK
- No further episodes
- Three collapsing episodes, gets stiff and after 5-10 seconds due to excitement.
- Coughing been controlled with codeine.
- Some weight loss
- Appetite good
- No vomiting or diarrhoea
- Sleeping respiratory rate <32
- Current medication
 - Pimobendan 2.5mg q12h
 - Codeine 1mg/kg PO Q8-12hrs PRN x 1 week as required.

- PE - Cardiac auscultation revealed a grade 4/6 systolic murmur. Bilateral murmur with increased grade now on the right Good pulse pressure. HR=PR=132 as for health status. No arrhythmia
- Initial Assessment – Syncope may be due to cough syncope (coughing combined with MMTVD), pulmonary hypertension, episodic cardiac arrhythmias, left atrial tear.
- Plan
- Repeat echocardiography.
- Echocardiography reveals
 - Echocardiography reveals emergent r-CHF
- Assessment
 - Echocardiographic parameters were suggestive of moderate pulmonary hypertension (based on TC regurgitant velocity) based on the estimated modal TR velocity as per the ACVIM PH Consensus Statement guidelines. Pulmonary hypertension in dogs is most commonly secondary to primary respiratory disease (e.g., chronic bronchitis, pulmonary fibrosis, or other forms of pulmonary interstitial disease). This is the most likely cause for Pablo's syncopal episodes.
- Treatment
 - Start Furosemide (frusemide) 2-6mg/kg q8-12h = 10mg q12h PO
 - Start Sildenafil 0.5-1.0mg/kg q8h (upper 0.5-2.7mg/kg VIN q8-24h) = 12.5mg q8h
 - Continue codeine 1mg/kg PO Q8-12hrs PRN x 1 week as required
 - Continue to monitor sleeping respiratory rate (and note whether there is a decrease since starting furosemide (frusemide))
 - Please take Pablo to your vet in 7-14 days to have his kidney markers checked to make sure he is coping well with the diuretic treatment.
 - Reassess 3 months and consider Spironolactone 2.0mg/kg q12-24h (ACVIM)

Pulmonary hypertension - case 3

- Benji
- Signalment - 1yo MN Cavoodle
- Hx - Presenting for episodes of collapse.
- Since last visit with neurology on 13.2.23, Benji had 2 episodes where his back legs drop, has really heavy breathing, tongue gets really pale (video seen by PB/BC). O reckon it's generally when he is aroused/excited (when mum gets home, he'll jump and run around).
- In total has had 5-6 of these episodes over the last month, prior to this nothing has been noted. O notes he doesn't lose consciousness, he just falls to the ground and struggles to get up. Each episode lasts about 1-2 minutes, O sits down with him to calm him down and settle him down. Afterwards O note his tongue will take a while to get back its normal colour, however after 5 minutes he's back to his usual self. No heart murmur/abnormal heart rhythm has been noted by vets previously according to O.
- Otherwise, Benji is a healthy dog - eats and drinks well, no vomiting or diarrhoea. Benji gets fed twice a day, usually just kibble
- PE - Cardiac auscultation revealed no systolic murmur. Good pulse pressure. HR=PR= as for health status. No arrhythmia Challenging to determine differential cyanosis with nail colour.
- Assessment
 - Collapsing episodes
 - In this particular case, the collapsing episodes are suggestive of acute transient HL paresis.

- May be cardiovascular, neurological less likely (previous neuro assessment NSA), metabolic (hypoglycaemia, pheochromocytoma), vascular or vagally induced (especially given that the patient seemed to retch prior to observed event)
- Cardiac
 - There is no evidence of any cardiac disease based on PE (no cardiac murmur or arrhythmia) but cannot exclude reversed patent ductus arteriosus in which the murmur may be low-grade or absent or pulmonary hypertension.
 - Echocardiography would further define, exclude structural heart disease and exclude or define possible pulmonary hypertension
- Plan
 - Thoracic radiography
 - Echocardiography
 - Electrocardiography
 - +/- Holter
- Thoracic radiography revealed
 - Cardiac changes may be consistent with right-sided cardiomegaly and distension of the main pulmonary artery - rotation may artifactually exaggerate these changes.
DDx: Tricuspid insufficiency, pulmonary hypertension (R>L shunt, dirofilariasis), Pulmonic stenosis
- Echocardiography reveals
 1. Moderate to severe right ventricular concentric hypertrophy in the absence of a right ventricular outflow tract obstruction such as pulmonic stenosis.
 2. Pulmonary hypertension, likely severe
 3. Probable reversed Patent ductus arteriosus
 - ECG shows sinus rhythm but deep S waves and electrocardiographic features consistent with R cardiomegaly.
 - PCV 65/67
- Assessment
 - Overall the leading DDx is reversed patent ductus arteriosus versus primary pulmonary hypertension. Whilst the former is suspected on echocardiography, the mild tongue cyanosis is atypical as one would expect differential cyanosis and a bubble contrast study is recommended to confirm if thoracic radiography is confirmed to be unremarkable.
- Contrast echocardiography reveals.
- Assessment
 - Confirmed reversed PDA
- Treatment
 - Start Sildenafil 20mg 0.5-2.5mg/kg q8h
 - Repeat echocardiography in 7 days to determine whether L-> R shunting has occurred
 - If not continue sildenafil and then phlebotomy on as necessary basis
 - Monitor PCV every 4 weeks
 - Maintain PCV between 60-65%
 - Phlebotomy as needed if hydroxyurea cannot maintain the desired PCV (low 60s)

Take home messages

- Have 'on radar' for patients with collapsing episodes.
- Difficult to diagnose so need high index of suspicion.
- Be thorough – a diagnosis through determination of underlying disease.

The coughing dog with a heart murmur

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I'd like to begin by acknowledging that the presentation is being delivered from the lands of the Kabi Kabi and Jinibara peoples and pay my respects to Elders past, present and emerging.

These notes may contain opinions that are that of the author.

Tracheal collapse/Bronchomalacia

- Progressive degenerative disease of the tracheal and bronchial cartilage rings
- Hypocellularity, decreased GAGs, chondroitin and calcium content of cartilage but aetiology still poorly understood - multifactorial (genetics, inflammatory, nutritional)
- Usually dogs with TC have BM as well but can see TC or BM alone- use of term TBM (nearly 80% of dogs with TC have concurrent BM) **Madron ACVIM 2009**
- BM newly defined as *regional to diffuse dynamic airway collapse of segmental and subsegmental bronchi with associated clinical signs due to airflow limitation* **Reneiro and Masseau The Vet Journal 273: 2021**
- Usually middle age toy breeds but can be young dogs/large breeds (BM)
- Static v dynamic – mostly dynamic but some have both (at least with BM) **Bottero et al. J Vet Intern Med 2013;27:840**
- Need to distinguish from other causes airway inflammation (idiopathic. CB; eosin bronchopneumonia etc) – often concurrent
- True prevalence not known but, in those dogs having bronchoscopies half had some form of airway collapse (21% TC; 47% BM) **Johnson and Pollard. J Vet Intern Med 2010;24:298**
- Brachycephalic dogs (having airway examinations) 87.5% had fixed bronchial collapse **DeLorenzi et J Am Vet Med Assoc 2009;235:835; Cote et al JAAMA 2022; 260 (13) 1**
- Clinical signs well known- coughing, honking, respiratory distress –if ET- inspiratory dyspnoea, if IT- expiratory dyspnoea
- 2 differing clinical syndromes – coughing versus obstructive disease
- Association with laryngeal disease (30%) **Johnson LR, Pollard RE. J Vet Intern Med. 2010;24(2):298–305.**
- Association with liver disease (46% increased liver enzymes) **Bauer NB et al. J Vet Intern Med. 2006 Jul-Aug;20(4):845-9.** – likely chronic hypoxia-induced
- Association with myxomatous mitral valve disease.....

.....which is really what this lecture is about.....

What about our/my real world?

- Classic scenario, a dog comes in with a cough and a heart murmur
- When I first graduated.....



- Cardiac was felt to be the most common cause of coughing i.e. CHF

- Start diuretics and job done
- But a clue to the future given that even James Herriot (I hope you remember who he is) said “Like all old dogs with a chronic heart weakness, he had an ever-present bronchitis” **Pers comm (well I read his books ;)**

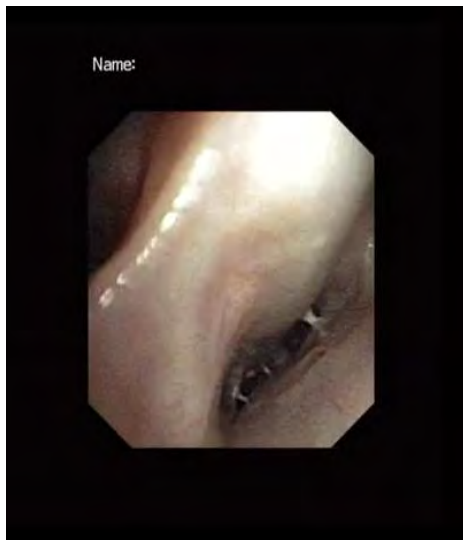
So why do we see coughing so commonly in heart disease?.....When dogs with a murmur, cough

- Pulmonary oedema? – well....
 - Cough reflex less convincing evidence for receptors deep in the resp tract
 - Not reported in human texts unless severe oedema nor seen in cats (at least not at “questioning rads” level)
 - Often no coughing in NCPO?
 - Less commonly see coughing in large dogs with heart disease unless fulminant pulmonary oedema/CHF
 - Coughing due to pulmonary oedema is always acute....not chronic unless superimposed
- Previous study showed CHF **not** associated with coughing in dogs with MMVD **Ferasin et al. J Vet Intern Med 2013; 27(2): 286-92**
- When dogs with a murmur, cough
- Used to think large L atrium compresses MSB – tempting maybe, maybe not and unlikely without concurrent disease - Hose and balloon analogy **Rishniw VIN**
- Airway collapse independent of left atrial size – looked at dogs with LAE and no LAE and found same incidence of bronchial collapse **Singh M et al. J Vet Intern Med. 2012 Mar-Apr ;26(2):312-9.**
- Left mainstem bronchus able to be seen radiographically clearer with LAE? **Kittleson VIN**



Compression mainstem bronchus

- However, now a recent study (2021) has identified a relationship between LA/Ao ratio measured echocardiographically (and VHS measured radiographically) and bronchial narrowing, supporting the contribution of cardiomegaly on airway collapse and heart size exacerbation of cough in dogs with heart murmur and cardiomegaly. Results confirmed a gradient association between heart size (estimated by vertebral heart scale and left atrial-to-aorta ratio) and bronchial narrowing, with the bronchi becoming narrower as heart size increased. **Lebastard et al. J Vet Intern Med. 2021;35:1509–1518).**
- When dogs with a murmur, cough
- It is very likely that any contribution of left atrial dilation on airway collapse is dependent on concurrent underlying bronchomalacia but the degree of left atrial dilation may exacerbate such a collapse. **But studies focused on just this pending**



- Confirmed what we suspected
- When dogs with a murmur, cough
- Nice because we all felt that this occurred....
- And somewhat interestingly...
- The opposite can also occur- stent MSB compresses LA causing emergent CHF
Dengate A et al J Small Anim Pract April 2014: 55:225
- When dogs with a murmur, cough
- Why then the connection between **chronic** coughing and heart disease..... James Herriot was right!
- Bronchomalacia and myxomatous mitral valve disease have a common signalment - small breed, middle age to older dogs
- Tempting to think of a common pathogenesis of cartilage defect

So how to distinguish- Coughing in cardiac patients- when is it heart (CHF) or DAD/TBM?

- Hx is important (normal SRR); PE is important (slow HR/sinus arrhythmia)
- When dogs with a murmur, cough
- But what is the 'gold standard' for Dxing I-CHF?
- SRR? – possibility highly sensitive but poor specificity (many other things cause increased SRR- stress, pain, pyrexia, other pulmonary disorders)
- Rads? – Not very sensitive, can define late pulmonary oedema (but still do not know whether cardiogenic or not)
- Thoracic u/s/VetBlue/TFAST? – B lines (rockets) can add support but not always specific
- When dogs with a murmur, cough
- Specialist echo? – probably best we have (my bias) - markers of increased LA pressure (mitral E max, E:A, PV:PA), grading of severity
- But....(I can hear you say) the dog responded to frusemide (furosemide) and/or pimobendan so it was CHF ..well.....
- Frusemide is antitussive **Sant'Ambrogio et al Eur Respir J. September 1993;6(8):1151-5.**
- Frusemide is a bronchodilator **Kian-Chung Ong et al Am J Respir Crit Care Med. May 2004;169(9):1028-33.**
- Pimobendan- effect on pulmonary hyperT/vasc **Atkinson KJ et al J Vet Intern Med 23(6) 2009 Nov-Dec: 1190-6** and bronchodilation **Shiga et al. Cardiovasc Drugs Ther. May 2002 16(3):259-63**
- Why is this important?!.....impact of premature diuretic treatment and activation of RAAS

- Please resist the temptation to start/increase frusemide based on coughing; ONLY if SRR increases or better still radiographic evidence of pulmonary oedema and then rigorous trial (next slide)
- Words that I never want to see again in a history
- So the only place for a frusemide trial
 - > document SRR is increased and subsequent normalisation of SRR in response to appropriate dose frusemide or
 - > document radiographic response to frusemide
 - > **not coughing**

My cardinal coughing commandments (or 7 breathly sins)

1. Thou shalt not ignore the grade of the heart murmur- if soft murmur very unlikely cardiac cough (talking about small dogs exception larger breeds – DCM)
2. Thou shalt not ignore the heart size- heart failure in small dogs must be associated with significant atrial enlargement (except acute rupture chordae tendinae)
3. Thou shalt not ignore HR and SRR- When in doubt SHR and SRR (and remember vagal tone).
4. Thou shalt not ignore thoracic radiography- pulm oedema, PV>PA but be careful- recent trick – lateralisation of oedema when acute or eccentric jet Diana et al JAVMA 2009; 235:1058
5. Thou shalt not over-interpret pulmonary crackles- hallmark oedema? More likely mucus in airways (bronchitis)
6. Thou shalt not forget to use a retrospectroscope- if coughing is chronic (and no Rx) unlikely cardiac. If coughing patient has survived for months without frusemide, or been on for a long time without titrating upward, it's probably not cardiac
7. Thou shalt consider echocardiography – ideally will define advanced disease and markers of increased left atrial pressure (increased PV:PA ratio, increased mitral E max and increased E:A ratio)

How to assess whether a heart is enlarged

- Eyeball – not good breed differences
- VHS Buchanan heart score
- LAS Left atrial size
- Modified LAS Modified left atrial size
- Thoracic inlet heart size

Case report 1

- Pablo
- Signalment -Age 14 years 2 months Sex Male Neutered Cross Breed
- Hx - Pablo is presenting for acute coughing spasms tonight – low grade chronic coughing 3 months
- Pablo was previously diagnosed with a heart murmur in Sept last year
He was evaluated at that time as an outpatient by a cardiologist (MMTVD B2) and started pimobendan. No re-evaluation since. He woke up very early this morning and was coughing and gagging for 30s at a time, occurred 2-3 times over a 40min period. He has had intermittent coughing spasms very intermittently, not like this - usually after eating. Owner has been keeping track of resting respiratory rate (<20)
No other known medical issues
No recent bloodwork
Eating and drinking well, no vomiting and diarrhoea
- PE - Cardiac auscultation revealed a grade 4/6 systolic murmur. Good pulse pressure. HR=PR=132 as for health status. No arrhythmia
- Initial Assessment
- Plan - Echocardiography, Thoracic radiography, SBP

- Echocardiography reveals:
 1. Myxomatous Mitral and Tricuspid Valvular Degeneration stage B2 (severe remodelling)
 2. Echocardiographic criteria for pimobendan benefit previously met (based on LA:Ao, nLVIDd EPIC criteria)
 3. Echocardiographic parameters not suggestive of increased left atrial pressure consistent with congestive heart failure (based on PV:PA, Mitral E max and E:A ratio)
 4. Echocardiographic parameters were suggestive of moderate pulmonary hypertension (based on TC regurgitant velocity) based on the estimated modal TR velocity as per the ACVIM Pulmonary hypertension
 5. MINE score is 11 which is indicative of severe disease.
 6. Progression since last assessment
Oct 2022 -> June 2023
LA 3.1cm -> 3.7cm
LA:Ao 2.2 -> 2.46
LVIDd 3.70cm -> 4.39
LVIDs 1.80cm -> 1.98cm
DIAGNOSIS:
Myxomatous mitral and tricuspid valvular disease stage B2 (severe)
MINE score 11 Severe
- Thoracic radiography reveals thoracic radiography did not support current pulmonary oedema. There was a significant bronchial pattern suggestive of chronic bronchitis.
- Assessment- The combination of a cough that has improved/resolved after antibiotics; no radiographic evidence of pulmonary oedema at the time of the cough, normal sleeping respiratory rate and lack of echocardiographic markers for increased left atrial pressure confidently exclude left-sided congestive heart failure. However, degree of LAD combined with likely bronchomalacia suggests compression of mainstem bronchus is likely.
- Treatment:
 - Continue current Rx
 - Pimobendan
 - Continue close monitoring sleeping respiratory rate
 - Reassess with repeat Echocardiography in 6 months unless clinically indicated sooner.
 - Consider cough suppressive treatment as necessary

Case report 2

- Stick
- Signalment - 9 years 4 months 3 days, 25.10kg FS Whippet X
- Hx- Heart murmur detected last visit 2 weeks, not present last November
- Coughing present from late April
 - Low grade morning and night
 - Terminal retch
 - Since April possible slight progressing
- Sleeping respiratory rate 30 and unchanged since starting furosemide (frusemide)
- Cough has changed in nature since starting furosemide (frusemide)
- Appetite good
- No vomiting no diarrhoea
- Urinating normally/ increased since furosemide (frusemide)
- Current medications
 - furosemide (frusemide) 40mg q12
- PE - Cardiac auscultation revealed grade I/VI systolic murmur. Good pulse pressure. HR=PR= as for health status. Sinus arrhythmia
- Assessment
- Plan - Echocardiography, Thoracic radiography

- Echocardiography revealed:
 1. Structurally normal heart
 2. Echocardiographic criteria for pimobendan benefit not met (based on LA:Ao, nLVIDd EPIC criteria)
 3. Echocardiographic parameters not suggestive of increased left atrial pressure consistent with congestive heart failure (based on PV:PA, Mitral E max and E:A ratio)
 4. Echocardiographic parameters for pulmonary hypertension could not be assessed due to lack of measurable tricuspid regurgitation as per the ACVIM Pulmonary hypertension
- DIAGNOSIS:
Structurally normal heart
- Radiography revealed no significant abnormalities
- Assessment - Based on our findings today, the coughing is not due to cardiac disease and the current furosemide (frusemide) treatment can be stopped. The coughing is most likely due to concurrent airway disease with viral disease being possible given the duration.
- Further investigation if the clinical signs of coughing recur or progress without a concurrent increase in sleeping respiratory rate or radiographic evidence of pulmonary oedema would include bronchoscopy +/- BAL.
- In the interim, it is reasonable to treat the coughing empirically.
- Plan:
 - Cease furosemide (frusemide) whilst monitoring SRR
 - Start doxycycline 250mg q24h for 10 days
 - Start Codeine 30mg every 8-12 hours IN ONE WEEK
 - If coughing persists or progresses with a normal sleeping respiratory rate, consider further investigation as thoracic CT, bronchoscopy and BAL
 - Owner to report progress 2 weeks

Case report 3

- Bella
- Signalment - 10 years 6 months, FS CKCS
- Hx - Coughing for last month and has settled down with medication
- Sleeping respiratory rate 24 prior to seeing ref vet and was 20 last night
- Heart murmur was detected at around time of coughing
- Heard fluid but not sure whether radiographs showed fluid
- Started frusemide and pimobendan
- Otherwise well, cough has improved slightly
- Gained weight recently
- Appetite good
- No vomiting apart from single episode a few weeks ago No diarrhoea
- Current medications furosemide 10mg q24h pimobendan 2.5mg q12h
- PE - Cardiac auscultation revealed grade IV/VI systolic murmur. Good pulse pressure. HR=PR= as for health status. Sinus arrhythmia
- Assessment
- Plan - Echocardiography, Thoracic radiography
- Echocardiography revealed:
 1. Myxomatous Mitral and Tricuspid Valvular Degeneration stage B1 (no cardiac remodelling)@
 2. Echocardiographic criteria for pimobendan benefit not met (based on LA:Ao, nLVIDd EPIC criteria)
 3. Echocardiographic parameters not suggestive of increased left atrial pressure consistent with congestive heart failure (based on PV:PA, Mitral E max and E:A ratio)
 4. Echocardiographic parameters were not suggestive of pulmonary hypertension (based on TC regurgitant velocity) based on the estimated modal TR velocity as per

the ACVIM PH Consensus Statement guidelines.

