that we have got to show our clients that we are an economic benefit rather than a salvage operator.

From the floor: One comment, I think it should be made clear that this program alone isn't going to accomplish anything. This fellow has got to do something himself.

Moderator: I think though, that if you work with herd health your relationship with this client just improves vastly over the years. At first he's calling you and then he wants you to come, he's glad to, he'll call and say what day are you coming? A lot of times when you are there maybe a couple of days before, he'll say, "You are coming Wednesday aren't you," or something like that, at least, in my area and they are looking for you to come.

From the floor: One thing you have got to realize is that everybody has a program to sell. You go out there hoping you can present it, tackle it and put a pitch in there to sell benefits. That's where veterinarians are very poor. They don't say a word about benefits. That's what the farmer is buying.

Chairman: How many of you are sending out a news information letter to your clients? I think they do this in a lot of other areas a lot more than we do in the midwest. I think maybe it's because we have so close proximity to our colleagues next door or sometimes the crossing of different clients, but in many areas they send out a lot of newsletters and information and I see nothing wrong with this at all.

There was a suggestion from the floor to try a pilot letter on mastitis and reproduction.

Pacific Area

Chairman: Dr. S. Smalley, Chandler, Arizona.
Practitioners: Dr. Robert Darlington, Snohomish, Washington.
Dr. Robert Abernathy, Duncan, British Columbia.
Clinicians: Dr. Otto Radostits, Saskatoon, Saskatchewan.
Dr. Robert Bushnell, Davis, California.
Subjects: Mastitis; calf diseases; abortions.

Dr. Darlington: On a large dairy with its calf mortality problems, we started several years ago force feeding colostrum, looking at colostrum antibody levels and running the zinc sulfate turbidity test on every calf in the herd. At that time they were milking about 700 cows. The management of this herd decided that a gallon of colostrum was the right amount to feed. We argued about whether these calves needed a gallon. One of the two farms involved was milking a thousand cows where all the calves were not run through the zinc sulfate test. We did look at the situation and they ran a control study for us. The night shed man gave a gallon regardless. The day shift man gave 2 quarts if the calf would nurse it out of a bottle and then put it into a esophageal tube if a calf did not nurse and those calves received 2 quarts. We had no losses in these calves in about 30 days, but we did have a difference in morbidity and I think it made us take another look at the volume of colostrum that we should be giving to these calves. Thirty-six of those calves received 2 quarts and of those, 7 had to be treated within the neonatal period for some condition. Seventeen calves received a gallon and none of those were sick. I realize that is a small number, but it is suggestive that volume of antibodies going into these calves did make a difference. To satisfy the need of whether or not you had to force feed or whether we could allow these calves to suck on their own, we maintained some bull calves and left them with the cows for four hours and ran zinc sulfate turbidity tests on those calves. Half of them had poor or none and half of them had good antibodies. I think this told us that it was necessary to force feed or make sure that calves did receive, in a lot of cases probably 3 quarts, rather than a gallon, but they did receive the volume force fed within the first 15 to 30 minutes. Going back after a year, there were 251 calves that we had data on, there were more calves that the zinc sulfate turbidity test had been run on. I went through and reviewed the health sheet to figure out the difference in the previous year of running the zinc sulfate test on morbidity and mortality. The percent that died was 2% for the good and 2% for the moderate, 11% on the poor and 24% on the none. One of our laboratory technicians in the hospital ran these samples and the herdsman or calf man would draw the samples and bring them to the office. The manager of the farm, just to check out the lab technician, continually ran in samples with 3 or 4 numbers all drawn out of the same calf to make sure our test was consistent. We were happy to find out that it was every time he checked us. When we got into figuring out the morbidity I took any calf that had been treated within the first 6 weeks for any condition, respiratory, scours or whatever and had received any medication and tabulated them for morbidity. There was only calves that lived through the 6 weeks, the mortality was out of this morbidity study so it is not a true morbidity. We ended up with a 20%, 40%, 60%, 54% morbidity of calves that had been treated for some

condition during the first 6 weeks. Today we are still using this as a management tool. All the cows are still being checked today on that farm as a management tool. If we start getting a lot of negative reports we just go up and tell the shed men that they are not doing their job.

Chairman: Bob Abernathy is going to have some comments on the vaccination program he uses in his dairies to minimize calf scours.

Dr. Abernathy: I wanted to say 2 or 3 things about some different areas. We didn't think today that we should talk a lot about colostrum because I think that probably everyone in the room realizes the importance of colostrum. Usually when I get into calf scour problem in one of my herds, and my herds are a lot smaller than Bob Dalington's herds, basically 40-50 cow herds, I usually talk to these dairymen and tell them it is extremely difficult to run a meter on a calf sucking off a cow's teat in the barnstalls. I try to encourage them to get 2 quarts of colostrum into them and basically we do that in problem herds by milking cows before they calve. We try to take 2 quarts of colostrum when they are in the barnstall before they calve and then give it to them with a Carnation bottle when they are on the ground, shortly after. Many of these cows suck extremely well when they are down on the ground, wet and when they are 20 minutes old. It is sometimes difficult to sell this to clients until you demonstrate it to them. In our practice we don't necessarily try to sell too many scour tablets. Most people who walk through the door are looking for scour tablets but I usually try to get the conversation turned around to some kind of a discussion on nutrition. We basically come up very strongly against milk replacers in our practice in replacement calves and we try to encourage people to feed 4 pounds of milk twice a day to dairy Holstein calves. We encourage people not to increase the 4 pounds twice a day and keep it level 4 pounds, twice a day straight through to weaning. We have also recently in the last year or two worked strongly in suggesting that the calves have water in front of them right from day one. I have found that less than 30% of my clients have water in front of their baby calves. In many of the baby calves are 10 weeks old before they have water in front of them. They are getting all of their water from the milk or the milk replacer that is offered to them twice a day. I have had some problem herds, two or three in fact in the last year where, during the last 2 or 3 years we have had great difficulty even when we were getting what I thought was adequate colostrum into the calves early enough. We still had weak calves in the first week that tended to die of something so that in those herds in the last year and a half we have started to vaccinate with IBR-BVD vaccine which in my practice area was never done in dairy cattle. It is absolutely amazing how in those herds recently after IBR and BVD vaccination while the cows are open, there has been a turn around in calf health.

Chairman: Otto has some comments on suckling versus force feeding colostrum and he also has some comments on the work he has been doing on with *E. coli* vaccine for calf scours.

Dr. Radostits: I would like to mix a little bit of some old information with some new information to give us some background on diseases of newborn calves. Perhaps we can also get some discussion going and hopefully get some comment on the two previous speakers and what they have related about calf problems. Dr. Darlington didn't really tell us about the diagnosis of those calves in his problem herds. Maybe he will get to that later. This is what I consider to be some new information we use now in western Canada in both beef and dairy calves on the agents of acute undifferentiated diarrhea in calves. If you run into an outbreak of scouring calves under 5 days of age we think there is a 95% chance that it is *E. coli.* If you have scouring calves 2-3- weeks of age we don't think it is *E. coli.* This is based on following herds for the last 8 or 9 years. So we now think that we have good information that the enterotoxigenic *E. coli* cause diarrhea primarily in calves under 5 days of age. That is useful information for the clinician in sorting out what he is trying to control on a farm. In the viral diseases that cause diarrhea in calves, Reo and Corona viruses occur 7-10 days of age and older and I believe that we are generating information that these beef calves on pasture, 3 weeks of age, running around with their tails up in the air, are probably the viral diarrheas. Similarly with dairy calves, we don't have as much information on that in dairy calves as we do in beef calves. But in general I think this is true. So that is important for treatment and control in dealing with scouring calves under 5 days of age, probably *E. coli* bacteremia and septicemia is a good possibility whereas in calves 10 days of age it is not. Therefore you would not have to treat them with antibiotics unless you thought you were dealing with a salmonellosis which occurs in older calves, two or three weeks of age. I have almost an evangelistic feel about this. I just want to leave this with you. If I try to fight infectious diseases in calves, I try to think about 3 principles when I am on the farm. We are going to emphasize colostrum here today. The first one is to reduce exposure of the calf to infectious agents. That means clean calving stalls, clean barns, sanitation, hygiene, you have all heard that before so that is what I look for when I am investigating a problem herd, that first principle. The second one is to establish and maintain a very high level of non-specific resistance namely through colostrum, so we look at that. The third one is to increase specific resistance maybe with some specific vaccines as management aids or tools to management in some cases and I will mention the work that we have been doing with an *E. coli* bacterin which will be available commercially in the next month or so. I think of those three principles when I am investigating outbreaks of infectious diarrhea in baby calves. Number two is being emphasized here today and number 3. Just to jump to number 2, colostrum, and establishing and maintaining a high level of non-specific resistance. That all starts with the nutrition of the dam. There was some very good British work

done in the last year or two that shows that if you compare the quantity and quality of colostrum from beef cows fed very poorly throughout the winter months, or fed a ration designed to lose body weight, compared to the colostrum quantity and quality in cows that are fed to maintain body weight there is quite a remarkable difference in colostrum quantity and quality which is reflected in the calves' colostral immunity and subsequently in its ability to look after infections.So nutrition of the dam is important. We push the idea in dairy herds to get colostrum into these calves in the first 2 to 5 hours and even before that. Even if that means force feeding colostrum and I was very glad to hear that Dr. Darlington is looking at that principle. What amounts? We say to producers, at least 5% of body weight in the first 2 to 5 hours. The amount of serum immunoglobulin achieved in the calf in 24 hours depends on a whole host of things which we took at veterinary college and I think sometimes we tend to forget. I just want to emphasize that when we talk about vaccines. Vaccines will not be the magic wand to control all scours. To vaccinate the cow, the calf still has to get that colostrum. So the amount of serum immuglobulin achieved in this calf at 24 hours of age depends on the immunoglobulin concentration in the colostrum and that depends upon nutrition and genetics. The amount of colostrum available depends upon how well the animal was fed and that usually is not a problem in dairy herds. The ingestion of colostrum depends on the vigor of the calf, maternal confirmation and maternal behavior if you leave the calf with the cow. If you force feed the calf colostrum then you don't worry about these 3 problems. The efficiency of absorption depends upon the age after birth which the calf first sucks, the amount ingested and the mothering ability. Something we shouldn't forget about and which makes the ingestion of large quantities of colostrum very early after birth very important is the absorption efficiency of colostrum. Overall it is only about 25-30%. The efficiency absorption of gamma globulins from colostrum at 24 hours goes down to 0. So the efficiency of absorption is not that great and you wonder if mother nature has made a mistake. It is very important to get as much colostrum into them as soon after birth as possible. Now what about force feeding colostrum? This is something that we have worked on for a while. We have asked the question many times, if you feed a calf colostrum by a stomach tube that goes into the rumen, does it stay there or does it go into the abomasum? If you feed calves 80 milliliters of colostrum per kilogram of body weight by 6 hours, let's say a 45 kg calf gets 3.6 liters and that is about 1 gallon of colostrum force feeding right after birth, the serum hemoglobin in those calves will go from .07 milligrams per milliliter at birth to 31.73 mg per milliliter on the average by 24 hours. That is a very high level of serum immuglobin in those calves. In summary, force feeding of colostrum either with a nipple if you can get it is even better or with a stomach tube or esophageal feeder (we use barium enema bags with plastic tubes) you can get a gallon of colostrum into these calves with reasonable ease. Let it run in by gravity flow, it gets down into the abomasum. If you give much less than that it may sit in the rumen. That is an important point. If you give much less than a liter, it doesn't get in there as quickly, and I am speculating that by distending the rumen it is pushing it into the abomasum. The rumen is very small in this baby calf. So if we get high levels of gamma globulin or colostrum into these calves right after birth, we are going to achieve very high levels of immunoglobulin. Maybe Bob can comment on this with respect to his calves.

