Chapter 14

Acute Systemic Conditions:
Diagnosis and Management of Sudden Death in Ruminants

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Introduction

Perhaps we'd better first **define** sudden death for the purposes of this discussion. One could start with: 'The discovery of dead, well-nourished non-perinatal animals within 2 days of them having been observed apparently healthy". One might quibble about the interval; it could depend upon the management system and species.

Gross necropsy findings

A sad feature of sudden deaths is that they are, by definition, unexpected and rarely observed, so the post-mortem interval is more likely to be long. Couple that with more rapid decomposition (see below), and it is understandable that post-mortem changes usually complicate the interpretation of the gross pathology in this class of mortality.

If there are any specific pathological findings, these will often be obscured by the more spectacular non-specific changes. To make matters worse, stockowners quite naturally believe that a dramatic event such as sudden death should be accompanied by equally dramatic gross necropsy findings, and they tend to question your diagnostic competence when you can't immediately point out to them the cause of death. They of course expect some clear-cut directions to be given to protect the remainder of the group from loss.

If the animals really have died quickly, with few if any signs observed (*ie*, the lack of observed illness is not merely the result of poor observation by an absentee manager), then certain assumptions can be made about the gross **necropsy findings**. The most important of these is the fact that most of the pathological changes will be probably be **unspectacular** and **non-specific**, since it usually takes time for classical text-book pathological changes to develop (think of abscesses, bronchopneumonia, etc).

Pulmonary congestion and oedema can be almost guaranteed, since blood flow to lungs reflexly increases with hypoxia. After death this congestion is exacerbated by gravity in the lower parts of the lung (**hypostasis**). Hypoxia and various substances like fibrin degradation products also damage the endothelium of pulmonary capillaries, which therefore tend to leak protein-rich fluid into the pulmonary interstitium and alveoli. This is **oedema**, whose most obvious feature is the stable foam produced when the fluid mixes with air and surfactant from the alveolar wall. The volume and tenacity of this foam can be an effective asphyxiant which rapidly finishes the animal off. We will all die with

some measure of pulmonary congestion and oedema (unless someone cuts our throat).

Rumenal tympany is likewise expected as a post-mortem change after sudden death; it will be enhanced by the fact that the animal's body temperature is either normal or elevated at the time of death, and the rumen is more likely to be more filled with fermentable ingesta than that of an inappetent animal dying more slowly.

Non-specific **intestinal changes** will be inevitable: the enzymes from enterocytes and bacteria loosen the villous epithelium within 10 minutes of death, and can even cause enterocytes to slough in the live animal if circulation in the bowel is sufficiently compromised (a common complication of circulatory shock). Since blood tends to pool irregularly in the bowel as the circulation fails, it is usual for parts of the small bowel in particular to show irregular and sometimes intense congestion. The lumen will always contain sloughed epithelium mixed with mucus (a very convincing facsimile of pus), and when this is mixed with blood that has oozed from the capillaries in a shocked individual, only an arrogant investigator would exclude haemorrhagic enteritis from the diagnostic possibilities without laboratory confirmation.

Serosal haemorrhage. Mention has been made above of the endothelial damage caused by hypoxia. Severe systemic illness also results in circulation of bacterial endotoxin and nasty substances absorbed from the bowel. All these insults prevent the endothelium from doing one of its most important functions, which is inhibition of the coagulation cascade. The resulting initiation of the clotting cycle (**disseminated intravascular coagulation**) results in rapid non-endothelial-mediated fibrinolysis (to keep the blood flowing), followed by rapid exhaustion of soluble clotting factors and then by spontaneous bleeding. This is usually most evident and spectacular on serous membranes such as pleura and epicardium. Perhaps the negative pressure in the thorax combines with agonal gasping to produce the pleural bleeding, while the poor old heart has to thrash away after everything else has closed down, so it can be excused for bleeding a little in the agonal period.

Thus, in a case of necrotic hepatitis (black disease), there will often be spectacular diffuse serosal congestion and haemorrhage, as well as pulmonary congestion and oedema, while the characteristic focus of necrotic liver, with its tell-tale fine haemorrhagic border, may be relatively small and lurking deep in the liver parenchyma and can be easily overlooked. To complicate the picture in this disease, the liver is notoriously susceptible to rapid post-mortem putrefaction that can rapidly obscure the pathognomonic lesion.

Causes of Sudden Death

Sudden death is broadly attributable to one or more of the following crudely classified causes: **acute infections**, **intoxications** and **physical** or **environmental disasters** such as electrocution, asphyxia and trauma, acute water deprivation and hyperthermia and the like. Of

these, only trauma is likely to be diagnosed easily at gross necropsy. So now we should look at the other causes in turn for strategies of diagnosis.

Acute infections

Clostridial infections are some of the most important cause of sudden death, and their diagnosis is difficult to confirm because of the rapidity with which saprophytic and pathogenic anaerobes proliferate after death. Routine bacteriological samples will usually contain these organisms, whether or not they are pathogenic, and they will tend to overgrow significant pathogens. In order for the microbiologist to get a better idea of the situation at the time of death, it is essential to include a panel of **impression smears** from likely parts of the carcass: from oedematous fascial planes in muscle, for example. Immunofluorescence can be used on these to make specific diagnoses, but even with simple Gram stains they can be very useful. Although the gut will obviously contain numerous bacteria of great variety, the importance of a smear of jejunal content for Gram-staining cannot be over-emphasised, since a surprisingly pure lawn of *Clostridium perfringens* can often be seen in direct smears of small bowel in cases of enterotoxaemia (absorption of epsilon toxin produced by this bacterium in the gut in sheep and calves). A segment of rib can also be a very useful submission, since it is slow to be invaded by post mortem invaders and can be easily chilled for transport.