DIAGNOSIS:

Myxomatous mitral and tricuspid valvular degeneration stage B1@

@Possible effect of pimobendan as per EPIC trial 2

- Radiography revealed no evidence pulmonary oedema
- Mild bronchial pattern and left mainstem bronchial compression
- Assessment
- Echocardiography showed myxomatous mitral valvular degeneration as expected but the cardiomegaly is mild (indeed the LVIDS and LVIDD are within normal limits). However, this is confounded by the current diuretic administration and likely underestimates real cardiac size. Despite this effect, it is very unlikely that the severe remodelling that accompanies left-sided congestive heart failure would normalise and so the echocardiographic changes today likely exclude previous left-sided congestive heart failure (as does the sleeping respiratory rate at that time)
- Other parameters of echocardiographic evidence for congestive heart failure such as PA:PA, Mitral E max and Mitral E:A ratio are not supportive for congestive heart failure.
- Given that previous left-sided congestive heart failure is unlikely and given that currently we are on minimal doses of furosemide, ceasing the current diuretic treatment is recommended based on the risk that furosemide use prior to congestive heart failure may risk activation of the RAAS that may progress the cardiac diseases.
- Bronchomalacia or other respiratory cause of coughing likely
- Plan
- Continue pimobendan even though technically stage B1 because current medications could be causing artifactual decrease in cardiac size. 2.5mg q12h
- Given that we are only currently on furosemide 10mg (1mg/kg) q24h and the echocardiographic changes are mild, wean/cease this whilst monitoring sleeping respiratory rate
- If the cough progresses/recurs with an normal sleeping respiratory rate consider Cough suppression (codeine/lomotil/maropitant/tramadol etc); bronchodilator (theophylline/terbutaline); consider inhalational therapy; diet to achieve at least 10% weight loss then reassess
- Outcome
- Weaned off furosemide, SRR remained < 24
- Cough controlled with as necessary use of cough suppression, owner found was somewhat seasonal

Case report 4

- Abby
- Signalment - 14 years 5 months FS Pomeranian 2.60kg
- Hx - - Abby had a funny turn last week (13/12/23).
 - Appetite has been quite variable
 - Diarrhoea 3 weeks ago and has settled down
 - Sleeping respiratory rate < 24 up until last week then > 36
 - Started coughing 1-week duration
 - No vomiting
 - Weight loss
 - Current medications
 - pimobendan 1.25Mg Chew GIVE 3/4 CHEW EVERY 12 HOURS. GIVE 1 HOUR BEFORE FOOD.
- PE - Cardiac auscultation revealed grade IV/VI systolic murmur. Good pulse pressure. HR=PR= as for health status. Sinus tachycardia
- Radiography revealed:
 1. Moderate left-sided cardiomegaly with congested pulmonary vessels and aveolar-interstitial pulmonary pattern (i.e. Cardiogenic pulmonary oedema) is consistent decompensated cardiac disease/ congestive heart failure.

- Post frusemide
- Echocardiography reveals:
 1. Myxomatous Mitral and Tricuspid Valvular Degeneration stage C (congestive heart failure)
 2. Echocardiographic criteria for pimobendan benefit met (based on LA:Ao, nLVIDd EPIC criteria)
 3. Echocardiographic parameters suggestive of increased left atrial pressure consistent with congestive heart failure (based on PV:PA, Mitral E max and E:A ratio)
 4. Echocardiographic parameters were suggestive of moderate pulmonary hypertension (based on TC regurgitant velocity) based on the estimated modal TR velocity as per the ACVIM PH Consensus Statement guidelines.
 5. MINE score is 8 which is indicative of severe disease.
- DIAGNOSIS:
Myxomatous mitral and tricuspid valvular disease stage C
MINE score 8 Severe
There is an apparent flail mitral leaflet with suspected visualisation of an untethered chordae tendineae, this is consistent with chordae tendineae rupture
- Assessment
- Treatment - Based on the diagnosis of congestive heart failure, the following cardiac medications are indicated in the longer term:
 - Frusemide - ~2mg/kg PO q12h
 - Pimobendan - 0.2-0.3 mg/kg PO q12h
- When eating and drinking normally (which may be following discharge):
 - Spironolactone (~2.0 mg/kg orally q24h) - This is now recommended as a standard adjunct in canine CHF treatment. The primary benefit of spironolactone in this context is aldosterone antagonism rather than diuresis. Start at a subsequent recheck if the kidney values are stable.
 - Benazepril^^ [0.25-0.5 mg/kg PO q12-24h] - Start when Abby's appetite has normalised.

Summary of my world

	CASE 1	CASE 2	CASE 3	CASE 4	CASE 5
Coughing	Low grade 3 months	4 weeks	1 month	2 years Worse last 2 months	1 week
HM	12 months 4/6	2 weeks 1/6*	4 weeks 4/6	4 years 5/6	2 years 4/6
SRR	<20*	30*	<24*	Subjectively increased last weeks	< 24 until week before > 36
Frusemide?	No	Yes	Yes	No	No
Echo	MMTVD B2 Severe	Normal heart	MMTVD B1	MMTVD C	MMTVD C RCT
Dx	As above + TBM + LAD compression	Viral tracheitis	As above + possible TBM	As above + TBM + LAD compression	As above
Outcome	Cough resolved with cough suppression	Cough resolved after stopping frusemide	SRR remained <24 after weaning frusemide, cough was seasonal	Good response to Rx LCHF	Good response Ultimately weaned off frusemide (azotaemia)

Take home messages

- Remain critical (skeptical even) of cardiac disease as a cause of coughing in those small breed patients with a murmur
- Ensure adequate evaluation has been done to say that is cardiac (i.e. definitive pulmonary oedema based on SRR, thoracic rads and response to frusemide trial) and not respiratory before treating as cardiac

CVS Group: expanding our network

Established in 1999, and with over 500 Veterinary practices, CVS Group is one of the largest veterinary service providers in the UK.

We are now looking to expand our network of quality, independent practices in Australia.

We focus on recommending and providing the best clinical care every time.

At CVS, we focus on recommending and providing the best clinical care to every patient, and we believe this is what drives the success of our practices.

We deliver the highest standard of healthcare and we empower our colleagues to make the right clinical decisions for our patients.

We support and develop our teams to be the best in their role, which includes access to industry-leading learning, education and development, supported by a diverse team of learning professionals and subject matter experts and an engaging educational platform.

CVS also shares best practice across our company and at the same time we give our veterinary professionals the independent freedom to make the best clinical decisions for their patients.



We are a great place to work and have a career.

Our vision is to be the veterinary company people most want to work for. This will help us attract the best people to enable us to achieve our purpose of delivering the best care to animals.

Our practices are backed by the strengths of operating within a group that is run at every level by vets, including on the Board of Directors.

Our general practice veterinarians are supported by a specialist team that offer unique expertise and advice to practices across the country, to continuously improve the standard of care that our patients receive.

Our unique Knowledge Hub gives our colleagues access to the industry's best learning, education and development - with a catalogue of over 450 courses, programmes and webinars. We also offer our colleagues free access to the British Small Animal Veterinary Association Library - home to all publications covering the spectrum of small animal veterinary practice.

The breadth of our business means there is a vast range of career opportunities. We are pleased to see many people developing their career across our company - for example in specialist roles, or in management. We have a track record of develop and promoting from within, and encourage secondment and permanent opportunities across our network of clinics both nationally and internationally.

We are also proud to offer funding to colleagues for veterinary clinical research that aims to benefit the animals under veterinary care, and research that supports the veterinary profession in providing the best possible care to animals.

In addition, all our practices and people are supported by experts in IT, HR, Health & Safety, Recruitment and Procurement - leaving veterinary colleagues free to focus on caring for our patients.

We provide great facilities and equipment.

In the UK all of our practices meet the rigorous standards of the Royal College of Veterinary Surgeons voluntary Practice Standards Scheme.

We are also investing up to AU\$90m million each

year in our practice facilities, clinical equipment and technology to improve standards of clinical care and our colleague and client experience.

In the UK, this has included rolling out a new minimum practice facility standard - specifically designed by our veterinary surgeons, nurses, practice managers, receptionists and patient care assistants - which includes well laid out clinical areas, modern office space and dedicated staff rooms.

It enables our vets to do the work they want to do, allows our vets to offer an increased range of services, and creates a nice place to work.

We take our responsibilities seriously including putting our people first.

Every important decision we make takes into consideration the impact it will have on our people. We care about supporting our colleagues' wellbeing and progress.

We have a network of pastoral support vets, mentors and Wellbeing Champions that help our



colleagues to look after their mental health, their career and to do their best work.

We care about being a truly inclusive organisation and have programmes to ensure that all of our colleagues feel welcome.

CVS places great importance on line manager conversations and has introduced check-ins to ensure all colleagues are getting the time and engagement needed from their manager. As well as providing feedback on performance and identifying development opportunities, these regular check-ins are designed to support wellbeing.

In addition, we have active Wellbeing Champions trained in "Supporting the Wellbeing of Your Team" and have introduced a Wellbeing Calendar for colleagues featuring topics such as resilience, exercise, sleep, stress. We have also launched a new CVS Refresh reward scheme where teams receive a weekly allowance to spend on treats or classes.

We track our performance by regularly measuring colleague engagement. Our employee Net Promoter Score (eNPS) reflects the number of people who would recommend us as a good place to work. It has been increasing since 2019

If you are interested.

We would love to speak with you regarding your succession plans.

If you are interested in your practice joining CVS we offer partial and full sale options.

Vendors find us an attractive option because we don't change their practice, practice name, clinical care and culture.

We also look after their team - maintaining and protecting their entitlements, wages and conditions.

In addition, we offer a quick and efficient process with completion and transfer of funds in as little as eight weeks.

As a stock market listed company we invest for the long-run, which means you can be assured that we are here for the long term. Being publicly listed also means that CVS is held accountable to public scrutiny, and there is more transparency than some other large, privately held groups.

Come and meet us or get in contact.

If you'd like to know more or are interested in discussing any aspect of CVS - and whether we would be a favourable option for you - we would be happy to meet for an initial, confidential chat.

Come and visit us at our stand 37 & 38 at the ASAV & VBG 2024 Conference, 12th-15th August 2024, Novotel Sunshine Coast Resort QLD.

Alternatively contact Nathan Micallef at CVS Australia on 0418 754 853 or email Nathan.Micallef@csvets.com.



Take a breath and **THRIVE.**



Vets Choice insurance for pets

Vets Choice insurance for pets and Guild Insurance are proud supporters of the Australian Veterinary Association (AVA) led THRIVE wellness initiative, which champions veterinarians and veterinary staff leading satisfying, prosperous, and healthy careers.

The THRIVE Mental Health and Suicide Prevention Framework, created in consultation with veterinary professionals supports well-being strategies that positively impact mental health outcomes for all. Supporting the THRIVE program aligns with our own philosophy for our customers – ‘Don’t go it alone’ and is a demonstration of our ongoing commitment to being there for those our communities rely on.

The framework outlines the 3 founding pillars of the THRIVE approach to addressing workplace mental health and suicide prevention.

1. **Prevent:** Preventing harm from psychosocial hazards in the industry.
2. **Promote:** Promoting strengths, capabilities, and the rewards of work.
3. **Protect:** Protecting us when we experience stress and are unwell.

The framework applies to everyone and asks us to remember that we’re all responsible for creating a work environment that is free from psychological risk and mental health stigma.

Together with the AVA, we encourage everyone to talk about the framework, explore each pillar and create ways of working that prevent harm, promote well-being and protect ourselves and each other. You can download the framework and access THRIVE resources at guildne.ws/THRIVE



Culture is key: How everyone can co-create more positive veterinary working environments

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What exactly is workplace culture?

Workplace culture is the network of shared values, beliefs and assumptions that connect people in workplaces, and impact their behaviour. When people work together over an extended time, culture can be an invisible but strong determinant of how and why things are done a certain way. It can have either positive or negative effects - a culture that supports positive workplace practices can enhance performance, while a dysfunctional one can hinder it.

Culture is what binds individuals at a workplace together and gives them a sense of belonging. It represents the unwritten rules that drive thoughts and behaviour so that individuals understand the 'way things are done' in their workplace. Culture can be contagious, and employees learn their culture in many ways, including through observation, training, leadership example and critical incidents, with people learning what to avoid (to reduce their anxiety) or what works well (to gain positive reinforcement). Strong cultures develop when the same people share experiences over an extended period, and frequent staff turnover can hinder this culture development. These strong cultures are not always positive, as undesirable behaviour can spread throughout the group, such as negativity and bullying.

Studies have shown that workplace culture impacts employee behaviour and overall performance.^{1,2} A positive relationship has been found between workplace culture and patient satisfaction in health-care settings, making it relevant to veterinary hospitals.¹

A system of workplace culture can develop in any veterinary hospital and significantly impact the wellbeing of all who work there. A culture that supports employee wellbeing can improve performance, while poor wellbeing culture can negatively impact it.^{2,3,4}

Why focus on veterinary workplace culture?

As a veterinary wellbeing coach, I am passionate about making a difference in the lives of all who work in the veterinary profession. I worked as a small animal veterinarian for 12 years and understand the wellbeing challenges facing all those who work in veterinary workplaces. I am often frustrated when my coaching clients are trying diligently to improve their wellbeing but are working in situations that are, at best, uncertain of how to support wellbeing, or at worst, are culturally toxic. Recent research has suggested interventions aimed at improving individual veterinary wellbeing. However, my experience with coaching veterinarians and their support staff tells me that using interventions at the individual level only addresses part of the problem. Changing the culture that they work in must also be part of the solution. I believe the workplaces within which many veterinarians and their support staff work have very negative cultures, and therefore, there is a great opportunity to impact the overall wellbeing of the profession by creating more culturally positive workplaces.

There have been concerns about low levels of veterinary wellbeing for decades. A review of veterinary workplace studies suggested that the profession is vulnerable to low levels of wellbeing and poor mental health.⁵ A recent worldwide survey identified a correlation between working in a veterinary practice and an increased risk of mental health problems.⁶ Compared to the general population, high levels of veterinary occupational stress contribute to an increased risk of depression, anxiety, burnout, and suicide.¹²

Throughout their career, veterinarians are exposed to numerous sources of workplace stress, with 80% describing their role as stressful.⁷ Pohl and colleagues identified numerous psychological stressors in veterinary practice, and Moses and colleagues found that 50% of surveyed veterinarians were often anxious or distressed about their work.^{5,8} Despite 80% of veterinarians in a study describing high levels of job satisfaction, 66% also had symptoms of burnout.⁹ Unfortunately, many of these identified stressors are inherent in veterinary work, and therefore difficult to manage or eradicate. Thankfully, some of the stressors facing veterinarians and their colleagues can be addressed and solved at the workplace level.^{10,11} These include barriers to reporting adverse events, inadequate training, challenging relationships with other team members, lack of appreciation, lack of autonomy, lack of respect, lack of support, stigmatisation of mental illness, poor management of negative patient outcomes, fear of making a mistake or client complaints, and poor leadership – all things that I believe we can improve with more positive veterinary workplace cultures.¹²

Researchers have identified attrition as an ongoing problem in the Australian veterinary profession, with 19% of respondents to a 2018 workforce survey stating they were considering leaving the profession within the next year.^{13,14} Possible contributors to attrition include workplace cultural problems such as bullying, lack of support, poor relationships with co-workers, and a toxic culture.¹² Simply increasing veterinary student numbers has not achieved the expected over-supply, and practical solutions are needed to increase retention, such as improving workplace culture.¹⁵

Researchers are also calling for workplace cultural change, suggesting that veterinary businesses provide environments that support wellbeing and create positive cultures.^{16,17,18,19} A positive culture has been identified as an important contributor to veterinary resilience and wellbeing; and a psychologically safe workplace culture allows staff to voice concerns and report adverse events.^{20,21,22,23,24} In contrast, Moore and colleagues found that a toxic veterinary team culture positively correlates with burnout and negatively correlates with job satisfaction.²⁵ A meta-analysis of studies researching interventions to decrease burnout in medical physicians, concluded that workplace change is more effective at supporting staff mental health than interventions aimed at the individual.²⁶ Healthcare researchers also found that workplace culture initiatives could promote engagement and decrease burnout in medical physicians; with positive correlations between patient outcomes and workplace culture suggested across multiple studies.^{27,28,29}

Professional bodies have also recognised the need for improvements in workplace culture. In the past eight years, national veterinary associations from Australia, Britain and America all have undertaken extensive analyses of the veterinary wellbeing issue. They identified poor workplace culture as associated with low levels of veterinarian wellbeing and have recommended that all veterinary workplaces develop positive working cultures.^{30,31,32}

Considering this evidence, I suggest that it is unfair to place sole responsibility for mental health on the individuals working in the veterinary profession. Wellbeing and retention programs must look at workplace solutions, as well as individual interventions. A practical and achievable goal is to improve the culture in all veterinary workplaces. Any defined group with a shared history can develop its own culture and the culture of a workplace can play a significant role in shaping the behaviour of its employees and impact their wellbeing. Not all workplace cultures are positive and understanding your own culture can help uncover problems that are impacting business success and employee satisfaction. Let's work together to co-create positive veterinary workplace cultures.

What does a positive workplace culture look like?

A positive workplace culture builds connection, respects boundaries, builds trust, and creates a psychologically safe environment. It enables great communication and encourages empathy. It provides meaningful work and encourages team collaboration, while supporting the growth of

individuals. A positive workplace culture supports the wellbeing of all who work there. But how can we define it in our own workplaces so that we can affect change?

Kim Cameron and colleagues developed their empirically based positive practices model to define and measure what actions contribute to a positive workplace culture.³ They identified six inherently good and virtuous workplace behaviours that can improve organisational effectiveness by bringing out the best in people. Table 1 describes the six positive practice dimensions, and their relevance in a veterinary workplace.

Research done by Cameron and associates (including in numerous healthcare settings) suggests that an increase in these six positive workplace practices can support positive emotions, resulting in improved employee wellbeing, work satisfaction, and performance.^{3,33,34} They suggest that the resulting changes in the workplace can include - decreased conflict, better decision making, better care of customers and fellow employees, improved patient care, decreased staff attrition, better customer satisfaction, and increased profitability.^{3,33,34} When individuals experience positive practices such as gratitude, it can increase positive emotions.^{33,35} The witnessing and promotion of good deeds creates a self-reinforcing upward spiral of positive emotions and behaviours, thus amplifying the positive effect.^{3,36} Positive practices can also buffer a workplace and its employees against the impact of adverse events, hence building resilience.^{3,36}

Changing workplace culture can be difficult and complex, so defining the desired culture with a model such as these six positive practices, provides structure and allows interventions to be designed to align employee behaviours with the new, desired, workplace culture.

Table 1
Positive Practice Dimensions and Definitions: Including Relevant Veterinary Research Supporting use of the positive practices model

Positive Practice Dimension	Definition	Relevance to Veterinary Workplaces
Caring	Interest in and responding to one another as friends.	Caring and compassionate colleagues are important coping and social resources for veterinarians. ^{7,19}
Compassionate support	Being kind, supporting those struggling. Honouring and supporting one another in their endeavours.	Support is important for veterinary wellbeing. ^{16,19}
Forgiveness	Avoiding blame, forgiving mistakes.	Forgiveness is key to safe reporting of significant events in veterinary hospitals and may decrease self-criticism and encourage help-seeking, while supporting excellent patient care. ^{16,22} A just culture may help protect against burnout in medical workplaces. ²⁷
Inspiration	Influencing and elevating others at work. Sharing enthusiasm and inspiring others by acknowledging the good they see in others.	Noticing that positives may counteract problem focus and negativity bias. ¹⁹
Meaning	Employees are motivated, renewed by, and see a purpose in their work.	Finding meaning at work is important to veterinary wellbeing and may protect against burnout. ^{19,37}

Respect, integrity, gratitude	Respectful, appreciative, trusting relationships are formed. Employees have confidence in one another.	Gratitude and positive emotions can help build veterinary resilience, improve coping skills, and increase wellbeing. ^{21,38} Appreciation was identified as important for veterinary wellbeing. ³¹ Respect and trust are important for psychological safety in veterinary workplaces. ³⁹
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Note. Positive practice dimensions and definitions from Cameron and colleagues³.

What about a toxic culture?

In contrast, a negative workplace culture is dysfunctional and dramatic. It allows harassment and bullying - with gossip, cliques, and ostracism of individuals; People use poor communication styles, and there is a lack of trust, respect, and psychological safety. In my experience, these types of veterinary workplaces tend to have a high turnover of staff, with low morale and poor employee wellbeing. They may even exhibit unethical behaviours. Sadly, many of us have worked in a place like this or know someone who has.

This type of toxic workplace culture has been associated with an increased risk of burnout. Moore and colleagues found that toxic veterinary team culture positively correlates with burnout and negatively correlates with job satisfaction.²⁵ Moreover, Moir and Van den Brink identified negative organisational culture as a key driver towards veterinarian burnout.¹⁶ This alone, should be enough to motivate us to build more positive veterinary workplaces.

