Practice Methods and Techniques

James J. Jarrett, *D.V.M., presiding*

Dr. Jarrett: Welcome to the first session of the Twelfth Annual Conference of Bovine Practitioners. First, I would like to welcome our colleagues from Mexico. We do have simultaneous translation. I am sorry we cannot translate Georgian! Speakers are asked to speak slowly.

Rumen Sampling Techniques:
Darby J. Moeller, *D.V.M.*
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Thank you very much. I work for Merck, Sharp, and Dome Research Laboratories and the last few years we have been evaluating a new antibiotic to prevent lactic acidosis in feedlot cattle. In doing so we pulled several thousand rumen samples and after finding it very difficult to do we found some methods to make this quite simple and I thought you might be interested in this technique - you might be interested in using it to monitor what is actually going on in the rumen and see if it has any relationship to any other diseases in the animal. We used a calf which we had made sick with lactic acidosis by holding off feed for 24 hours and then putting back on feed on a 85% concentrate ration. In the past we simply used a large tube using a foick speculum and sliding the large tube down into the rumen. We would agitate the tube or we would not agitate the tube and it really was quite difficult. The results were variable as far as getting a rumen sample. If it takes very long to get a rumen sample you are going to get a lot of saliva which will dramatically change the pH in the rumen. We were looking not only for pH but we were sending these samples in for lactic acid and VFA tests. So we had to strain these samples through cheese cloth and got to be quite a task. The first calf had a pH of 4.1 and it died about two hours later. The technique that we finally devised is equipment, except for the rumen strainer, that probably all of you have. On the end of the tube there is a little piece of black tape with a rumen strainer attached. This is available from Precision Instruments, Lincoln, Nebraska. All you do is slide the little rumen strainer into a plastic tube that fits and then slide the plastic tube into a bigger tube. And this you can get by going to a hardware store and working with the tubes until you get ones the right size. Then you insert it through to frick speculum and attach the one end to the bottom end of an equine stomach pump. It will fit perfectly - you slide the tube down into the rumen. It takes about three or four pumps and you will get a nice clear sample into the tube. This made all the difference in the world to us because we were running pHs on these samples and then freezing them immediately and then sending them in for VFAs and lactic acid tests. But it is a real easy technique to use. The pH meter which we used to use is expensive and I am sure not many of you would want to invest money in it. The Corning Model III, a couple of years ago cost $150 - I don't know what it costs now. The only bad thing about it is that the probe breaks very easily so you really need to order an extra probe because someone will end up breaking one! I thought you might be interested in this because it is one way of monitoring the rumen. The pH tells you a lot about the rumen because as the pH goes down, the lactic acid levels go up. We have found that any animal with a rumen pH below 5.5 pH was getting sick, 5.0 they would have a severe diarrhea, and a 4.1 - that is the lowest level I have had an animal live! But, it just may be a useful tool to use in finding out what is going on in the rumen.

Differential Diagnosis of CNS Problems
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Many of you were present today at our seminar when we talked about BVD in cattle. We spent the entire day talking about the problems of BVD and the losses that were incurred from it. I am sure you will recognize that we never did mention the severe losses from CNS - Central Nervous System Disorders. Well, that is the topic we will cover now the differential diagnosis of central nervous system disorders in cattle. Although we do not consider these conditions to cause large losses, they certainly can. I would like to review this with you just shortly to show you what can happen. First of all a case in southwestern Kansas - 548 calves with respiratory problems; not too bad of a problem -30 dead! I am not trying to tell you that CNS is a number one cattle disease, but I am sure that all of you at the seminar must feel that BVD must be very close and when we go to the seminars tomorrow, we will find that respiratory disorders must be number one. I am just saying that this can be a very serious situation and we are going to look at a few of the problems. I will list my top ten and go through them briefly with just a comment or two about each one and how I feel we can do a better job of diagnosing them. (1) We need a better history, a very good history. (2) We need to do a good necropsy, (3) we need to submit the proper tissue for examination to a laboratory. We see *polioencephalomalacia* most often; it is acute, the animals are usually blind, they isolate themselves once they go into these CNS type symptoms such as tremors

and recumbency and there is usually little hope for them. The downer animal's head is pulled back, or if in lateral recumbency, the eyes are rolled back. I think most of us would agree this is typical of polio. Grossly, necropsy lesions are primarily in the cerebral cortex and histopathologically there is laminar cortical necropsy. The typical "polio" brain can very readily be diagnosed from gross necropsy by identifying the cortico-laminar without histopathological examination. (2) *TEME - thrombo embolic meningoencephalitis.* The animals are usually not blind; many times it follows a respiratory outbreak, probably more frequently than not. Many times the animals are merely found dead with no symptoms whatsoever. This is a little difficult to discern. Typical of this condition is a knuckling type of a gait and the hocks tend to knuckle over. The gross lesions may reveal a thrombus that has formed in the cerebral cortex. Histopathology would present the typical thrombosis with an embolism that can be seen grossly and is quite diagnostic.