What is the nature of the protection provided by colostral immunoglobulin? Two broad categories, systemic protection and local protection in the gut. If you get colostral immunoglobulin in the calves they are protected against coliform septicemia and from pneumonia in early life but not necessarily later on, unless they start producing their own immunoglobulins at 6-8 weeks of age and they are protected from dying from diarrhea. We now know that there is a very nice relationship between the levels of gamma globulin in the calf 24 hours of age whether or not it dies from diarrhea. The calf that has high levels of gamma globulin can develop diarrhea but it virtually needs no treatment whereas calves with less gamma globulin are very susceptible to dying from diarrhea regardless of the cause. That is a very intriguing phenomenon. The second point is that colostrum provides protection in the gut. It prevents infectious diarrhea if there is specific antibody there. That is the important thing and that is what the vaccines do. If you inject *E. coli* vaccine into cows they develop antibody against those *E. coli* and the calf subsequently becomes exposed to those same homologous *E. coli* it does not get diarrhea. There is an important distinction there. *We need specific antibody in that colostrum to prevent that calf from getting diarrhea;* specific *E. coli* antibody, specific reo-virus antibody, specific corona virus antibody. If that specific antibody is not there, the calf may still develop diarrhea but he gets over it very quickly if he has a high level. And I think that explains why diarrhea is so common in our beef herds yet a much smaller percentage die. Something we musn't forget is the duration of passive immunity which is not very long. The IgG which is in large quantities in colostrum lasts about 60 days and most calves have to start synthesizing their immunoglobulin and these other immunoglobulins, IgM, IgA are gone by 21 days. That means that our sanitation and hygiene has to be very good and this correlates well with the incidence of pneumonia. We don't see much pneumonia in calves 2 or 3 weeks of age. When do we see it? 6-8 weeks of age in our dairy calves. They don't synthesize their own immunoglobulins and when they are exposed to those antigens they will come down with pneumonia.

Practical recommendations to maximize the transfer of colostrum immunoglobulin? The first point we should remember is we should try to mimic the natural situation if we can. Try to minimize getting away from that. If we are going to leave the calf with the dam for a while, we want to

develop a good neonatal calf relationship in the first 24 hours that will promote the absorption of gamma globulin from colostrum. Unfortunately in many of our dairies now we are taking the calf away from the cow and raising him in an individual stall. I think we have to do a better job for this calf to achieve high levels of immuglobulin. Number 3, force feeding colostrum. Will the owners force feed colostrum? Yes, they will, our small herds will but I am wondering if the large herds will. And a clean environment — that is very important. You mustn't forget about that because you can break down any immunity. The third principle is the increase in the specific resistance of this newborn calf. We could vaccinate the dam, that's nothing new, we tried *E. coli* vaccines 20 years ago and thought they had some limited success but it lost popularity. We had some wrecks. Norden brought out the reo-virus vaccine a few years ago. Vaccinate the cow for what? Well, *E. coli,* salmonella, rota virus, IBR, BVD, you get asked these questions every day. Should I vaccinate the dam? Should I vaccinate the calf against these antigens? Dr. Abernathy mentioned BVD. In 8 or 9 years of tracking several of the herds we have never had a BVD virus in calves under 4 months of age. I don't think we have any evidence that it is a cause of acute diarrhea in baby calves. Maybe, but I haven't seen it. I don't think we have any evidence that IBR virus itself is the cause of explosive outbreaks of diarrhea. I think the bad ones are *E. coli,* salmonella and rota virus. Salmonella is not so important unless you are working with intensified herds. *E. coli* and rota virus are the important ones. While we get into vaccines we have to look at the cost, benefits, risks and the logistical problems. In Saskatoon, 5 or 6 years ago, we developed what we call VIDO which is the Veterinary Infectious Disease Organization and they received a 5 year mandate to work on calf scours. In the last 2 years Steve Akers and his research group have culminated their work in the production of a *E. coli* bacterin which a laboratory in Toronto is producing and it will be available in January 1980. I am just going to summarize this. Based on the herds we have worked with in Western Canada and in herds in which enteric coli bacillosis was a problem, the K 99 positive antigen was common to many of these outbreaks. So they have made a bacterin which is a formalinized killed bacterin and when you inject it into the cows it produces antibodies which are transferred to the calf and prevents attachment and colonization of *E. coli* in the gut. That is a step forward. These cows are vaccinated at 6 weeks and 3 weeks before calving. There may be a logistical problem in that people will forget to do it. It is another thing you have to remember to do. These times are reasonably critical, 6 weeks and 3 weeks before calving. It shows excellent promise providing the *E. coli* which are causing the diarrhea are K99 positive *E. coli.* I want to emphasize we think this is a management tool to be used with discretion. We emphasize to our owners that this is not a magic wand to prevent all scours in calves but if we could reduce the scours caused by K99 positive *E. coli* we could make headway. I have tried to emphasize the importance of colostrum, some new information on the efficacy of force feeding colostrum and something on specific resistance of calves.

Question: I want to ask Dr. Darlington a question about the kinds of diagnoses and what was the major disease in these calves? Can you give us some kind of idea of morbidity and mortality?

Dr. Darlington: In that herd situation it was primarily respiratory and as far as the cause I can't give it. Very few scours. The morbidity was mainly pneumonia.

Question: Did you have those calves on milk replacer or whole milk?

Answer: Whole milk.

Question: All the way through? You didn't switch them? At what age was the pneumonia?

Answer: Anywhere from 2 weeks on.

Question: How much milk were you giving them?

Answer: 2 quarts twice a day, nipple bottle.

Question: What kind of housing did you use?

Answer: Individual stalls, wood floor, in a building. I am not sure how much bearing this has on those calves after weaning when they are in group pens, we also had some pneumonia in them and on culture we found Pasteurella as well as mycoplasma organisms. Those calves on whole milk were being fed mastitis milk also, if I am not mistaken, although to my knowledge we never did while I was there, or subsequently culture mycoplasma from the cow's milk. So even though we had some pneumonia there was some mycoplasma involved but it was probably not because of the mastitis milk.

Question: Are most of your calves on milk and not milk replacer? The majority of our calves are on milk replacer.

Answer: My recommendation to the dairymen is that in my opinion he cannot afford to feed milk replacer at least in the first ten days. There have been herds that have been having continual scouring problems where they are treating 50-75% of the calves and have totally eliminated this syndrome by waiting until after 10 days before they start feeding milk replacer. In my experience you can expect a change in the consistency in that stool 48 to 72 hours after you switch over to milk replacer and that calf cannot utilize non-milk protein until it is about 3 weeks of age. I like to get it half way there anyway before we start having protein deficiency on his diet. And in most herds, if they will save all their waste milk they will have enough milk.

Question: In calves that have respiratory problems after weaning have you ever used Nasalgen or Pasteurella bacterin? And if so, with what kind of results?

Answer: Varying results with Nasalgen day two, day three, repeated just prior to weaning. We used pasteurella bacterins on a weekly basis with very limited response. With Nasalgen, it has been a sporadic situation as far as we think we are getting a lot of response and other times we don't. It is interesting in another herd we were having a respiratory break in their calf facility and we went through and vaccinated with Nasalgen in the face of the outbreak with

about 130-140 calves involved. The fellow ran out of Nasalgen and he got down to the larger group of calves which were about 5 months of age and didn't bother going through those with Nasalgen. Virology showed we had an adenovirus and his losses were all in the group that he did not vaccinate with Nasalgen. He lost about 20% of them.

Question: Have you had similar experiences with it?

Dr. Abernathy: Yes, we have several herds that I am involved in. We have a lot of respiratory problems before weaning and after weaning and we run into a lot of coughing. Very few symptoms other than when you move young calves after weaning you get coughing and when you get stress situations such as wet and cold we run into more respiratory symptoms other than the coughing and quite high fevers that are readily treatable with sulfas or tetracyclines. I cure them and a month later, with more stress, they breakdown again. For this type of syndrome Dr. Jarrett gave me a Triangle 4 product which is IBR, P13, and the Pasteurellas. We seem to have excellent results and are able to completely eliminate the coughing problem and these repeat problems. We can combine the Nasalgen IBR, P13 with the Pasteurella of less expensive products and get the same kind of results I am getting under this other program in after-weaning calves but we would like to cut down the expense.

Question: At what age are you giving this product?

Answer: We are giving Triangle 4 at two weeks before weaning and also at weaning. We also give it to the dry cows approximately 2 or 3 weeks before calving.

Question: Just a couple of different questions for Dr. Radostits but I am not going to try to hold him to it and one comment. The comment first, you said something about large dairymen and whether or not they would be able to, or want to, force feed calves colostrum. In my experience if they lose enough calves they are going to do it. To carry this thing one possible step further, I see on at least 3 or 4 different diaries, my partners have also, where good colostrum feeding takes place at least by our standards and the calves do well until over into the 10-15 day age. Then they get a little bit of diarrhea and the first thing a farmer does is probably pull back on the milk and maybe they are feeding milk replacer. This can occur anywhere from ten to twenty-four days and a lot of these calves just wither up and die. No diagnosis, but by the time we get to them they have already got every antibiotic in the world in them and the best treatment seems to be try to keep them from pulling back on the milk and to continue feeding them. They are in fact starving them to death. I wonder if Dr. Radostits has any comment on this?

Dr. Radostits: The whole milk or the milk replacer is an area that interests me. Do you think that you see more of this syndrome in calves fed replacer than on whole milk?

Answer: I heard you talk on milk replacers often and we stress a *good* milk replacer. We also try to keep them on whole milk hopefully up to 2 weeks if we can.

Dr. Radostits: If they are giving it up to 2 weeks of age there is a good chance they should be able to handle milk replacer even though it is not of the best quality.

From the floor: It doesn't seem to be so much a milk replacer quality as maybe some virus or other things are just hitting them. With scours, by giving them less fluid, where in fact they need more fluid, it seems sometimes they are starving them to death. We have run into problems with this and on occasions where we can convince the people to continue to feed them a gallon or get them back up to a gallon as soon as possible they seem to work their way out of it.

Dr. Radostits: I have seen that so many times. I think milk replacer quality is one possibility. If that isn't the possibility then the viral diarrheas would have to be considered. I don't think it is *E. coli.* You should have been able to pick up whether it was salmonella. These reo viruses and corona viruses tear off the epithelial cells and villi cells and it takes anywhere from 4 to 8 days for them to migrate back. If they don't migrate back normally, the calf ends up with villous atrophy and continues to scour. The owner keeps pulling back on milk, and the animals starve to death. The reason he is pulling back is because, when he feeds them milk, they scour because they have villous atrophy.

Questions: Were you able to get any information from detailed post mortem examinations?

Answer: No.

From the floor: Most of the time this was a deal where they would talk to me about it and there wouldn't be any dead calves. I think a standard recommendation would be to feed them more milk and work their way out of it. A lot of times I wouldn't even see the dead calf, but they would tell me the calf had died.

Dr. Radostits: Well, I think with viral diarrhea you can expect them to scour from 4 to 7 days depending on what the owner does in that period. He may bring them back or make things worse. What we recommend for half-dead calves is to feed them, if you can, smaller amounts more frequently. And they would digest a higher component of that smaller amount than giving them larger amounts.

Chairman: I want to ask the people in the audience, what their average morbidity of scours is in their practice. The reason I am asking is we have several clients that have mortality rates under good management of 5% or less but morbidity rates of scours in the area of 70%. I was interested in Dr. Radostits' comment on the morbidity rate even though the calves do not die and I am wondering if we can get some idea if that is common for other people or not.

Dr. Abernathy: Under some feeding conditions we had 90% scouring rates and with all types of intensive therapy they scour for 10 days or 14 days anyway. In other dairies it is not quite that high. It also depends on whether we are feeding whole milk or milk replacer as to what happens during that period. (Do you have a higher incidence with milk replacer?) I think we do. But I think we have also a high incidence of scours with whole milk.

Chairman: Question concerning this viral diarrhea: I have this herd that has a high incidence of corona virus in its

calves and I would say quite routinely. Do you have any experience with vaccination of cows for that? I know Norden has a product they are pushing now for cow vaccination.

