

Field pathology – techniques, findings and diagnostic options

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Abstract

Necropsies are an integral part of health programs in cattle feeding operations. Multiple benefits include better methods of fine tuning prevention and treatment programs. Disease processes are made visible and can be used in the very important process of training cattle handling personnel. Efficient use of time is enhanced by practical necropsy procedures. Guidelines are provided for necropsy interpretation and differential processes. Necropsy tips include use of necropsies to evaluate vaccine effectiveness, evaluation of pneumonias as dynamic processes, recognition of different types of interstitial pneumonias, and clarification of relationships between acidosis and other digestive diseases. Diseases of the central nervous system can be differentiated through necropsy observations supplemented with judicious use of laboratory assistance.

Key words: necropsy, pathology, pneumonia, IAP, bloat

Résumé

Les nécropsies font partie intégrante des programmes de santé dans les exploitations d'engraissement des bovins. Il en découle bien des bénéfices incluant de meilleures méthodes pour raffiner les programmes de prévention et de traitement. Les mécanismes causant la maladie deviennent alors visibles et peuvent être utilisés pour la formation très importante du personnel manipulant le bétail. Des procédures pratiques de nécropsie améliorent l'utilisation du temps. Des directives sont fournies pour l'interprétation de la nécropsie et des processus différentiels. On conseille d'utiliser la nécropsie pour l'évaluation de l'efficacité des vaccins, l'évaluation de la pneumonie en tant que processus dynamique, reconnaître les différents types de pneumonie interstitielle et la clarification de la relation entre l'acidose et les autres maladies digestives. Il est possible de différencier les maladies du système nerveux avec les observations de la nécropsie complétées avec l'utilisation judicieuse du laboratoire.

Introduction

Field necropsies offer numerous benefits to the practicing veterinarian and the livestock owner. The most productive approach is to conduct multiple necropsies to monitor disease patterns and causes of deaths, as well as responses to processing programs, treatment programs, and overall management. This requires that we keep it practical. What do I want to know? Is there some oddball disease or is there an economically significant problem? Practicality means utilizing time efficiently and recording information so findings can be later compared and compiled. It's better to do an accelerated necropsy on several animals than to spend an hour doing a veterinary school sanctioned perfect necropsy. A typical program should involve cowboys and doctor crew, and should put on a show and teach. The show gets onlookers' attention and teaching is a never ending process in the feedyard. It improves competency and maintains a focus on the health management objectives. Cowboy necropsies are not as good as necropsies done by veterinarians, but they're a lot better than no necropsies, and they help teach if used properly. Email necropsies may serve a purpose, but I often can't figure out what's going on even when I'm standing there with a knife in my hand. I need to feel it, compare it, evaluate the time of death, and even smell it.

Necropsy Technique

Technique is only important if it improves efficiency and outcomes. It may be practical to emphasize certain systems. Consider history, and then perhaps choose to save time by not looking for thyroid, adrenal, or pancreas. Necropsies should be made easy through use of appropriate instruments. Knife sharpening is worth the time it takes to become proficient using whatever technique is chosen; good references are available.⁵ Find a preferred way of opening the thorax using whatever equipment you prefer, whether it is simply a knife for smaller carcasses or a mechanical aid for heavier cattle.

The principle of brain removal is to get it out of the skull using axe, saw or water pressure. We need a usable specimen, not a “perfect” specimen. Don’t hesitate to use a reciprocating saw, especially if you’re an older person.

Systems Evaluation

Keep the real etiology in mind. Don’t get distracted by counting bugs; they’re generally all in the feedlot somewhere. Think about the vaccines that really work versus those that “sort of” work, versus those that don’t work. Make your own lists. Consider this when evaluating necropsy findings. Work = *Clostridium chauvoei*, infectious bovine rhinotracheitis (IBR); sort of work = bovine viral diarrhea (BVD), bovine respiratory syncytial virus (BRSV), *Pasteurella multocida*, *parainfluenza virus 3* (PI3); don’t work = *Clostridium perfringens* A, B, C.