(3) *Nervous coccidiosis* - this is not very well understood. It is a condition that is seen quite readily and often. There is an article in the 1976 MVP by Julian Kennedy that pretty well describes coccidiosis. When we think of this disease we usually picture the old steer that is just bleeding from the rectum. These rarely, in our experiences, have turned into the nervous type. On histopathology we can see various stages of these organisms in the crypts and the complete erosion and necrosis of areas of the colon. Nervous coccidiosis may follow a very acute case of diarrhea, which may not even be bloody. It may hit three or four animals in a herd, may attack very suddenly and the only symptom that is noticed is that if the animals are moved very suddenly, one of them will go down very quickly and go into convulsions. We can make a tentative diagnosis as to just what we are dealing with. I do not know of any treatment for them; I have never saved any. I have tried many supportive treatments with not much results. They have very nearly the same type of tremors as polio, but I think we would have to associate it more with the onset after a little excitement phase and that could be the way we can differentiate it. There are no gross CNS lesions. Obviously, if we did a good necropsy, we would find hemorrhages in the cecum and colon. The location is unknown as far as the brain is concerned. It is probably a biochemical disease that certainly does not show up on histopathology.

(4) *Listeriosis* - Many times these animals will be seen to circle in one direction and isolate themselves. It usually is associated with the feeding of silage or haylage. Gross lesions of the brain are not observed. Histopathology reveals microabscesses in the brain stem. (5) *Hepatic encephalopathy* - This has been pretty well described in a number of species, but not very well in cattle. When the liver is completely destroyed, a lot of things happen. The brain is very vascular and it just does not receive enough nutrients. So, hence the encephalopathy. The symptoms are similar to polio. One animal was blind, wandered off, got through the fence and fell into a pond. The next one acted nearly like rabies - bellowed, went down with a saw horse gait. The owner found a couple of them dead every morning. Some calves look pretty normal and then they go into severe convulsions and die in about four or five hours. There are two forms: acute and chronic. Some of them live through the initial attack. The attack may really not be noticed and they will do real well for maybe two or three weeks. Then all of a sudden they will exhibit severe symptoms. They will go down and die. A good necropsy is very important to differentiate it from polio and TEME. Severe hemorrhages occur in subscapular and subcervical nodes and throughout the kidney areas. There is massive edema, and a very fatty liver that floats in formalin. Good necropsy can certainly reveal massive hemorrhages and edema. The folds of the abomasum are enlarged due to edema. I do not know of any other type of condition that causes this type of edema and hemorrhage in the bovine animal except a severe hepatic disease, one that just totally destroys the liver. As far as the brain is concerned, there are no gross lesions. Microscopically however, we can discern some changes in the white mater and in the brain stem. It is a vascularization type of change. This has been described in ammonia toxicity which will produce very nearly the same thing as can be seen here. Spongiform degeneration is another name for it. This is in the white tracts where you can see large vacuoles.

(6) *Moldy feed,* or unpacked silage, can produce symptoms very similar to polio and it can be very drastic. I do not know how many of these we see that we do not recognize. I am sure that this does happen over and over again and it is just diagnosed as a CNS condition, and since it is an individual animal, that is about all that happens. I think that this is something that we could keep in the back of our minds and by following those steps of doing a good necropsy, and submitting some samples we could differentiate it.

(7) *Acute lead poisoning* - here is where a real good history is important. They can present very nearly the same signs. The gross lesions would be basically none. If we were going to examine them we would look in the cerebral cortex for some lead inclusions, better yet, we would submit liver samples and if we get over 20 parts per million of lead, I think that is considered diagnostic.

(8) *Rabies* - A condition not considered very common in the bovine animal but it certainly can occur. Something that is first and foremost in our minds when we see one of these animals. Any of these that I have described could certainly be mistaken for rabies or *visa versa.* They exhibit very nearly the same signs, usually bellowing and this type of reaction associated with rabies but this can be true in any CNS condition. Gross lesions are basically none. The Purkingie cells are the ones we have examined histologically for inclusions bodies. A better method is to use the fluorescent antibody (FA) test. When we are doing a necropsy, we put half the brain in formalin and the other half is frozen so that

we would certainly be covering about all the avenues.