Enterotoxaemia will always present diagnostic difficulties, since its definitive diagnosis rests on the demonstration that sufficient ε toxin has been absorbed from the gut to fatally affect the animal. Unfortunately, methods of detection of this toxin are only sensitive enough to detect its presence in the gut lumen, where, unfortunately, it may be produced after death by *C. perfringens* type D. Until we develop methods of detection of the toxin in fluids such as aqueous humor or cerebrospinal fluid, probably the most confident diagnosis of this disease is made on the histological demonstration of a peculiar periarteriolar oedema in certain parts of the brain, at least in sheep. Recent work has shown that this brain lesion is also seen in naturally occurring cases of enterotoxaemia in goats, and in experimental enterotoxaemia in cattle. So brain should always be submitted from all cases of suspected enterotoxaemia, no matter what the species.

Smears taken at necropsy are of supreme importance in cases of **anthrax**, of course. Ideally, anthrax is suspected on the basis of history of being endemic in the area and of sudden death with bloodstained discharge from several orifices, and a blood smear will be made from an ear vein before the carcass is disturbed, and stained with new methylene blue and examined on the spot for the large bacilli with metachromatic capsules. In practice, of course, this is rarely if ever done: few field people carry a suitable microscope or stains to field necropsies, or have the confidence to interpret such preparations under field conditions. So the necropsy will probably go ahead and the cause of death (with luck) will be suspected on finding the huge, pulpy black spleen. Realisation

will then be accompanied by a nasty sinking feeling as the zoonotic risks are remembered from long-past lectures, and frantic efforts at personal clean-up and carcass incineration will get under way (probably on a day of maximum fire danger). None of this worst-case scenario should, however, distract from the fact that the smear made in the field will usually be better than one made in the lab from fermenting internal organs, even if the smears have to go to the diagnostic laboratory to be examined. So **always submit direct smears**.

The same sorts of principles apply to cases of sudden death caused by **piroplasmosis**, which will also have large dark blackberry-jam spleens. Peripheral blood smears are just as important in these cases; in particular, the smear should be of **capillary** blood, since erythrocytes parasitised by *Babesia bovis* tend to stick to endothelium of cutaneous and cerebral capillaries. In a rotten carcass dead of babesiosis, scraping the tip of the tail until it is moist, then squeezing capillary blood to the surface, will yield smears in which the parasites can be seen or their antigens demonstrated by immunofluorescence. Better still is to examine the brain: even in a rotten carcass, the gray matter of the brain will usually show the deep pink blush of erythrocyte-plugged capillaries in *B. bovis* infection, making this one of the few specific gross changes that will survive putrefaction in these cases. The other textbook features of babesiosis (red urine, haemoglobinuric nephrosis) may easily be lost by bladder emptying or destruction by rot. Cerebral tissue tends to be much better preserved than internal organs.

Investigation of **intoxications** is dealt with in another session of these workshops.

Physical and environmental catastrophes

History is of course critically important in these investigations. An outbreak of sudden deaths in a group of Victorian Angus cattle, which had recently been transported to a Queensland feedlot in summer, initially puzzled investigators because there were no deaths in Brahman cattle at the same time in the same feedlot. Perhaps some plague endemic in the Queensland Brahmans had infected the poor Victorians? Turned out that the watering system had broken down during a weekend heat wave, and the stoic Brahmans were better acclimatized to the extreme conditions.

Five dairy cows were found dead in one corner of a holding yard. They'd been heard bawling frantically seconds before, and survivors were still careering around the metal enclosure. The owner in that instance had the presence of mind to check the wiring in the adjacent shed; otherwise he might have been electrocuted, too. He said he made the diagnosis because "The bellowing had exactly the same pitch as that of a cow that once got an electric shock in the bails" and he'd never forgotten it. Some things they don't teach in vet school.

A Strategy for Investigators of Sudden Death

The notes above show that there are some extra stresses placed on the investigator of sudden death; presumably that is why television scriptwriters seem to be endlessly obsessed with them. What is really useful in these circumstances is a flexible but logical investigative framework upon which to hang the investigation; a planned sequence that can nevertheless be modified to suit the circumstances. What follows is one version which might reduce the regrettable omissions that so often confound diagnosis.

Animal/s reported dead by owner/manager, who wants you to attend

During the first phone contact, reel off a list of the questions below, to be answered when you get to the property (this will allow checking of records, if necessary: better than guessing, and wasting time on the phone):

Questions

- Age, breed, sex, numbers & origin of animal/s and cohorts.
- Duration of current location.
- Feed management.
- Water.
- Most recent introductions.
- Vaccination history.
- Treatment.
- Precise timetable of current event.
- How are the cohort animals?
  (*i.e.*, manager to go and check them, and have a good look at the environment, while awaiting your arrival).

Action when you get there

- Look at any survivors that are showing clinical signs.
- Think about alternatives to routine necropsy (*i.e.* for the anthrax cases).
- Get stuck into necropsies (owner can answer the questions at the same time).
- At display stage of necropsy, stop and try to select which option of the three basic **causes** is the best bet.
- Take samples, with emphasis according to most likely cause.
- Advise on management of survivors according to most likely cause (*e.g.* alternative pasture/rations when poisoning is, on balance, the most likely option). Some sort of advice is nearly always appropriate before you leave the property.

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