more to demonstrate their importance than we have in this country, but perhaps over the next year or two we will be able to do some catching up because there are several stations in this country where work is being done with mycoplasmas in cattle respiratory disease. Some of the ways that mycoplasmas could possibly cause respiratory disease are certainly the close association with the cell sometimes appears to lead to a toxic effect which is not associated with a toxin but simply with the whole organism itself. Then there are exotoxins produced by some mycoplasmas and certainly metabolites produced by some mycoplasmas which, because of the close association with the cell, cause cellular-changes and breakdown with interference with wholesale nutrition. There is competition for certain nutriments. There could also be a change in antigenicity which would lead to immune response which could cause lesions. Most importantly, I want to point out today the immune suppression which has been proven with mycoplasmas from other host species but among those from cattle only with *mycoplasma bovis.* Certainly it is an important immuno-suppressant of the cellular immune system in cattle. This work has been shown most clearly at California and I think it is something which we will be hearing a lot more about as far as the possible predisposition by mycoplasmas to bovine respiratory disease. We wonder sometimes why in experimental infections we don't see a disease which looks like what we call a mycoplasma infection in cattle. We feel, though, because there is an increase in mycoplasma antibodies, that there is a good chance that they are involved and certainly because we see respiratory disease go into arthritis from which we can isolate mycoplasma that they probably are involved and then certainly Dr. Hjerpe among others has shown that antibiotics such as tylosin which are quite effective against mycoplasmas are not effective against pasteurella and are effective in at least 40% of shipping fevers. He attributes that probably to their mycoplasma cytal activity. We also know from analogy to other host species that they are quite likely of importance.

Discussion

Question: Have these fat cattle, that you just showed a picture of, that are showing atypical interstitial pneumonia, have those animals been on Rumensin?

Answer: No.

Question: If you are involved in a problem with atypical interstitial pneumonia and there happen to be three or four animals showing signs at the same time, different degrees--some rather severe and some milder--is there anything that you could do to help in treating these or to tell the owner?

Answer: I think it's best to prepare the owner for the worst. First say that the three or four animals all might die, certainly the more severely will and that he may want to consider slaughtering the one or two that are worse and if he'd like to chance treating the less severe ones, while there is no specific treatment, you could try dexmethazone and antibiotics, as a treatment for this. I had one animal that I thought was going to die that I gave very large doses of Vitamin C, and it recovered.

Question: These animals had been on alfalfa hay, would it be a good idea to change the feed?

Answer: Probably it would be a good idea to change feed, even if it meant buying some different hay. I would suggest changing the hay for a few days anyway.

Question: Could you slaughter these at the regular packing plant?

Answer: I think perhaps with the veterinary inspection there they might not comprehend what this type of pneumonia is and may condemn it ante-mortem. So better to slaughter it privately.

Question: A large number of cattle in the west are vaccinated for lepto, what is the justification?

Answer: There were some clinical cases of lepto in South Texas this year, more than they had seen in some time, testimonials from oldtime practitioners state that lepto is a significant problem and can be a severe clinical disease in unprotected cattle. Probably, *lepto. pomona* and *grippotyphosa.* Vaccination does prevent this and as a testimony to the merits of vaccination, we don't see clinical lepto to any great extent in cattle. In dairy herds, lepto can be a fertility problem. We have diagnosed *lepto hardjo* infection in some dairy herds in our area, characterized by infertility, not by outright abortions. Febrile response with some clinical illness, some anorexia and infertility problems in the herd in general which did respond to vaccination. Confirmed by titer rises between acute and convalescent samples in the clinically affected animal.

Question: Is there any difference histologically between these three different types of interstitial pneumonia?

Answer: I think there is but I'm not a histopatholigist at all. Dr. Jensen says that he believes that you can differentiate histologically whether the reaction might be due to hypersensitivity reaction or dietary, that you may be able to tell this on histopathology.

Question: Dr. Frey is behind the podium here so I'd like to ask him. Could you elaborate more on your gnotobiotic calves and your attempt to reproduce shipping fever by injecting the different known etiological agents, and what were some of your findings?

Answer: I could tell you briefly what I had on the few slides remaining which did deal with the combination infections. We didn't know quite where to begin on combination infections because there are endless

permutations that one can choose from. But, because we did pick up some sero conversions to *mycoplasma bovis* which I mentioned can be immuno depressant and because we had picked up respiratory syncytial (RS) sero conversion in the same groups of calves, incidentally the sero conversion to respiratory syncytial virus was 100% and it was only in one group with the *mycoplasma bovis.* Those were perhaps a little more severe, so we thought that maybe we should try that as a combination to start with. We did give *mycoplasma bovis* about a week before the respiratory syncitial virus to take advantage of the immuno-depressing action of *mycoplasma bovis,* if any, in a calf, and then followed with respiratory syncytial virus and in two calves neither one became sick at all. These were black, white-face calves and we had seen a number of other times where we gave some of the cross breds, or those not largely Hereford, respiratory syncytial virus with no effect from the inoculaton. We followed in two other calves with respiratory syncytial virus first and this time hoping to take advantage of the damage it appears to cause to the mucociliary apparatus in the respiratory tract, waited about eight days until we got the febrile response, which is about the time we get it in the calves. We do not always get it, and as I mentioned in the two black, white-faces which we used earlier, and which incidentally may have had a positive, or may have had antibodies, because at that time we did not have a serology test which was suitable for RS virus. But, at any rate, in these two Herefords we did know that they were negative to RS virus, we gave RS virus, took their temperature twice a day and followed with *mycoplasma bovis* about eight days later. One of those calves was a cross-bred and she was polled, did not get sick at all. The other calf probably would have died if we hadn't killed it about four or five days later. We did see pretty good lesions in that calf. That's about as far as we've gotten in combination infections.