(9) *Pseudo-rabies* - this has been considered rather important lately, more so in swine and particularly in cattle that are associated with swine. We do not have many of those producers anymore. It is ordinarily associated with the usual mad itch type sign but it can produce some of the very basic CNS signs that could be very readily confused with any of these other disorders. A non-suppurative encephalitis is seen on histopathology. Again the FA examination is a little more reliable. It can be done very readily on this other half that was not formalized and give us quick accurate diagnosis.

(10) *Intracranial abscesses* - these do occur and many times are in just a single individual animal that turns up with a very strange CNS disorder. When we split the skull, we find an abscess that pretty well explains it. Basically the same symptoms occur as in the others. Gross lesions could certainly be variable: they can be in the cerebral hemisphere, and I have seen a number of these down in the brain stem area. Histopathology would show the abscesses; many times they can be seen grossly and taking up a very large portion of the brain. And another one that fits along with that is *intracranial lymphoma* - everyone of these that I have seen exhibit some type of "pop-eye" symptom along with CNS signs and very often we see lymphoma cells on hispathology. The lesion can be variable in any place in the brain.

Briefly then, these are the ten conditions - Polio can be mistaken for many of these others. TEME would be associated with respiratory problems and knuckling, but not being blind like polio; nervous coccidiosis occurs in a type of outbreak - and so on down. Basically there are three things: get a good history, get the tissue, it is not too hard to do, it does take a little effort, but any kind of a meat saw can sure separate the skull (the animal does not need to be skinned). Split that skull, you do not even have to saw clear through, put one half in formalin and freeze the other half. Get a real good specimen and come up with a very diagnosis, at least a healthier doagnosis! Again, history, necropsy, and then submit the proper tissue.

Simplified Bacteriology for Veterinary Practice

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This is the smartest group of veterinarians I have ever addressed. Normally speaking, up in Minnesota, this talk takes three days! I will show you pictures of the major equipment from a very small practice in Wisconsin such as the incubator, a little alcohol burner, some of the test tubes with some of the media, a microscope, and this practice has a nice little autoclave. They prepare their media in little bottles and store them in the refrigerator until they are ready to use them. Now the equipment that you see here is 90% of what you need to do microbiology. Just before they need the plates, they heat up the media that is in the bottles and pour their plates, blood agar plates and also mannitol media. Large plates are used for susceptibility testing. They have a API 20 E system. A lot of these systems are coming on the market, this is the one I would recommend most highly. It is a little system that allows you to identify bacteria with a great deal of accuracy and minimum amount of investment in reagents. This particular system will run ten biochemical tests and you get a code by giving numbers to these plusses and you get these numbers and then you look up in the little code book and identify the bacteria. Some strips have ten tests and others have 20. These sell for about a little less than $2.00 a strip. The reagents you need come with the API system when you buy it. This practice had just two days of work sitting there on the counter, very near the front of the building. It is where the farmers that come in can see them by looking around the corner and they mentioned to me how interested the farmers are in the results of the bacteriology. They come up and say, "Where is mine?" and they can show it to them.

If you are going to do microbiology in practice, the best thing to do is to limit your goals and use an organ system approach. The three you can use in bovine medicine are mastitis, pneumonia, and scours. If you can cover these three areas you are going to have some success and if you use your microbiology to govern your antibiotic usage, you are going to have something that is really going to help you in practice which is really what I am up here to tell you about. You are going to improve your therapeutic success, particularly when you have a lot of antibody resistance, you are going to know which antibiotic to use. As I looked over the records from this practice, they would have no idea how to treat many of these cases. Another thing you find by doing susceptibility testing is that it reduces your costs. You have a tendency in practice to go for rather expensive, heavy treatment when you do not know which drug to use. You can find yourself going back to some of the old drugs that are a little bit cheaper. You will reduce the development of resistance when you are working with large groups of animals, big herds. You use a lot of antibiotics and do not know what to use, you will use quite a bit of combinations and this will lead to the development of resistance.

The final point I would like to make is that when you do susceptibility testing, you put the animal health program in the hands of the veterinarian. There is no way they can get this service except through you when you can do this.

With the large antibiotic plates you can use a dozen discs. You can pick four colonies from your plate, original culture, and put them in broth and you let that incubate a few hours until you get a medium density - this technique is called the Kirby Bower technique. You would have to find this written up, I am just recommending it. After you get the right density of growth for a few hours of growth in the broth, you swab it on to these real large plates. The large plates allow you to put about a dozen discs on - if you use a small plate you can only put four discs on it to get really good results. So, you get your discs on and sometimes you have to push