Medical-Surgical Case Work-Up

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Editor's Note: This is a presentation dealing with the decision process “to cut, or not to cut” on several specific cases. Pros and cons of surgery as well as the resulting findings and ultimate outcome are presented in a lively discussion format. Some case reports have been omitted due to lack of space.

Dr. Smith: We heard yesterday afternoon that clinical medicine bases itself on probabilities—the probability of a certain disease state being present or the probability of a certain surgical approach or medical therapy working or not working. Basically I would like to share with you two objectives that we hope to gain this afternoon. The first is to share with you some cases which we have found, we meaning Sheila and myself, interesting and challenging at the University of Wisconsin. The second objective, which perhaps is more important, is to gain from you and you gain from each other, information which, either we don’t know or your cohorts might advise you that you already don’t know! I think if we come to these continuing education-type format sessions and do not take back more than we come with that we are, in fact, the losers. In most cases we are not the losers. So we expect and certainly request participation from the audience if you have something in each individual case to offer. Basically we will be going through these cases with three things in mind. The first question, and probably the least important, is: whether we cut or don’t cut. In other words, to cut or not to cut, even though that is listed in the brochures as being an important fact, but it is probably not the most important. We will early on establish whether we should cut or not cut. Secondly, we will establish the time of surgery. I think this is one of the most important things that we need to discuss. Relative to the medical/surgical aspects of the case, when is the appropriate time to cut? After a certain amount of medical therapy, or early on in the case? Finally, how to cut, what approach to use. What anticipated surgical procedure do we expect would be most likely to provide positive results for the cow? Finally, but certainly not of least importance, 1 would like to acknowledge the fact that all of these cases have come from the University of Wisconsin and some of you have sent some of them to us. We are very appreciative of the fact that you have been able to provide us with these teaching cases for the students, ourselves, and obviously in a situation like this, for our fellow practitioners. To those of you from Wisconsin and northern Illinois, it could very well be that one of them is in fact yours!

Case No. One

This case we have entitled “cow with colic,” a 3-year-old, 1,550 lb. Holstein cow, which is bigger than average. This particular cow calved two weeks previously, no problems, unassisted delivery. Last night she ate her meal normally. When approached at 5 o’clock this morning by the farmer when he went out to milk the cows, she would not get up. He called his veterinarian about 8 o’clock this morning. Later on in the morning, the veterinarian saw the cow. At that time she was still reluctant to stand. She showed evidence of colic by virtue of stretching out when she was in lateral recumbency. She showed some signs of abdominal distention, particularly on the right side. She had no evidence of having passed any feces during the night or early in the morning. The veterinarian was able to get her to stand. He noticed that she had signs of colic, characterized by kicking at the abdomen and the desire to lie down again.

The veterinarian said that he had a cow which he thought had a twist around the mesentery. Certainly, by virtue of the history, it seemed a very logical provisional diagnosis. The cow was put on a truck and shipped in to us with the history which you have just heard. When the cow arrived, we noticed she had a normal temperature, pulse, and respiratory rate. She was depressed and moderately dehydrated, probably a 7-8% dehydration based on skin touch and degree of anophthalmus. She had abdominal distention on the left side. It looked like a pear on the left and a little like an apple on the right, so it was a mild abdominal distention which was in the ventral abdomen on the right side. She had shown signs of colic at home, but on arrival at the clinic, they were no longer apparent. The colic signs which we did note were those of perhaps stretching of the legs and a slight crampiness so they were not as obvious as they had been previously. She had a marked area of sharp tympanitic resonance on the right side of the abdomen, at the 10th or 11th intercostal space, back to the middle part of the paralumbar fossa. On percussion of the abdomen, we were able to detect the presence of a small amount of fluid within that ventral area. We did a rectal examination and were able to identify a large segment of bowel which was distended. This large segment of bowel included the small intestine and large intestine, probably involving both cecum and proximal colon. This distention was moderate in intensity and encompassed approximately 2/3 of the right side of the abdomen. Now we have a cow which had shown signs of dramatic colic at home to the point that she was not able or willing to stand up. By the time she had a truck ride of approximately one hour, she was not showing the same severity of colic but showed some abdominal distention, depression, dehydration, fairly large "ping" on the right side, possible fluid within that "ping," and a rectal examination revealed distention of the large and small...
intestine. The one thing that is missing from the discussion so far concerns fecal passage. This cow had passed a small amount of feces on the truck. She had not passed any during the night or up to that point when she had been put on the truck. On rectal examination, we also raked a small amount of fairly soft feces out of the rectum. At the time of the examination, we questioned the owner with regard to specific feed changes. This was a Monday that we saw the cow, and he had between Friday and Saturday changed from haylage to grass hay, and then back on Sunday to alfalfa in round bales. During that 2-3 day period he had two significant roughage feed changes. He mixed his grain once a week. The last time they had mixed the grain, which was the latter part of the previous week, they had fed from the bottom of the bin and he said there may have been some mold as they had mixed that grain together. At this point we had a provisional diagnosis, but we wanted to do some laboratory work.