We've done quite a few more single infections with RS virus and have maybe 50% response with moderate to severe illness and in the other half of the calves what I would call mild response with usually a febrile response usually no higher than 104.2 and in some of the more severe ones we get 105-106.

Question: Have you done any work with pasteurella?

Answer: Well, the next step and on about the last slide I had those two steps, and then the third one was the combination of all three, pasteurela, M. bovis, and RS virus. Probably I'm not sure in what order, but maybe it would have been M bovis first, then RS virus, then pasteurelia.

Other people, like Dr. Smith of Montana State have done quite a lot of work with RS virus...and about all I can say is that from the work in this country, we do know that calves need to be at least two months and preferably four months of ages before one gets a good response to this virus, and other than that we don't know a lot about the virus in calves.

From the floor: I think if you are talking about experimental challenges with infectious agents in an attempt to reproduce something that looks like shipping fever pnuemonia, I am prejudiced for sure because I've been successful with only one way so far and that is to find a serologically negative calf to *hemophilus somnus,* use a culture of it that is virulent and has been kept in chicken embryos, where I know the virulence. One group tried to follow this procedure this last fall. They didn't think they could break controls on a trial. They were planning to have 5 controls when they got done, but they thought just to be safe they'd have 10 and they killed all ten flat out with H. somnus intravenous challenge with less that 100 bacteria. If you want to do something with a pathogen try Hemophilus.

Question: Do you have any more information on the use of Rumensin in pulmonary emphysema cows?

Answer: Maybe someone here has more up-to-date information on that.

Question: Have you ever seen or observed any animal get emphysema when they have access to Rumensin?

Answer: I haven't, this relationship with monensin and emphysema is something more recent, in the last year or so. So when I used to see this a couple of years ago I never thought of looking in to it.

Question: This practitioner has seen a problem in pastured cattle where, following a dry pasture and then rain where the pasture has become a little more lush, that several weeks later he would have some of these cows dying from clostridial infection, and he was wondering if I had any experience with that.

Answer: I have not. I don't know whether there is any relationship or not.

Panelist: Only one question is that I have felt that once in a while cattle on lush pasture were more susceptible to clostridial infection not due to strictly anoxia but possibly hypomagnesemia. Some of those pastures with high potassium levels have a tendency toward hypomagnesemic. And there may be some metabolic reasons for increased clostridial susceptibility in a hypomagnesemic cow.

Question: In the interstitial pneumonia feedlot cattle, is the mortality as high in the recently weaned calves as it is in the yearling cattle?

Answer: Mortality isn't as high, because in this problem in recently weaned calves, there may be a 60, 70, or 90% morbidity and the mortality may be only 3, 4, or 5%. Whereas, in yearling cattle the morbidity is very low, maybe 1 or 2 animals in a pen and the mortality is almost 100%.

Question: Are pasteurella bacterins still used widely?

Answer: As far as I can tell, in my contact with practitioners, some swear by them and some swear at them. But, I'd say that about half of them that I work with probably do use pasteurella bacterins. What's your experience, Ned?

Dr. Brown: I think that we have seen some practitioners that have used them consistently and feel that in comparisons in feedlot instances off the truck, one or two doses, that they feel that they are very beneficial. I do this on the basis of morbidity and mortality information and performance of the penned cattle. Let's say the isolated herd

of purebred Herefords that has never encountered anything in their life except fresh air and sunshine and they are the healthiest calves around, there's been no movement of cattle back in this direction and they are highly susceptible the use of pasteurella bacterins, the old Bar 3 or Bar 4 program, with killed IBR and two doses of pasteurella was one of the most favorable ways of preconditioning that calf.

Question: In the list of causes of sudden death in feedlot cattle that you surveyed, did you see any *Clostridium perfringens,* type D.

Answer: At that feedlot in yearling cattle it did not seem to be a problem. I'm sure that it occurs in some feedlot animals, no question about it but there is very little work done to reproduce enterotoxemia in cattle. Many of the case histories that you read about where sudden death was perhaps caused by *Clostridium perfringens* type D in cattle were kind of circumstantial evidence. In other words, cattle died and they thought it was due to enterotoxemia, they vaccinated and the problem seemed to stop. With that in mind at CSU, we did attempt to reproduce type D enterotoxemia in cattle, we were able to reproduce it, the signs, and the lesions. All I'm saying is that in the yearling cattle we did not observe it but I'm sure that it could be a problem in some feedlots under certain conditions.