Creating a positive culture in your veterinary workplace

Changing a workplace's culture is a complex and challenging process, but studies suggest it is possible.^{3,40} It takes time, patience, great leadership, and motivated team members. Important steps to facilitate change include:

1. Assess and measure your existing workplace culture and levels of team member wellbeing. Determine what you are already doing well and where there might be room for improvement. It is critical that team members feel safe enough to be honest during this process – this may require an anonymous survey.
2. Define the desired culture with input from all team members and create a clear vision of what you want to achieve. Then align required behaviours with this culture, making changes as needed. This may include removing members of the team who repeatedly behave in ways that do not support the desired culture.
3. Ensure team members understand why culture change is needed, so they believe in the process.
4. Maintain a stable workforce to encourage a strong culture and, when necessary, select new employees that are a cultural fit and support their socialisation (assist them in learning the culture).
5. Leaders can foster positive practices by modelling behaviour and spreading a positive culture. They play an essential role in explaining the value of any changes that are being implemented. Leadership can make or break change – they must model the desired behaviours, and lead by example.
6. Training team members to support the new culture. This training should be ongoing and positive – one team meeting is not enough.

Some suggested and evidence-based interventions that can build a positive veterinary workplace culture include:

- Strengthen caring between team members by fostering social connections at work and with social events^{3,20,41}
- Encourage compassionate support for each other by improving team-members emotional intelligence and empathy with training^{3,20,41}
- Facilitate compassionate support by providing structured support systems, assigning mentors, and encouraging help seeking^{16,19,20,41}
- Demonstrate compassionate support and promote psychological safety by providing a safe space for team members to discuss challenging cases/issues^{42,43}
- Model forgiveness for mistakes, and teach self-forgiveness with self-compassion training⁴⁴
- Leadership training to encourage leaders to inspire others to be more positive⁴⁵
- Enhance meaning at work by supportive job-crafting¹⁹
- Increase appreciation from management to team members, and between team members^{41,46}
- Leadership training in positive wellbeing practices to build organisational resilience^{47,48}
- Leadership training in coaching skills⁴⁹
- Embed a wellness program across the organisation^{20,30}
- Leadership should model and encourage positive wellbeing practices that they want instituted (such as taking lunch-breaks)¹³
- Endorse and spread the positive culture with positive artefacts around the workplace⁵⁰
- Build autonomy by including all stakeholders in workplace culture decisions²⁰

Why creating a positive culture is key to successful veterinary workplaces

The combination of individual wellbeing challenges with societal and workplace stressors, is taking a toll on individual veterinarians and the entire profession. Veterinary workplace culture and team effectiveness can impact the job satisfaction and wellbeing of all who work there. The AVA defines workplace culture as the values, traditions, beliefs, interactions, behaviours, and attitudes in a veterinary hospital – with 25% of 2479 Australian veterinarians surveyed in 2021 not satisfied with their workplace culture.⁵¹ Given the positive return on investment for wellbeing spending, and ongoing attrition from the profession, managers and Corporates should be motivated to support wellbeing to retain staff.⁵²

Therefore, I believe that now is the perfect time to create a positive cultural shift in all veterinary workplaces. A positive workplace culture leads to engaged, connected, and high-performing teams. This results in happy workplaces and satisfied customers. I look forward to helping you create your own plan for positive cultural change in your workplace, during my presentation.

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Managing strong emotions in veterinary workplaces

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What are emotions?

Unfortunately, there is no consensus on the definition of an 'emotion'. The American Psychological Association defines emotions as: *"conscious mental reactions (such as anger or fear) subjectively experienced as strong feelings usually directed toward a specific object and typically accompanied by physiological and behavioural changes in the body"*. Most experts do agree that there are neural systems dedicated to processing emotions, and that emotions motivate both thoughts and actions.

Emotions are functional and are thought to have evolved in man to mobilise us to deal quickly with important events. They occur as a rapid, short-lived, and involuntary response to a trigger. A trigger can be a thought or experience that we sense is important to our welfare. This leads to a physiological response, with resultant associated thoughts and behaviours.

Simply put, when an emotion occurs it can elicit change in our physiology, feelings, thoughts, and behaviours. These changes can be either positive or negative (or both). Emotions are powerful things and should not be underestimated. Learning how to manage emotional situations in the workplace is a skill that cannot be under-valued.

What is emotional intelligence?

Building our own emotional intelligence (EI), and the EI of our team members, can assist us in managing the strong emotions that commonly occur in veterinary workplaces. The term emotional intelligence has been around for about 60 years but became popularised after the book "Emotional intelligence" by Daniel Goleman was released in 1995. There are several definitions of EI, with Salovey and Mayer defining it as "the ability to monitor one's own and other's feelings and emotions, to discriminate amongst them, and to use this information to guide one's thinking and action".¹

Goleman and his colleagues describe four core skills that contribute to EI.² The first two skills are about how we relate to ourselves, while the second two are about how we relate to others.

1. *Self-awareness*. This is about being aware of and understanding your own emotions. What are they, and how are they impacting you and those around you?
2. *Self-management*. This is about managing your own emotional response well. What do you do with your own emotions? How are you reacting? Are you responding thoughtfully, or reacting without thinking?
3. *Social awareness*. This is about reading and understanding the emotions of other people? Do you show empathy? Can you see the other person's perspective in emotional situations?
4. *Relationship management*. This is about positively managing inter-personal, emotionally charged situations. In emotional situations can you find common ground and build connection. Can you manage conflict and de-escalate the situation? Do you inspire and influence other people? Can you shift the emotions of others in a positive way?

EI is a type of intelligence that can be measured. One instrument is the MSCEIT (Mayer Salovey Caruso Emotional Intelligence Test) which measures how we perceive, understand, think about, and manage emotions.³

While some people are more naturally emotionally intelligent, I believe that it is a muscle that most people can exercise and strengthen (and studies support this). Improving our EI starts with self-awareness, and then self-management. If we don't know what emotion was triggered, why it was triggered, and how it impacted us, it is difficult to know how to manage it. Without self-awareness and self-management of our own emotions, we can't read the emotions of others and manage tough emotional situations. Building EI starts with getting touchy-feely with our own emotions – something that many people find challenging.

Why is emotional intelligence important?

High EI is important to everyone as it has been linked to increased positive emotions and happiness, increased health and wellbeing, better quality relationships, greater empathy, lower stress levels, improved resilience and coping skills, better leadership potential, enhanced conflict resolution and improved job performance.¹⁻⁵

In contrast, someone with low levels of EI may be more easily stressed, more at risk of anxiety and depression, less capable of managing their own negative emotions, and have greater difficulty dealing with strong emotions in others.¹⁻⁵

EI is important to professional success, job satisfaction and interpersonal functioning in the often emotionally demanding veterinary workplace. A recent cross-sectional study by Wells and colleagues found a statistically significant, negative correlation between EI and SAD (stress, anxiety, and depression) in veterinary students.⁶ As EI decreased, their risk of SAD increased, and vice versa. Numerous studies have found associations between high EI and psychological wellbeing and resilience, while Bell and colleagues found EI to be one of the most important capabilities for veterinary employment (with the inclusion of emotional competence in the VetSet2Go employability framework).^{7,8}

Emotional self-awareness

Emotional Self-Awareness is the ability to understand your own emotions and their effects on you and on your performance. You know what you are feeling and why—and how it helps or hurts what you are trying to do. For some people this is easier said than done – they know they are emotional (they feel the physiological reaction), but they can't label or clearly describe what emotion they are feeling. If this is you, then you need to practice this skill in less emotionally stressful moments. It can be helpful to use an app, such as the mood meter, which prompts you to name the emotion you are feeling during different times in the day and think about the impact it is having. This helps to improve your emotional vocabulary. Another option is to use an emotion wheel to help you accurately name your emotions. As you can see below in the emotion wheel, being “angry” can mean a lot of different things – ask yourself am I frustrated by what just happened, or I am hurt, or am I feeling threatened – all quite different emotions. Similarly, being happy can mean different things – it could be you're feeling love, amusement, or playfulness.



Part of emotional intelligence (EI) is understanding

Veterinarians, when compared to the general population, experience more negative emotions at work, so it is important that we learn how to manage these emotions effectively to support our own wellbeing, and the wellbeing of those around us.^{9,10}

Interestingly, Spitznagel and colleagues, while researching things that predict stress and burnout in veterinarians, discovered that the veterinarian's reaction to these stress-inducing encounters was as important as how frequently they occurred.¹¹ In other words, how veterinarians and their team members feel about workplace stressors, is as important as how much stress they have. This is telling, as while we often can't control the number of stressful moments in our day, we can control how we feel about them. That takes emotional self-management.

Suppressing your strong negative emotions (wearing a 'positive' mask), while sometimes necessary during a veterinary workday, is associated with poor health and wellbeing outcomes. That type of surface acting, where your surface doesn't match how you are feeling, is associated with in-authenticity and burnout.¹² In the long-term, aligning your observable emotions and behaviour with your internal experience, is healthier.

We will explore more deeply specific techniques to manage strong emotions in veterinary workplaces during my presentation. The following are evidence-based, and may be helpful:

- Understand what your emotional triggers are and prepare a plan to handle these situations when they occur.
- Be proactive about potentially stressful situations and do something before the emotion is fully felt - avoid the situation (if possible), change the situation when you can, shift your focus onto something else, or change how you think about it.
- Reappraise the situation and consider the other person's point of view – take a different perspective.
- Take a physiological break to decrease the stress response – mindful breathing exercises, stretching, going for a walk, practising mindfulness.
- Insert a pause before saying or doing anything. Take a moment to question your thoughts and ponder the situation from all sides before reacting. Then you are more likely to respond thoughtfully.
- Debrief tough emotional situations so that you can process what you felt and learn from the situation. This allows you to remove the mask at an appropriate time. This can be done verbally with a trusted friend or colleague or written down if debriefing alone. Using a structured debrief can be useful. Think about the 3 WHATS when debriefing:
WHAT happened? Stick to the facts.
So WHAT? Why is it important?
Now WHAT? What did you learn? What does this mean?
- Manage your chronic stress to decrease your emotional reactivity over time, using techniques such as:
 - o Practice mindfulness
 - o Improve your physical wellbeing by getting enough sleep, eating well, and exercising
 - o Build quality connections with other people
 - o Take breaks and holidays
 - o Set boundaries to maintain work/life balance
 - o Be self-compassionate – treat yourself with the same kindness and care you would give to a dear friend or loved-one
 - o Write about your emotions to debrief once you get home

Enhancing our own positive emotions

The work of Baumeister and colleagues identified that humans have a strong negativity bias – they spot the bad things easier than the good.¹³ Veterinary work encourages clinicians to look for problems, further strengthening this bias and increasing negative

emotions.^{9,10} To combat this negativity bias, it is important for veterinarians and their team to consciously notice the positives in their lives and to feel positive emotions such as joy, gratitude, and hope.

Barbara Fredrickson's (2001) "broaden-and-build" theory suggests there are both short and long-term benefits to increasing how much we feel positive emotions.¹⁴ The theory posits that positive emotions broaden people's attention and thinking in the moment and build enduring personal resources over time that can be drawn upon later to help manage future issues.^{14,17,18} These psychological and social resources can help build a buffer against the stressors in veterinary work and minimise any lingering effects of negative emotions.^{14,15} Positive emotions can create a positive spiral leading to improved psychological resilience, better coping, and increased wellbeing.¹⁴⁻¹⁶ Positive emotions not only feel good in the moment, they may contribute to people's happiness and wellbeing in the future.¹⁷

One of the great things about positive emotions is that they can be contagious (just as negative emotions can spread also). Some of the ways that you can feel and spread positive emotions in your workplace include:

- Practice gratitude
- List 3 things that went well in every workday
- Savour everyone's successes at work, and be proud of your/their accomplishments
- Get outside for a moment and spend time in nature
- Relax with music/podcasts/books in your breaks
- Appreciate the people you work with and your clients
- Admire a job well done
- Enjoy time with your animal patients
- Have fun with your colleagues
- Achieve a new professional milestone

Social awareness and relationship management

Once you have strengthened your EI muscle with increased emotional self-awareness and self-management, you will find that your ability to read the emotions of others will be easier. Being able to manage emotionally charged situations at work is a foundational skill. Imagine being able to read that a client is frustrated and confused, and then being able to calm them down by acknowledging their perspective and answering all their concerns.

Listening to their concerns, showing empathy for their point of view, and communicating compassionately can all contribute to these tough emotional situations going well, resulting in a happy client. We will learn more about these skills during my workshop on communication.

Why strengthening your emotional intelligence is worth the effort

Having high EI allows you to recognize and label the emotions in yourself and others. It means you are comfortable expressing and discussing your feelings and you allow yourself to truly feel a range of emotions. You understand your own triggers and can show composure and self-control when required. You can take responsibility for your own emotions and advocate for your own needs when necessary. You can spot tough situations before they escalate, and settle conflicts. You can handle the difficult conversations well.

High EI is associated with job satisfaction, good interpersonal functioning, leadership ability, and overall success in life. We can all improve ours – it just takes practice. Take every small emotional moment as an opportunity to practice your self-awareness and self-management, and watch your EI grow.

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SCIENCE DID THAT.

Diagnosing canine heart failure in practice: Combining clinical examination, radiography, echo and TFAST

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The decision to start cardiac medication involves a lifelong commitment that can have significant negative financial and health impacts if they are initiated unnecessarily, and reduced life expectancy if identified belatedly. In this lecture we cover the physical examination, clinical history and in-practice imaging findings that research shows can substantially improve your ability to identify patients in, and at risk of, heart failure.

Clinical history

In small breed dogs, DMVD usually progresses slowly over months to years with a variable rate of progression so that 50% or less actually die of their DMVD.

Can we identify which of our DMVD patients are more likely go on to develop heart failure? Are there historical and clinical findings that increase our suspicion that there is heart failure present, or imminent?

In a longitudinal study of 244 dogs with DMVD (Lopez-Alvarez et al 2015) it was found that several commonly observed historical and clinical findings in dogs with DMVD are independently predictive of a worse outcome. These signs were: history of cough; exercise intolerance; decreased appetite; breathlessness/tachypnoea; and syncope in conjunction with physical examination findings of heart murmur intensity louder than III/VI and absence of respiratory sinus arrhythmia. When multiple of these findings was detected there was a greater likelihood of progression to cardiac death, and if the number of these findings increased over time, this also greatly increased the likelihood the patient would die from their disease.

These authors concluded that small breed dogs with DMVD tend to develop several, sometimes subtle, clinical signs before they develop CHF and that this data can be obtained in general veterinary practice before performing any ancillary diagnostic tests that can help the clinician's subsequent decisions regarding the patient.

I am sure that the inclusion of cough in this list will have raised some questions for the reader, as the association between cough, heart disease and heart failure can be controversial. Coughing is not correlated with pulmonary oedema unless it is severe but is often associated with left cardiomegaly and concurrent airway disease (Ferasin et al 2013). A cough can be present in patients with preclinical DMVD and those with CHF but is usually caused by the intersection of cardiomegaly and airway disease rather than oedema. It is interesting that despite this, it was one of the independent predictors of poorer outcome, possibly because it indicates severe left heart enlargement.

Heart murmur

Degenerative mitral valve disease is characterized by mitral valve insufficiency, so the first clinical finding is an apical heart murmur. Heart failure will not develop until the patient has progressed into advanced pre-clinical (Stage B2) disease, and this classification has the criterion of a heart murmur of grade 3 or more. The exception to this rule will be dogs with dilated cardiomyopathy, in which the mitral insufficiency due to annular stretch develops late in the disease and may cause only a soft murmur. It is important to be consistent with auscultation technique and heart murmur grading.

A summary from cardiologist Dr Fiona Campbell is provided below:

Cardiac auscultation technique in dogs and cats (Campbell 2013)

1. Place palms over the left and right thorax to identify if a thrill is present.
2. Use palms to locate the apex beat (the place where the heartbeat feels strongest, on the left thoracic wall).
3. Place the diaphragm of the stethoscope over the apex beat and listen.
4. Count the heart rate.
5. Evaluate heart rhythm: regular, regularly irregular, irregularly irregular?
6. Assess synchronicity of the right femoral pulse with the heartbeat.
7. Determine if a murmur is present. If so, consider the intensity and timing. Then, gradually move the stethoscope craniodorsal (over about 2 rib spaces) and identify how murmur intensity changes. If murmur gets softer, the location of the murmur is **apical**; if murmur gets louder, the location is at the **base**.

Grading heart murmurs (Campbell 2013)

- I Very soft murmur that is not immediately audible but can be heard only after careful auscultation in a quiet environment.
- II Soft murmur that is audible with careful auscultation.
- III Moderate murmur immediately audible with auscultation.
- IV Loud murmur without a thrill.
- V Loud murmur with a palpable thrill.
- VI Audible with stethoscope held slightly off the chest wall.

Having a copy of this available in your consult or treatment room is helpful so that everyone in your practice is consistent.

Respiratory rate

The most sensitive test for onset of congestive heart failure in a patient with confirmed mitral insufficiency or DCM is increase in the sleeping respiratory rate. A diagnostic cut-off of 41 and 34 per minute in dogs with MVD and DCM, respectively, was useful in the prediction of presence or absence of CHF with high accuracy (sensitivity and specificity between 92 and 100%) (Schober *et al*, 2010). Remember there are other causes of increased respiratory rate so this applies specifically to patients with known left heart disease, at risk of heart failure.

Sleeping respiratory rate is more sensitive than radiographs, NT-proBNP, TFAST or echo and it is free and easy.

All owners of dogs with a heart murmur $\geq$ III/VI should be taught to monitor their pet's respiratory rate. This one piece of information they can provide will be so helpful for you when making decisions about management.

Thoracic radiographs

After you have evaluated history and physical examination findings, if there is a loud left apical heart murmur and clinical history of one or more of cough, reduced appetite, reduced exercise tolerance or increased respiratory rate, then the next most useful test is radiography.

You do not need an echocardiogram to diagnose heart failure. Echo is useful to stage a heart murmur earlier in the disease, and identify any complications such as pulmonary hypertension, but radiographs will provide you with answers to the questions of cough and respiratory signs that cannot be determined any other way (except CT, which is overkill in most cases).

The objectives of the radiographic examination of animals for heart failure are to: establish the diagnosis of CHF; determine the severity of CHF; discriminate between right and left congestive heart failure; differentiate between CHF and other diseases causing similar signs; and document the course of the disease and its response to therapy.

Thoracic radiographic technique

It is impossible to overemphasise the importance of good radiographic technique, especially for diagnosis of congestive heart failure. At best, bad positioning of thoracic films makes it harder to read them; at worst it creates artifactual changes that are misinterpreted as pathology, sometimes leading to many years of unnecessary (and expensive) cardiac medications. Rotation alters all cardiac parameters and makes accurate interpretation of cardiac size and shape impossible.

Remember - air is your friend. You must do everything you can to maximise pulmonary inflation. This means NO GENERAL ANAESTHESIA! Light sedation and good positioning aids are nearly always adequate.

DO NOT hold patients for the exposure unless you have a genuine emergency. Dyspnoeic cats are my exception, but you would be better off stabilising them before attempting radiography. Frequently sandbags suffice. If for some reason you must anaesthetise your patient, then you need to induce them in STERNAL recumbency and perform the VD/DV first and manually inflate the lungs for the exposure (gently). Keep them in sternal between lateral exposures to minimise the atelectasis that occurs almost immediately with anaesthesia.

During anaesthesia patients do not take deep breaths so it is important to manually inflate the lungs (physiologic deep breath) by squeezing the bag. This requires someone to gown up and be in the room. As long as they stand back from the primary beam, radiation exposure to the staff member should be negligible.

Of course, if the patient is anaesthetised there is no excuse for poor positioning! Extend the legs and correct for rotation.

Artifacts of poor positioning and their impact on the diagnosis of heart failure

• Rotation - lateral radiographs

If the sternum and dorsal spinous processes aren't similar heights off the table then the rotation of the chest projects the spine ventrally. This makes the heart appear very tall. The terminal trachea is also swung around so it looks like it is deviated dorsally, again giving the appearance of a large heart. I've also seen this misinterpreted as heart base or cranial mediastinal mass effects.

• Rotation - VD/DV radiographs.

If the dorsal spinous processes project beyond the vertebral bodies then the radiograph is too rotated for interpretation of the cardiac shape. Things like the left auricular bulge, main pulmonary artery and aortic arch are impossible to properly evaluate in a rotated radiograph. It also makes the cranial mediastinal width impossible to determine and the middle lobes hard to interpret.

• Underinflation

Shallow breaths are always very challenging, and conscious patients can be frustrating to catch at maximum inspiration. Anaesthetised patients without positive pressure ventilation are the worst, usually with abysmal lung volume.

Pulmonary underinflation leads to a small caudal lobe area, making the heart look bigger, and increased interstitial opacity due to all those un-inflated alveoli contributing to pulmonary density. When under-inflation and rotation appear together (and they really are common bedfellows) in small, often overweight dogs, the combination of an artifactually large appearance to the heart and increased pulmonary opacity are very often mistaken for left sided heart failure.

How to overcome these problems

1. Take time and teach your staff to get great radiographs. Palpate the sternum and dorsal spinous processes to ensure they are level, extend the forelimbs.
2. Routinely obtain right and left lateral and DV radiographs of your cardiac patients. VD is useful if you can't get the dog straight in DV, and all 4 is ideal (if your patient is not severely compromised).
3. Invest in foam wedges, troughs and sandbags - you cannot get good radiographs and practice good radiation safety without positioning aids.
4. Lightly sedate your patients. This is completely acceptable in heart patients, just use appropriate pharmacology. We use butorphanol, around 0.2mg/kg.