It indicated the packed cell volume was 32% and the total protein concentration was 8.3 grams per deciliter. Remember, this cow was moderately dehydrated and a slight elevation of total protein probably reflects hemoconcentration. The 32% packed cell volume is relatively low if you consider the fact that she was dehydrated, still probably within the normal range. Her white blood cell count, almost 20,000 per microliter, is high, and the predominance of segmented neutrophils is also high. You must remember now that the cows we see in the referral hospital have been transported, so there is a very high likelihood that part at least of this leukocytosis and neutrophilia might be associated with endogenous corticoid release from the truck ride. Part of it might be associated with the same endogenous corticoid release associated with the colic condition. However, it is likely, or at least possible, that this cow may have also had an inflammatory condition which could partly count for that high blood cell count, although it is not really showing up dramatically in the fibrinogen which at 500 mg per deciliter is certainly well within the normal range. Her serum chloride concentration, as well as potassium and sodium, were normal. So if there was an upset, it was not severe.

We had basically five “ruleouts” that we considered strongly—mesenteric group torsion was certainly one which we considered since the cow was put on the trailer to come to the clinic. That was a primary “rule-out.” What were the chances of having a mesenteric root torsion? I think it is more common than we in referral hospitals will be able to identify because the condition is so acute. If you will look at the right side of the intestinal tract of a cow, you will notice the small intestine around the outside of the mesentery. With torsion of the mesenteric root we have volvulus of the majority of both the small and the large intestinal tract. So the entire jejunum, ileum and cecum, and most of the proximal colon, twist around the root of the mesentery. Certain parts at the top, because they are suspended within the combined mesenteries do not twist. These cows appear as round in shape because they have tremendously rapid distention of the small and large intestines. It is a very dramatic disease, rapidly fatal if uncorrected. Another condition which we also considered very strongly was volvulus of the distal phalange of the small intestine which is that area where the mesentery is longest. What happens is that the area where you have a long mesentery will twist about itself, and cause, of course, a strangulation obstruction, a closed loop obstruction. She either had a torsion at the root of the mesentery, or a volvulus of just the distal phalange of the mesentery. Another possibility, although less likely, would be some sort of rupture or other cause in which we had peritonitis established over, in this case, the spiral colon area, causing ilius in both the small and large intestine. We did not do a peritoneal tap. The reason being that we had already established a provisional diagnosis which we were comfortable with, and the cow was distended enough that we did not feel we wanted to risk puncturing the distended viscosa. Lower down on the list, but nevertheless, still of consideration, was abomasal volvulus. The reason we didn’t think it was abomasal volvulus is because in those cows, the “ping” is further cranially, usually you can feel, if they are that large, the outline of the greater curvature of the abomasum on rectal palpation, and we certainly could not do that in this particular cow. We did not consider small intestinal obstructions such as intussusception, the reason being on rectal examination you can feel large loops of intestine. Intussusception, invariably, in adult cattle, involves the small intestine. If you feel a distended cecum or colon, chances of an intussusception in an adult cow are very close to zero.

Let us just discuss briefly what we would have done, or what we will do if we take this cow to surgery. We can do either a standing approach in which we operate from the right side. We run the risk of the cow lying down if she is still in colic, or we have a twist in the mesentery and have to exteriorize the bowel, or we can do a lateral recumbency with the cow on her left side, preferably with supports under her hip and shoulder to provide a pocket for the distended rumen to fall into. Therefore, our distention on the right side will not preclude abdominal closure or good exteriorization of various parts of the bowel. In the list of “rule-outs” that we considered, I did not talk about enteritis or indigestion. They are medical conditions and they certainly were very high on the list—indigestion was very high on the list in this particular cow. Basically this was one cow where we had to decide to cut or not to cut.