Dr. Radostits: We have no experience with it but we are going to use it next spring in beef herds. We have no experience with dairy herds at all, the same firm has the oral vaccine, and they suggest that it can be given parenterally. It makes some sense if you can successfully increase specific antibodies in colostrum. I am not selling Norden vaccine. Calves get that protection for the first week or ten days that gets them over the critical period. The only problem with that is that 90% of our cows on a worldwide basis have antibodies to certainly reo virus, maybe less percentage to corona virus, so you are really vaccinating those cows to protect 10% of the calves.

Chairman: I had that question myself when I talked to Norden people and their comment was that vaccine was primarily developed for use in beef cattle where they couldn't control force feeding in colostrum in getting the vaccine into the calf as soon after birth as possible. They prefer giving it to the cow.

Question: I want to ask a question in regard to what you just said there about it protecting only 10%.What about the quality of the antibody level in fact in the colostrum? Do you increase the quality of antibodies by vaccination? Maybe those 90% don't have a high enough titer that they are really passing on an anamnestic response.

Dr. Radostits: It increases the level of antibodies of the whole herd. We don't know how much antibodies are required in the intestinal tract to protect calves from diarrhea. Only 10% of the calves have a complete absence of the antibodies involved so that is the guideline. It also goes along with the morbidity and outbreaks due to virus diarrhea, 10-15% of the calves will get it. In closed herds where they have never had exposure to the virus it is a lot higher.

Question: Dr. Radostits I have a question in regard to colostrum in beef cattle, I think the things that you have pointed out there are very important for all cattle but now the thing you brought up about beef cattle not getting colostrum because they couldn't insure it — in your area where you have a lot of beef cattle, do you have any programs to encourage beef men to particularly force feed colostrum to their first calf heifers since their colostrum levels are going to be lower, regardless of nutrition sometimes?

Dr. Radostits: That is an excellent question. I tell veterinary students that it is easy for us to say to the dairymen to forcefeed colostrum to your calves like Dr. Darlington does and like some of us are doing now because you can milk the cow and give the colostrum to that calf. You have control of that situation. The beef man, who is calving 200-300 calves and gets up in the morning and there are 7 more calves on the ground doesn't really know the levels of immunoglobulin in those calves or how much colostrum the calves have ingested. We've had an opportunity for the last five years to work on a large ranch in British Columbia in which they calve out about 1200-1400 two year old heifers every year. We take students up there and we calve them out. We really went up there in the first instance to look at the calf diarrhea. Of all the heifers that we calve out, we try to get colostrum from them right in the calving clinic and we force feed it to the calf. We measure gamma globulins in those calves in which we force fed colostrum available first of all to 2 year old heifers at partuition. And this is 76-77, the data are very similar for the rest of them. This is the amount in milliliters and 52% of those heifers in 1976 had less than 500 milliliters of colostrum available by milking out with oxytocin. 44% had 500-10000, only 3% 1000-2000. In our earlier remarks we said that this 45 KG calf needs 3.6 liters or 3600 milliliters of colostrum to get adequate levels of gamma globulin. It is impossible for those calves to get that amount from these heifers. And they are similar for all the five years. 22% of these calves born naturally from those heifers are hypogammaglobulinemic, severely hypogammablobulinemic. If the management happens to be good enough and they spread those cow-calf pairs out within 24 hours of birth on dry hills so they are not exposing that calf to the infectious agents, my first principle, those calves are probably just getting enough colostrum to prevent coliform septicemia. to answer your question specifically, how do you get the beef man to force feed colostrum? If he is having a problem he has to tie his heifers and calves up, milk them and force feed the calf, or assist the calf at birth to make sure he gets up and sucks within 15 minutes. The Hereford cows are taking 2 to 3 hours to get on their feet to suck. We have a lot of real interesting information like that. About 22-25% are hypogammaglobulinemic.

From the floor: I have one comment on the *E. coli* bacterin you were discussing. I have several herds that have had severe problems with coliform septicemia with quite large death losses and we went in to attempt to make up an autogenous bacterin and we made up a separate one for each herd involved and tried using this and injecting dry cows. We've been able to completely eliminate the septicemic part of the problem where we got death losses. We did continue during the first 5 days of the calf's life. We still run into diarrhea in other words we still have enteritis, but it is rather easily treated and the morbidity is very low so we are very pleased with the autogenous bacterin and the *E. coli* that we have been using.

Question: Do you know if it contains the K99 positive *E. coli?*

Answer: In order to select the *E. coli* from which we make the bacterin, we must isolate the same *E. coli* in at least 6 different calves in at least 3 different organs of that calf's body. In other words, to isolate it out of the intestinal tract we don't consider that to be enough, but if we can get it out of the lung and spleen and the intestinal tract and can take out 6 different calves in that dairy we go ahead and make up a bacterin. But as far as whether it has the K99 ion in it, I have

no idea.

Dr. Radostits: In our work and in work done in other parts of the world, K99 positive antigen is now turning out to be a common antigen to the most common sero types of *E. coli* that are causing colibacillosis outbreaks in calves. We still get some sero types out in left field but we're getting most of them with the K99 positive antigens.

Question: Does this same bacterin work for coliform mastitis?

Answer: The same herds have coliform mastitis. I have been told so many times that it wouldn't work. I haven't tried it, but we are running a trial right now and are going to find out.

Dr. Abernathy: How many people check for lungworms on these coughing calves when they are 3 or 4 weeks old? I didn't and I am suprised. A year ago I didn't do that on coughing housed calves, but I do now and I suppose now in our practice we are seeing about 30% positive on coughing, relatively strong, calves that cough when you stir them around just like you were talking about here a while ago. We have come up with a surprising incidence in our practice and we have diagnosed it down to 10 days of age and the life cycle if you read it in a book shouldn't be like that but obviously the only way that I can figure it out is that it is going across the placenta and these calves obviously have mature lungworms and they are coughing larvae in ten days and I would challenge you to start checking these calves. I am in coastal British Columbia where there are relatively mild temperatures and we have big lungworm problems in our calves, no matter whether they are inside or outside. I also challenge you to identify the parasites if you see them. If you see a lungworm don't relax, go ahead and identify it and find out what it is because levamisol won't work if it is not dictyocaulus. My other comment is that I am a Canadian so that this vaccine that is going to come out is going to be available for me but I just wondered if this stuff is going to be available in the United States and whether it is going to be another smuggled product!

Chairman: **I think we better get on to our next topic which is abortion and Dr. Darlington is going to go over the causes of abortion and Dr. Bushnell will have some comments.**

Dr. Darlington: I guess everybody has his problems with abortions. It seems no matter where a bunch of veterinarians are working on cattle together, the comments always go back to abortion sooner or later. I would like first to throw out some causes in my practice that I see for abortion and if somebody has the answers, I would certainly appreciate knowing them. We see a higher percent of twin abortions than we do single. For years if an animal aborted twins at 6 months or 7 months I was never alarmed about a cause or anything else and just assumed the fact that we are going to have more twins abort. Now I don't know if that is a real assumption that is just because it is twins, it is an injury between the two or what, but I do find that as an incidence of abortion and a cause for it. I see a lot of cows that are milking 65-75 lbs. a day 7½ months with calf, they dry them off and they dump the calf 3-4 days later,. I believe that is a steriod or a hormonal inbalance due to the ceasing of milking. Trucking we see occasionally injury, the obvious one that the tractor hits her or she is closed in on a gate real hard and you have got injury both to the cow and the aborted fetus. Errors in drug use we have made a majority of our herdsmen knowledgeable enough not to be using dexamethazone or azimicin or some of these products in treating cows that are with calf but every once in a while we do come up with an error in drug use and dump the calf. Unfortunately probably most of the ones that we end up even after we exhaust all of our laboratory diagnosis come back with idiopathic abortions. We see an occasional IBR abortion. Not a great number of them but we do see a few. I have had a BVD isolate out of a 60 day aborted fetus. It takes a very observant dairyman to find those for you, but that was done several years ago in Washington State. We see some vibrio in bull bred herds. I recommend that even those herds using clean-up bulls vaccinate for vibrio. We have not seen any Trich in our area, but we do see some Lepto, at least we think we see some Lepto. Hardjo, at least serologically we have not been able to isolate it. Serologically it has been a problem for the last 4 or 5 years and seems to be an increasing problem. I have been working with Beecham Laboratories lately on a new 8 way Lepto vaccine and a 4-way Lepto vaccine that they are working on I can only tell you that we have not solved any of our abortions. We have one herd in particular that we have had a lot of abortions in and we still haven't got it solved. We have attempted to isolate lepto out of it. We continually get high hardjo titers out of it. We get some others and so we went back out and started trapping rats and we have been very successful. We have found a lot of ictero out of the rats and we have been very successful. We have found a lot of ictero out of the rats and ictero out of the cows but every rat on the farm has been positive. We have been able to culture that.

Dr. Bushnell: Well, this is certainly a tough subject and the only thing that I can offer is some observations by other veterinarians and some of ourselves through the school and through extension service. Steve said we should first say something about what the accepted incidence of embryonic death or fetal death is and that is a tough one. You probably would have an idea within your own practice. It would appear that it would fall somewhere within 2-5%, depending upon the herd and the area. The only thing that I can offer in that regard is the fact that we have had the opportunity lately to do some additional pregnancy examinations in beef herds that we have had on some foothill abortion trials and in five different herds in the various ares of the state it is obvious that we have early embryonic death in these beef herds that probably approaches 5-10%. Alright now, what is the cause of this? Here are herds that are naturally bred. They are herds that are vaccinating for vibrio. What are some of the reasons? And certainly even though we are vaccinating for some of these diseases, we are still going to experience some of them but we have not been able to relate all these instances

to specific diseases. It does appear that early embryonic death is something that occurs both in dairy and beef herds. As far as outward abortion goes, we are talking now about fetal death 5, 6, 7, months, either prior to or during the dry period and the observations are interesting that abortions occur at drying off in these very high producting cows. I can't offer any reason for that. I would like to suggest that, in terms of talking about vaccination particularly with the multiple leptos, this observation was made in the midwest and by a few practitioners in California, the antigen mass has something to do perhaps with abortion. We vaccinate heavily pregnant cows with larger and larger antigen masses and we may be precipitating some of these abortions which to me is an interesting concept so my question to Bob now is, if that is true with 5 way, what happens if we go to 8 way? It is a consideration that we are vaccinating these cows close to the time of drying off or during the dry period and we may be precipitating some of these problems with some larger masses of antigens. In terms of how we are going to diagnose these things we have blood tests available to us. Paired serums have always been frustrating to me and I think to other people, and I am not sure we ever really diagnosed an abortion with paired serums. Culture is a very difficult thing and I would like to ask Dr. Radostits if he considers an isolation of the virus in the fetus, such as BVD, automatically to be the cause of the abortion or do we have to relate it to some pathological lesion? Because BVD is very common and it can be isolated from both fetuses, vaginal discharges and other areas of the cow at least in our dairies in California, it is very difficult to relate these to individual disease problems. I might say that an area that is of interest as far as embryonic death is the sanitation used in insemination. It has been our observation on several dairies that those people that are still using ampules and have their own stall boxes no longer prepare it with fresh ice and water but they leave the thaw box sitting in the refrigerator and you go in there and there is a slime about an inch deep and you can culture corynbacteriam, various streptococci, pseudomonas and other organisms out of these things. So you wonder sometimes that even though we can't say specifically this is the cause of early embryonic death, I think one would do well to investigate insemination practices and thaw techniques and so forth in regards to sterility. We even found, in a few dairies, ice cubes that they were using in the house were heavily contaminated with pathogens, for example, ducks were swimming up in the well water the people were drinking and they had problems everywhere. But specifically we found this in the fact that we are getting this out of the thaw boxes in relation to insemination. The other thing that is of interest is that the semen itself, even though the AI companies are doing much better jobs of putting up semen, there is always a potential for pathogens. It brings to mind here that several years ago we encountered a situation with embryonic death, infertility, and a certain percentage of mummies and we isolated a candidia out of the semen. The same candidia was traced back to the dye the AI company was using to color their semen, so there is always a potential for contaminated semen and that is something you should consider in the case of early embryonic death, infertility and mummification within your practice.