TFAST

In the emergency setting, or even just to help refine a differential diagnosis list, a thoracic ultrasound evaluation is very useful, but should ideally be used in conjunction with radiography, not exclusively. This is because a lot of pathology can present with dyspnoea and B lines, not just heart failure, and radiographs will help you determine this.

A basic TFAST will evaluate the pleural space for effusion (or pneumothorax) and a few sites for peripheral pulmonary pathology. Any fluid or cells in the alveoli or interstitium (alveolar-interstitial syndrome or "AIS") will transmit sound which then strikes the aerated lung on the far side that creates a ring down/reverberation artifact, causing "B-lines" (AKA rockets, comet tail artifacts). It is important to remember that a lot of different pathology will cause these, not just cardiogenic oedema. Other causes of AIS and B lines include non-cardiogenic oedema, interstitial pneumonitis, pulmonary hypertension, bronchitis, ARDS, haemorrhage and neoplasia. If the patient has been in lateral recumbency for a while and then sits up, you will also see B lines that disappear over a few minutes due to atelectasis. Typically CHF will cause more B lines in the dorsal/caudal lungs than the cranioventral lungs. If the dog is severely affected, there is likely to be pathology that extends to the lung periphery but if it is mildly affected then TFAST is unlikely to be useful.

If you can include a basic evaluation of the heart to look for left atrial enlargement this will greatly enhance your ability to diagnose heart failure, but remember that there are plenty of dogs with heart disease and respiratory disease so a big atrium does not necessarily mean heart failure. It would be a good indication for diuretics in the emergency setting, however. Once the patient is stable some thoracic radiographs can help make sure there isn't other thoracic pathology present. Don't forget that diuretics are anti-tussive, so a cough often responds in the short term, no matter the cause. Always monitor respiratory rate to determine response to treatment.

Echocardiography

The most useful time for echocardiography is when a grade 3/6 murmur is first diagnosed. This will confirm the diagnosis and stage the disease so pimobendan can be commenced if appropriate. I would not delay echo if it is accessible and the owner can afford it, because I do see some patients with a newly diagnosed grade 3 murmur that already have severe left heart enlargement.

If congestive heart failure is diagnosed in the clinic before echo, appropriate therapy needs to be initiated and the patient stabilised. At this stage, echo is appropriate to confirm the

cause of the heart failure and look for complicating factors such as pulmonary hypertension.

Medium and large breed dogs do get DMVD and will often have poorer systolic function than affected small breed dogs. Systolic function is usually intermediate between normal and DCM, and they can often develop severe CHF. While treatment for heart failure due to DMVD or DCM is essentially the same, there are greater risks of arrhythmia in DCM and often a poorer prognosis, so a diagnosis helps with deciding on additional tests such as ECG/Holter monitor, dietary evaluation, and with managing client expectations.

In young patients, congenital anomalies such as PDA are naturally the major rule-out. If thoracic radiographs are negative for heart failure but there is cardiomegaly or you are concerned about a heart murmur, echocardiography is also indicated.

Predicting CHF with echocardiography

In patients with DMVD, systolic function is typically preserved but the left atrium dilates in order to normalise the pressure in the lungs while it becomes increasingly overloaded with regurgitated blood and increased blood volume due to the activation of the RAA system. Heart failure develops when the ability of the left atrium to enlarge is exhausted and the left atrial pressure rises, precipitating pulmonary oedema.

Predicting left heart failure with echo in DMVD patients aims to identify parameters that are associated with increased left atrial pressure (>20 mm Hg) and left ventricular filling pressure (> 15 mmHg). There is complex interplay between these parameters in the face of increased left atrial volume, diastolic dysfunction and the compensatory mechanisms instigated by reducing stroke volume. This is what makes diagnosis of heart failure so difficult with echocardiography. There are several parameters that have been found to have fair but variable sensitivity and specificity for prediction of heart failure. The most useful parameters are listed below, and the more of these identified the greater the risk of heart failure. Importantly, these are not diagnostic of CHF and we always need to correlate with clinical evaluation (RRR) and, if possible, thoracic radiographs.

Echocardiographic parameters predictive of elevated LA filling pressure, a marker of congestive heart failure in the dog

MV E peak >1.25 m/s

E/E' >12 (E/E' value >9.1 indicated a 95% probability of LAP >20 mmHg.

Inversely, an E/E' value <6 indicated a 95% probability of LAP <20 mm Hg.

IVRT <45 ms

E/IVRT >2.5

PV/PA value >1.7 in 2D predicted CHF with an accuracy >90%, a sensitivity of 96%, and a specificity of 91%; and >1.6 in MM predicted CHF

LA:Ao > 2.5

TDI derived Tei index > 0.8 (lateral wall)

PWD derived Tei index >0.75 (lateral wall)

If these are not present, the dog is unlikely to be in heart failure. If some are identified, the dog is possibly in failure but should be correlated with clinical signs, SRR and thoracic radiographs.

Of all the sonographic studies, echocardiography is the most reliant on good technique and knowledge to ensure accurate results, and interpretation of the findings requires specialised training and much experience. You should be very careful interpreting results of non-specialist cardiac examinations.

So is my patient in heart failure?

Are the clinical signs consistent?

Heart failure is characterised by clinical signs of exercise intolerance and increased respiratory rate and effort due to pulmonary oedema. If you are diagnosing CHF based on radiographs of a patient that does not have consistent clinical signs you should be questioning your diagnosis.

Is the heart enlarged?

Normal VHS is generally <10.7V, up to 11V for Labradors, Cavalier King Charles spaniels, greyhounds and boxers. The trachea should deviate ventrally away from the spine. The carina elevates as the heart becomes taller. Normal craniocaudal dimension should be 2.5 intercostal spaces for deep chested dog breeds and up to 3.5 intercostal spaces in barrel-chested breeds.

Although less common, it is possible to have heart disease without heart enlargement (especially in diseases that do not cause volume overload, such as hypertrophic cardiomyopathy, pulmonic and aortic stenosis, or with acute chordae tendinae rupture). Cardiomegaly is a better indicator of volume overload disease (eg mitral valve disease, dilated cardiomyopathy) and can be measured to monitor progression of disease. In Cavalier King Charles Spaniels with mitral valve disease, rate of change of VHS has been shown to be predictive of onset of congestive heart failure, with rate of change per month increasing significantly in the last year before onset of CHF (Lord et al 2011). That's a lot of radiographs though...

Is the left atrium enlarged?

Many times this has helped me with the interpretation of some challenging radiographs. In most of our patients, left sided heart failure develops only after significant cardiac decompensation and left sided volume overload. This means the left atrium should be severely enlarged. In dogs this is observed radiographically as a wedge opacity at the caudodorsal heart border and often a circular opacity superimposed centrally on the caudal cardiac silhouette on the VD/DV. A VLAS > 2.6 is fairly reliable, and >3 is very sensitive indicator of significant left atrial enlargement.

If the caudodorsal heart border is normal it is extremely unlikely that the patient has left heart failure. The exception to this rule is if a patient has developed fulminant heart failure after rupture of a chordae tendinae relatively early in the disease process. These dogs can have relatively small hearts but should have a very loud murmur.

On echo/TFAST/POCUS, LA:Ao ratio >1.7 is abnormal, > 2 CHF more likely, > 2.5 is strongly supportive.

Are the pulmonary veins enlarged?

In my experience, large pulmonary veins are not consistently visible in patients with congestive heart failure. If you see them, it lends weight to the diagnosis, but if you don't this does not mean the dog isn't in heart failure.

In the early stages of heart failure there is a general increase in size and visibility of the small veins in the lung. This is easiest to appreciate if you have an earlier thoracic radiograph for comparison. There are also semi-objective measurements for vein size. The cranial lobe vessels are evaluated on the lateral radiograph and in the 4th intercostal space the vein should be less than the diameter of the proximal part of the 4th rib. The caudal lobe veins are seen on the VD/DV and theoretically should be less than the diameter of the 9th rib where they intersect. All veins should be similar in size to the paired artery.

TIP - caudal lobe vessels are really hard to see on a VD because the caudal lungs are poorly inflated when the dog is on its back. If you are interested in ruling out CHF then get a DV too. If there is significant oedema it will be difficult to make out the vessels due to increased pulmonary opacity. Don't get thrown by the different shape of the heart on the DV.

Is there evidence of cardiogenic pulmonary oedema?

Fluid in the interstitium causes hazy increase in opacity with preserved vascular markings, progressing to flooding of the alveoli and an alveolar pattern where the vessels are masked and you may see some air bronchograms.

In dogs, this occurs first close to the left atrium, hence the “hilar” distribution described in texts. This is due to a combination of increased hydrostatic pressure, variations in capillary length and lymphatic drainage. It progresses in the caudal lobes next, more severe towards the hilus of the lobes and less severe in the periphery (unlike non-cardiogenic oedema). The pattern is usually symmetrical, or slightly worse in the right caudal lobe than the left.

In large breed dogs, the distribution can sometimes be more patchy, have a broncho-interstitial appearance, and can settle dependently, which can be a trap. Look for the left atrial enlargement to help direct towards oedema.

What if you can't decide?

You can administer a dose of frusemide and repeat the radiographs 3-6 hours later. Cardiogenic oedema will improve, sometimes this is spectacular. If you have previous thoracic radiographs of the patient then review them for comparison. If the changes are really subtle and the clinical signs aren't severe, then you can always have the owners monitor SRR for a few days and report back. If it is < 30 / min consistently then you are unlikely to be dealing with CHF.

Get help from a radiologist

These days general practitioners are blessed with a plethora of teleradiology providers that can help with interpretation. I have two pieces of advice for you with regard the use of teleradiology:

1. If you have taken rubbish radiographs then the best radiologist in the world will struggle to give you useful information, so take care every time and get the best images you can. You owe it to your patients.
2. Provide great history and signalment and always provide the clinical question your imaging study is aiming to answer. Be sure to say if the patient has been treated with anything (especially diuretics), and if there has been any response to treatment. Don't just cut and paste your clinical record – summarise the clinical exam findings and distil the history into pertinent facts.

A good interpretation should give you more than a list of differentials, it should give you the most likely differential and why, and what you could do to refine the differential list. You won't get any of this if you don't provide good history. I like to speak with the clinician and discuss the patient and radiographic findings and many times this has significantly improved accuracy and patient outcomes.

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Imaging and interpreting lungs better: Make the most of your radiographs and when do you really need CT?

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Recent research into lung anatomy and pathophysiology and the growing use of CT to investigate lung disease are changing the way we interpret radiographs.

In this lecture there are tips to gain confidence in navigating the lung, how to refine the differential diagnosis list with lesion distribution and a brief look at bronchial and bronchiolar disease and the use of CT.

1. Lung gross anatomy

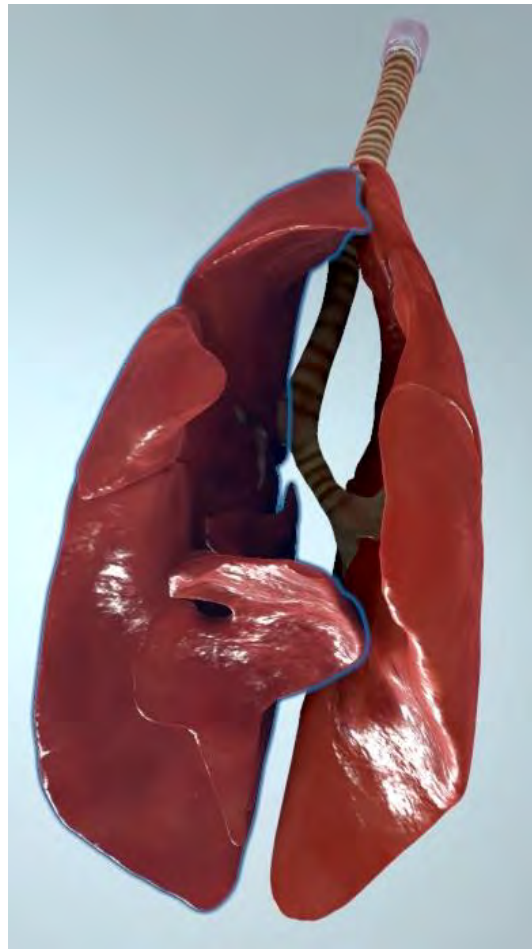
The first secret to improving your skills in interpretation of lung disease is to go back to basics and understand the anatomy of the lung. The lobar anatomy and bronchial tree are highly predictable and when you use it as a road map you can navigate the lung with confidence. The following figures show the lobar anatomy of the dog. The junctions between the lobes are important to recognise because these will correlate with fissures in the presence of pleural effusion, and with lobar signs when we have consolidating lung disease. Recognising these is one of my top tips to help localise disease and refine differential diagnosis lists, particularly useful with identifying pneumonia, lung lobe torsion and lung masses. We will review several case examples in the lecture.



Figure 1. Plastinated canine right lung lobes (heart, left lung lobe removed) (Credit: Online Veterinary Anatomy Museum)



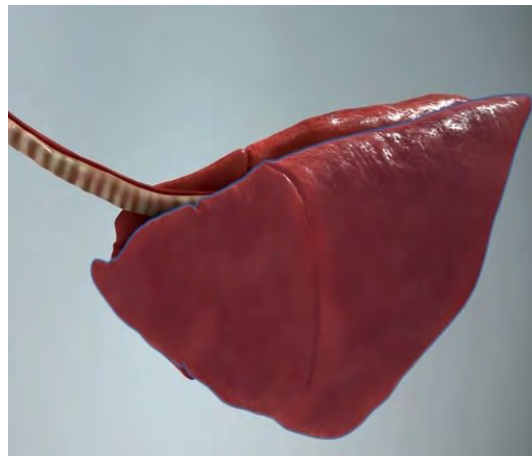
a



b



c



d

Figure 2. Canine lung models generated in EasyAnatomy

- Craniocaudal view of the canine cranial lungs, looking at the thoracic inlet
- Ventrodorsal oblique view – the right lungs including the accessory lobe are outlined in blue. Note the deep fold that envelops the caudal vena cava. That is why accessory lobe consolidation masks the caudal vena cava.
- Pleural surface of the right lungs. Note the cardiac notch – the natural window in the lung through which the heart is accessed for echocardiography.
- Pleural surface of the left lungs. Note the small fissure between the cranial and caudal subsections of the cranial lobe. This extends internally as a pleural distinction so that the cranial portion can become consolidated separate from the caudal subsection.

2. Lung radiographic anatomy

The normal structures visible on a radiograph are the trachea, mainstem and lobar bronchi, pulmonary veins and arteries. Other than a small amount of fat in the mediastinum and pleural space, the only contrast in the thorax is provided by the air within the airways and alveoli, which is why diagnostic thoracic radiographs maximise air in the lung. Anything that displaces air will increase opacity. This can occur within the alveoli, in the interstitial space or in the vessels or bronchial walls.

While not visible on every radiograph, the bronchial tree is important to identify and trace to the best of your ability, ensuring evaluation of all lobes systematically. Practise tracing these on every set of radiographs. Also trace the lobar margins in your mind (they won't be visible when normal).

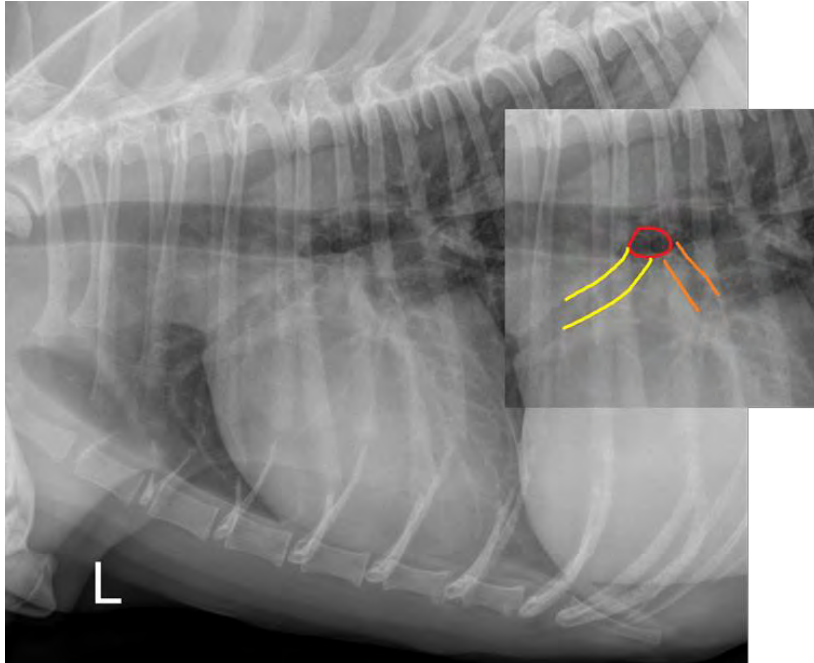


Figure 3. Left lateral radiograph of a dog, optimal for imaging the right hemithorax. The right cranial lobe bronchus (yellow outline) and middle lobe (orange outline) are seen arising separately from the carina (red outline).

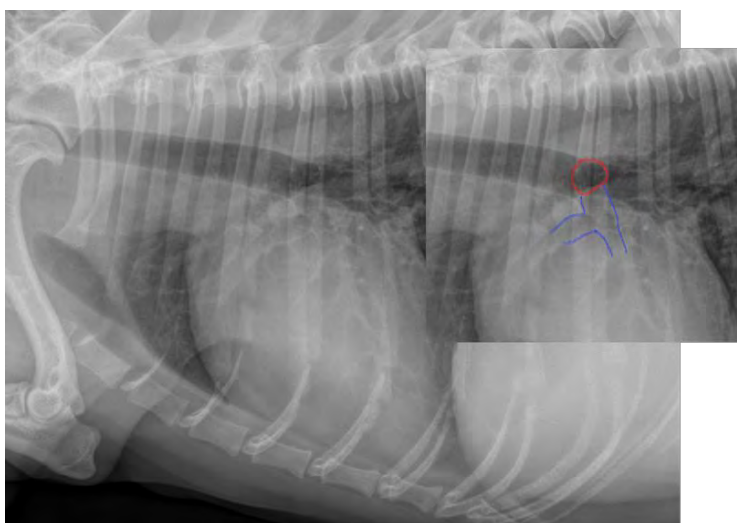


Figure 4. Right lateral radiograph, optimal for imaging the left hemithorax. The carina is circled in red in the detail inset, trace it ventrally and you will see the short common left cranial lobe bronchus that then branches into cranial and caudal subsections (outlined in blue). Also visible is the right cranial lobe bronchus tracking cranially from the carina.

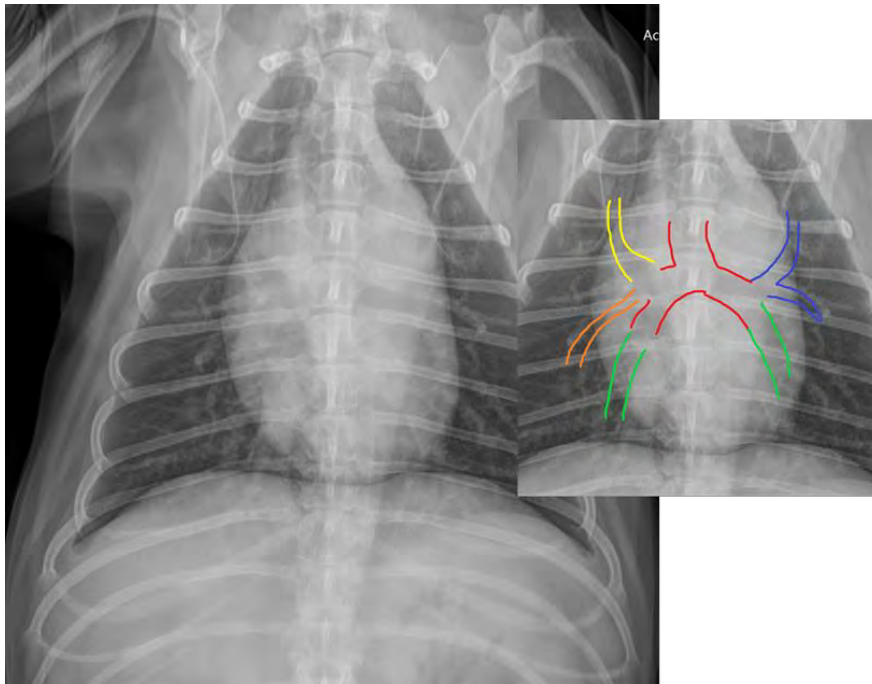


Figure 5. VD radiograph of a dog thorax. The trachea and mainstem bronchi are outlined in red, the caudal lobes in green. Yellow is right cranial lobe, orange right middle lobe, blue left cranial lobe. The accessory lobe bronchus is not visible in this patient.

When evaluating the lower airways, the normal number of bronchial rings visible is not defined. Scant limited literature suggests that there is mild increase in parenchymal mineralisation and equivocal bronchial wall thickening with age in dogs, variably detectable with radiography and CT. Many digital systems create a lot of small rings in the lung parenchyma as well as the soft tissues, likely detector artifact, and some structures other than bronchial walls can cause rings on a radiograph.

