Investigating Sudden Death in Livestock

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Sudden death = no premonitory clinical signs.
Sudden death is one of the most common presentations of disease in ruminant livestock, partly due to the extensive nature of the husbandry and subsequent lack of observation but also due to the number of causes of rapid death that can occur. A quick search of the Cornell University’s ‘Cornell Consultant’ program reveals that for cattle there are a possible 224 differentials when sudden death is the only presenting sign and this is excluding the unique plant poisonings that occur in Australia but not in the USA.

A starting point to investigations into all ruminant disease is to understand your area, the husbandry practices that apply, and the pasture types that are available. For instance, in a summer dominant rainfall area with subsequent summer growing pasture species plant poisonings do tend to occur well into winter when stock are hungry, protein and energy deficient, and poisonous plants such as Green Cestrum are the only available green pick. So sudden death investigations at this time would always include a history of access to areas where there might be cestrum. Similarly, supplement feeding with urea is common through winter if standing dry feed is available and so urea poisoning is more common.

The use of pathology is very important in verifying a tentative diagnosis based on clinical signs and history but when investigating death in livestock it is rarely economically feasible to go on a “fishing expedition” for all possible causes if there is no initial suspicion on the cause of death. It is not out of the question for lab fees to reach $1000 in a complex case. It is important to stage testing so as to rule in or out the most likely causes first and then sequentially get to the least likely. In a vast majority of cases deaths cease as quickly as they occurred and owners become less likely to want to pursue a diagnosis at their expense. A thorough history along with clinical signs and post mortem findings is usually enough to have certain diseases in mind. If you suspect nitrate poisoning, test for it first and if negative, move on to the next most likely. Seeking advice from another veterinarian on which way to go can also be invaluable.

Necropsy of ruminants.
There are published versions of how to best approach the necropsy of ruminants but I have found that you need to develop a technique that best suits yourself. We are not all patient, methodical and thorough operators but that doesn’t mean we cannot find the answer. A few tips I have are:

1. Having the carcase with its left side down is probably the easiest way to view the abdominal contents without the rumen getting in the way. However, for certain conditions it may work the other way around. The spleen and reticulum are best approached with the left side uppermost so if you suspect lead poisoning (lead accumulates in the reticulum) or tick fever (enlarged spleen) you may want the carcase with the right side down.
2. Take the time to get the carcase to the display stage with both upper-most legs reflected back and the ribs reflected. Don’t be scared to use an axe for cutting through the ribs, especially the dorsal section.
3. Aqueous humour is an excellent sample in cases of sudden death for things like urea poisoning, nitrate poisoning, calcium and magnesium levels, ketosis, acidosis, and even anthrax can be tested. 0.5mls is enough to perform most tests.
4. Brain extraction is often worth the trouble. The brain often has characteristic changes suggestive of certain causes such as PEM, lead poisoning, enterotoxaemia in sheep, and many others.
5. Grab a sample of fresh spleen as it is a good substitute for blood in a dead animal. Can be used for virology amongst other things.
6. Petechial and spot haemorrhages around a carcase are not suggestive of anything other than that the animal was very unwell prior to death (not surprisingly). You’ll see these haemorrhages often on the epicardium and endocardium but you can see them elsewhere also. Don’t read too much into it but if you can’t find anything else it’s something to tell the producer. It can be one of the signs of enterotoxaemia.
7. If there’s nothing obvious on autopsy, think about metabolic conditions – hypocalcaemia, hypomagnesaemia, urea poisoning, nitrate poisoning, clostridial diseases.

Pathology test results are often academic by the time they are received and it is prudent to communicate this to clients. Up to 7 days for histopathology results in many cases can be expected and longer if testing is staged. A decision often needs to be made at the time about how to prevent further problems in the remaining at-risk stock. Most often this decision requires whether or not to move the animals from the paddock that they are in and/or whether something needs to be changed with their nutrition. Most of the time we are dealing with one-off cases of death, in which case there is little cause for panic. When deaths start to increase in number then a decision needs to be made about what sort of disease process is going on. For instance, a case recently investigated involved post weaning lambs moved from the weaning yards to a new paddock and then subsequently lambs were dying in the new paddock. Hypocalcaemia exacerbated by the initial mustering, yarding, change of nutrition and movement to the new paddock caused the deaths. Further movement to another paddock may have resulted in even more deaths but the initial instinct was that the new paddock was the problem. Similarly infectious conditions may be exacerbated by mustering, yarding, or being moved into close proximity to one another and a paddock shift would rarely provide a cure. However, in other situations, especially nutritional deficiencies and plant poisonings, a paddock shift is the best course of action. Nutritional stress plays an important role in disease susceptibility (eg coccidiosis in lambs) and a move to better feed conditions or supplying supplement feed can be much more economical than mass treatment of mobs.