Another area is iodine and feeding of EEDI to dairy cattle. Here again, observations made by practitioners in California in the Chino area where there was a lot of embryonic deaths, abortions, and endometritis showed that they were related to feeding of high levels of EEDI in the feed and they did some field work and showed indeed that levels fed were very high. Now in the last few years there has been considerable documentation come forth showing that EEDI is an immune depressant. It depresses the lympocytes which then would have some effect on the macrophages and the macrophage is of course a primary cell in defense mechanism in both the uterus and the mammary gland so that there is some basis for it. Also high levels of EEDI can suppress Vitamin A and it increases the neutrophils. It also acts like a corticosteroid stimulant so therefore there is a basis for some of the chronic disease entities that we see in these herds that are on high EEDI. How do you find out if the herd is high in EEDI and iodine? Well in our experience it has been very difficult. We got into this because, of not only our interest in EEDI in the feed, but also iodine in the santitizing procedure we are using. It was very difficult to investigate a feed or call a feed company and find out what they are putting in the feed. The amount you will measure in the feed or the history just won't correlate with what possibly the cows are getting because either they are not going to admit or they are not labeling properly what they are putting in there. Much more, possibly dairymen are feeding them alternative sources such as free choice EEDI in the corral. So the easier way to find this out is by taking the tank milk sample, measuring the EEDI in the tank milk and that tank milk sample should be below 500 micrograms per mil. If you have been showing levels of 750 and above micrograms per mil, the animals are probably ingesting a level far greater than they need to be and you can get immunodepression at that point. Often times you will find these dairies are running 1500 - 2000 micrograms. So we developed this as more or less a routine in some of this research and withdrew the iodine. Cutting down usually sometimes gives you what you think are dramatic results. Other times you can't see any results. There again it is one of those nebulous things but is certainly simething to consider and there is a way to measure it. The big question comes up in herds where the owner is using you for rectal palpatation pregnancy determinations. They go on non-return rates and you often wonder if you are getting embryonic death because of the non-return rates or in these 42-45 day cycles, are these missed heat? One way you can discover this of course is by running a progestin level in the milk and we have begun to do that recently. We have done this in some of the herds and what we do is we have the dairymen, through the practitioner, freeze a sample of milk at the time he inseminates the cow and he saves and he gets 15, 20, 30 of these samples and then he sends them to us and

we run a progesterone level. We have found as high as 30% of these cows are in the luteal phase when they are inseminating them. So this will give you some handle on these types of herds that aren't using it quite so closely to find out whether they are actually having early embryonic deaths or they are actually finding missed heat periods. Another method that has been used in diagnosis is taking problem cows to slaughter. I think this is something that can often eliminate a problem. Other times you don't find anything out, but I think it is a good procedure if you have not been able to find out particularly in fertility problems, embryonic deaths and some of these sort of things and you can't really palpate clearly a deficiency in the uteri or ovaries. Taking these to slaughter, looking at them grossly, looking at them histologically and doing some follow up cultures sometimes can be informative.

I think leptospirosis and the role that it plays both in outward fetal abortion and embryonic death is a question in all of our minds. It is important to know for the practitioner and for us also within the school. We have embarked upon some additional investigations in the last year and some of the things that we have found out is that with vaccination you can get extremely high titers that can be confused with infection and therefore since our 5 way lepto vaccines have come on the market I think that many of the lepto abortions in these herds have become confused because we are told initially that we cannot produce as high titers in vaccination as we do get from natural disease and this is not the case. But you can differentiate the anitbody that you produce from natural disease is called IgG and the anitbody that you produce from vaccination is IgM. Therefore you can differentiate these if you can get someone to break these two elements out and we ran a trial and Jim Glosser of Montana did this. Even though the total neutralizing titer is high, if you break out the IgM which is the vaccination titer, then you can tell those animals which actually went through the infection from those that did not. We just offer this as a means to further differentiate at least serologically the possibility of high lepto titers in these herds. Now the other thing is that even though we can measure a titer it is impossible to differentiate some leptospirosis infections serologically. The only way we can do it is by isolation. We sent some of these sera to Kitty Seltser at CDC. Kitty says, and others that are involved with Lepto, to really find out the sero variety of lepto you have in your herd, you have to make an isolation. Now this is not as easy as it seems. We are attempting to do this in about 12 herds and I will just give you some tips that if you want to try to pick an isolation from recently aborted cattle, the urine is the best place to go but you will increase your opportunities for isolation if you inject the cow with lasix and take at least the second voiding of urine or wait at least until the urine is a very light color. You can increase the chances of isolation by some 90% if you take the recently voided urine that comes from the injection of Lasix. The other thing is that you should do your dilutions in the field. As strange as it may seem, the more you dilute the urine, the more likely you are to make some isolate which seems just opposite of what we thought originally. So I would encourage some of you who do get involved. If we are really going to define the lepto problem we need to start making more isolations, because there have been only 6 serovarieties of lepto isolated in cattle in the United States and most of them not too frequently. We are already facing the fact that we are going to have a 5 antigen vaccine and now we are going to have an 8 antigen vaccine and the more we vaccinate, the more we are going to confuse the picture. The harder and harder it is going to become to diagnose our problems so I think that as people involved with the industry we are going to have to move and try to make some isolations and try to get these infections identified.

Question: Would you go over that serology again?

Answer: When the lepto vaccines were only dealing with Pomona I was told, and I think most of the people thought, that we could differentiate actual infection from vaccination by titers by the degree of the titer, particularly the microagglutination test. But in recent years since we have these multiple antigen vaccines we are finding that actually we are producing titers from the vaccination that are in the same area as infection titers so that once these vaccinations have been applied and you are still having abortion problems it is very difficult to go in and try to determine which sero variety you have because now you produce so much antibody from vaccination. The other thing is that you have so much cross reaction within the hebdomadis group that we start to say now we have hardjo or now we have something else because this titer is higher. For example, according to Kitty Selter at CDC you can have a 1-1600 for hardjo and you can have a 1-40 for hardjo on a particular animal, for instance, and you can't necessarily say that is a hardjo infection. That can very well be a hardjo infection. There is no relationship between the height of the titer in that particular group of organisms and the one that is actually causing the disease which I had assumed previously.

From the Floor: Let me just mention a couple of things in Virginia where we have been hunting for Lepto. One is, and I got this from L. Hanson, especially if you have grippo titers, hunt for raccoons. We have had some, and this is hard to believe, but we had one herd that was having problems in their heifers with high grippo titers and abortions and there weren't any racoons around the place. You would only see them occasionally, but up in the silo there were some 20 coons that lived there. They jumped over on the unloaders that would come around. This herd was getting infected from their urine up in that silo so if you get grippo titers, hunt for coons. I have had two herds now and they are the only two herds that I have seen. They both had coons up in the silo. There is a person at VPI that works a lot with lepto and the best transport media you could probably get, it is fairly inexpensive and it is also maybe a relatively good diagnostic test, just when you are out there, if you have some small hamsters, inject them right there. The hamster is an excellent isolation tool. They are relatively inexpensive and

if they get lepto it reproduces so fast in them that they die within about a week. There is a real good chance that there was lepto in that urine. We are getting the hampsters and putting them into the cages and just take them out or we have just been giving them to the practitioners and they will do them and then bring them back at their convenience and it has worked out. You know you can get pomona with no problem, and hardjo. Use a TB syringe and a real short needle and inject about a cc of urine into the hamster's belly.

Question: How do you identify the organism after the hamster dies?

Answer: If they die you have to cut them open and take one kidney and put it in formalin. Take another kidney and refrigerate it and send it to the lab. The hamsters that are infected on histology show lepto in the kidneys and then you can make a positive isolation.

Question: Where do you send them to get them identified?

Answer: To somebody who works with Lepto. We happen to have somebody who does work with lepto at VPI. But if I were doing it myself I would save that one kidney and you can freeze it and you know if you freeze it you would be all right for the isolation and send the other kidney for a histo. You can get a histo done almost any place. You can get a silver stain done on the kidney but it works out in practice that if it were positive on histo they were positive in that other kidney, so it is a relatively simple thing. It is not hard to get hamsters and it is easy to pull out kidneys and send one for examination and then you wait for your reports before you go through all the rest of your routine. If the hamsters don't die within about 6 days, or so, they probably don't have lepto.

Question: Does anyone from the Pacific area know where we might send these hamsters and get a diagnosis?

Answer: The only place that I can say that you can get an accurate analysis would be Kitty Seltser at CDC and how much of this material she would take I don't know. If you sent her positive material she would, but if you sent her a lot of diagnostic material I doubt if she would. We tried some hamsters a couple of years ago and we didn't get anything and the reason is we were using urine direct and the dilution method that we mentioned is really important. Evidently the smaller number of organisms that you inject both into the media and the hamster the chances of isolation are greater and that is why they dilute these things out to about 10 to 3, 10 to 4.

If you can have some hamsters, just go out to the farm and give them lasix and collect the urine right there. Take it and squirt it into the hamsters.

From the floor: The thing that amazes me is that it seems to be such a hardy organism in the environment, being spread from cow to cow and yet it is one of the toughest things to isolate. It is harder than any virus.

Question: Are any of these 5-way vaccines more antigenic than others? We here stories that one is much more antigenic and causes more problems in your diagnosis than others.

Answer: We ran the two 5-way and three 3-way vaccines in this trial. We have all the data back but we haven't analyzed them. My impression is that we are not going to find differences between brands of vaccine, but within vaccines there are certain groups that are less antigenic than others. For example, your *pomona ictero* and *canicola* are good antigens but your grippo and hardjo tend to be poor antigens as far as we measure. We are only measuring circulating antibody, and those two antigens seem to be the weaker antigens. I can't say at this time if there is a difference between groups of vaccines. I have heard speculation that there could be and Jim Glosser of Montana was telling me a very easy way to find out the antigenic masses in a given vaccine is to just send it in and have a protein analysis run. Those with the higher protein levels are the ones with higher antigen mass. I have never done it but we are going to try it.

I am just talking about total antigenic mass. If you have 5 antigens in there you have got more antigens than if you have 3 antigens. But in terms *of hardjo* let us say if you put in the same antigenic mass that you did for pomona I am just suspecting that the measurable antibody seems to be less. Hardjo is the one organism that they haven't developed a good test for even within the laboratories. It is just one of those things that is added and they keep as high a mass as they can but as far as a routine lab evaluation they are unable to do it, so just on the basis of our trial, the circulating antibody, the IgM was much less from the *hardjo* vaccines and the grippo vaccines than it was for the other three.

Panelist: It seems it might depend on how well they filtered the vaccines before they made it and how much cultural medium might be in the vaccine too. I have some questions about progesterone assays. If you find out that a high percent of the cows they bred on multiple service had elevated levels of progesterone, can you say this is a reflection of embryonic death or just poor heat detection?

Answer: Well, the only thing that you could say is that the cows were in the luteal phase and that if they selected the cows in the luteal phase and bred them, particularly 30% of the cows, undoubtedly the heat detection system is relatively poor. But in order to really point this out, what you really need to do is go back and take another sample 5 days later. We take one sample, and then come back 5 days later and take a second sample to see if she has gone into the follicular stage or not or if the progestational luteal phase stays the same. The point being, if they are selecting those cows for breeding at the follicular stage, the progesterone should be very low, 30% of the cattle are not in the follicular stage to be bred, in fact they are well outside the follicular stage.

Question: If they are in the follicular stage and progesterone levels are low and it is an abnormal multiple of 21, you assume then there has probably been an embryonic death?

Answer: You are showing, in truth, that the dairyman is doing a poor job of heat detection which helps you to differentiate it from embryonic death.

Answer: I have 4 groups that are on milk progesterone levels for pregnancy diagnosis and all of these have milk

drawn on them the day they are in heat and then the milk on 21 so that we have 2 levels of progesterone to compare and therefore if the milk progesterone level has to be low on the day the cow is bred then it has to be high on day 21 in order to call her pregnant. And even with that in my 4 herds we have about a 10% error factor in these cows that are called pregnant on milk progesterone and then I call them not pregnant later. So I think that in order to justify milk progesterone you have to do two milk samples and I think that is the way it is done in England where they take milk on the day the cow is bred.