2.2 Radiographs of a bronchial tree cast - the surprising role of the bronchioles

In a short communication published in 2023, Scrivani and Percival described the radiographic and CT appearance of two silicone casts of canine bronchial trees housed in the Cornell veterinary anatomy museum. The appearance of the larger airways (trachea, principal, lobar and segmental bronchi) was of tapering, branching and radiating opacities as would be expected. What was unexpected, however was the effect the casts of the smaller airways – the bronchioles (~ 0.5 mm luminal diameter) – had on the radiographic and CT images. Bronchioles are the small airways with no cartilage in their walls and they deliver air to the alveoli. A terminal bronchiole feeds a pulmonary acinus, the smallest functional unit in the carnivore lung.

In the radiographs of the silicone cast of the canine bronchial trees, the bronchioles produced a speckled pattern resembling micronodules and created the illusion of thick, small-diameter rings that mimicked thickened bronchiole walls. This created the appearance that conforms to the schema of a bronchial lung pattern with increased numbers of thickened small gauge rings in the pulmonary periphery (Fig.6). Of course, the silicone casts had no gas in the airway lumens so this pattern in a live patient would in fact represent occluded bronchioles rather than thickened, air-filled bronchi. Blood vessels also probably contribute to this pattern in the live patient.

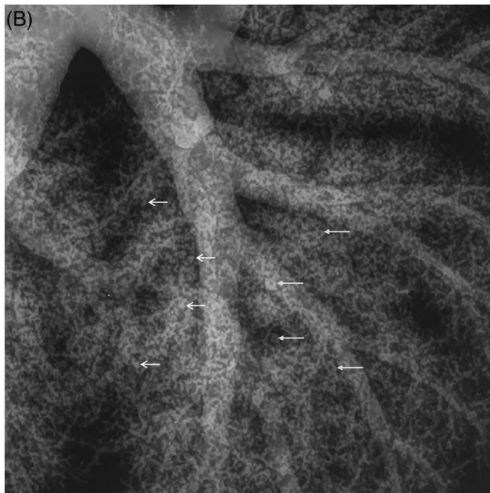


Figure 6. Reproduced from Scrivani and Percival (2023). Detail of a radiograph of a small dog bronchial tree silicone cast. The arrows highlight small gauge rings created by superimposition of the tiny bronchiolar plugs. This can mimic thickened bronchiole walls.

This radiographic pattern would, therefore, be expected in diseases where bronchiole lumens are occluded by thickened walls, mucus, cellular infiltrates etc (bronchiolitis). Also, the presence of peribronchovascular micronodules caused by lymphatic or haematogenous (miliary) spread of disease can mimic bronchiolar plugging and create this radiographic appearance, thus explaining the presence of thick, small gauge rings in miliary lung patterns (Fig. 6).

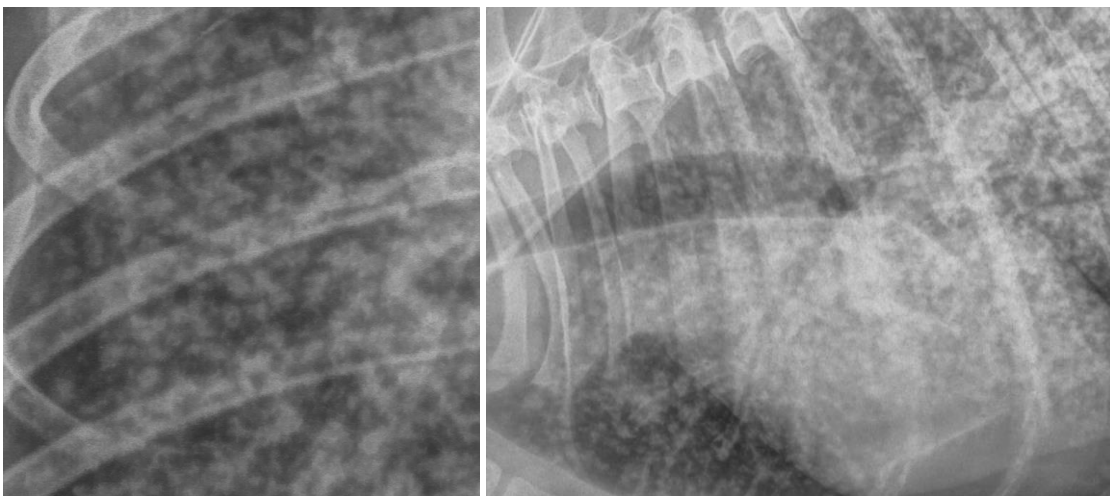


Figure 7. Details from thoracic radiographic study of a 9 year old kelpie dog with a lung mass and diffuse miliary lung pattern, attributed to bronchoalveolar carcinoma metastasis. Note the thick ring opacities mimicking thickened bronchiolar walls, likely caused by micronodules in the peribronchovascular space.

3. Refining your differential list

3.1 Localise your lung disease

While recognising lung patterns is helpful, the location of the pathology in the lung is more useful when deciding on the likely cause of the disease. Ventral distribution of alveolar and interstitial disease is very typical of aspiration-related disease and lobar pneumonia. If there is consolidation dorsal to the carina, this is almost never primary pneumonia and you should be looking for other diseases such as lung lobe torsion, pulmonary masses that cause obstructive consolidation, or rare multifocal disease such as haemorrhage or haematogenous pneumonia. Concurrent thoracic findings such as left cardiomegaly, thoracic lymph node enlargement, pleural effusion will also help refine your differential diagnosis.

Cardiogenic oedema can appear as interstitial, alveolar or bronchointerstitial pattern, but its distribution is nearly always hilar and caudal. Large breed dogs can have more patchy and ventral distribution so the presence of a large left atrium should raise oedema up the list with several different distributions.

Conditions with diffuse distribution are chronic bronchitis, asthma, film artifacts of underinflation or quantum mottle. The first question to ask when you have a diffuse interstitial pattern – is this technique related?

Conditions arising from the bronchial tree or bronchovascular bundle are bronchocentric. This can include nodules associated with bronchial plugging, which are important not to mistake for neoplasia, but this may not be possible with radiographs alone.

4. Bronchial or Bronchiolar disease?

We know that larger airway disease can cause bronchial wall thickening with an air-filled lumen that will create larger gauge ring and linear opacities branching and radiating from the pulmonary hilus.

Bronchial disease reported in the dog is predominantly acute and chronic bronchitis and dynamic airway disease. Eosinophilic bronchopulmonary disease is an extension of interstitial lung disease that can manifest in a primarily bronchial form. In cats, bronchial disease is usually limited to asthma and infectious or inflammatory bronchitis.

Diseases affecting the bronchioles, however, will create tiny, thickened rings and miliary type patterns. This raises the question – what diseases affect the bronchioles? Outside of histopathology lectures, have we ever considered these as clinically important anatomic structures? Bronchiolar disease can be primary, usually in response to an inhaled pathogen or irritant, can develop as an extension of disease from the larger airways or from the structures of the pulmonary interstitium (interstitial lung diseases).

5. Bronchiolar diseases in cats

Bronchiolar diseases cause significant morbidity in people and are poorly described and diagnosed in our veterinary patients, however with increasing use of CT and lung biopsy, our understanding of bronchiolar disease is gaining rapidly. While there is almost no literature on the conditions in dogs, Reinero et al (2019) published a pivotal review of bronchiolar disorders in cats, with a proposed classification system as follows:

- Primary bronchiolar disorders
 - o Constrictive/obliterative bronchiolitis
 - o Mineral dust airway disease
 - o Infectious bronchiolitis
 - o Other primary bronchiolar variants
- Secondary bronchiolar disorders
 - o Extension of large airway disease
 - Asthma
 - Chronic bronchitis
 - Parasitic bronchitis
 - Bronchiectasis
 - o Component of interstitial lung diseases
 - Bronchiolitis obliterans with organising pneumonia/cryptogenic organising pneumonia
 - Bronchiolocentric interstitial pneumonia/airway centered interstitial fibrosis

In the cases described in this review, it was shown that progressive or poorly responsive signs of bronchial disease were often explained by extension of the disease process into the bronchioles.

5.1 Diagnosis of bronchiolar disease is made with CT, BAL and lung biopsy

Diagnosis of bronchiolar disease or bronchiolar involvement can be challenging and requires a combination of detailed clinical history, diagnostic imaging, BAL and histopathology. The radiographic appearance is highly variable, with reported normal radiographs, bronchial, bronchointerstitial, mixed alveolar and bronchointerstitial lung patterns, many with nodules (Hahn et al 2018, Wylie et al 2019, Phillips et al 2000). These radiographic patterns have a wide range of differential diagnoses that importantly include neoplasia which can pre-emptively curtail diagnostic workup if it is assumed that prognosis is poor.

The distinction between bronchiolar disease and interstitial lung diseases is often based on CT imaging characteristics which can locate the pathology within, adjacent to, or distant from the airways or bronchovascular bundles. This helps refine the differential diagnosis list and direct the collection of samples for pathology.

5.2 CT findings in bronchiolar disease of cats

5.2.1 Cats and the “tree in bud” sign

The “tree in bud” sign is a hallmark CT finding of bronchiolar disease in cats. It describes the CT appearance of multiple areas of linear branching pattern with terminal and adjacent nodules, resembling a budding tree. Using a maximum intensity projection (MIP) can improve conspicuity. This sign is most often created by bronchioles filled with exudate or mucus, bronchiolar wall thickening, or bronchovascular interstitial infiltration (eg by lymphoma). While a normal bronchiole is 0.5 – 2 mm in diameter, some of these plugs can be very large, reported up to 9 mm, and can create significant nodular opacities on thoracic radiographs. These are distributed as satellites to a lobar bronchus, or when very severe can affect the segmental or lobar bronchus. On radiographs the end-on plugged bronchi and bronchioles take on a nodular appearance, however on CT the true tubular shape of many of these abnormalities is better able to be appreciated (Hahn et al 2019). It is of critical importance to recognise these or be aware of their potential, as the alternative differential is usually metastatic neoplasia which frequently leads to euthanasia.

Infectious disease is an important mimic of non-infectious inflammatory and fibrotic disease with the same CT findings, and depending on the type and extent of infection, may be treatable with a better prognosis and potential for cure (Reinero et al 2019) so a BAL for cytology and culture, or lung biopsy, can be critical tests for optimal patient outcomes.

Other CT findings described in these patients include bronchial wall thickening (very common), peribronchial ground glass densities, parenchymal bands, air trapping and reduced local arterial perfusion leading to mosaic perfusion patterns.

6. Bronchiolar disease in dogs

Bronchiolar disease is limited to sparse case reports, and interestingly, the tree in bud sign has not been described in the imaging (based on my search of the literature). It remains to be seen whether a similar or different spectrum of bronchiolar conditions will be described in dogs as have been in cats, but there is no doubt that once we have an awareness of their existence we are far more likely to recognise them in our patients in future.

7. So when do we need CT?

Thoracic radiographs remain the first line of imaging in small animal patients. Don't feel you always need CT, however you should be thinking about CT for evaluation of lower airway disease, particularly when clinical signs are progressive or poorly responsive to treatment. Early diagnosis of bronchiolar disease may prevent or slow progression to fatal fibrosis. CT is very useful when there are complex mixed patterns, and with poorly defined nodules, where it will assist with differentiating inflammatory from neoplastic disease, guide sampling, and assist with prognosis. With thoracic masses CT is often essential for accurate

localisation within the lung or mediastinum and is the test of choice for staging and surgical planning.

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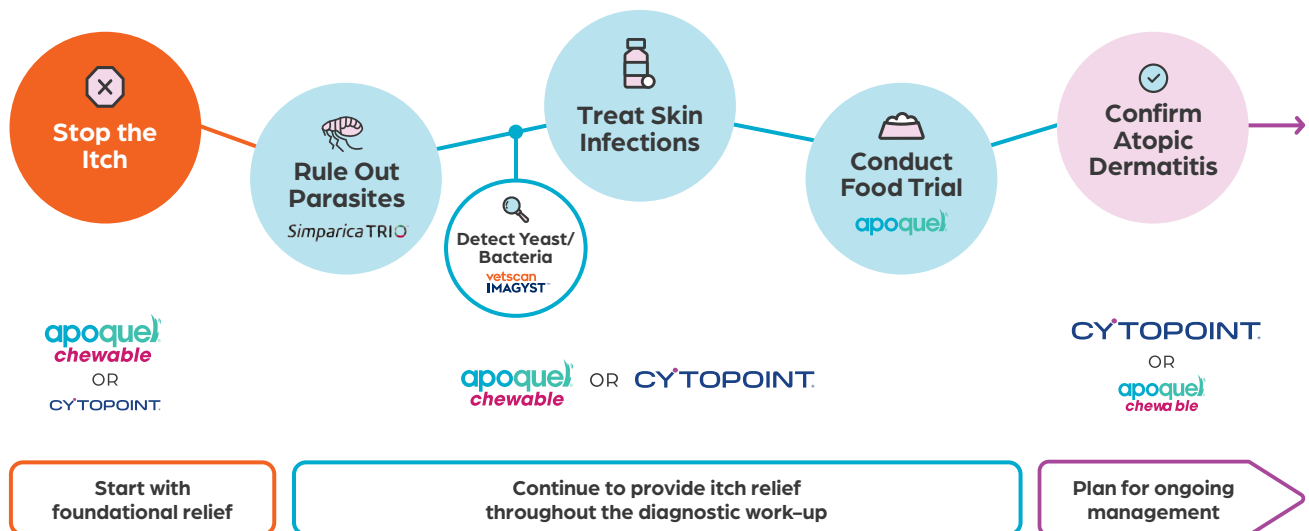
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Aerodigestive disorders: How many of our respiratory sign patients actually have oesophageal disease?

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One of the most important advances in respiratory medicine in the last decade has been the understanding of the link between laryngeal and oesophageal dysfunction and chronic respiratory signs. More and more research in this area proves how important it is for practitioners to be aware of aerodigestive disease and how to investigate it further.

Aerodigestive disorders

The aerodigestive tract encompasses respiratory and upper digestive structures including the pharynx (nasopharynx, oropharynx and laryngopharynx), larynx, bronchi/bronchioles, pulmonary parenchyma, oesophagus, and oesophageal gastric junction. The term aerodigestive disorder covers a wide range of conditions that develop due to complex interaction between the airways and alimentary system (aspiration associated respiratory syndromes). Typically, they involve failure in airway protection, abnormal swallowing, gastro-oesophageal reflux, or a combination of these, resulting in aspiration and respiratory disease.

In people, aerodigestive disorders contribute to the pathogenesis and progression of respiratory conditions including chronic cough, asthma, bronchiolitis, idiopathic pulmonary fibrosis (IPF), and chronic obstructive pulmonary disease (Grobman 2023). Treating the underlying alimentary dysfunction is important to improving patient outcomes, however the alimentary signs are often lacking and therefore can go unrecognised.

In veterinary patients, aspiration pneumonia is the most recognised of the aerodigestive disorders but there are many others such as laryngitis/pharyngitis, otitis media (via reflux into the Eustachian tubes), bronchiolitis, ARDS, sleep apnoea, laryngeal paralysis and a wide range of bronchial and interstitial lung disease.

Chronic cough

Investigating chronic cough is common in veterinary practice and typically involves thoracic radiography, advancing to CT, bronchoscopy and BAL when feasible or available. In many patients, treatment trials are instigated with no investigation due to cost constraints in our clients.

Thoracic radiographs are very insensitive for diagnosing the most common causes of coughing in our patients (lower airway inflammation and dynamic airway disease) but are important for excluding other causes such as heart failure, lung masses, pneumonia and severe airway or interstitial lung disease.

Like the situation in human medicine, we are now recognising that many patients with chronic cough have subclinical oesophageal or swallowing disorders causing or contributing to respiratory signs. Coughing may be elicited by stimulation of the oesophageal-bronchial reflex which involves vagally-mediated bronchoconstriction secondary to acidic stimulation of the distal oesophagus. Extra-oesophageal reflux of acid or non-acid gastric secretions (pepsin etc) to the larynx or pharynx can also trigger a cough. If there is failure of airway protection then aspiration into the trachea will trigger coughing and can perpetuate chronic lower airway inflammation. Often there is a complex interplay between airway and oesophageal disease, forming a cycle of exacerbation between upper airway obstruction, increased gastric reflux, aspiration and airway inflammation and so on. This is particularly evident in patient with BOAS, hiatal hernia, megaesophagus or chronic vomiting.

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Hopper, B - Aerodigestive disorders: How many of our respiratory sign patients actually have oesophageal disease?

Videofluoroscopic swallow studies (VFSS)

Diagnosis of these conditions requires functional investigation of swallowing, breathing and oesophageal motility which is achieved with videofluoroscopy swallow studies (VFSS). This test is restricted to specialty practice but an understanding of its use and benefits is important for general practitioners so they can advise clients and recognise when it will be of benefit in their patients.

VFSS need to be performed in the conscious animal, ideally in a normal physiological position (standing). A thorough evaluation includes assessment of the oral (prehension difficulties), the oropharyngeal/cricopharyngeal phase (coordination of bolus movement from the oral cavity to oesophagus) and the oesophageal/gastro-oesophageal phase (movement of the bolus through the oesophagus into the stomach). Each phase must be evaluated using liquid barium, soft food/barium and kibble/barium mixtures for a thorough assessment. Radiographic oesophagrams are suboptimal for evaluation of focal or intermittent pathology (which can be missed) and poor for evaluation of the oropharyngeal phase.

Coughing in dogs with no oesophageal or gastrointestinal clinical signs

Recent investigations have shown that a large proportion of dogs (25/31, 81%) presenting for chronic cough with no reported oesophageal or gastrointestinal clinical signs had VFSS abnormalities causing or contributing to their respiratory signs (Grobman et al 2019). In this lecture we will review the VFSS technique and some of the conditions it can identify that are important causes of aerodigestive disease.

Conclusions

While VFSS is not available to most practitioners, identifying dogs with disorders of the pharynx, oesophagus and stomach as a primary source or contributor to their respiratory signs is imperative if we are to understand and treat them effectively. This condition is common in dogs with unremarkable thoracic radiographs and no gastrointestinal signs, and diagnosis requires multimodal and multidisciplinary investigation that starts in general practice.

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The mediastinum: Recognising and diagnosing disease in this space

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The mediastinum is the mysterious thoracic space that contains all the important stuff but is so hard to evaluate on a radiograph. In this lecture we will use cross sectional imaging to simplify the anatomic complexities of the mediastinum and de-mystify signs of disease in this space to really elevate your radiographic interpretation skills.

Anatomy of the mediastinum

The mediastinum is a potential space bounded by the reflections of the right and left pleural sacs, which themselves consist of a continuous transition between mediastinal, diaphragmatic, costal and pulmonary pleura. In most dogs and cats, the mediastinum is fenestrated, allowing free communication between the two pleural sacs. The mediastinal space is continuous with the fascial planes of the neck at the thoracic inlet, and the retroperitoneal space through the aortic hiatus, important to remember for causes and radiographic signs of pathology in this space.

The mediastinum is often divided roughly into three parts: the cranial mediastinum being cranial to the heart, the caudal mediastinum caudal to the heart, and the rarely used term middle mediastinum, containing the heart. We also divide it into dorsal and ventral portions.

The mediastinum contains all the structures that pass into or through the thorax, including the heart and major blood vessels, trachea, oesophagus, lymph nodes, nerves (vagus, phrenic), thoracic duct and thymus.

Radiographic anatomy of the mediastinum

Mediastinal reflections

There are two reflections of the mediastinal pleura that can be recognised on a radiograph: the cranioventral; and caudoventral mediastinal reflections (Fig. 1, 2 and 3). The sternal lymph nodes, internal thoracic arteries and veins, and thymus live in the cranioventral reflection. The thymus causes mild to moderate widening of the cranioventral reflection which takes on a “sail” shape on the VD, mimicking a jib on a yacht, until about 6 months of age. There is usually just a little bit of fat in the caudoventral reflection. The sternal lymph nodes drain not only the thoracic wall and ventral thorax, but also the cranioventral abdomen and peritoneal cavity. The caudal mediastinum appears radiographically as an almost insignificant structure, visualised as a thin web of tissue in the caudal thorax on the left side, between the heart and diaphragm. The mediastinum is, like the pleural space, a potential space that may become wide when there is free mediastinal fluid.

A third, the vena caval mediastinal reflection, is the fold that envelops the caudal vena cava as it traverses from the diaphragmatic caval hiatus to the right atrium. This is only recognised on CT (Fig. 3).

The remainder of the mediastinum is recognised by its structures.

Cranial mediastinum

The dorsal portion of the cranial mediastinum is bounded by the paired longus colli muscles running ventral to the spine, the cranial vena cava on the right and brachiocephalic trunk and left subclavian artery on the left (Figs 2, 4). The trachea in this portion is usually slightly deviated to the right due to the left aortic arch structures. Other structures in the cranial

dorsal mediastinum are the oesophagus and the cranial mediastinal lymph nodes, which drain the head and neck and connect with the sternal and tracheobronchial lymph nodes.

The normal width of the cranial mediastinum varies. On the ventrodorsal or dorsoventral projections, the mediastinum is superimposed on the spine, and the width is usually less than approximately two times the width of the vertebra. In obese patients, the cranial mediastinum may be widened by fat so evaluating the amount of subcutaneous fat along the thoracic wall can help determine if there is likely to be a large amount of mediastinal fat.

