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Bovine Respiratory Disease & Diagnostic Veterinary Medicine (Managing Respiratory Diseases in the Herd)

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Year in and year out, diseases of the respiratory system are a major cause of illness and death in cattle from 6 weeks to two years of age. Sadly, this is as true today as it was 30 years ago despite development of new and improved vaccines, new broad spectrum antibiotics, and increased fundamental knowledge as to the cause of disease. WHY? I don't have the answer and I doubt if anyone does. As a pathologist, I often see firsthand the devastating effects that bacteria can have in the lungs of cattle that die from respiratory disease complex or shipping fever. I often wonder to myself, "They know this calf had a bacterial pneumonia, why did they send it in?" "What do I need to know to do a better job?" Is there some missing bit of information that will tie this all together?" As a pathologist, I also see lungs from cattle suspected of shipping fever that might have looked abnormal in the field but with no evidence of pneumonia when examined microscopically. Obviously there is a disconnect somewhere. This presentation will focus on shipping fever and will mention some conditions that can be mistaken for bovine respiratory disease. I am a diagnostic pathologist so will not dwell on vaccinations or treatment; that is the purview of other animal health professionals, especially your veterinarian. Diagnostic considerations will be a constant theme. There are a number of reasons for performing diagnostics on diseased animals; the most important is learning.

The focus of today's presentation will be on LEARNING; what can we learn from diseased animals and each other.

The bovine respiratory disease complex (BRD, shipping fever) is a multifactorial process. There are many variables that come together to cause disease. No two scenarios are exactly alike but they often culminate in severe and fatal bacterial pneumonia. Perhaps it is these complexities that stymie our efforts to control BRD. A lot of my experience has been as a pathologist examining respiratory disease in feedlot, not range cattle. The variables, viruses, and bacteria are the same whether or not the cattle are pastured or confined in a feedlot; the feedlot environment simply magnifies the different participants. The feedlot is also a pretty good laboratory for a better understanding of BRD. All of us, producers as well as practicing veterinarians, laboratory diagnosticians, and a host of other animal health professionals should strive to share our knowledge at every opportunity; it is only through learning from each other that we can make better strides in controlling BRD.

Bovine Respiratory Disease Complex – SHIPPING FEVER

The Pathogens

When shipping fever is mentioned one of our first thoughts is viral infection. Viruses are an important component of BRD but are not necessarily present in all outbreaks of respiratory disease. I doubt if I can tell you much that you already don't know. The major viruses are IBR (bovine herpesvirus type 1), parainfluenzavirus-3, respiratory syncytial virus, and bovine virus diarrhea. BVD is not generally considered a primary respiratory pathogen in the sense of the other viruses just mentioned; it is more likely responsible for crippling the immune system. The industry is making pretty good strides at eliminating calves persistently infected with BVD virus but it is still out there and persistent infected calfs serve as a source of infection for herd mates. Other viruses may play a role in shipping fever: bovine respiratory coronavirus and rarely bovine adenovirus may be involved in some cases but the school is still out on these.

As you all know, bacterial pneumonia is the real killer in shipping fever. These names are also very familiar to you; the most important being *Mannheimia hemolytica*, *Pasteurella multocida*, *Histophilus somni* (formerly *Hemophilus somnus*), and *Mycoplasma spp. (bovis).* Historically, the number of fatal cases has been dominated by *M. hemolytica* followed by *P. multocida* > *Histophilus somni*. *Mycoplasma* infections on the other hand tend to be sporadic but often explosive with large numbers of animals experiencing respiratory disease and joint infections. There are years according to some studies, however, where this relatively prevalence is thrown out of kilter; in 1998 the number of cases at the Oklahoma Animal Disease Diagnostic Lab was dominated by *Histophilus*; why is just conjecture, another unknown when it comes to BRD.

The Vaccines

Vaccines for BRD are hopefully being improved, developed, and marketed at a steady rate but, being a pathologist, I'm certainly uncomfortable discussing vaccines. I do tell students at the University of Wyoming that there are over 60 different vaccines marketed as an aid in the prevention of BRD. I can appreciate why the choice of vaccines for a herd health program can be confusing. Your veterinarian is the best source of regional information concerning the proper selection of vaccines, when and how to use them, and other guidelines. It is important that vaccines on the farm or ranch be handled and used properly. The best vaccine on the market is of little benefit if mishandled, misused, or given at the wrong time.

The Antibiotics

Like vaccines, several new antibiotics have been marketed in recent years. These antibiotics are efficacious against a broad range of bacterial pathogens. Use of these powerful antibiotics is often of little benefit if given too late in the course of disease when damage to the lung is far advanced. Failure to diagnose disease early and to aggressively institute antibiotic therapy effectively is the most common cause of treatment failure. Importantly, a trend of bacterial pathogen's developing antibiotic resistance noted 10 to 15 years ago is

reversing itself. I would like to believe that this is due to the more judicious use of these drugs by the cattle industry; congratulations.