Dr. Abernathy: Our trial in British Columbia is a university project and it is all at the government's expense. The herds that we have on the program are free. I was told that the machine is expensive and the chemistry expensive and the technician is expensive and the samples are about $5.50 each.

From the floor: I could palpate a lot of cows for $5.50 each! Do you have any comments on the price?

Dr. Abernathy: Our tests currently are run at the university and the cost that we transmit back is about $2.00 a sample. However, it is one of those things if you could stimulate enough interest you can build a demand through volume and you could get this thing done at a much more reasonable rate. I think also there is a kit coming on the market.

From the floor: There are about 3 commercial laboratories doing it. One is the Cornell, the cost is $2.50 per sample, the turnaround time is 48 hours from when they receive it and when they send it out. So a lot of that depends on how long it is in the mail. They do have kits available that all you have to do is put the milk in and they are ready to mail off.

Question: What is the normal incidence of abortion and when should one be concerned?

Answer: The comment that was made last night with Leon Weaver's study and from Minnesota and Colorado all hit in close on 6 to 10½% and I think that is very realistic and we are talking about primarily embryonic deaths, and not abortions. If we add to that our twins and occasional whatever happens I think that 12% abortion figure is not that far out of line in most commercial areas.

Question: Does everybody agree with that?

Answer: That might be true in the dairy situation. Rather than call that a normal level of embryonic death let's back up to maybe some beef cattle and beef heifers that are as reproductively clean that we could look at essentially in a commercial breeding program that Dr. Rice and I were involved in where we did pregnancy diagnoses at approximately 35-40 days on every one of them, over 1035 head in all. I forgot the exact percentage now but about 3.4% of those had lost their fetuses by the time they were palpated a second time after they had been moved from one geographic location to another to a new ranch and then they were reexamined at a later state in the fall again and that was what the embryonic loss was at that time. The palpation technique was slipping of the membrances and all of the diagnosis was either done primarily by myself and Dr. Rice did a fair number of them in that situation. So I think that on that basis we don't want to be deluded into thinking that a high embryonic mortality is normal. Maybe we need to put a little more attention into finding out why we are losing that many fetuses.

Question: Was it from one herd?

Answer: Yes, all one herd and they were all artifically bred.

Answer: These 5 herds that we did I think would average more than 5-10% embryonic deaths between 45-50 day period up to about 3-4 months period. There is a 2 month span there. There was embryonic death of about 5.0%. These were also all heifers.

Answer: In our practice we see a variation from about 3% to about 12% and almost like everything else if somebody can have 3% everybody should be able to have 3%. There must be a reason why they aren't getting 3% but it is a little hard to find out what it is.

Question: You said that feeding high levels of EEDI would cause immuno-depression. What levels are you talking about and you also mentioned that less than 500 micrograms per ml in the tank milk was normal. Now is that free iodine or is that EEDI or what are you measuring there and where do you have it measured also?

Answer: NRC recommendations are, I think, .5ppm in the ration. When you are adding 50 milligrams per cow per day you are going to wind up with about 25ppm in the ration.

Dr. Bushnell: I think in the dairies that we estimated where they are adding supposedly 50 mg of iodine we would come out with about 25 ppm in the grain ration. It is added in the grain ration. 50 milligrams EEDI is the recommended low level dosage for hoof rot control per head per day. But we find in our dairies that many of them will have 100-125 mg per head per day so you can very easily meet all the iodine requirements and end up with less than 500 micrograms per liter in the milk. It is the same as 500 parts per million in the milk. You would have to find a commercial lab; we do it in our own laboratory at Davis. Iodine level is a very difficult thing to measure but some of the human labs probably would come the closest to doing it.

Question: I don't know if I should bring it up but what about calf losses or abortions with BVD vaccine in dry cows? Does anybody have anything on that?

Answer: Tim and I talked about this last year and I had a large, well managed herd that was having high calf mortality problem and we made many changes and this was one of the changes that we made. We started vaccinating the cows three weeks before they freshened with no diagnostic information to go with, but it was empirical and I don't know if it was finally time for them to stop dying or if it worked but the mortality last year was running around 50% and it is now down to 5%.

Question: Had those cows been previously vaccinated?

Answer: They were vaccinated whentheyfreshened on an

annual basis but the rationale that I was using was that, you can rationalize anything anyway, I was trying to vaccinate the calf in utero figuring that the BVD virus presence might be causing some immunesuppression and that was the reason they were getting the scours.

Answer: I simply wouldn't do it if the cow had never been vaccinated before.

Answer: The calf is immunecompetent to BVD at 150 days so there should not be any problem as far as causing abortion.

In Europe, the vaccination of dry cows during the last trimester of pregnancy is practiced very commonly and as Steve said the calf is immunocompetent at that age with well-developed active immunity and that is easily diagnosed at birth, pre-colostral.

Question: You are talking about that one virus.

Answer: We are talking about BVD virus. They are certainly not immunocompetent to IBR virus.

Question: Anybody here from Idaho or Oregon where they tested this and I don't recall all the details but I think their conclusion was that this was not of any benefit. Does anybody know more details about it?

Answer: There are 2 studies going on right now. At Ohio, and this is both a close examination of the records out of Ohio of one big practice where they had documented everything on this scour business with BVD vaccination usually on open cows and you know switching different cows to give it to them during the dry period. It really looks at the moment that there are definite advantages to it. And I know in Virginia we have got probably at least 10 thousand cows or so with one practice that does have real good records. We have some real good records and there is a tremendous difference, in the calf. That is the primary reason for doing it. It is not the cow so much but the calf problems. Calf morbidity and mortality, before and after, but over a large number of cows and this has been for about 2 years now. In one of these particular herds it has made a significant difference. You can really see it when you stop vaccinating or when the farmer forgets to vaccinate. Now we are only talking about NADL strain also. Only use NADL strain or the Singer strain but don't use that Oregon strain.

Question: Tim, on those samples you have cows that were vaccinated for BVD previously, you are vaccinating those cows at drying off or about 3 weeks prior to calving. Are their calves born with high titer?

Answer: All the work is not in yet, but it's not coming out that way. At least for titers...but you see this is the whole problem with BVD, they don't run high titers. You would expect them to be born with resistance to challenge whether it is antibody or CMI. See they are being vacciated, the virus infects the calf.

Question: You are injecting this vaccine into the uterus?

Answer: No, we are injecting it into the cow. And you would say well the cow has an immunity, this is the question, if she is already immunized, you will stop the spread of that vaccine before it ever gets to the uterus. That is the question. Right? I cannot explain it well, but it does not seem to work like that there is enough that seems to be getting through. This is preliminary stuff yet, we will have to wait until we get enough numbers, we only have about 50 so far and I know Vernon only has about 50 at the moment.

Chairman: **Our next topic is mycoplasma mastitis. Dr. Abernathy is going to discuss his experience with one herd that had myco mastitis problems and then Dr. Bushnell is going to follow up and discuss his experience with mycoplasma in California. We are using mycoplasma as a model to get involved in controlling the mastitis situation in a herd that has a problem.**

Dr. Abernathy: I am going to introduce the problem of mycoplasma mastitis by discussing my experience and then we will get on and learn from Dr. Bushnell. I have a client that bit off a little more than he could chew a while ago and he had a pretty good 50 cow purebred herd and had a lot of production when I first started working with him. He also had a friend who had a 150 cows and it seemed to be more successful so my client decided to increase the herd. At that point he had a struggle with the dairy inspector and got turned down on a rather small herd transfer and a small quota transfer so he got mad and he went to a politician and the politician got things turned around for him. He went out and bought 90 more cows and lots of quota and from that point on there were all kinds of problems. First of all there was a labor problem, then there was a nutritional problem and then there was a housing problem, a quality problem and a milking parlor problem, just about every problem that was possible. Most of his attempts to satisfy dropping production were to go out and buy more cheap heifers and he went out and he bought cheaper and cheaper heifers and more and more. For his 8000 lbs of quota, where he should have needed 150-160 cows, he all of a sudden had about 300, none of them producing well. So I came in when the bulk tank was hitting consistently over 200,000 and I think the bulk tank was well over 200,000 for about 3 months in a row so he was not only being faced with poor production but he was being faced with being shut down by the dairy so we got involved originally because there were a lot of strept ag. quarters and I started with CMT and culture them out. Meanwhile he kept on buying more cheap heifers. So I recognized the problem as something different when we had 12 new quarters one morning and none of them were sick. I am not very much of a believer in the Cornell method of sending all my samples to the lab with the bulk tank just to find what kind of general population we have in the herd to start with. When we received the samples back, about 20 quarters I think, 15 of them had no growth and that's all the lab told me. So I phoned the lab and then I sent more cultures and said we better start doing something about mycoplasma and I started making some phone calls. Mycoplasma had never been diagnosed in Western Canada to my knowledge at that point and mycoplasma mastitis had never been diagnosed in Washington or Oregon state to the best of my knowledge as well. So I phoned around and I kept

phoning farther and farther and finally talked to Dr. Jasper at Davis and he was immensely helpful. Basically we did composite cow samples every week until the herd was negative and all the positive cows went to slaughter. We then sampled every month for another 18 months and basically what we did in the herd is, we went to an extreme sanitation measure. From what Dr. Jasper told me the spread of mycoplasma is almost totally in the parlor. It is almost totally from teat to teat or udder to udder or milk to milk from cow to cow. So we dipped everything, including washing hands between cows, and that is extremely difficult to do. We got milkers to wash hands. We dipped the cluster, we dipped teats and we instituted the use of paper towels.

We organized the herd into groups according to mycoplasma status and I think we had 4 or 5 groups. We organized heifers, fresh heifers into one group, fresh cows into another group and we quit doing the CMT and that was the one thing that probably I decided without someone else telling me. I got the impression in this parlor that the fellow running the CMT was really spreading it around and mostly because he was a speed artist at how fast he could do a CMT and he was going from cow to cow as fast as he possible could. He was doing a CMT on an individual cow and then reading it off in about 30 seconds and going on to the next. I got the impression that he probably created most of the spread from cow to cow. So we stopped that. We culled about 120 cows of this herd in about 6 weeks. In our experience in this farm the only transmission on this farm was in the parlor. We looked at a lot of other things, two or three months after the problem, to try to figure out whether there was some evidence of transmission or whether we had mycoplasma in some other place but we never were able to be successful in doing it. We had a regulatory veterinarian that walked in and swabbed noses about 10-12 weeks later and he swabbed vaginal cervical swabs. He really alarmed me because he got mycoplasma growing on all of these nasal swabs and when we thought we had the herd negative. Subsequently the mycoplasma identified from the genital tract was *mycoplasma bovigenitalium*. It was different, we were dealing with *mycoplamsa bovis* in the udder and the mycoplasma that was diagnosed from the nasal swab was something different again. The *mycoplasma bovis* that we were dealing with in the udder just disappeared by the measures we took in eliminating it in the parlor. The challenge is the masses of samples that you are sending to a lab, I suppose. You should check and be sure somebody is looking for mycoplasma and you should especially start looking for mycoplasma when you get a lot of no growths, especially from cows that appear not to be ill with rather dramatic secretion changes.