Dr. McGuirk: A couple of questions first. Given the feeding history, were there any other cows on the farm similarly, or otherwise affected? The next question would be when you look to the “ping” on the right side, if you hold the stethoscope in a single spot and you thump her in a single spot, does the pitch of the “ping” vary or is it consistent? It varies, it goes up and down. My interpretation of that, when looking at cows is that is probably tubular gut that I’m listening to the “ping” in and not the abomasum. My general experience has been that abomasal torsions or displacements, left or right, (obviously there’s no tubular gut on the left), don’t vary over the very small period of time you are listening to them, whereas “pings” associated with tubular gut will vary over time. The pitch changes as the gut contracts and relaxes. So that makes me think “ping” is in fact what you hear in the tubular gut, and that we don’t have an abomasal problem. Do I want to “cut” or “not cut?” I want to “cut.” One of the marks of a good clinician is using all available information. But secondly, because first of all you have the acuteness of it, at least as far as the dairyman knew she was all right last night. This morning she began to show signs, but from the early morning signs she was not very distended, and by the time you saw her five hours later she was really distended and had become quite dehydrated over a short period of time and she was actively painful. I would say of all the
Dr. Smith: Certainly you wouldn’t get too much argument out of me on that rationalization of “yes” to “cut” and how to “cut.” Unfortunately we looked at it slightly differently. The reason we did that, if you look at the pros and cons once again, is that I don’t think this is a case where there is a clear cut answer. We looked at the fact that she had severe colic at home. She had much less colic in the clinic. If it was a torsion of the mesenteric root, we would have expected that to have progressed or the cow would be virtually dead. She was more dehydrated and more distended, so that’s going back towards cutting, but she was not in colic or very minimally so when she arrived. Secondly, she did not have that evidence of passage of fecal material which I think was a good sign in terms of starting to pass things through. If I had seen this cow on the farm and was only there once and had to make a decision, I would have “cut” her. In a clinic where you can watch her virtually minute by minute almost, then it is a different situation. It depends on where you are, but basically because the colic was becoming less severe, the cow seemed to look a little bit brighter than what we had heard at home, although I didn’t really give you that information, we decided to wait. During the period of the next two hours, the cow essentially very quickly reverted back to what is normal, so we did not “cut” this cow. We never did find if there was anything going on inside of significance in terms of surgical relationships, but I think this is a classical type of situation where you are tossing up between indigestion, severe indigestion, or a twist of some sort. I think both are right. If you “cut” a cow like this you would probably not kill her. You would probably not do her any harm. If you didn’t “cut” her and left the farm without any good, watchful eye, you might in fact, make the wrong choice. This is the only case where we have a clear cut situation where the decision is to “cut” or “not to cut.” In this situation we decided to wait. In fact, the cow did have what we diagnosed as indigestion. Bob Whitlock, what are your comments?

Dr. Whitlock: Did you do a rectal later on?

Dr. Smith: Following her on later that afternoon and evening, she showed less evidence of distention and she started to pass loose feces over the next 24-hour period. So the answer is, the distention relieved itself, the rectal palpation of distention was certainly less apparent later on.

Dr. Smith: Whitlock’s point, in case you didn’t hear that, was that this cow may have had a home cecal rotation, which by virtue of the truck ride, shook free and untwisted sufficiently that fecal material could pass. I could certainly attest to that because we are doing a research project in which we are identifying on the farm cervical cecal volvulus cases and by the time they get to the clinic, the odd one will have corrected, so the point is well taken. My experience, however, is that those cows are usually not in acute colic early on. That, however, does not preclude it.

Question: Was any medication given by the veterinarian?

Dr. Smith: No medication was given by the veterinarian at home or by the clinic, except for fluid therapy early on.

Question: On a farm, should we decide not to “cut” her, what about medical therapy?

Dr. McGuirk: In this particular case, given the abnormalities that you see on laboratory and from physical examination of this cow, the major problem here is one of dehydration and a slight tendency to alkalosis. With relatively normal electrolytes, slight increase in rumen pH, I probably also would have instituted some fluid therapy and you could argue in this case that oral fluid therapy, if you’ve seen a colic start to relieve itself, would have been appropriate. I probably would have elected intravenously here because of the GI distention. That would have been my first line of therapy. Perhaps watching the rumen pH over time to make sure that it is correcting itself, and if it didn’t maybe return the rumen pH to normal by transplant of normal rumen fluid or an acidifier like low concentration of acetic acid, but that probably would have been a little more long term. The fluid therapy would have been what I would instituted first.

Question: With a mesenteric torsion, would not the cecum be displaced?

Dr. Smith: Most certainly it is displaced, but that doesn’t mean that it can’t be lost from palpation.

Question: How is the cecum displaced?

Dr. Smith: The cecum could be displaced centrally, cranially, or dorsally, so I don’t think that could be a definitive rule-out.

Question: Will mesenteric root torsion correct itself?

Dr. Smith: We could stand here all afternoon and discuss this back and forth. My personal feeling is “no.” I am sure cecal rotations will correct themselves. If you have, however, a true torsion of the mesentery, and I’m talking about 180° or greater, I think edema forms so rapidly that they will not correct themselves. Surgically trying to untwist them is so difficult that it is just inconceivable that they could just untwist themselves. Now that is not to say that people might not have had clinical experience and could argue otherwise. I don’t think there is any way to definitely demonstrate that. My personal feeling is “no,” but I certainly can’t say that with any degree of certainty.