Thought should always be given to sacrificing an animal that is presenting as very unwell or at least typical of the syndrome. A fresh post mortem exam can be your best avenue to a diagnosis. It can be a difficult judgement to make and even in large flocks of sheep owners may be reluctant to sacrifice an animal. Younger vets can be less confident that they will find something on autopsy and may shy away from advising this. Blood tests are often of little value compared to fresh and fixed tissues. My theory is it is better to do something rather than nothing and routine blood tests in ruminants can often be unrewarding.

Differentials
Having a likely list of possible causes comes with experience but there is generally a finite number of differentials for sudden death that encompass greater than 95% of cases. In my region the top differential diagnoses for sudden death in no specific order are:

1. **Pneumonia.**
   Can be found dead and reported to be “fine yesterday”. A history of some sort of stress, usually transport but co-mingling is also a common cause especially in weaners.

2. **Haemonchosis.**
   Worms are usually on the minds of sheep producers all the time but occasionally they are caught by putting sheep back onto heavily contaminated pastures or by using ineffective drenches.

3. **Lead poisoning.**
   A market issue due to residue concerns as well as a killer. Blindness is the most common reported clinical sign if seen alive. Edta blood is best sample from live cattle, fresh kidney in dead animals but can often find lead in the reticulum (left side approach).
4. Urolithiasis

Shouldn’t necessarily be a sudden death syndrome but sheep are often found dead rather than visualised as unwell. Often mistaken for intestinal parasites and drenching doesn’t fix the problem. Much work has been done to evaluate the best treatment but adding salt to increase water intake seems to be the simplest solution.

5. Metabolic

A history of mustering or held off feed can be suggestive.
6. **Plant poisonings**
   Generally stock need to be hungry so held in yards without feed or transported or just starving.
   Sometimes curiosity plays a part for naïve stock.

7. **Nitrate and cyanide poisoning**
   Both cause an inability to oxygenate the body but different mechanisms. Nitrate causes chocolate coloured blood, cyanide cause bright red blood. Stressed plants (cultivated, frosted, wilted) are most dangerous for nitrate. Regrowth of grain sorghum crops is probably the most dangerous for cyanide.
8. Bloat and acidosis

Frothy rumen contents, clinical signs in live animals, and the presence of a “bloat line” in the carcase are good confirmatory signs for bloat.

9. Urea poisoning

Convulsions may have been noted prior to death. Aqueous humour will confirm either urea or ammonia levels but be mindful that levels will change post mortem. Access to urea most important.

10. Clostridials

Very hard to definitively get an answer, especially in cattle but in sheep a positive glucose test on urine, fibrin clots in the pericardial fluid, haemorrhages outside and inside the heart, and lesion in the brain are all good ways to convince yourself of the diagnosis. Remember the vaccine only lasts about 3 months so repeat vaccination is often required, especially for lambs on crops.
Fibrin clot in pericardial fluid is suggestive of pulpy kidney.

11. Septicaemia

Could probably be included with clostridials but is always a possibility and bugs like anthrax and salmonella should be considered.

Erysipelas in a grower pig

Notifiables

There is no good reason to not look for notifiable diseases when applicable. The cost of testing is covered by the state government for endemic notifiable diseases and all testing, including to exclude other causes and find a diagnosis, is covered when looking for exotic notifiables. There is now a carcase-side anthrax test kit which is relatively simple to use and can be used up to 48hrs post mortem.

Anthrax test kit – a negative result

Other notifiables to consider in cases of sudden death might include babesiosis (tick fever) and possibly theileria for cattle. Bluetongue should be considered for sheep.

Conclusion
The most important elements of dealing with sudden death in livestock is not necessarily to know each disease intimately, but more to understand the process to work up the disease. By definition these cases are presenting with no or minimal premonitory signs and so the differentials in the first instance are similar for most cases. Often the diagnosis is evident from the history – signalment of the animals, location, available plant species, feed conditions, management practices – combined with any clinical signs that may have been observed. After this it is a process of staged testing to get the answer by the most economical and sensible means. Understanding the diagnostic tests and their sensitivities/specificities will help you determine what a sensible approach is and what is a “fishing expedition”. For example, in my experience it is not very worthwhile to chase positive diagnoses for either epsilon toxin or botulinum toxin in individual cases unless it is for academic reasons. Whilst a positive result is very helpful, a negative result does not exclude the disease. Similarly, the importance of the outcome to the client needs to be weighed up. As long as the deaths have stopped, the result, beyond excluding notifiable and important diseases, may not be in the financial interests of the client to pursue, and more tests will add more cost. Ruling out those things which could ultimately decimate a herd/flock or cause ongoing financial and production losses is the most important to thing to get to first. Ruling out notifiables is important both for regional, state and national biosecurity but also important statistically to satisfy markets that we are looking for notifiable diseases.