Dr. Bushnell: Certainly all the things that Dr. Abernathy just described would be quite typical of what we would see with mycoplasma in our herds. Just to add to the information that he mentioned, there is more than one type. There are 6 different mycoplasmas that potentially we can deal with. The clinical disease is not equal in all of them. It is not even with the same type. Bovis tends to be the most pathogenic and I think that is probably what you had in the udder. As he mentioned it is found in other membranes, particularly the respiratory membranes of calves. The first question you encounter when you find mycoplasma in a herd is the degree of spread, how long it has been there and you have to make a decision whether you are going to cull, as Bob mentioned, or whether you ar going to try to live with the disease. Either way, you can develop a successful program depending upon how the dairymen will cooperate with you. I think when a mycoplasma initially breaks out in a dairy, because it is acutely clinical, that most of the cases will be in the hospital string, or they will be identified clinically and you won't find too many cases outside that clinical group. A few, but not too many, whereas after mycoplasma has been in a herd for a while, it gets into the dry cows. There are latent shedders in the herd and so forth. It is a little more difficult to find every animal that is infected, it is a little more difficult to spot these latent shedders. Certainly it is not easy to isolate from the dry cow, even though we culture all dry cows in a situation like this, or the fresh cow. So that is the first decision you have to make if you get in very early in the outbreak and the clinical cases are pretty well grouped and there doesn't seem to be a lot of spread through the herd. I think you are better off culling, as Bob did in his herd. and going to the extreme sanitary measures and so forth to make sure the spread doesn't continue. I think it is an udder contact. I think that initially where it is spread is in the hospital pen or in the treatment of cows. Infusion of cows I think is the primary spread factor. Then you get secondary spread through the milking machine but certainly infusing dry cows and treating clinical cases are the primary methods of spread. We can have spontaneous mycoplasma occur as far as udder infection goes in the herd because these organisms are found in the vagina, and you can isolate it from the urine. In fact some of these are in higher numbers in the urine than in the vagina. Now what I think happens is that when an animal urinates, particulary a heifer, when you are milking her or treating her or doing something else to her, there is very commonly contamination of the urine on to the udder. So I think it is a mechanical transmission that initially gets that first case going. Treat that first case. Then you have tremendous numbers shed in the milk then you have secondary transmission via the hands and milking equipment and so forth. In this case, probably they purchased infected animals and part of the problem with the disease is a latent shedder. We found cows three years later infected that probably were infected at the initial outbreak and we have cultured them as high as 8 times and three years down the road after 8 cultures we found them positive. So these have been latently infected cows all this period of time and that is what makes it more difficult to get those last few cows out of there in some of these chronic infected herds. Sanitation, segregation and culling really are the basics to controlling mycoplasma.

Question: You said that culling, especially if you caught it

early, was the best way. But for instance, in a pure bred dairy, somebody has just bought a $30,000 cow, how do you deal with her? Can you bring her back to your regular herd after drying her off or what do you suggest?

Answer: Well certainly, antibiotic therapy hasn't shown any efficacy in curing the disease. If they are going to recover, they are going to recover on their own and a good percentage of these will do it depending on the type of mycoplasma you have. But if you have a valuable animal like that, if you were to dry that animal off and keep her and see how she freshens, the chances are 50-50. Clinically she might come in clean but would still be a shedder. Now we have had very high producing cows come back producing 100-120lbs. of milk and we have been able to isolate mycoplasma out of three of the quarters and yet they were fairly badly infected when they went dry. So you do get recovery and we do have herds that are maintaining mycoplasma strains, but if you do that you are at high risk and you have to maintain them, and identify them permanently. Once they are mycoplasma cows, they come back to that string, we enforce sanitation and milk that group of cows last and so forth. So it does take some stringent measures to be sure it doesn't spread throughout the herd but it is being successfully done.

That is one of the things that we recommend because very recently we have had the opportunity to go in and swab more units and the disinfecting procedures. If you get a cow that is shedding high numbers, there are just millions of those things in the milking unit, in fact, even with our backflushing even though we are doing a good job, and I always thought mycoplasma was a fairly easy microorganism to kill, you have to maintain a fairly high level of iodine and a fairly accurate flushing system to really eliminate all the organisms that are there, so the milking unit becomes a very important means of spreading. If you can concentrate on the unit even in the hospital barn, if you can isolate these cows to a group and your fresh cow area and your hospital barn area where these cows are coming in and initially don't know whether it is mycoplasma or something else, in that area it is very important that you dip your units and use very stringent disinfectant. That is where you can keep the cycle going.

Question: Would you specify some of those concentrations of iodine for us?

Answer: Well, if you are just doing static dipping in a bucket, if you are going to dip a high number of units, you want to have about 200 parts, if you are going to dip a unit ten or 15 times probably 100 parts of iodine, but no less than 100 parts in a bucket. We usually go to 200. In backflushing, we always recommended if you have a mechanically well-designed system, you use 25 parts but in some of these we just by chance happended to have a system in some dairies where we had some breaks and they were encouraged to kick that up to 50 parts or greater. But this is with a rinse system behind it. During a clinical outbreak you should be flushing the unit at least 50 parts and if you are going in the bucket, 100-200 parts.

Question: What about the pH of the water?

Answer: The pH of the water is really very important. In our area we have a lot of alkaline waters and iodine is less effective in alkaline waters and that is part of the reason you have to go to higher parts of iodine because you have to get the pH down and lot of these disinfectants have only so much phosphoric acid in them. Right now we are looking at the factor of pH in levels of iodine. But I still think that even though we are able to get the pH down around to about 3.5 which is desirable, 3.5-4, we are still looking at least 25 partsin our systems.

Question: Can you pick it up in the bulk tank as some people suggest?

Answer: Certainly if you are going to set up a system where you are going to monitor herds the bulk tank is a good thing to include. I think there are 2 dangers, however. First, depending on the number of cows and where the clinical disease appears the bulk tank is not 100%. You can have several cows in the hospital string for example and no cows in the milking herd shedding initially in the outbreak and you won't find it in the bulk tank. The other thing is you can pick up a form which is not a pathogenic mycoplasma. This can be easily confused if you are not doing FA's and identifying the organisms. You could pick up some non-pathogenic mycoplasmas in the tank. If you are just doing routine lab work, you could confuse it with pathogens.

Answer: One other problem I think that I got involved with mycoplasma was about 6-8 months ago or maybe longer. We came up with positive bulk tanks samples and at this point we got shaken up. We did every cow on the dairy and this was a 1200 cow dairy and we did not come up with a single mycoplasma. We sent the sample to Davis and it was *bovis*. Since then Dr. Smalley is doing the culturing for us and he has bulk tank samples that were positive a couple more times and yet we still have never culled a cow with *bovis* or with any kind of mycoplasma out of the dairy. I think we have to watch and not get overly concerned but realize we may have a problem and watch it but in this case we don't really know. We may have a cow on the place that has mycoplasma or we may not.

Answer: I can't relate to this specific herd but this is not an uncommon thing to happen. If you are doing just bulk tank as a routine, and you find it, the next place to go immediately is to your clinical cases. If you go that route rather than the whole herd and really establish that you have clinical disease that is the second step. It is going to show itself in clinical cases, because if you don't have a clinical disease you really don't have a problem.

Answer: We monitor a majority of our bulk tanks on a monthly basis and they will come up positive one month and you go back and recheck it and it's negative. There will be very few colonies. The philosophy that I have taken, be it right or wrong, is just monitor that tank. We look at clinical cows and we start to see some colonies on the tank but I don't get too worried until I start to see a significant number of colonies on the tank. If there are only 4 or 5 colonies on that tank it is not a big deal but if you get 50 colonies, there is

something definitely going on.

Question: Is if characteristic of more than one of these kinds that the cow simply dried up?

Answer: I think that you can see that with all of it. It is a very acute, swollen udder, with heavy garget or sandy milk as they describe it or she goes to the other extreme and she just becomes agalactic. I think it can happen with any of the various types. The cure rate is probably higher with some of the others than it is with *bovis*. *Bovis* seems to be the one that is hardest to control and gives you the most severe clinical disease and the most pathology. You can't really group things, because if you go in expecting something you will be fooled, because it's always going to be a little atypical. You are always going to find atypical cows. In general, all of the 5 types can appear very similar.

Question: How do you determine how far it is, whether it is just in your new clinical cases, or how well established it is in your herd?

Answer: In some herds it can be so common to have abnormal milk that they just assume that is just the way cows are and there can be a real high percentage of cows in the milking string being milked and still be infected with mycoplasma. As for treatment procedures, I know that it is possible to have a real high incidence of mycoplasma mastitis without using intra mammary therapy as a source of spread. This one dairy that you came and visited had a milking vacuum of 16 inches and some other problems, such as real poor sanitation, so I think that even without intramammary therapy you can have high spread. Again, we have seen this same thing of bulk tanks going positive and then immediately culturing strings and have them all come up negative and even having the total bulk tank come up negative. My feelings are like Dr. Smalley's. If you are culturing all clinical cases and you are not coming up with any and you occasionally have a bulk tank go positive, they are probably practicing good enough sanitation that it won't become an epidemic problem.

Question: Can you rule out laboratory error?

Answer: You can't always rule out the possibility of a collection error or a laboratory error because mycoplasmas sheds such huge numbers and we have seen this happen that when you are collecting more than one sample either from tanks or from cows you can get cross contamination even on your plate. If you try to put too many organisms on your plate you can get some contamination. So this is always something to be aware of. I remember one instance where 20 cows all of a sudden came up in this dairy that were not clinical. They should not have been there because they were out of a clean string and all 20 samples were contaminated. We went back and sampled those cows. He had sampled one clinical case and then he had sampled all these other cows and he had enough organisms there on his hands to contaminate every one of those samples. This happens. Numbers on the plate is one criterion you can use. If you find very low numbers and you go back and don't find clinical cases, I am not always sure that you can be sure that those organisms were in that particular sample.

Question: What about the udder wash water?

Answer: In the udder wash water that you use prior to milking, really what you are after there are coliforms and there we are usually looking at 75-100 parts. The 75-100 parts you would use for coliforms would certainly handle the mycoplasma and you sample the dry cows you can expect to pick up a number in a dry pen. You can culture it out. I don't know what the percentage would be, but let's say that you had 10 cows out there and by culturing dry cows you probably would pick up 5 of them. On those other 5 freshning then you might pick up another couple of them and those other 3 you might not find until somewhere in the lactation cycle. So your isolation attempts in the dry period and the fresh period do not seem to be the best time to get them, but you better pick up as many of those as you can ahead of time. The other thing that you can go on is abnormal secretions. A lot of these cows are known in mycoplasma strings where we maintain the cows. These cows come in and the secretions will be abnormal. We have always graded the secretions in the lab and we look at them and if they have garget or this sort of thing we make a note of it because those cows very often culture negatively with mycoplasma in their history. But the herdman will say, "you know that cow, the milk really didn't look right when I sampled her, but it was negative on culture." You never go on a negative culture once she is positive. You always accept the fact that she is positive. But it is just our ability to isolate it out or the activity of the organism at that time varies. I don't know of any dry cow preparations, and we have sampled cows that have been treated with everything, that will knock the organism out during the dry period or the fresh period.

Question: I just wondered on Steve's monitoring program how often when you grow mycoplasma you identify it as *bovis*? Do you push it that far or do you just call it mycoplasma when it grows on the plate?

Answer: Whenever we have a new herd that we haven't seen mycoplasma-like colonies on it, we send it off to be identified. I would say that probably 95% of the time if we think it is mycoplasma it is *mycoplasma bovis*. Every once in a while we have an error.

Answer: I think I can say from the herd that I have been dealing with it is a problem in the culturing technique. We have cultured it at different labs. We have gotten positive *mycoplasma bovis* out of the 3 different labs, and yet in between, we come up with negative bulk tank samples along with the positive. We do have *mycoplasma bovis* coming out of these tanks. Where it is coming from, I don't know, but it has been identified and from enough different sources I am confident that we have *mycoplasma bovis* there.

From the floor: I have a couple of comments. One of the first herds in which we had a problem with mycoplama was a herd somewhat like yours tht had a *strept agalactiae* problem and it was about a 400-500 cow herd and they had 30 cows with mastitis accumulated over a short period of

time. It just happened, this is speculation of course, that at the same time they were having a calf pneumonia problem and the same man that was caring for the calves and pneumonia problem and the same man that was caring for the calves and treating the calves was doing all of the intramammary treatment on the cows. Against my recommendation they were using penicillin to inject the udder to treat the cows rather than a commercial tube and this fellow did not have the best sanitary habits and I am sure that is the way the mycoplasma got introduced because when they first looked at it, we thought of *strept agalactiae* treatment but they wouldn't respond to treatment. We had mycoplasma. Another thing that has been a benefit in our practice is looking at this bulk tank on a monthly basis both for the bacterial content and the mycoplasma content. It has been a good method for us to get more involved in the control program of mastitis in the dairy because you get something concrete to deal with and you can get involved in doing system analysis on a regular basis and really get more involved in the whole mastitis program.