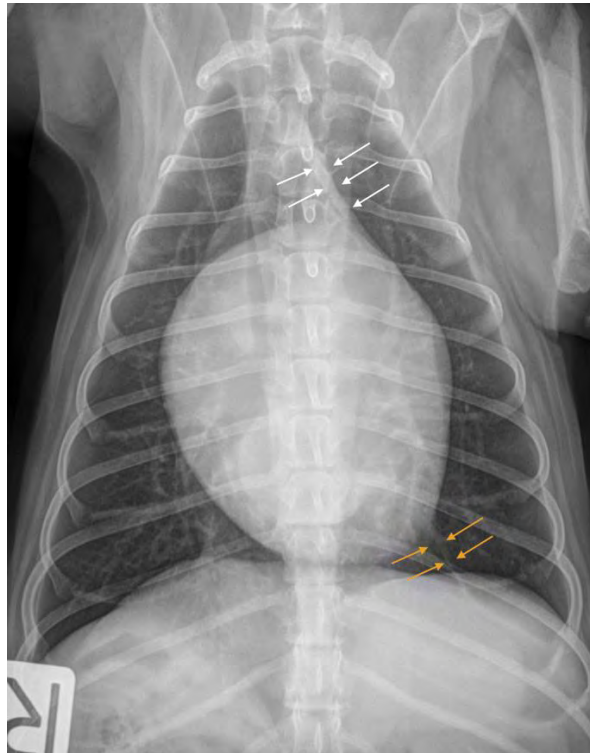


Fig 1. VD radiograph of a dog with left cardiomegaly. The cranioventral mediastinal reflection is marked with white arrows. The caudoventral mediastinal reflection is marked with yellow arrows.

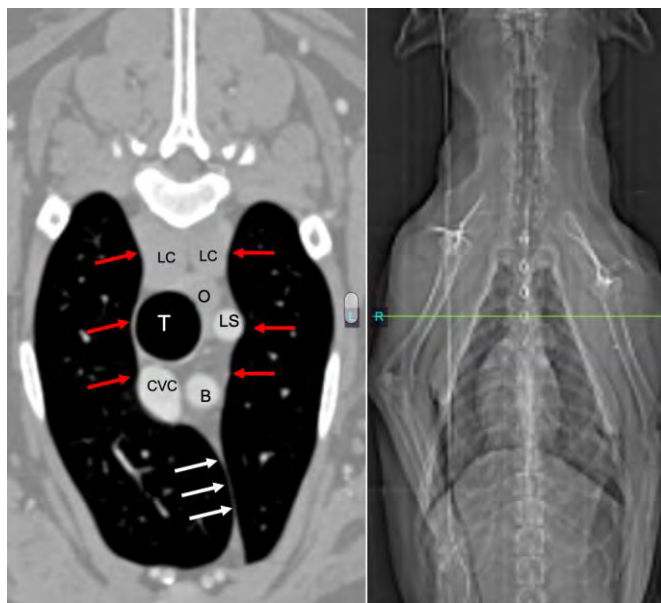


Fig 2. Transverse contrast enhanced CT image of the cranial mediastinum, soft tissue window, at the level of the reference line indicated on the DV topogram to the right of the figure. Right is displayed to the left of the image. The red arrows outline the dorsal cranial mediastinum which is bounded by the paired longus colli muscles (LM), trachea (T), oesophagus (O), cranial vena cava (CVC) on the right and brachiocephalic trunk (B) and left subclavian artery (LS) on the left. The white arrows indicate the obliquely oriented cranioventral reflection, separating the left and right cranial lung lobes.

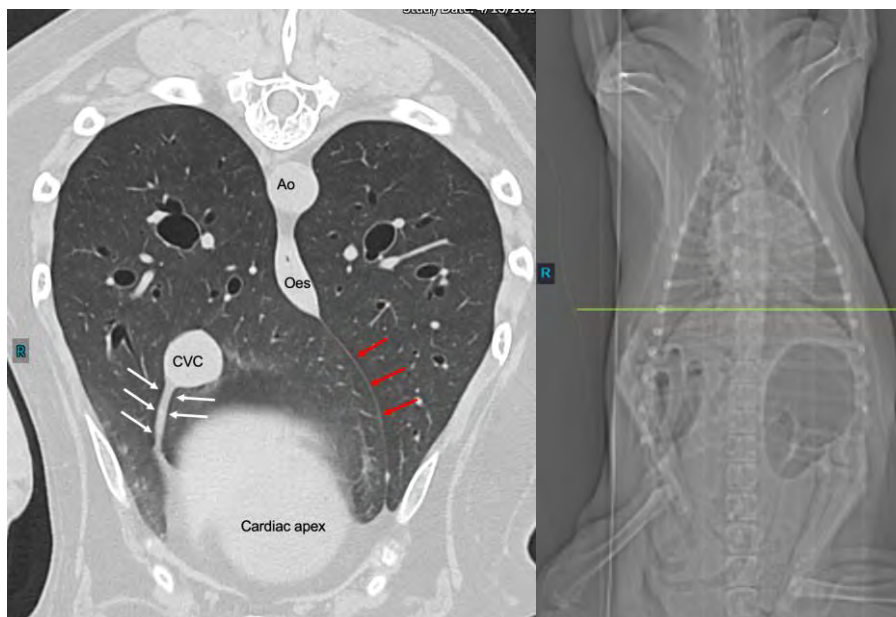


Fig. 3 Transverse CT displayed in lung window at the level of the caudal mediastinum. The white arrows indicate the caudal vena cava mediastinal reflection which indents into the accessory lung lobe. The red arrows indicate the caudal ventral mediastinal reflection forming the boundary between the accessory lung lobe and the left caudal lung lobe. There is fat in the ventral mediastinum, surrounding the heart. CVC: caudal vena cava, Ao: Aorta, Oes: Oesophagus.



Fig 4. VD radiograph detail of the cranial mediastinum. The white arrows indicate the lateral margins of the dorsal mediastinum.

Middle mediastinum

This is the cardiac domain. The descending aorta forms the left lateral boundary of the dorsal middle mediastinum and enlargement (post stenotic dilation, patent ductus arteriosus, aneurysm) will cause a bulge in this contour. The right lateral margin is not readily identified. The cardiac margins form the lateral margins of the ventral middle mediastinum. Tracheobronchial (hilar) lymph nodes that drain the lungs and communicate with the cranial mediastinal nodes are distributed around the trachea and mainstem bronchi over the base of the heart (Figs 5, 6).

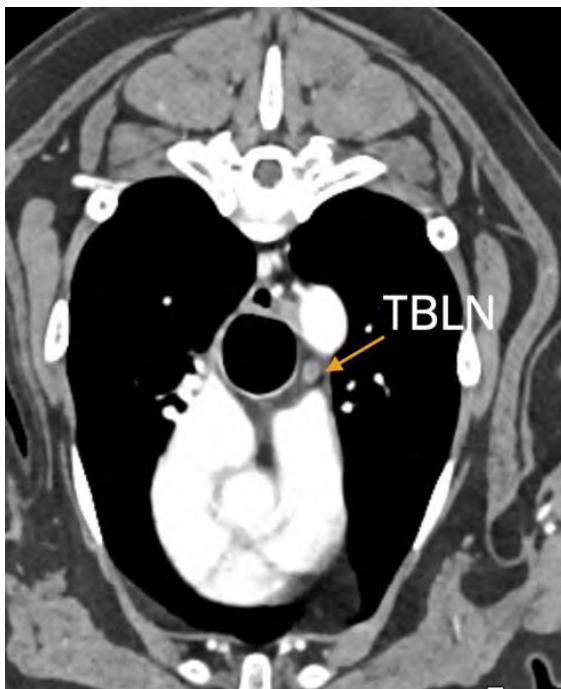


Fig 5. Transverse CT at the heart base, contrast enhanced, displayed in a soft tissue window. One of the tracheobronchial lymph nodes (TBLN) is labelled between the pulmonary trunk, tracheal and aorta.

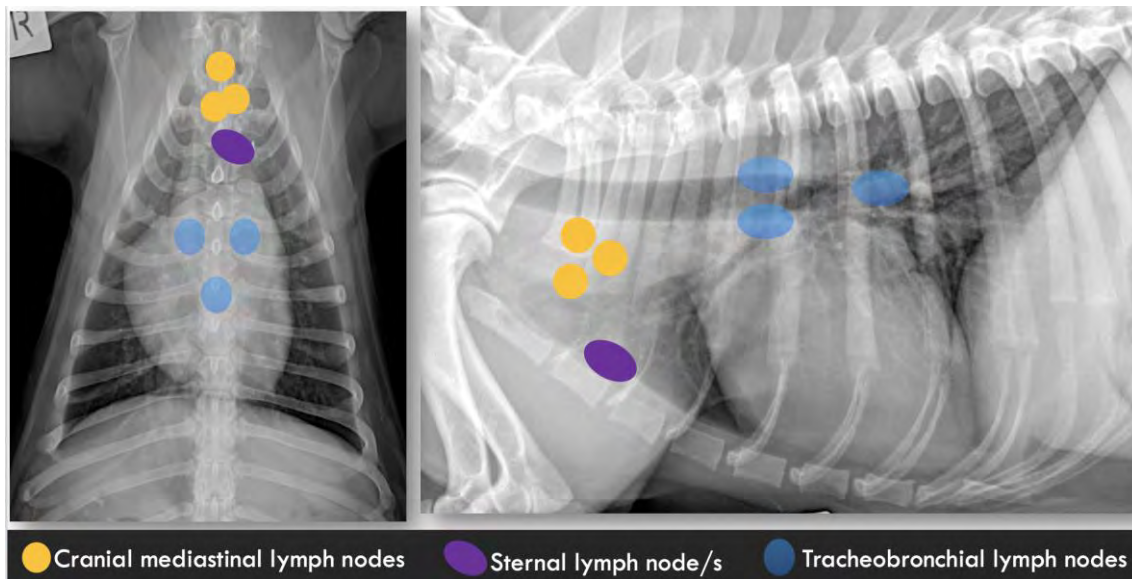


Fig 6. VD and right lateral radiographs of a dog thorax with the stylised position of the thoracic lymph nodes marked per the key. Note the coloured shapes are much larger than the normal nodes, for clarity.

Caudal mediastinum

The caudal mediastinal structures visible on a radiograph are the aorta, the oesophagus and the caudal vena cava. Other structures present but only distinguished with CT include the azygous vein, the thoracic duct and the vagus nerve.

When dilated, the oesophagus expands the lateral margins of the caudal mediastinum and is often visible extending beyond the shadow of the overlying spine and sternum. A small amount of fluid opacity can be present normally in the caudal thoracic oesophagus, particularly in patients that are anaesthetised or sedated, and particularly in left lateral recumbency.

Adjacent to the oesophagus is a closed mesothelial lined cavity that is a cranial extension of the omental bursa that gets separated from the peritoneal cavity when the diaphragm forms. It is to the right of the oesophagus, between the heart base and the diaphragm. If this space is contaminated (e.g. by oesophageal perforation, migrating plant material etc) it can develop empyema (formerly referred to as a para-oesophageal abscess). This is an important diagnosis to make as the serous cavity is not continuous with the pleura so lavage won't be effective treatment. It needs to be specifically identified, opened and lavaged at surgery.

Mediastinal space pathology

Pneumomediastinum

Air can accumulate in the mediastinum from rupture of the trachea, mainstem bronchus or oesophagus, can track into the chest from the subcutaneous tissues of the neck and axilla, e.g. secondary to pharyngeal stick injury, staking injury, dog bite, motor vehicle accident, surgery, or from the lung. The latter can be very mild and only detectable on CT, or moderately severe.

Spontaneous pneumomediastinum is more commonly identified in sight hounds but has been reported in many breeds. Pulmonary interstitial emphysema develops due to gas leaking from alveoli into the surrounding interstitial lung tissue, and this tracks along the bronchial tree to the mediastinum in a process called the Macklin effect. Pulmonary

interstitial emphysema can be detected on CT as gas within the pulmonary vascular sheaths but is usually too mild to be detected on radiographs. It is more prevalent in sighthounds (Sánchez Jiménez *et al* 2024) but can occur in many breeds, in dogs with and without pulmonary pathology. Pulmonary interstitial emphysema has been reported to be a reliable early indicator of barotrauma in CoVID-19 patients being mechanically ventilated.

If a severe spontaneous pneumomediastinum develops this is more likely to be associated with severe pulmonary disease, such as fibrosis or interstitial pneumonia. I have seen it develop secondary to paraquat poisoning and it has been reported in a young Dalmatian with severe fibrosing interstitial pneumonia.

Air in the mediastinum can rupture the pleura and progress to cause pneumothorax, but a pneumothorax will never progress to a pneumomediastinum. Air in the mediastinum can track caudally into the retroperitoneal space and cause pneumoretroperitoneum which is not usually of clinical consequence.

Radiographic signs of pneumomediastinum

Most of the mediastinal structures are not individually visible due to them silhouetting with one another. Pneumomediastinum allows visualisation of the cranial mediastinal vascular structures, the oesophageal and tracheal serosal surfaces, the aorta margins, and sometimes the heart base structures like aortic and pulmonary trunk. If the air is tracking into the thorax from the neck, there will be evidence of subcutaneous or fascial emphysema.

Pneumomediastinum can cause tachypnoea and discomfort, but is not life threatening. If it progresses to pneumothorax, however then there is the potential for serious clinical disease.

Radiographic signs of mediastinitis

Mediastinal effusion is rare, predominantly associated with severe oesophageal disease (usually rupture) in which case there may be a combination of air and fluid. The radiographic appearance can be very complex, depending on the positioning of the patient and the gravitational effect on the fluid and air. Fluid in this space will reduce the sharpness of the margins (cardiac, cranial vena cava etc) and create a wispy, murky appearance to the radiograph. This usually progresses to a pleural effusion quite rapidly.

Caudal mediastinal/para-oesophageal empyema will form a discrete soft tissue opacity mass effect adjacent to and silhouetting with the oesophagus. It may silhouette with the diaphragm if large.

Mediastinal masses

Mediastinal masses most commonly arise from lymph nodes (lymphoma), thymus (thymoma, thymic lymphoma) and ectopic thyroid tissue (carcinoma). Neuroendocrine tumours are relatively common at the heart base (aortic body tumours also known as chemodectomas) and have a predilection for brachycephalic breeds. Non-neoplastic disease is less common but can consist of reactive lymphadenopathy, branchial cyst, oesophageal disease (polyp, neoplasm, parasitic granuloma), hernia, abscess/empyema, haematoma and aneurysm.

Cranial mediastinal masses can be difficult to differentiate from pulmonary origin masses (Ruby *et al* 2020) but there are some useful features that can help localise a mass to the mediastinum.

1. Mediastinal masses are typically on midline
2. They will displace a mediastinal structure/s
3. They may create an “extrapleural” sign caused by the pleura draping over the mass.

4. They will not have air bronchograms or margins that form acute angles with the thoracic wall.
5. They commonly arise in the location of the cranioventral reflection

Once you have localised a lesion to the mediastinum, next localise it *within* the mediastinum (eg cranioventral, craniodorsal, heart based, caudal dorsal etc.). The mass displacement effect on other structures can help achieve this. For example, an enlarged tracheobronchial lymph node is identical in location to an enlarged left atrium but the lymph node will depress the mainstem bronchi whereas an enlarged left atrium will dorsally elevate the bronchi. With a region isolated, there is a finite number of structures in that space than can cause the mass.

Computed tomography is far more accurate and sensitive for the evaluation of mediastinal and pleural structures, and is almost invariably beneficial for further diagnostic workup, staging and surgical planning.

Thoracic ultrasound is ideal for guidance of sampling as this has less complications and takes significantly less time than with CT guidance. It can be surprisingly difficult to differentiate mass origin with ultrasound however there are signs such as vascular and bronchial markings and shred signs that indicate pulmonary origin. Static masses that do not move with respiration are more likely mediastinal or pericardial in origin.

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Pleural effusion clinical pathology: How to get the most out of your samples

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Specialist Clinical Pathologist – Antech Diagnostics

Pleural space disease in the dog and cat can be extremely difficult to manage and diagnose due to concern regarding patient stability and the ability to perform accurate imaging, diagnostic, and therapeutic techniques in patients with respiratory compromise. Despite this, respiratory dysfunction is an emergent condition that if left unresolved can lead to severe physiologic compromise and even death, so imaging, diagnostics, and therapeutic interventions must be performed but are often dictated more by the patient's condition and stability than the clinician's or owner's decisions or convenience. The clinician with a case management plan.

Pleural space disease can be caused by fluid, air, soft tissue, or abdominal organs within the pleural space and most often presents as patients with a shallow breathing pattern, tachypnoea, and paradoxical breathing. On auscultation there is often reduced breath/adventitious sounds and the location of the decreased sound can help to differentiate causes (i.e. ventral indicates pleural effusion is most likely whereas dorsal indicates pneumothorax is most likely). Pleural effusion is commonly seen with cardiac disease, pyothorax, chylous effusion, neoplasia, and hypoalbuminaemia. Pneumothorax is most often seen secondary to trauma, foreign body, neoplasia, or bulla rupture. Diaphragmatic hernias are most commonly seen secondary to trauma and may also cause pleural effusion.

As pleural effusions are predominantly a secondary condition, a thorough investigation beyond fluid analysis should always be performed and include a full physical examination +/- baseline CBC/biochemistry/UA and thoracic imaging (radiography / ultrasound) to evaluate for primary disease states (e.g. cardiac murmur/enlargement, jugular distension, neoplasia, coagulopathy, inflammatory disease, fever, uveitis, abdominal enlargement/fluid accumulation, hypoalbuminaemia, intrathoracic mass lesions). However, pleural fluid analysis is a simple but powerful diagnostic test which at a minimum narrows differential diagnoses and often definitively diagnoses the cause for the pleural effusion. As therapeutic thoracocentesis is sometimes one of the first steps in stabilizing these patients it is also a test often performed early in case management.

When collected, pleural fluid should be collected into EDTA tubes (for cell counts and cytology), clot tubes (for biochemistry testing on the fluid) and culture devices if septic processes are suspected / identified. At a minimum nucleated cell and RBC counts, fluid total protein and cytology slide evaluation should be performed with some advocating for more biochemical tests to be performed as they provide more accurate fluid classification (e.g. fluid albumin, fluid cholesterol, fluid triglyceride, fluid glucose, fluid LDH activity).

Cell counts, both nucleated and RBC, are routinely performed on automated haematology analysers; however, the presentation will highlight when this can lead you astray if you are performing these counts in house. In the reference laboratory when the counts are discrepant, manual methods are used for confirmation. Also note, nucleated cell differential on fluids should always be performed from cytology slides as the cells in the effusion are not all "normal" cells present in blood (e.g. mesothelial cells, macrophages) and the analyser differential, if reported, will be inaccurate.

Total protein measurement via refractometer is often sufficient but if more accurate measurements (e.g. lipaemic sample) or additional testing is required, biochemistry analysers can be used (i.e. total protein, albumin, cholesterol/triglycerides, creatinine).

Cytology preparations are used to determine nucleated cell differential which is used in fluid classification and also to assess for any atypical cell populations (e.g. carcinoma, large immature lymphocytes, mast cells) which may provide clues as to the cause for the effusion and patient's presentation. If fluid is to be sent to a laboratory, slides made in-house at the time of collection are very much appreciated as the cell morphology is better and some changes can be better interpreted as storage artefact is ruled out (e.g. erythrophagocytosis must indicate intrathoracic haemorrhage rather than possible storage lesion). If the fluid is clear, consider concentrating the sample prior to making a slide (i.e. centrifuge as per urine sediment and then prepare slide from the cellular material at the bottom).

Historically, based on the nucleated cell count and total protein measurements, pleural fluid in veterinary medicine has been classified into one of three categories: pure transudate; modified transudate; or exudate. Recently, discussion has started around whether we could be more accurate with our fluid classification by using a more mechanistic classification (i.e. transudate vs exudate only with specific fluid entities defined with additional testing i.e. chylous, haemorrhagic, neoplastic) or even using human classification methods (i.e. pleural fluid protein: serum fluid protein and pleural fluid LDH); however, as yet no one specific method has surpassed the historical classification method.

This historical fluid classification is primarily reliant on fluid protein and nucleated cell count with RBC, fluid characteristics, cell differential, and specific cytologic findings (e.g. microorganisms, foreign material) used to further narrow the classification (see table at the end of the proceedings).

Pure transudates are seen with decreased colloidal oncotic pressure and increased hydrostatic pressure. Pure transudates in the thoracic cavity are most often seen with hypoalbuminaemia and volume overload but can also be seen in early cardiac disease. Hypoalbuminaemia often causes effusion concurrently in the abdominal cavity and perhaps pulmonary and tissue oedema.

Modified transudates are relatively nonspecific and are seen with increased venous hydrostatic pressure and vascular protein leakage. Common causes in the thoracic cavity include cardiac disease, neoplasia, parenchymal inflammation, feline infectious peritonitis, and lung lobe torsion (although often many mesothelial cells are noted with this).