An Overview of Things Routinely Found on Gross Necropsy

BRD Bronchopneumonia and Stages from Fibrinous to Chronic

Typical bronchopneumonia (shipping fever) is the biggest killer in the feedlot, therefore it deserves the most attention. Bronchopneumonia is not a timed event—lesions develop at different rates, so we can stage the process but not easily assign a precise timeline.² We often need laboratory backup, but all the bugs are in the average feedlot. There is usually no biosecurity in typical cattle operations; therefore, try to determine what’s important. Get a feel for the predominant problems, then submit specimens for microbiology with specific questions for the diagnostic laboratory. Use the gross and laboratory information to adjust programs, realizing the decisions are more subjective than objective. Teach cowboys—use antibacterial sensitivities to discuss their role in decision making, but don’t overlook other inputs such as treatment success. This may help with regulatory issues in some cases.

Pleuritis

The bugs don’t read the book, so don’t try to identify them by looking at a general lesion. Pleuritis is the result of inflammation which can be caused by multiple organisms. I can’t look at a fibrinous pleuritis and name the causative organism. Chronics may suggest a review of the health performance profile. A category of “progressive” can be useful in contemplating why an initial treatment success can end in failure.

Peritonitis

There are multiple causes of peritonitis. It is often useful to evaluate chronicity of the lesions. Acute inflam-

mation with reddening, but little fibrin, may reflect acidosis. Suppuration may suggest organisms penetrating the abdomen through injury or enteric defect. Chronic fibrous lesions tell us the injury occurred some time ago, such as in hardware survivors.

Feedlot Bloat

Is it acidosis or bloat or DOES IT MATTER? Did someone see it die? If not, the differentiation of ante-mortem and postmortem can be difficult.¹ Bloat lines can develop after death, and pelvic edema may be suggestive but is not specific. The real value may be in backing up and looking for the real causes including ration, bunk management, and environment.⁹ Always check rumen pH. The pH strips are adequate because the objective is an approximation. There is often postmortem change in pH, but this has been overstressed. Recent actual feedlot studies demonstrate the rumen pH typically varies only about 0.2 points in the few hours after death.⁵ Checking rumen pH routinely will establish meaningful patterns. At the least, a high pH may forecast urea problems.

AIP (Atypical Interstitial Pneumonia)

Primary interstitial pneumonia in the feedlot can be roughly divided into the classic tryptophan/3-methylindole or “fog fever” form, and a less understood but frequently seen form that is most common in heavy feedlot cattle. This form is a combination of lesions. Many of these have been previously treated and many have mixed lesions including bronchopneumonia, interstitial pneumonia, and bronchiolitis.⁴ Distribution is usually “patchy”, giving a less homogenous gross appearance, and often the different presentations all present in 1 microscopic field.¹⁰

There has been some evidence of a spike in occurrence coinciding with more digestive deads, possibly related to typical acidosis events in heavy cattle. The fact that 50% or more of eructated rumen gases are typically inhaled has led to speculation that these gases may be involved in the pathogenesis. However, the impact of rumen gases, particularly H₂S, is very difficult to verify. Only sudden death due to anoxia with high H₂S levels has been reported in cattle, and studies in rodents have demonstrated acute alveolar edema.⁸

Congestive Heart Failure (Right Heart Failure)

These are fairly common in heavier steers and can look a lot like pneumonia. Most of these seem to get pulled and treated as pneumonia cases. We see more at higher altitudes, but these are related to pulmonary hypertension and the individual physiology is just as important as altitude.³ Perhaps these are similar to genetic expression associated with various respiratory and cardiovascular outcomes in humans.

Diphtheria (Necrotic Laryngitis)

These are often called calf diphtheria, but can occur in a variety of cattle sizes that are sometimes well into the feeding period. The precise etiology and pathogenesis remain confusing. Something predisposes the animal to *Fusobacterium necrophorum*, and study is made more difficult because most seem to get pretty bad before they are spotted. IBR virus is a fairly frequent finding, but any role is not clearly defined. Consider recrudescence.

Tracheal Edema (Honkers)

One looks at the trachea to find it, but then what? Causes are obscure, but at least we know the cause of death.

An Overview on What Things Might be Less Commonly Encountered

BVD—Mucosal Disease

Mucosal disease is not the real problem. Acute exposure and persistently infected cattle (PI) are the source of economic loss. A percentage of PI cattle end up being the source of significant failures. Acute BVD can produce digestive lesions, but acute lesions are rarely encountered in southwestern feedlots. The most costly impact is through immunosuppression, which is probably very common but can't be fully measured. It takes a lot of imagination to find microscopic lesions in the lungs that are caused by BVD, so any losses are likely the result of immunosuppression. So, if I own the cattle, I don't vaccinate incoming stressed cattle. This is popularly regarded as heresy. There is no good documented support for the concept that repeated vaccinations somehow find a sweet spot in resistance.