Diagnostics for BRD

Performing diagnostic tests for cases of BRD is the only way for producers, practicing veterinarians, and diagnostic laboratory personnel to learn. The learning process can begin at the onset of clinical illness but does not have to end with the death of an animal. The learning process also begins with the producer. Whether or not the learning process begins and ends with the rancher is up to his/her discretion but it can continue on to involve detailed and yes, somewhat costly, diagnostic laboratory procedures. Is the benefit worth the cost? This is something the producer will need to answer for themselves. It is unlikely that cattle producers at the level of the farm or ranch budget for extensive diagnostics. If your budget and appetite for diagnostics is limited, you can still benefit from on-the-ranch necropsies but the potential for learning, although a definite plus, represents only the tip of the iceberg. What price can we put on learning?

It is imperative that diagnostics for viral infection be done early in the course of clinical disease; many viruses such as PI-3 and RSV are present only during the early stages, if you wait until an animal has died the virus may no longer be recovered and there may be no definitive residual lesions of viral infection when the lung is examined microscopically by a pathologist. What then are possible tests for viruses that can be used at the onset of infection? First, there is no substitute for isolation of the virus. For this, deep nasal or pharyngeal (throat) swabs are samples of choice. It is important to get the swabs sopping wet with secretions and send the swabs chilled in a sterile container (you can break a cotton-tipped wooden swab off in a blood tube or a zip-lock plastic bag). Not to be crude but a virologist colleague of mine recommends using tampons instead of cotton tipped swabs because of their increased absorbency. The downside of virus isolation is the turn-around time that can be up to 2 or 3 weeks. Depending on the laboratory's capability and resources, a sensitive test to detect the virus is the polymerase chain reaction that can be performed within a matter of hours. Another different method of testing is serology. Blood samples need to be taken at the onset of clinical illness and another sample 10 to 14 days later; a four-fold rise in antibody titer is a good indication of viral infection. Again, however, the downside is waiting 2 weeks for results. Even though virus isolation and serology require several days, results may help you and your veterinarian make management decisions for future years.

As already mentioned, bacterial pneumonia is the immediate cause of death in the great majority of BRD cases. *Mannhemia hemolytica* and *P. multocida* as well as *Histophilus somni* are common inhabitants of the nasal passages and, in my opinion, a positive culture tells you little regarding the cause of the bacterial pneumonia. *Mycoplasma* are less common in the nasal passages, some reports indicating only 3-6% of healthy cattle harbor the organism but again, a cause-effect relationship is difficult to establish. If you'll take advantage of the opportunity, you the producer can learn a lot from gross evaluation of the lungs of dead cattle, even to the point of identifying the most likely bacterial pathogen and in some cases, getting an estimate of the duration of the pneumonia. The two most common bacteria, *M. hemolytica* and *P. multocida* are the easiest to differentiate and will be used here

as an example. In many respects, these two bacteria represent the extremes of fatal pneumonia in cattle. *Mannhemia hemolytica* causes an acute, rapidly progressing, fulminating pneumonia. The affected animals are obviously very sick and many cases will die from 3 to 7 days after the onset of illness. If treatment is delayed, even if the animals recover, there will be considerable residual lung damage. At the opposite extreme, the pneumonia caused by *P. multocida* tends to be insidious and develops more slowly. It is difficult for these animals to be identified as clinically ill. As a result, the pneumonia is already in the more chronic stages when antibiotics are first given, often resulting in treatment failure. Again, if the animal lives there will be residual damage. The gross pneumonic lesions typify these two extremes. Commonalities include a relatively sharp line of demarcation between the firm, discolored anterior and ventral areas of pneumonia and the adjacent more normal lung tissue. This is where the similarities stop. With *M. hemolytica*, as much as 80% of the lung can be affected. **The most striking difference, however, is that the dark mottled pneumonic areas** bulge above the level **of the more normal adjacent lung tissue.** Other features include often copious deposits of yellow, friable exudate (called fibrin) on the surface of the lung and distension of the septae that separate individual lung lobules with yellow fluid and fibrin similar to that on the lung surface. Contrast this with the pneumonia caused by *P. multocida* which typically affects less than 50-60% of the anterior and ventral lung in fatal cases. Again, **the most striking difference indicating *P. multocida* is that the firm, affected lung is uniformly plum-colored or purple and collapsed below the level of the adjacent more normal areas.** There is often little to no fibrin on the lung surfaces and interlobular septae are not distended with exudate or fluid. Now that you are empowered with this new knowledge, how do you validate your diagnosis and why is this distinction important? The only way validate your observations is to submit samples of the affected lung to a laboratory for culture and, preferably, for microscopic examination by a pathologist. Obviously, if the bacterial culture agrees with your assessment, you are on solid footing and you have learned a little from this presentation. But what if no bacteria are cultured; often the case if the calf had been previously treated with antibiotics? This is where the pathologist can be of value. Microscopic lesions are pretty distinctive also and the pathologist should be able to point you to either *M. hemolytica* or *P. multocida*. There are also additional points of confusion. There are cases where both of these or other combinations of bacteria are cultured from diseased lung. Usually, gross evaluation will favor one or the other but lesions of *M. hemolytica* often dominate because this is the more fulminant pathogen. The pathologist should also be able to help in this regard as microscopically there will be some areas that suggest *P. multocida* and others that are more typical of *M. hemolytica*. Now to answer the second question posed above, why is this information important? Remember, *M. hemolytica* causes an acute fulminant infection. Are you watching the cattle often and close enough; are you treating early and aggressively? Additionally, there is a better chance of identifying predisposing viral infection because these animals often die in the more acute stages. If you are seeing animals dying of *P. multocida* pneumonia, you likely did not detect the clinically ill animals early enough in the infection for effective antibiotic treatment. You may need to look a little harder and closer. Additionally, since many of these animals die in the later chronic stages of disease, if viruses were present they are likely long gone.