Exudates are seen with increased capillary permeability secondary to inflammation, infection (including bacterial infection and feline infectious peritonitis), and neoplasia.

Chylous effusions are a specific effusion that would often be categorized as a modified transudate on protein and cell count alone; however, the milky, opaque fluid characteristics combined with a predominance of small lymphocytes and elevated fluid triglycerides allows this effusion to be separated as a specific entity. Chylous effusions are seen with cardiac disease, intrathoracic neoplasia (particularly those affecting the lymph nodes i.e. lymphoma, thymoma, metastatic disease invading lymph nodes), as an idiopathic phenomenon, with traumatic rupture of the thoracic duct, diaphragmatic hernia, and chronic coughing.

Haemorrhagic effusions are diagnosed if there are $>1.0 \times 10^9/L$ erythrocytes or a PCV $>5\%$ in the effusion and to confirm intrathoracic haemorrhage (rather than contamination from vascular spaces during aspiration) it is preferred to see erythrophagocytosis or haemosiderin within the macrophages in the effusion. Haemorrhagic effusions can be seen with trauma, coagulopathies, ischaemic/necrotic lesions, and neoplasia.

Feline infectious peritonitis effusions can be very classic in their presentation as they often have a disproportionately high protein measurement compared to the cellular component (i.e. protein $>40g/L$ but with a cell count $<5.0 \times 10^9/L$). This can also be seen on the

cytology slides with few cells present amid a thick or stippled eosinophilic proteinaceous background. These are an effusion type which can benefit from extra diagnostic testing including fluid albumin : globulin measurement, fluid immunocytochemistry for feline coronavirus within macrophages and feline coronavirus PCR evaluation.

Effusion Type	Colour/Turbidity	TP (g/L)	WBC (x10 ⁹ /L)	Predominant cell type
General Conditions				
Transudate/Low protein transudate	Colourless/Clear	< 25	<1.5-2.5	Mesothelial cells, mononuclear cells, few neutrophils
Modified transudate/High protein transudate	Lt. yellow to apricot/clear to cloudy	≥ 25	< 5.0	Mesothelial cells, mononuclear cells, few neutrophils
Exudate	Apricot to tan/cloudy	> 30	>5.0	Neutrophils Non-degenerate: non-septic Degenerate: septic
Specific Conditions				
Chylous	White/opaque	> 25	Variable	Acute: small lymphocytes Chronic: Mixed population Rare mast cells
Neoplastic	Lt. yellow to apricot/clear to cloudy	> 25	Variable	Neoplastic cells +/- inflammation, mesothelium
Haemorrhagic	Pink to red/cloudy	> 30	> 1.0	RBCs and WBCs proportional to blood. Erythrophagocytosis; Haemosiderin/haematoidin
Feline Infectious Peritonitis	Straw to slightly cloudy, viscous	>25 often >40	<5.0	Often background eosinophilic stipples and crescents (protein), predominantly neutrophils (often vacuolated), macrophages, lymphocytes

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An update on pneumonia in dogs and cats

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Pneumonia is defined in the human field as an infection of the lung parenchyma by one or more pathogens. This is distinct from pneumonitis, which specifically refers to inflammation of the lung parenchyma due to injury (trauma, caustic substances, stomach acids). Pneumonitis is often classified as non-infectious, however damage to the tissue may predispose to bacteria or other pathogens colonising the lungs. This distinction is important as some patients may appear to have pneumonia however, they may only have pneumonitis. Pneumonia can be associated with the inhalation of pathogens through liquids or solids (aspiration pneumonia) or may be spread through other avenues e.g., haematogenous spread (bloodstream), pleural extension, intrathoracic extension.

Bacterial pneumonia is the most common type of pneumonia we see in practice. There is probably a larger variation of disease within this clinical picture than we understand. This may depend on the type of bacteria present, the number, the variation, the lobe/lobes affected, the state of the systemic immune response, the state of the local (pulmonary/bronchial) immune response and the level of accompanying pneumonitis. This is why sometimes these patients may do well as outpatients while others require intensive care and hospitalisation. 1/3 of pneumonia cases are caused by multiple organisms and 20% of cases involve anaerobic pathogens.

Species of bacteria we most commonly see are *E. coli*, *Streptococci*, *Klebsiella*, *Pasteurella*, *Staphylococci*, *Mycoplasma*, and *Bordetella*. Cats rarely develop pneumonia in clinical practice, canine patients are far more common.

The spectrum of presentation is diverse and depends on a variety of factors as discussed above. Signs of bacterial pneumonia can include cough, nasal discharge, exercise intolerance, respiratory distress (tachypnoea, dyspnoea), lethargy, fever, and anorexia. Normothermia does not rule out bacterial pneumonia and some patients may only present with pyrexia on presentation. Thus, 3-view thoracic radiographs are always important when investigating pyrexia of unknown origin. Physical exam may be normal or consist of increased crackles on lung auscultation, increased bronchovesicular sounds, wheezing or even cyanosis.

When investigating pneumonia, it's important to evaluate a CBC, biochemistry, and UA for each patient. This often does not allow us to diagnose pneumonia, but it rules out important causes of the pneumonia in that patient. CBC is vital to assess the number of neutrophils present and the morphology (based on a blood smear). A neutrophilia with a left shift or neutropenia would indicate a more severe disease. An evaluation of the oxygenation status of the patient is vital to ensure the patient is stable and doesn't require supplemental oxygen. This can be achieved through pulse-oximeter readings or an arterial blood gas measurement. The most vital test for pneumonia is thoracic imaging. Radiographs are readily available in general practice (however CT sometimes has its place). Evidence of an alveolar pattern is supportive of pneumonia. Differentials for this could include other fluids (oedema-cardiogenic, non-cardiogenic), blood (coagulopathy, trauma), or inflammation (pneumonitis). The only definitive way to diagnose pneumonia is through the culturing of fluid from the lungs through BAL/trans tracheal wash. A clinical picture, the appropriate bloodwork changes, physical exam findings and the distribution of the alveolar infiltrates in the lungs (e.g., bacterial pneumonia is most common to present in the ventral lung fields: the right middle lung lobe being the most common) is supportive of pneumonia and should ideally prompt the use of antibiotics. Sometimes in early cases of pneumonia

the radiographic pattern may be interstitial or there may be no pattern at all. In these cases, a repeat set of thoracic radiographs can be helpful in 12-24 hours as the pattern begins to evolve on radiographs and can lag behind the clinical picture. It can be helpful to run a CRP in some cases. CRP or C-reactive protein is an inflammatory marker. It can be increased in cases of infection, inflammation, or neoplasia anywhere in the body. Increases can tell us that the patient might be undergoing one of these processes and in the right clinical picture can be supportive of pneumonia. It can also be used as a tool to track the disease process and discern when to stop antibiotics. In some studies, CRP was shown to resolve faster than radiographic resolution of disease for pneumonia and a normal CRP prompts the cessation of antibiotics.

ISCAID guidelines discuss in detail the treatment of pneumonia. Typically, in stable patients, an antibiotic can be chosen such as amoxicillin. For more severe cases treatment with amoxicillin-clavulanate acid and a fluoroquinolone may be required. In some circumstances aerosolization of antibiotics is appropriate. It's important not to forget other areas of supportive care such as fluid therapy/hydration, nutritional needs, anti-nausea medications, treatment of the underlying cause that leads to pneumonia, pain relief if appropriate, nebulisation for airway humidification and regular movement/ambulation (i.e. do not maintain constant recumbency) to ensure adequate recovery.

Generally, for simple cases of pneumonia, 1-2 weeks is adequate for treatment. Monitoring is advised with CRP where appropriate.

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Respiratory noise and character: How the sounds and patterns your patients make can lead you to a diagnosis

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When assessing a patient with respiratory disease, before we reach for fancy diagnostics like CT and bronchoscopy, there are often many clues we can take from the character of breathing and the sounds that the patient makes, which can help us to achieve a diagnosis sooner. Furthermore, some of these sounds and changes may indicate intestinal disease and not airway disease. Ensuring we understand what our patients are presenting for is vital to ensure we use the appropriate diagnostics to achieve a diagnosis.

In the modern world, we have the advantage of video technology and smartphones that allow us to identify changes in our patients. This is a vital tool for respiratory disease diagnosis as often the description from an owner can lead us down a different diagnostic pathway. Asking an owner to film the event before any diagnostics can be incredibly helpful.

With respiratory patients, we aim to achieve a diagnosis in the least invasive manner possible. This is particularly important given that these patients may sometimes be unstable and unable to tolerate diagnostics, such as radiographs or general anaesthesia for CT/bronchoscopy. Once we have localised the disease, we can then perform the necessary diagnostics (if any required) to achieve a diagnosis and formulate a treatment plan.

Signalment is often overlooked for many patients. In internal medicine common things occur commonly and these common things are more common in the breeds that the disease is more common in... have I lost you? Put simply your differential list can change based on the age, sex and breed of the patient presenting for respiratory disease. Some examples are below:

- Brachycephalic patients such as English bulldogs, Pugs, French Bulldogs may be more likely to present with upper respiratory tract disease or gastro-intestinal disease related to these changes (aerodigestive disease).
- Dolichocephalic breeds presenting with nasal signs are more likely to have nasal tumours or nasal aspergillosis (fungal rhinitis)
- Younger animals are more likely to present with infectious disease or immune-mediated disease, older animals are more likely to present with neoplasia.
- Breeds such as West Highland White Terriers are predisposed to conditions such as pulmonary fibrosis (lower airway disease).
- Siamese cats are more likely to present with feline asthma.

Some common mistakes that are made when we confuse:

- Vomiting (indicating gastrointestinal disease) vs retching (indicating pharyngeal, oropharyngeal disease) vs regurgitation (passive expulsion of material indicating oesophageal disease).
- Coughing vs retching in the feline patient
- Stertorous breathing vs stridorous breathing.
- Respiratory distress vs look-a-like respiratory distress syndromes (from systemic pH imbalances, stress, pain, and abdominal disease).
- Reverse sneezing vs respiratory distress.
- Wheeze vs a cough
- Retching/gagging vs coughing

It's also important to identify the character of breathing. It is important to note if the breathing changes are:

- Intermittent or continuous
- Low or high-pitched noise
- Impacting movement/mobility
- Associated with postural changes/open mouth breathing
- Rate
- Effort
- Abdominal input and synchrony
- Inspiratory or expiratory effort
- Fatigability

All these clues can help us understand the location of the disease as well as allow us to input early and later-stage therapeutics to help these patients.

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Talking tubes: Understanding oesophageal dysfunction in dogs and cats

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The oesophagus is often the forgotten organ of the gastrointestinal system. Its role however in gastro-intestinal health is vital to ensure both appropriate ingestion and metabolism of nutrients and to avoid respiratory tract compromise. Its main role is in deglutition (swallowing) and it's at the final stage of this complex pathway between the brain, nerves, hormones, and muscles. The bolus transfer from the oral cavity to the stomach is dictated by pressures (positive pressure in front, negative pressure behind) that signals organ and nerve function to occur and propels the bolus towards the stomach. Dysfunction in the anatomical or functional ability of the oesophagus (and/or its adjacent organs/muscle) results in dysphagia (an inability to swallow). Clinically this can result in severe malnutrition and aspiration pneumonia and if left untreated will often be fatal. Dysphagia is far more common in dogs than cats. Most commonly in cats, structural diseases of the oropharynx are the cause of dysphagia including ulcers, masses, gingivostomatitis and oesophageal strictures.

The swallowing process involves 31 pairs of striated muscles, 5 cranial nerves (sensory and motor branches) and the swallowing centre in the brain. There are 4 stages in total. The final involves the oesophagus. The outer layer of the oesophagus is connective tissue with a staggered blood supply. These are the two reasons why the oesophagus heals poorly after surgical intervention/trauma. In dogs, the entire muscular component of the oesophagus is striated muscle, in cats the distal 1/3 is replaced by smooth muscle. This becomes important when selecting medications to manipulate oesophageal movement.

Typically, a patient presenting with oesophageal disease may retch, gag, hypersalivate, or regurgitate. They may cough within a short time of drinking water. They may have deep swallowing/seem painful when swallowing food or water. They may have recurrent pneumonia. Sometimes the disease process may involve multiple areas of the swallowing pathway and may not be fully isolated to the oesophagus. Furthermore, oesophageal dysfunction may be a part of a wider systemic disease process.

For these reasons, a full physical and neurological exam should be performed on these patients. Furthermore, full laboratory assessment is often indicated including measurement of CBC, biochemistry panel and UA. Looking at analytes such as CK can be helpful to rule out myopathies as a cause. Often a thyroid level and a cortisol (baseline) are indicated to rule out hypothyroidism or hypoadrenocorticism as a cause. If suspecting Myasthenia gravis, an acetylcholine esterase inhibitor antibody is indicated.

Thoracic radiographs are important to rule out a number of findings:

- Extra-thoracic or intra-thoracic disease leading to structural impairment of oesophageal motility.
- Dilated oesophagus or a megaesophagus.
- Mediastinal or pulmonary pathology that may lead to a paraneoplastic disease process and oesophageal motility disorders (e.g. a thymoma-inducing myasthenia leading to oesophageal dysmotility).

It's important to note that if the oesophagus appears normal on radiographs (i.e. not dilated) this does not rule out dysmotility and further investigation may be required, such as with a swallow study/fluoroscopy.

Treatment of oesophageal disorders often involves resolving the underlying disease process, the addition of pro-motility medications (such as cisapride) and medications that help relax the LES (Sildenafil), as well as environmental changes such as upright feeding. Failure to support these patients appropriately can result in repetitive aspiration pneumonia events and death.

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Diet-associated dilated cardiomyopathy (DCM): Update

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Dilated cardiomyopathy (DCM) in both dogs and cats encompass a variety of causes with the most common being primary (genetically linked) DCM found in certain breeds such as Doberman Pinschers, Boxers, Irish Wolfhounds, and Great Danes, but also include toxic, infectious/inflammatory myocarditis, metabolic/dietary and idiopathic etiologies.

Diet-associated DCM was a common cause of feline DCM due to taurine deficiency until 1987 when studies showed taurine supplementation resulted in dramatic improvement both clinically and echocardiographically in these cats.¹ Consequently, taurine supplementation in commercial cat food became the norm and this etiology grew increasingly rare apart from patients who are fed home-cooked, vegetarian, or non-mainstream diets. Similarly in dogs, certain breeds such as American Cocker Spaniels² and Golden Retrievers³ have been shown to be predisposed to taurine deficient DCM, as well as large breed dogs fed lamb/rice diets (e.g. Newfoundlands⁴), Dalmatians with low protein diets⁵ etc.

In 2018, growing reports of breeds not previously considered to be predisposed to developing DCM from veterinary cardiologists in the U.S., prompted the U.S. Food and Drug Administration (FDA) to issue a public notice, and began a multidisciplinary investigation into the causes of such increase in incidence. The FDA analyses showed that >90% of diets fed to these patients were labeled as 'grain-free', and majority of these 'non-traditional (NT)' diets contained higher proportions of legumes/pulse ingredients (e.g. peas, lentils, chickpeas), and to a lesser extent potatoes/sweet potatoes compared to traditional diets. Further metabolomic analysis comparing top brands of NT diets fed to reported DCM dogs vs. traditional diets not clinically linked to reported cases identified >100 compounds that are either higher or lower than traditional diets, with peas being the leading possible ingredient associated with the disease.⁶ Apart from two studies that specifically examined Golden Retrievers fed a NT diet^{7,8} which has shown an association with taurine deficiency, other studies looking at breeds apart from Golden Retrievers have not shown such consistent findings.^{6,9-11} Taurine deficiency therefore does not appear to play a major role in this cohort of patients. Although majority of cases are associated with dogs, it is also noteworthy to point out that a small number of feline cases have also been reported.

These observations subsequently led to several retrospective and prospective studies over the past few years that sought to shed light on the clinical, biochemical, and echocardiographic changes observed in dogs being fed NT diets. From the limited studies performed thus far, it appears the disease phenotype exists in a spectrum with longer duration of diet being fed, potentially leading to more progressive phenotypic changes, and subsequently a worse outcome.¹² Increased left ventricular chamber dimensions and systolic dysfunction were consistently reported across studies with some patients progressing towards congestive failure development. Yet, improvement in both echocardiographic measurements and reversal of clinical progression to a point of reducing or discontinuation of medical therapy has also been reported in some cases post diet change.^{7,8,11-15} Studies examining pre-clinical dogs of different breeds further reported an increase in ventricular arrhythmia frequency¹⁶ and elevated cardiac troponin I levels in dogs on NT diet suggesting the possibility of early cardiomyocyte injury, although such changes are subtle and clinical importance are yet to be determined.¹⁰

Comparison across different studies however remains a challenge as many of them are retrospective in nature, and the definition and duration of feeding 'NT diet' are inconsistent

across reports. The exact causative factor of this disease phenotype also remains unknown. An accurate picture of this disease process is thus still unclear at this point. Nonetheless, it is important for clinicians to always obtain a thorough diet history when assessing cardiac patients. Until clearer evidence of the exact causative factor of this disease process is available, dietary alteration to ones that do not contain high proportions of pulses and/or changing to grain inclusive diets are worth considering at this point in patients who demonstrate echocardiographic evidence of myocardial dysfunction. If patients are suspected to have diet-associated DCM, cardiology referral is highly recommended for accurate assessment of myocardial function with echocardiogram +/- ECG/taurine testing to guide appropriate monitoring and therapy.

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Effect of systemic disorders on the heart: Feline

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Although we are accustomed to categorizing disease processes according to specific body systems, in reality, most diseases and subsequent treatment decisions we make often have implications on other organs. The heart, and its extension the cardiovascular system, is no different and often is susceptible to significant collateral consequences that may itself, become the major determination of the patient's ongoing morbidity and even mortality. This discussion aims to outline the most common systemic conditions in our feline patients that may have major consequences involving the heart.

Senescence

Similar to humans, older cats commonly have reduced ventricular compliance and delayed relaxation (diastolic dysfunction). This may make them susceptible to fluid overload when administered intravenous fluid therapy. Acquired cardiomyopathy also becomes more common as cats become older with median age for diagnosing HCM to be 7.4 years old with overall risk of all-cardiovascular death for HCM patients significantly higher as the patient gets older (28.5% - 10 years post diagnosis vs. 6.7% - 1 year post diagnosis).¹ Cats aged ≥ 9 years old that are undergoing interventions (e.g. general anaesthesia, fluid therapy, extended-release glucocorticoid) that may precipitate congestive heart failure (CHF) should therefore be considered undergoing an echocardiographic exam.²

Hyperthyroidism is common in older cats and may exacerbate underlying cardiomyopathic process. Thyroid hormone exerts both direct effects on the cardiomyocyte and indirect effects of upregulating β -receptors causing enhanced adrenergic activity. This leads to an increase in stroke volume and cardiac output via positive chronotropy, inotropy and lusitropy. Systemic hypertension is also observed between 10-23% of cats with hyperthyroidism and in nearly 25% of hyperthyroid cats that were normotensive at the time of diagnosis, hypertension may develop after euthyroid state has been achieved.^{3,4} Clinically, hyperthyroidism may cause ventricular hypertrophy, tachyarrhythmias, intracardiac conduction abnormalities, and congestive heart failure in some cats (especially when underlying primary cardiomyopathic process is already present). Hence, serum thyroxine levels should be assessed in cats with abnormal cardiac auscultation/echocardiographic findings who are 6 years or older.²

Cardiovascular-renal disorders (CvRD) describe diseases or medication/toxin-induced damage to the kidney and/or the cardiovascular system which leads to an abnormal interaction of the two systems and possible ongoing detriment of each other. These disorders can be divided into subgroups based on the primary insult to be either cardiovascular (CvRD_H) or renal (CvRD_K) in origin, or whether both systems are concurrently diseased/affected by other disease process/drugs/toxin (CvRD_O).⁵ The pathophysiology of CvRD likely involves a complex interaction of hemodynamic changes, neurohormonal activation (sympathetic nervous system, RAAS), and reactive oxygen species, but a clear understanding of the nature and prevalence of CvRD remains lacking in our veterinary patients. Common examples of CvRD in the feline patient involving different subgroups include systemic hypertension, which may be caused by primary renal disease, endocrinopathies (e.g. hyperaldosteronism/hyperthyroidism), or neoplasia (e.g. pheochromocytoma) with serious consequences of target organ damage often involving multiple organ systems including the heart (left ventricular hypertrophy, diastolic dysfunction, possible congestive failure and rare cases of aortic aneurysm/dissection), kidneys (progressive renal dysfunction and proteinuria), as well as eyes (retinopathy/choroidopathy), and the brain (stroke, encephalopathy). Appropriate

management of patients therefore requires careful consideration and frequent monitoring of all systems involved.