Question: Do you always use an iodine type disinfectant?

Answer: There are other disinfectants that are effective. Chlorine is certainly effective. I think the reason we have gone to iodines is because the multiple use that we have for them. We know they are effective. They are more effective on organic matter than chlorine and you can also use them in about a 4 to 1 ratio so we are talking about a 100 parts iodine, 300-400 parts chlorine. Chlorine is more caustic and irritating and very hard on liners. With the iodines liner life actually increases, whereas with chlorine the line life drops off dramatically. This is why we have gone to iodine. Dr. Jasper led me to believe that probably 10-15% of the cows that come through the dry period are shedders and we would get a cure rate of maybe 85% of the cows or is a little bit higher than that as far as incidence of cows still shedding after the dry period.

Dr. Bushnell: It depends a lot upon the herd and the cows and the organism. I would say that you would be very lucky if you came up with 10 or 15%. I would say that most of the work that we have done would show closer to 50%. You can re-isolate. The longer you follow those cows, the more times you do it, the higher the percentage will come.

Chairman: **Dr. Darlington is going to start out discussing his experiences with coliform mastitis and then Dr. Radostitis is going to give some comments on treatment.**

Dr. Darlington: In my area in the State of Washington we see both Klebsiella and *E. coli* and we see an occasional Aerobacter. I had a couple of slides on an autopsy of a Klebsiella cow. A Jersey farmer brought in 3 cows and lost them. All of them had been sick less than 12 or 13 hours from the time they first noticed the problem and I think if we look at the autopsy we can get an idea of the intensity of the situation and the difficulty in treating even those that don't die that rapidly. There were ectocardial and endocardial hemorrhages; characteristic, classic, abomasal ulcer, hemorrhages in the abomasum due to enterotoic shock from the coliforms either from a mastitis or occasionally from a metritis. When you look at the abomasum it really gives you an idea of how intensive your therapy has to be and which way you go for therapy for coliform. The primary problem that we have in our area is with Kelbsiella but we do see a lot of *E. coli* and I was interested in the work that came out in 1975 on the bacteria levels in bedding as far as the incidence of mastitis. We have cultured wood chips and shavings and sawdust going into the dairies and almost universally in our area they are positive for Kelbsiella. Pseudomonas are present in them as well even though you don't see it as a mastitis problem. Except for the killed dried coming from the furniture factories and door factories they are almost universally positive for Klebsiella. When the numbers of coliforms backeria in the bedding reached 10^6 or higher the incidence of coliform mastitis increased. I think in looking back at some of the herd situation where we see coliforms and other places where we have not seen them, the type of bedding, the way it was handled, the number of times that they level the stalls starts to made some sense as far as bacteria counts are concerned. In one herd we had a situation where they stopped leveling the free stalls and got out of a coliform mastitis problem. Thinking about bacteria counts, probably the surface area of the bedding had the lower bacteria counts. An inch or 2 inches down, it had the moisture and also the warmth in a bedding pack and we came through with a rake once a day and raked it right up over where the cow was going to lie her udder down on it and we got the population on the udder, went to the parlor and didn't wash it all off and injected it back to the quarter. I have had them done by sending them to a diagnostic lab that is doing water counts for bacteria. Don Jasper had been doing the same work and maybe Dr. Bushnell was involved in it too in California, Showing the incidence of bedding. Bacteria counts 10^6 or greater had a high incidence or had an increased incidence of coliform mastitis. In this study there was a great number of samples, percent of the samples in the wet bedding, but in that bedding it never reached that 10^6 figure that was reported in 1975 as being somewhat of a magic number where you had problems or you didn't have problems.

As far as treatment for coliform and Klebsiella mastitis, mine is the same. Probably the thing that I feel that is the most important is early detecting and milking that cow every hour. It is getting rid of the bacteria and eliminating the endotoxin released from the cow so we do not get this histamine reaction which I believe causes the abomasal upset and makes that cow extremely ill. We do use tetracyclines and sulfa-dimethoxine IV. We use a lot of antihistamines. I will give 40cc's and repeat that at 4 hour intervals. Recently we started using Banamine. I started using Banamine on a scouring calf that is down and out, he will just get up and walk away for you. I tried 3cc of Banamine on a 5 day old calf that was down and extremely depressed and I can't say that it saved them all but I would say that everyone that I have given it to has gotten up and ate and some of them died

a week later but it has been quite a dramatic effect and that is because it is a prostaglandin inhibitor I believe.They also say that it will block the endotoxic shock. With that information I started using it on coliform mastitis cases and I think we are getting some response. We are thinking about going on a higher dosage. We are using the same dosages in the equine, 1cc per 100 lb body weight. The other prostraglandin inhibitor we have used quite a bit on these coliform cows is aspirin, and I think we have some benefit there. Genocin has been used, 35cc IV, one dosage, and that is with mixed emotions. The herdsmen in our area are adept at giving fluids and at times on some of these cows they will give 4-5 gallons of Eltraad IV-4000 and we seem to get some benefit there.

Dr. Radostits: I would like to make some comments about the clinical management of peracute coliform mastitis as we see it in our Saskatoon Veterinary Teaching clinic. We see 15-20 cases a year. Unsually it is the peracute ones that are referred in to our clinic by our ambulatory people. The case mortality rate over the last 10 years is about 60%. I am talking about the peracute cases of the cows which are very, very ill. What are the problems as I see them? Well, I believe that the owners are not seeing them early enough but that may not be their fault. Maybe the unique feature of coliform mastitis, based on some recent work, is the organisms are multiplying in the mammary gland for 12-24 hours before the cow really gets sick and they have multiplied to such massive numbers that the cow really cannot control them. Some good works suggest that. These cows have peracute toxemia and that is difficult to treat. I have a problem deciding with confidence which antibiotics to use and by which route, how much, how often. The economics of treatment is starting to bother me. After two weeks we sent home a cow that we treated for coliform mastits and the bill was $342.00. She survived so that was not too bad. The complications are many. Maybe it is the complications that are killing these cows that go down but we know tht many of them go down, maybe dislocate their hip. They develop more mastitis, decubitus ulcers, and pulmonary edema. I can't understand that but these cows will start breathing heavily about 3 days after they have been in the clinic, then they get necrosis of the muscles. I would suspect if we could avoid these complications it might improve my survival rate.

Just a few words on epidemiology. We have heard many times that pre-existing cell count in the milk is very important. If these cows have a very low cell count they seem to be very susceptible to peracute mastitis. More than 90% of the peracute ones that we see in our clinic are within 2 days of calving. We rarely see it before calving. It is usually within 2 or 3 days after calving. We see it most commonly in high producing cows in our area even though we don't have a high incidence of high producing cows. It is most common in our better herds which are on our mastitis control program. By far the peak incidence is during the winter months when our cows are housed. Most of our cows are bedded on cereal grain straws. It is pretty rare to get any barn around there with shavings or sawdust. Whether it is associated with poor milking sanitation in the herds we deal with is doubltful because I say they are in our better herds. Herds that we are quite proud of. We think they are doing a good job. I believe we have to know something about pathogenesis before we treat it. The organism gets in the mammary glands and proliferates. Some recent British work shows that maybe that organism can remain in the gland for some time. American work has shown that too. So we really don't know how long that organism has been around. There is good evidence that it may remain latent for 30-40 days. In any event, it elaborates its toxin, produces and endotoxemia, and very marked leukopenia and neutropenia. It just drains the cow's neutrophil reserves, produces mastitis, toxemia and interesting enough a diarrhea. I don't know if that is an endotoxic diarrhea. These cows dehydrate and become very weak and recumbent. About 50% of the cows dehydrate and become very weak and recumbent. About 50% of the cows which they send into me at the clinic or into our Food Animal Clinic are recumbent. These 1600 lb. cows have to be loaded with a forklift and trucked into the clinic. Well, how do we treat them? The antimicrobials we use parenterally. Most of the time we have been using chloramphenicol at the dose rate of 25-50mg per kg body weight every 12 hours IV. We have not used tetracycline for some time now because the *E. coli* that we isolate from these cows are resistant to tetracycline but what we have used for the last year in about 1/3 of these cows is Trivectrim, that is trimethaprine potentiated sulfonamide. I understand you don't have this available in this country. A tremendous drug for the treatment of *E. coli*, salmonella. It is used extensively in Australia and Canada now. It is going to gradually take the place of chloramphemicol, a very useful drug. The *E. coli* that we get from calves and cows are over 97% sensitive to this drug. So you should have this. You should put pressure on your government to allow this to be used. If you don't like chloramphenicol and can't get it, try trimethaprine, potentiated sulfanilamide, either sulfadoxine or sulfadiazene. I am sure it is available for small animals and horses. Oxytocin, well we tried to strip out the quarter with multiple injections of oxytocin and that is nothing new. Very often it is not very rewarding. Intramammary? We inject chloramphenicol which is the same product we give intravenously. We put 5 grams of that product in 500 milliliters of distilled water and put in the infected quarter. This has been determined vary arbitrarily. This is an attempt to improve the survival rate. I don't really know if that does any good in the mammary gland. We are in the process of trying to evaluate that. It might be all absorbed by the mammary gland, but I don't know. It is an attempt to get an antibody into that gland cistern where the organisms are multiplying. Fluid therapy, this is something that we perhaps over do because I am convinced that fluid therapy is necessary if we are going to save these cows. Undoubtedly we overtreat these cows compared to what you would do in a farm situation, compared to what our ambulatory people

would do on a farm situation. We use massive quantities of fluid. We think it is very important in saving cows. A 440 Kg cow will get 40-60 liters of fluid in a 24 hour period, 20-30 liters in the first 4-6 hours particulary if they are badly dehydrated and recumbent. So we put in indwelling catheters into their veins and keep the fluids at them through a 5 gallon plastic jug around the clock until we get them on their feet, which usually takes about 2 days. So antibiotics, fluid therapy, corticosteroids, what is the rationale for that? In summary the rationale is to treat endotoxemia. you know that corticosteroids do about 7 or 8 different things in trucked animals. We have been trying to evaluate this. I did a retrospective analysis on all the records of the cows treated for coliform mastitis in our clinic over the last ten years and roughly 55% are not treated with corticosteroids and 45% are. The survival rates are both the same. Maybe we are not using enough. The clinicians use different levels. One milligram per 1-5 kg of body weight IV every 24 hours. I don't know what levels of cortiosteroids should be used in a big mature cow for the treatment of shock. I just don't have any thumb rule, because the pharmacodynamics have never been done. The small animal people tell us that we should be using 30 mg per kg body weight every 12 hours. I would just have to say, I don't know. It makes some pharmacologic sense to me to be looking at corticosteroids. We are going to start using massive doses in our cows to see if that will make any difference.