Treatment for BRD

Again, not that I need to reinforce the point, but diagnostic labs and specifically pathologists are really not in a position to make treatment decisions or recommendations. Positive bacterial cultures include antibiotic sensitivity testing but this is to be used as a guide to selection of an appropriate treatment regime by you and your veterinarian; not as an absolute. The three rules of antibiotic therapy remain valid today, even with the newer antibiotics that have come on the market:

- Proper early diagnosis
- Selection of an appropriate antibiotic
- Proper dosage, route, and treatment schedule

Another recommendation is that effective treatment should be accomplished with as little stress to the cattle as your management capabilities will allow. Some of these animals are clinging to life by only a thread; added stress may send them into the abyss. When the situation becomes sufficiently dire, metaphylaxis or mass medication may be the best or only option but make sure there are adequate, justifiable reasons for the added stress and expense of treating the entire herd. Here, we can also learn a little from the feedlot situation but you and your veterinarian should develop an algorithm that works for you.

- Some indications
    - Past history of high morbidity (sickness) and mortality (death) – this can be cattle from a certain origin that have a history of BRD under certain conditions, i.e. southern cattle or recently weaned calves exposed to extremes of cold wet weather +/- nutritional concerns.
    - Over 10% pulled for treatment plus a death loss on any one day
    - Over 25% pulled for treatment plus death losses in any 3-5 day period

Conditions That Might be Mistaken for BRD

There are many conditions that can be mistaken for bovine respiratory disease. The clinical signs that make us suspicious commonly include difficult or rapid shallow respiration with the head held low and extended. These include metabolic conditions such as acidosis. As some of you know, the lung helps regulate pH in the body by exhaling carbon dioxide. In severe acidosis, there is rapid respiration in an effort to exhale more CO_2 in an effort to correct the imbalance. Other causes include, but are not limited to, pain, fever of any cause, and left-sided heart failure. This is one reason why perceived clinical respiratory illness should prompt closer examination and death losses should be followed by a necropsy early on; to help rule in or rule out BRD as the cause of death. It is important to approach the examination or necropsy with no preconceived idea as to the underlying cause. If the evidence fits so be it, but if it doesn't don't force a diagnosis. Gross lesions at the time of necropsy can fool you also. Gaseous distension of the lung (emphysema) is not uncommon with severe labored respiration as a near terminal event no matter what the underlying cause. Congestion and accumulation of fluid in the lung (reddened, heavy, wet 'edematous') is also commonly observed as a near terminal event and can be especially prominent in congestive heart failure. The old adage, "if it's not firm it's not pneumonia" are words to live by. In a

pinch, if there is still confusion, a piece of severely pneumonic lung will not float in water but congested and edematous (red and wet) lungs will float.

"Mortui vivos docent"

These words, "Let the dead teach the living", are as appropriate and meaningful to cattle producers and ranching operations today as they were to the ancient Latin scholars who tried to develop some meaning from and understanding of disease. Like these Latin scholars; we should learn from our past experiences with animal disease and from each other. Sure, producers can learn a lot from veterinarians and animal disease diagnosticians but it doesn't stop there; they can also learn from their diseased animals. More importantly, veterinarians and diagnosticians can learn a lot from producers. Veterinarians and diagnostic labs learn from producers who share their knowledge and who are willing to pursue diagnostic testing.