Alteration in hydration status changes cardiac chamber sizes and wall thickness which may easily be misdiagnosed as diseased if not taken into consideration. Hypovolemia may cause pseudohypertrophy of the left ventricular wall, and reduced left ventricular internal diameters as well as left atrial dimensions. On the other hand, IV fluid therapy may cause intravascular volume expansion, leading to an increased diastolic left ventricular internal diameters, fractional shortening and left atrial size.⁶

Systemic inflammations may lead to rare myocarditis which can manifest as various cardiomyopathic phenotypes. Myocarditis caused by *Streptococcus canis*, toxoplasmosis, feline infectious peritonitis (FIP) caused by feline coronavirus⁷, *Bartonellosis*^{8,9}, feline immunodeficiency virus (FIV)¹⁰ have all been reported, and panleukopenia viral genome has also been found in cats with spontaneous cardiomyopathy and myocarditis.¹¹ Sequelae may range from CHF/aortic thromboemboli/sudden death to transient myocardial thickening (TMT) where reverse remodeling of cardiac structural changes and discontinuation of diuretics may occur.¹² Prognosis largely depends on success in primary disease management and reversibility of cardiac changes.

Corticosteroid administration associated CHF has been reported in a case series¹³ where cats who have received systemic steroids (mainly methylprednisolone, but also oral prednisolone and parenteral dexamethasone, betamethasone and triamcinolone) subsequently developed CHF days-weeks later with echocardiographic evidences of HCM phenotype and left atrial enlargement. Some of these cats (7/12) survived >1 year with successful weaning off of diuretics therapy and reverse remodeling of left heart changes. Two subsequent studies^{14,15} examining small cohorts of cats receiving either methylprednisolone or oral budesonide/prednisolone re-echoed a week later showed various changes in bloodwork and echo exams that suggested plasma volume expansion post steroid administration with the later study also showing increase in left heart chamber dimensions. The pathogenesis of such changes however remains unclear, but caution is warranted when systemic steroids is administered to cats, especially those that have suspected underlying cardiomyopathy.

Electrolyte imbalance specifically potassium derangements may cause serious arrhythmia in cats. Hypokalemia lowers the resting membrane potential, decreases cardiomyocyte excitability, and prolongs repolarization, with the latter more important in arrhythmogenesis (e.g. VPCs, QT prolongation, AV dissociation). Hyperkalemia induced ECG changes are severity dependent. Mild hyperkalemia (5.6-6.5 mEq/L) shortens repolarization, causing shortened QT interval, peaked/tented T waves; when K⁺ reaches 6.6-7.5mEq/L, cell-cell conduction becomes affected, widening QRS complexes and decreasing R wave amplitude; Moderate-severe hyperkalemia (7-8.5mEq/L) prolongs PR interval/P waves are absent, sinoventricular rhythm may be present as conduction between SA node-AV node remains; If K⁺ >8.5 mEq/L then patient is at risk of cardiac arrest with sine wave/slow ventricular escape rhythm, eventually resulting in ventricular fibrillation or escape rhythm with pulseless electrical activity.¹⁵

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Feline aortic thromboembolism (FATE): Updates on management

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Aortic thromboembolism (ATE) is one of the potentially life threatening sequelae associated with severe cardiac diseases in cats. According to the REVEAL study, approximately 11% of cats with hypertrophic cardiomyopathy (HCM) may succumb to this complication over their lifetime.¹

The presence of left atrial enlargement is the most important risk factor predisposing cats to developing an intracardiac thrombus with blood stasis and possible endothelial injury/hypersensitive platelet being likely pathomechanisms that are specific to cats. On echocardiography, the presence of spontaneous contrast (smoke) representing clumping of red blood cells (negative prognostic factor) may be appreciated, and at times, visible thrombus within the left auricle can also be seen. It is however important to note that the absence of intracardiac thrombus does not exclude the diagnosis of ATE in cats.

Dislodgement of thrombus and downstream embolization (most commonly in the terminal aortic bifurcation) causes acute blood flow occlusion resulting the clinical signs abbreviated by the 5P's: acute Pain, Pallor, Poikilothermia (cold to touch), Paresis/paralysis, Pulselessness, and Pallor. Furthermore, paw pads and nail beds will appear cyanotic and gastrocnemius muscle may be firm and painful. Patients may appear in distress and tachypnoeic (either from elevated sympathetic tone and/or concurrent congestive failure). If thrombus is small, embolization to systemic arterial branches (subclavian/brachial, coronary, mesenteric arteries) may also occur resulting in forelimb ischemia, renal, myocardial, intracranial and intestinal infarcts.

Cardiogenic ATE is diagnosed based on clinical signs and echocardiographic evidence of left atrial enlargement. Doppler signal will be absent in affected limb if complete arterial occlusion is present. Venous sampling of affected limb(s) may have comparatively lower blood glucose and higher lactate than systemic venous sample. Serum biochemistry will also show markedly elevated creatinine kinase, ALT and AST levels secondary to myolysis. Thoracic radiographs should be performed once patient is stable to assess for concurrent left sided congestive failure (pulmonary edema/pleural effusion).

Treatment is centred around pain relief, thromboprophylaxis, treatment of underlying cardiac disease and nursing care. Analgesia should be provided immediately with opioids (methadone, buprenorphine, or fentanyl CRI/transdermal) and be continued for at least the first 24 hours. Thromboprophylaxis should be provided to reduce risk of ATE recurrence with low molecular weight heparin (dalteparin, enoxaparin) immediately then continued on factor Xa inhibitor (rivaroxaban, apixaban) as well as antiplatelet therapy (clopidogrel). Thrombolysis relies on patient's own intrinsic system as thrombolytic therapy has not shown to be effective in increasing survival and may pose risk of reperfusion syndrome.^{2,3}

ATE is usually associated with a poor prognosis with traditional rates of survival to discharge between 33-39% (euthanasia rates in these studies were 24-35%) although this is improving with more recent cases improving survival up to 73%. The condition holds a much better prognosis if only one limb is affected or the cat has only partial loss of limb function. Low rectal temperature tends to be a negative prognostic indicator.^{4,5} In terms of long term prognosis, cats can potentially have complete recovery of limb function. However, some have residual neurological deficits or tendon contracture. Patients should be rechecked within 1-2 weeks post ATE event to assess for distal limb necrosis, electrolyte levels, treatment compliance and improvement in neuromuscular function. Sleeping

respiratory rates should also be monitored for development/recurrent congestive failure. Ischemia may lead to tissue necrosis, necessitating surgery. Recurrence of ATE is estimated to be 24-45% although dual thromboprophylaxis with rivaroxaban and clopidogrel together may suggest lower recurrence rate^{6,7}, however, this has yet to be confirmed with properly designed prospective trials. The presence of congestive failure does not have a negative impact on survival to discharge but does have a negative prediction for long-term survival (median survival time for cats presenting with signs of congestive heart failure is 77 days, whereas the median survival for cats without these signs is 223 days).⁵ Most cats have poor long-term survival rates and usually, die from cardiac disease rather than recurrent ATE.

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Interventional treatment modalities for mitral valve disease

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Management of myxomatous mitral valve disease in dogs have traditionally been mainly dependent on conservative medical therapy. However, over the past decade with the advancement in imaging technology, surgical techniques and refinement of human interventional strategies, management of MMVD in dogs have undergone a gradual paradigm shift with various interventional treatment modalities becoming increasingly available around the world. This talk discusses these interventional treatment options including left atrial decompression (LAD), open heart mitral valve repair (MVR), and transcatheter edge-to-edge repair (TEER) with a focus on the latter.

LAD is a minimally invasive procedure for palliative management of dogs with severe stage C/D MMVD that is not well controlled on medical therapy alone. The procedure is performed percutaneously via jugular venous access and the left atrium is accessed via transeptal puncture. Balloon dilatation ensues creating an iatrogenic atrial septal defect (IASD) that aims to reduce left atrial pressure. LAD has been successfully performed in canine patients¹ but patients are at risk of spontaneous closure of the IASD (20%)² which is also reported in human patients as well as development of right sided systemic congestive failure due to the consequent increase in right atrial pressure (36%). The ideal timing of when this procedure should be performed is still yet unknown given the scarcity of literature but preliminary data suggests a survival outcome that is at least comparable if not improved then medical therapy alone.

Open heart MVR in dogs mirrors the technique used in human patients with mitral annuloplasty to reduce size of commissure, excision of ruptured chordae tendinae and placement of artificial chords made with expanded polytetrafluorethylene (ePTFE) to support valve leaflets. The success rate of open heart MVR is high in selected centres around the world³, however, the need for cardiopulmonary bypass and highly trained personnel, and the associated cost limits its ability to be performed widely across the world.

TEER has been established in human patients via the Mitral-Clip for nearly 20 years as a minimally invasive procedures for patients where surgical repair under cardiopulmonary bypass is unsuitable. It utilizes an implantable device inserted percutaneously to appose the anterior and posterior mitral leaflets to improve leaflet coaptation and minimize regurgitant volume. It has been shown to confer comparable survival benefits as traditional surgical approach. The V-Clamp is the recent veterinary adaption of the Mitra-Clip.⁴ The device is delivered by transapical puncture under fluoroscopic and 3D transoesophageal echocardiogram guidance in a hybrid approach via a mini-thoracotomy. Preliminary follow up data reports a procedural success rate of 95% in 40 dogs with TEER performed with no perioperative mortality, as well as a 9-month post-op survival of >87%.² The procedure offers an alternative to medical management for canine patients with severe stage B2, C and D MMVD patients. However, long-term survival studies which have demonstrated superior outcomes in human patients have not yet been available in dogs.

Successful interventional therapy on dogs with mitral valve disease (whether open or minimally invasive) depends largely on a collaborative team of highly trained personnels including cardiologists, surgeons, anesthesiologists, critical care specialists, technicians and support staffs. Although there are yet many questions unanswered such as ideal patient selection criteria, risk factors determining successful surgical outcomes, long term outcome when compared to medical therapy/between different interventional therapy etc.,

the increasing availability of these therapeutic options being available across various centers around the world will continue to allow us to learn and perfect these techniques.

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Overview on diagnosing feline cardiomyopathies

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Feline cardiomyopathy includes a heterogeneous group of disorders in which despite years of research both in the human and feline world, remains largely unclear in regards to its etiopathogenesis and natural history. The prognosis and clinical presentation may vary widely between individual cats, and the lack of large scale prospective clinical trials makes it challenging to provide solid evidence-based clinical decisions.

The most recent ACVIM consensus statement on feline cardiomyopathies¹ proposed a classification system whereby cardiomyopathies are grouped into specific morphological and functional phenotypes without assuming the underlying cause. These include: hypertrophic cardiomyopathy (HCM), restrictive cardiomyopathy (RCM), dilated cardiomyopathy (DCM), arrhythmogenic right ventricular cardiomyopathy (ARVC), and nonspecific phenotype (NSC) (previously known as unclassified cardiomyopathy).

The key for this new classification system is that it recognizes the limitations in which certain phenotypes may include cats with known causes (e.g. hyperthyroidism, systemic hypertension, diet etc.) and unknown causes (e.g. idiopathic); and that disease phenotype may change as comorbidities develop or disease severity progresses over time (e.g. a cat with HCM that develops regional LV wall hypokinesis from an infarct may show systolic dysfunction and segmental myocardial thinning towards the end stage and assumes a DCM phenotype).

A new staging system similar to dogs with mitral valve disease has also been adapted to provide guidance on approach to diagnosis and management based on available evidences:

- Stage A: Breeds that are predisposed to cardiomyopathy (e.g. Maine Coon, Ragdoll, Sphynx etc.)
- Stage B1: There is evidence of myocardial disease but risk of imminent CHF and arterial thromboemboli (ATE) is low
- Stage B2: There is evidence of myocardial and risk of CHF/ATE (mainly dependent on presence of left atrial enlargement)
- Stage C: Cats with current or previous CHF/ATE
- Stage D: Cats with CHF refractory to treatment

HCM is characterised by concentric or asymmetric hypertrophy of the left ventricular (LV) wall and/or interventricular septum (IVS) in the absence of a dilated LV chamber. Diagnosis of primary/idiopathic HCM is made by the documentation of an IVS or LV free wall thickness (at any location) of >6mm during diastole, in the absence of other causes of myocardial thickening (systemic hypertension, hyperthyroid, pseudohypertrophy secondary to hypovolemia, inflammation/infection/transient myocardial thickening). Because myocardial thickening can be asymmetric, it is recommended that serial measurements of the above structures be performed via a two-dimensional echocardiographic image (confirmed by at least 2 views - short and long axis) rather than relying on measurements obtained by M-mode echocardiography alone.

DCM phenotype is characterised by LV systolic dysfunction of the ventricular myocardium (which may or may not be reduced in thickness) with progressive increase in ventricular systolic and subsequently diastolic dimensions. DCM phenotype may also be caused by multiple etiologies, including diet, inflammatory/infectious myocarditis, tachycardia-induced

cardiomyopathy etc. The most common cause of DCM phenotype in cats however remains to be idiopathic.

RCM may be a discrete entity or a progression of another form of cardiomyopathy. RCM is characterised by impaired diastolic filling (as indicated by an often dilated left, and occasionally right atrium), where the left ventricle has minimal signs of hypertrophy or systolic dysfunction. Patchy endomyocardial fibrosis is sometimes observed ("endomyocardial" form) which may be so severe as to obliterate the distal portions of the LV chamber. In some cats, the scarring affects mostly the myocardium ("myocardial" form).

ARVC is characterised by replacement of the myocardium of the right ventricle with fibrous and/or fatty tissue (the left side may also become involved in advanced cases). This leads to marked ventricular wall thinning. The exact cause of this disease is unknown. However, it is theorized that it may be initiated by an inflammatory condition, which then leads to secondary deposition of fibrofatty tissue as an end-stage process. The echocardiographic changes usually consist of marked RA and RV dilatation, and patients often have ventricular arrhythmia.

NSC represent a heterogeneous group of disease and is characterised by phenotypes that do not fit within the above classifications. They may likely be progressive (or regressive) forms of the other cardiomyopathic phenotypes. Given its heterogeneity, according to the consensus guidelines, patients with atypical echocardiographic presentation should be given the diagnosis of "NSC phenotype" followed by a description of the echo observation (For example, the cat is diagnosed with NSC phenotype characterised by LV eccentric hypertrophy, focal septal thickening, and poor systolic function.).

Cats with symptomatic cardiomyopathy usually present with respiratory signs due to either pulmonary edema and/or pleural effusion, or neurological signs due to ATE. Some may have auscultatory abnormalities (murmur/gallop/dysrhythmia). However, the absence of such abnormalities does not exclude clinically significant cardiac disease.

Thoracic radiography is mainly used for assessing presence of pulmonary infiltrates when patients are presented with tachypnoea as the radiographic findings relating to heart chamber enlargement is variable. Important reminder that pulmonary edema in cats does not tend to follow the "perihilar" rule and may be patchy or diffuse throughout any or all of the lung fields.

The use of thoracic ultrasound is mainly for identifying presence of pleural effusion. Although ring down artefacts ("B-lines") may be appreciated when pulmonary edema is present, it is important to note that this finding may be present in other types of pulmonary disease and is not specific to pulmonary edema alone.

Cardiac biomarkers (e.g. NTproBNP) may be used either as 1. a screening test for subclinical myocardial disease in cats with abnormal physical exam findings. This has reasonable specificity (80-100%), but relatively poor sensitivity so it cannot be used to exclude the presence of disease, nor can it differentiate between normal vs. mild-moderate disease if levels are within normal reference ranges; or 2. To differentiate cardiac from non-cardiac causes of dyspnoea with a bedside testing sensitivity ranging between 94-100%. In other words, if a dyspneic cat has a negative NTproBNP test, then CHF is highly unlikely; however, if the NTproBNP test is positive, then other testing is required to confirm cardiogenic causes (e.g. echo to identify left atrial enlargement, presence of pericardial effusion, thoracic radiographs showing cardiomegaly and left auricular bulge etc.).²

Echocardiography remains the most sensitive and specific modality with which to diagnose and classify feline cardiomyopathies, as well as assess risk of congestive failure or

thromboemboli formation with the presence of left atrial enlargement and intracardiac thrombus/spontaneous contrast.

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The effect of systemic disorders on the heart in the dog

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Although we are accustomed to categorizing disease processes according to specific body systems, in reality most diseases and subsequent treatment decisions we make often have implications on other organs. The heart and its extension, the cardiovascular system, is no different and often is susceptible to significant collateral consequences that may itself become the major determination of the patient's ongoing morbidity and even mortality. This discussion aims to outline some of the systemic conditions in our canine patients that may have major consequences involving the heart.

Gastric dilation-volvulus (GDV)

GDV influences on cardiac function include shock, cardiac arrhythmias and myocardial dysfunction. All of which contribute to early morbidity and mortality in GDV patients. Pathophysiology is mainly attributable to decreased tissue oxygen delivery, circulating inflammatory cytokines and shock, leading to a net effect of reduction in cardiac performance. Necropsy findings have shown myocardial lesions including myocardial degeneration, fibrosis and necrosis. Ventricular arrhythmias occur in ~40-70% of GDV patients and cardiac troponin I (cTnI) elevation is detected in all dogs with severe ECG abnormalities, with significant correlation between level of cTnI elevation and ECG severity and patient outcome.¹ It is therefore recommended to monitor ECG continuously during the period of hospitalization and instigate antiarrhythmic treatment if malignant ventricular arrhythmia is detected. Some patients may require further treatment such as positive inotropic support if myocardial dysfunction is present.

High output cardiac failure

High output CHF is ultimately caused by a decrease in systemic vascular resistance secondary to an underlying disease process, leading to activation of neurohormonal systems (e.g. RAAS, SNS, ADH) causing increased volume retention and overwhelming the cardiac function despite the lack of underlying cardiac pathology. Although it is rare it is an important condition to recognize as most of the time, by treating the underlying disease process, high-output CHF may resolve on its own. Conditions that may cause high output CHF include: chronic anemia, systemic arteriovenous fistulas, liver cirrhosis, renal disease, Paget's disease, Beriberi, chronic pulmonary disease, sepsis, and pregnancy. In dogs, chronic anemia is the most common cause of high-output cardiac failure.²

Hypothyroidism

Thyroid hormone exerts both direct effects on the cardiomyocyte and indirect effects of upregulating β -receptors causing enhanced adrenergic activity. In the hypothyroid state, various cardiac effects are manifested including overexpression of certain myosin heavy chain isoform subtypes, reduced sarcoplasmic reticulum Ca-ATPase activity, reduced beta adrenergic receptor number and responsiveness, and accumulation of mucopolysaccharide as well as fibrosis within the myocardial interstitium.² Furthermore, severe hypothyroidism can also impair myocardial blood flow causing systolic dysfunction and increase peripheral vascular resistance. Clinically, it is manifested as bradyarrhythmia (P wave prolongation, low QRS amplitude, inverted T waves, sinus bradycardia and rarely AV blocks), weak precordial beat, left ventricular wall thinning, and myocardial dysfunction. Thyroid supplementation may reverse most cardiac signs.

Hypoadrenocorticism

Within the cardiovascular system, cortisol maintains vascular integrity and responsiveness to circulating vasoconstrictors and aldosterone plays an important role in sodium and potassium transport and extracellular volume homeostasis. Patients with severe hypoadrenocorticism may present with hypovolemic shock and hypotension during an Addisonian crisis due to volume depletion from aldosterone deficiency, and hyperkalemia may cause further bradyarrhythmia including atrial standstill, sinus bradycardia, atrial/ventricular extrasystole, and second/third degree AV block. Systolic function and dilated cardiomyopathy (DCM) phenotype with concurrent CHF have also been documented in dogs with typical³ and atypical⁴ hypoadrenocorticism, with recovery following therapy.

Pheochromocytoma

Although not very common, adrenal medullary tumors may have significant cardiovascular consequences both due to their ability to locally invade the cranial vena cava and cause obstruction that may lead to systemic congestive failure, as well as the functional aspect of excessive catecholamine secretion leading to systemic hypertension, tachyarrhythmia (most commonly sinus tachycardia) and gross/microscopic structural cardiac pathology.^{5,6}

Myocarditis

There are many documented causes of myocarditis in the veterinary literature including non-infectious causes: such as pharmacologic agents (e.g. doxorubicin), toxins (e.g. rodenticide, cholecalciferol), immunologic diseases (e.g. IMHA), trauma, heat stroke, hemodynamic shock (e.g. GDV), neoplastic (peritumor inflammation/primary cardiac tumors, cardiac metastasis or paraneoplastic inflammation) and infectious causes including protozoan (e.g. Chagas myocarditis caused by *Trypanosoma cruzi*, leishmaniasis, toxoplasmosis, neosporosis, babesiosis, hepatozoonosis), parasitic (e.g. dirofilariasis, angiostrongylosis), viral (e.g. parvovirus, West Nile Virus, herpes virus, distemper, coronavirus), bacterial (e.g. Lyme carditis, bartonellosis, ehrlichiosis, leptospirosis, staphylococcus, streptococcus etc.) and fungal disease.⁷ Definitive diagnosis requires endomyocardial biopsy and therefore most diagnosis is a presumptive diagnosis based on clinical suspicion. Patients with myocarditis often have arrhythmias (ventricular arrhythmia being most common) requiring antiarrhythmic therapy, and some may have structural pathology demonstrating a dilated cardiomyopathy (DCM) phenotype that will benefit from positive inotropes such as pimobendan. A markedly elevated serum cardiac troponin I is highly suggestive of acute myocardial injury and inflammation. Some patients may have residual myocardial disease despite treatment of underlying systemic cause requiring long-term cardiac medication.

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