Leptospirosis

Leptospirosis is very hard to diagnose. There are no good simple tests that work much better than histopathology. Mere presence of leptospires without speciation or lesions can be misleading. This is an example of the hazards of polymerase chain reaction (PCR).

Redwater (Bacillary Hemoglobinuria)

This can be an extremely small lesion, and can be missed. It may be possible to confirm with histopathology if it is found. The vaccine should have worked. The clostridia are probably always around, so look for the cause of liver damage.

Blackleg

Vaccine is effective! If found in fed cattle, there is usually trauma. Don't be too precise in expecting textbook lesions. You can't always find it grossly, by histopathology or by culture. Fluorescent antibody tests

may be most practical, but be careful as false positives can occur.

Hepatitis

Look for old fluke damage. Plant toxins like pyrrolizidine alkaloids can find their way into feedyards.

Acidosis-Grain Overload

"Digestive deads" are perhaps the most costly cause of death loss and reduced performance, partly because it affects heavy cattle, is very common, and is frequently fatal. We need to keep monitoring the problem with necropsies, including developing a feel for rumen pH, and need to work with nutritionists toward solving the bunk management issues. The diagnostics are not clear cut. This is one of those cases where history and necropsy (primarily to eliminate other obvious causes of death) need to come together.⁹ Don't lose sight of the cause if there is a bit of interstitial pneumonia. Don't worry too much about antemortem vs postmortem bloat, or "is it acidosis or bloat?". At this stage, it's all a feed-related syndrome, and the answers are in the management plan plus execution.

TEME (Thromboembolic Meningoencephalitis)

This is pretty much a gross diagnosis after the brain is removed. Feedlots classify these as "brainers", and they are pretty hard to sort without a necropsy. The differential frequently requires lab support if there are no typical brain or heart lesions suggestive of *Histophilus somni* infection. Polio is pretty hard to diagnose with gross examination, and even with histopath it can be difficult to diagnose in some cases. CNS disease can also result from invasion of the meninges by a variety of bacteria.

Tuberculosis

This disease is not common. If you find typical gritty lesions, decide what to do next. This may involve a call to regulatory officials. This is not a pleasant thought, but it is essential.

Hydronephrosis and Pyelonephritis

These are usually not a big problem to diagnose, and they are a sporadic finding. These may be a cause of death, but there is not much to do other than say "There, I found it". Be alert for dragged-in acorn poisoning and do histopathology if suspicious, especially during droughts when cattle are more prone to eat less palatable feeds.

Vena Cava Syndrome/Metastatic Pneumonia

The lungs often reveal liver abscess vascular invasion leading to metastatic pneumonia. Full abscess rupture means a rapid death, so it pays to include a thorough liver evaluation in the necropsy.

Hardware (Traumatic Reticulopericarditis)

Always feel for adhesions anterior to the reticulum, and consider this as a cause for heart failure. Hardware can set off a more extensive peritonitis, often much later. The most important concern is that a large number of these suggest a hardware source in the system, like after a destructive storm.

Parasitic Infection

Unless there are lots of them, do fecal flotations and guestimate the value of deworming. Try floating suspected mucosal scrapings in water. If nematodes are readily visible, there is considerable infestation.

Intestinal Intussusception

This is an easy gross diagnosis to define cause of death, but was there an enteric problem that predisposed the animal to the problem?

Findings that Trigger the Need for More Extensive Diagnostic Work

Unfamiliar Lesions

We tend to develop a comfort zone, and unusual lesions in more than 1 animal should send us looking for help. This is especially true if the possibility of a foreign animal disease or regulated disease enters the differential.

Confirm Postmortem Diagnosis

The lesions may suggest a disease, but they less often define etiology. That may be important in adjusting programs for prevention and treatment. We need to periodically check to make sure we're not starting to believe our own stories. Communicate with the diagnostic laboratory to practicalize and economize—never run tests if you don't know what you are going to do with the results.

More Specific Diagnosis

When it gets past sporadic, more work needs to be done. Heart failure is a good example. Is there an

underlying cause such as monensin, gossypol or dietary defects?

Feed Related Toxicity

Monitor sulfur levels as preventive medicine, especially with some of the variables introduced with ethanol byproducts. Levels over 0.4% dietary sulfur are correlated with increased polioencephalomalacia incidence.⁷ Try to get management to do routine feed sampling. Without that, submit feed samples when observations are unexplained.

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