Bob has told me to comment about antihistamines and Banamine. I used to use antihistamines a long time ago. I don't use them anymore. Probably thought they were not doing them any good. I don't see the rationale for using antihistamines. If you have a very sick cow, if there is histamine release your antihistmines are probably not going to be of much value because the antihistamines work by competitive antagonism. So the histamines are already there. But it is difficult for me to prove that they don't do any good. you can't prove the absence of a relationship, you can't prove that antihistamines don't work. So I don't argue with the practitioners anymore. If they want to use it, fine. What about Banamine, a prostaglandin inhibitor? Yes, that may make some sense. However, if you look at the pathogenesis and what these cows look at necropsy there is edema of the mammary glands in these acute cases. There is diffuse necrosis. There is probably extensive endotoxemia and the Israeli who has worked on this is showing very nice levels of endotoxin in the blood of these cows with coliform mastitis. The endotoxins are being absorbed into the circulation all the time. That is what you are fighting. My theory is we are going to have to fight this disease based on controlling that endotoxin. The problem with that endotoxin is tht it is a very large molecule. A very, very large molecule which in the cow probably like other mammalian enzymes the kidneys cannot excrete and get rid of quickly. Theoretically we have to get at that endotoxin some how in the systemic circulation or the mammary glands. In our clinic, cows stay an average of 4 days. If we don't make any progress after 4 days, we usually recommend killing them, particulary if they are down. Hospital stay costs 8 dollars a day. We are not making any money on that. Antibiotics: a 500 kg cow using 25 grams of chloramphenicol a day at 75-80 cents a gram, costs $20 a day, that is $80 4 days, $120. We usually do some lab work. Usually just simple hemograms to see whether or not there is a leukopenia and how severe it is and whether or not the cow is responding. We find that very useful as a prognostic aid which says, yes, the cow is finally coming back with a regenerated left shift or she isn't. Oxytocin for a few days will cost you $12; intramammary 16 dollars. Professional services, what is my time worth? I cost it out at 1-2 hours a day at $25 an hour. I know I spend much more time just bedding those cows down and keeping them rolled over. I could probably hire someone to do that. I spend a lot of time re-assessing these cows every day, or my resident does or some veterinarian does. That is $100-200. I spend 42¢ a day on catheters. I spend $68 on cortico steroids. Total cost $448. I mentioned that cow, two weeks ago, we sent home, cost $342. I am sure we had much more into that cow than $342 and then what if the cow died? I am into it for $342. I think the treatment of coliform mastitis is very, very difficult. I think the solution is to try to control it. We need urgently some research to try to minimize the incidence of coliform mastitis. Once you are presented with a peracute form I think it is very difficult to treat it. My survival rate has been very poor and I have what I consider to be ideal conditions. I am talking about the peracute forms. The very, very sick cows, not acute or chronic forms. We probably treat many of those everyday with a much higher survival rate. Survival rate for me in the peracute form has not changed in 20 years.

Question: Do you find that the heifers seem to survive it a lot better than the older cows?

Answer: I don't really know, I would have to go back and check my records. You don't see it very often in 2 year old heifers. My recollection is that I see it most commonly in mature cows.

From the floor: My experience has been I find that if I have a young heifer that has it I have a good fighting chance. But if it is an old, high producing cow or say 3rd or 4th calf or beyond, my chances whatever I do is to little avail but it seems with a heifer I have a good chance.

Dr. Darlington: I am seeing coliform mastitis in the cow that has been fresh for 30-75, 80 days; she is in peak production. She is one of the best cows in the herd and she is really pouring that milk out. It is not the fresh cow. We very seldom see an acute form in a fresh cow. It is that cow that is down the road and at peak production.

Question: Do you save those cows that get coliform mastitis right after calving?

Answer: Very, very seldom.

Question: Do you get it in herds using dry cow therapy?

Answer: My information comes more from Dr. John Woods, some work he had done. I know his interpretation was that cows that had very low incidence of other types of mastitis were much more likely to have coliform mastitis

than cows within quite an infected herd. So if you are in a good dry cow treatment program you have other types in a very low incidence and you are much more apt to have coliform mastitis than any other herd. I know that is what I have seen in my practice. Two of the cleanest herds I have got have by far the most coliform. I see it right after calving. The tendency is to be a very high producing cow, generally a "leaker." My luck in treating this cow results in basically getting to her extremely quickly, almost to the point that every 15 minutes you are not there that she is not treated after it is discovered, it hurts. In other words, if you wait 12 hours that is 12 hours wasted that could cost the cow's life. One other comment, I have used quite a bit of genocin and I have had very good luck at 10cc's IV rather than your level of 35. I don't know if I am lucky or what, but I have had good luck at that level. Give that generally for four days. I was just told by another practitioner that if you give it twice the first day you will do much better. But I have been giving it once a day, 10cc for 4 days and having excellent luck. I had this tip from practitioners from Wisconsin and they are having luck with it also. One other treatment that you didn't mention that I think is a good treatment is calcium on these downer cows. I think most of us have heard of that. I am not saying that they have milk fever, but I think that in conjunction with these other treatments we do quite a bit of good with calcium.

Question: If you put medication intramammarily, do you still strip out as often as possible?

Answer: We put it in, strip all day, and then put it in at night before we leave the cow.

Dr. Abernathy: I use calcium routinely on all of my coliform mastitis cows. I think they are hypocalcemic and I monitor calcium intravenously very closely. I also run about 2 bottles of about 50% dextrose into all of them just routinely because I think if we flood their liver with dextrose it just gives them something to work with for a while because these cows are totally anorexic. I also use B-vitamins and then I use whatever brand of antibiotics. I go along with Bob that we try to milk them as frequently as possible.

Dr. Radostits: They are hypocalcemic. So are overload cows but overload cows don't survive if you give them calcium. I just can't buy that. I have seen too many clinicians at our college kill coliform cows by giving them calcium. There is no way I will give calcium to a coliform cow. There is calcium in the fluids at a physiologic level going in there. But if you keep giving calcium to coliform cows you will have them keel over. These cows will not survive, you don't increase your survival rate by giving them calcium borogluconate just because of the calcium in there. The other thing is that sick cows that are toxic with mastitis do not become hypocalcemic to the point that they are going to die form hypocalcemia. Sick cows don't develop milk fever. It is those healthy cows with a big bag of milk that develop milk fever.

From the floor: Yes, but some of them get up after you give them calcium.

Dr. Radostits: They don't have coliform mastitis. That's the problem. If they are getting up and responding and I could have talked about the differential diagnosis of coliform mastitis. I think you are treating milk fevers, not coliform mastitis.

Question: Is there a relationship between positive CMT and coliform mastitis?

Dr. Bushnell: We had the opportunity to follow coliform herds for some period of time. We see coliform maybe a little bit differently than has been projected here. When you actually follow a herd you realize there is a tremendous number of coliform infections that occur. Only a percentage of these are peracute. Those generally are the Klebsiellas and they are usually the bad guys. As far as infection rates, where we follow these herds and show an increase in CMT's related to organisms, we have streps or staphs in these herds. In most of those, about 90% of the increase in CMT's are due to micrococcus or *staph. epidermis,* occasionally strep. A very small percentage are related to your coliforms but on the other hand about 90% of your clinical disease are related to coliforms. So what that tells us is that this is what happens I think when you get rid of *strep* and *staph.* There is still infection taking place due to mechanical things and just the fact that we are milking cows. If a lot of the micrococci and low pathogenic coliforms are getting in there we are not seeing much clinical disease but the real peracute ones that get in there that shows us this real clinical disease. This is what you are referring to. The infections that occur. Most of them are non-pathogenic type organisms infections. Well, I think the relationship is that coliform is probably one of the few organisms that is fairly well controlled with a level of leukocytes, I think Schalm showed us that quite well. If you have 500,000 cells in there you can control coliform infections. If you get rid of strep and staph you are having a higher percentage of the cows that don't. But a lot of these herds that have a lot of strep. and staph also have a lot of concurrent coliform infections and it is when you remove the strep and the staph the coliform becomes more obvious, but they probably slightly increase in numbers. Most of the infections going on are these non-pathogenic type organisms that are just causing transient problems which are probably good because they get in there and they cause a little increase in CMT and the coliform probably does not get in there. Dr. Louise Owens is married to a dairyman and so she has an in. She worked with Dr. Schalm when she was going to school at Davis and they were using endotoxin to treat chronic cases. She has used endotoxin to treat coliform mastitis now for about 3 years. We have followed all these cases with her. 90% of the coliforms in that herd she treats with no antibiotics, just endotoxin and she gets a good response. It brings out a tremendous flush of leukocytes, the milk thickens and then the animal recovers. It doesn't work against Klebsiella. You get a real peracute and occasional peracute *E. coli* or Klebsiella and it is absolutely of no benefit. But against your routine case of coliform mastitis, a little hard quarter, milder types, it works beautifully. 0.5

milligrams of Difco endotoxin diluted in saline and injected into the quarter with 90% response. These are all clinical cases, these are acute. They are not the real hot peracute ones you are describing. What we would do is put more in the Klebsiella category. They are certainly acute coliform mastitis. Probably under normal practice circumstances the veterinarian would not see a high percentage of this type of patient. They would be treated with an antibiotic tube or something like this and passed off to some other type of infection. Most of them are not that real peracute with a high fever. The other thing of interest is that we actually established a coliform mastitis with Klebsiella and we followed that cow for 2 days and it was interesting to note that the first thing that goes before temperature or anything else is the appetite. And then they will start to get this viscous stringy udder secretion and then the temperature will go up and you will actually see clinical evidence in the quarter before you have any rise in leukocyte counts. They pass tremendous volumes of urine very early in the disease and you can just watch them dehydrate right before your eyes. I think this is part of the endotoxin effect that they are urinating. This is one of the larger reasons, besides the fever, for the dehydration. The other thing of interest was that in this particular cow, whe had a non ag. strept low pathogenic chronic infection in one quarter. This was a heifer by the way. Within a matter of 12-24 hours after we injected her, she started reaching to the Klebsiella infection and that infection just took off. We just developed millions of non-ag streps in that one quarter. So this gets back again probably to your philosophy and really treating these cows heavily. Not only is it the infection of the endotoxin itself you are trying to get against, but it is also probably setting off all types of other chronic low grade infections within that cow.

Dr. Abernathy: Let me just put a little nutritional aspect into this. In our country we feed a lot of brewer's grain and one thing that I have noticed is that especially with summertime coliform mastitis, I don't know how many of you feed brewer's, but if it gets more than about a week old or so, at least in our country, you can feed anything in the total mix ration. You can grind up garbage and eat it great and I can't document what's in it but I am sure we are looking at loads of mycotoxins. I don't know what they are, and also clostridia. In these cows on this old aged brewer's, the dry matter intakes drop off and milk production starts dropping off because of that but coliform mastitis starts to increase in the better herds, ones that have their counts down around 200,000 or so. It is cyclic with feeding of this old brewer's. It is more of a problem in the summertime because it starts rotting faster in the summer. When that brewer's starts to smell, or starts to get rotten and you are feeding it to the cows, whether is is mycotoxins or clostridial toxins or whatever from any immune suppression standpoint, problems arise. That is something that has seemed to be valid observation.

Dr. Bushnell: That's interesting because we have had dairymen that know every correlation with certain feeds, feeding beet pulp, so we got into this thing of looking at various feeds in relationship to various coliform outbreaks and what we found, and I don't know if it was a direct relationship or indirect, is that our by-product feeds in California, namely beet pulp and cottonseed meal or whole cottonseed, are tremendously loaded with Klebsiella. There are millions of Klebsiella in these seeds. We worry about our bedding coming in and we worry about the cleanliness of the milk, but in California at least we start feeding calf pellets and so forth at day one and we have cultured these pellets and your by-product feeds that are in those pellets. Often you will find tremendous numbers of Klebsiella. You can also go to your water troughs where the grain has gotten in the water troughs and the Klebsiella are in there by the millions. So we talk about shavings. I tend to downgrade shavings. In California I don't really think that is our big problem. There are other problems related to coliform mastitis. But certainly the potential for coming in through our feeds, or by-product feeds for putting Klebsiella in the bedding or anywhere you want to put it is tremendous. I just wonder when you mentioned yeast if you don't have the same thing in your by-product feeds.

Question: I am curious what laboratory measures you are using to confirm the diagnosis specifically bacteriology, and cultural sensitivity, hematology, measuring calcium-phosphorus levels on cows you suspect are hypocalcemic, are any of you doing any of these?

Dr. Darlington: As far as bacteriology, in our office we are gunning an identification with API 21 we do get besides Klebsiella pneumonia an occasional different Kleb-organism out of some of these clinical mastitis cases. To digress a little bit from that, I might mention in regards to the better herd situation, in 1963, I saw my first case of Klebsiella mastitis in a 600 cow herd that had better than average management and dropped about 18 cows in about 3-4 weeks. I was a new practitioner, just bought the practice from somebody else and that somebody else still happened to be in town and so I had him working with me on it. Otherwise I would not have had the honor of working for that farm after that. But in checking the literature, in checking around the United States, I found two places that had experienced Klebsiella mastitis at that time. One of them was an institution or a school farm in Pennsylvania the other was Carnation farms in Carnation, Washington, both very highly managed operations and I think our commercial dairymen today have approached what those people were doing 15 and 16 years ago and we were getting the cell counts downs. They were stopping that first line of defense. I think it is a management problem that has to be addressed to the industry and in the parlor situation.