Question: Where was the pulse rate recorded?

Dr. Smith: The pulse recorded was in the clinic. We will talk more about pulse rate later on. So that’s a good point, Sheila is going to discuss pulse rate relative to vagal indigestion and other conditions later on. She will have some other information which many of you will not have, perhaps.

Question: What type of pulse elevation could you expect with indigestion?

Dr. Smith: I would say there is a wide range. Indigestion can go from a normal appearing, normal pulse cow, to a cow with severe colic and with a very high pulse rate. They can be extremely painful. Generally, however, for a fairly short period, from an hour to 4-5 hours.

Question: Would calcium be of benefit in this cow?

Dr. Smith: It is difficult to evaluate that. We did not actually run a calcium test. If there was some evidence that this condition was precipitated by hypocalcemia, for example, precipitated by her being in heat, or something like that, then you might envision that calcium therapy could be of benefit.

Question: (Dr. Weeks): What about novin?

Dr. Smith: It is supposed to have some antispasmodic activity and certainly analgesic properties. I doubt it would be of very much
benefit in a cow that was in severe colic. It may have some clinical efficacy, although it is doubtful, I think, in many situations, in a cow, unless it is just a mild colic. So I would say a big question mark.

Question: Was there any follow-up on the white blood cell count?
Dr. Smith: No. We do not have a follow-up on this particular cow. This was a "budget" case so we didn't follow up on that.

Question: How did you rule out peritonitis?
Dr. Smith: We, in fact, did not rule out peritonitis on the basis of the physical examination. We did not do a peritoneal tap because of the distention. We did not feel the chance of getting clear peritoneal fluid warranted it. Obviously, because we didn't explore it we could not rule it out. The fact that the cow recovered so rapidly is good circumstantial evidence that she did not have peritonitis. The two most likely causes of peritonitis would be, of course, type 3 or type 4 abomasal ulcer disease and traumatic reticuloperitonitis. There are others, of course, as well. But in all likelihood, we didn't feel that this cow had peritonitis because of her rapid response and, in fact, she has not had a relapse in the three or four months since admission.

Question: Was there a pain response on rectal palpation?
Answer: There was no pain response on deep rectal palpation or on xiphoid pressure. This cow was hauled 35-45 miles, as I recall, in about an hour's drive.

Question: This high white cell count, could it reflect a stress reaction from the truck ride?
Dr. Smith: I think it would be very, very unusual for a three-year-old cow to show that much of a stress response just from a truck ride. So I would couple it with the truck ride, plus the stress of the colic, plus, probably, some inflammation associated with some part of the bowel. Those are just suppositions. There are a lot of unanswered questions in this cow. But isn't that the way it is always?

Dr. Whitlock: What about strychnine?
Dr. Smith: Yes, especially if it is a group of animals. That was one of Dr. Fetchel's original points in the original discussion. A very important point. Were there other animals involved? Certainly with these feed changes if it's strychnine or if it is indigestion, you would expect the possibility at least more than one animal being involved. Certainly that isn't always the case.

Case No. Two

This case involves a 6-year-old cow, which from the back, looked a bit like an apple on the left and a pear on the right. This cow had a very interesting history, having calved four months ago. She exhibited, early on, signs of traumatic reticuloperitonitis, characterized by fever and abdominal pain. This was shortly after calving. She was treated with antibiotics. Response was good for approximately one month. A month after calving, she was explored, a rumenotomy was made by the referring veterinarian. He felt there might be an abscess in the cranial part of the abdomen. However, he did not find any evidence, either externally (of the rumen), or from inside the rumen. He saw no direct evidence of penetration by wire. He had good response to the surgery. The cow did well. She was also treated with antibiotics for a period of time and seemed to respond well for another two months, at which time she showed recurrence of what he felt was peritonitis, but she had classic signs of vagal indigestion which we don't need to discuss any further. The distention on the left side was dramatic, the low distension on the right side was dramatic. This cow's temperature was 101.2°F, pulse rate 40 per minute, which many of you recognize as being one of the classic signs of vagal indigestion. Sheila will talk about that later. The respiratory rate was 20 per minute. The rumen was distended and had a lot of fluid in it. The cow had marked abdominal pain on xiphoid pressure. The PCV was 32% and the total protein concentration 9 grams per deciliter, which is high and does reflect the possibility, at least, of inflammation or abscesses within the peritoneal cavity. The white blood cell count was high normal, 64% being segmented leucocytes. Fibrinogen was still within normal limits, but a little higher than you might expect. Blood gases were normal with the exception of whatever you might interpret, although it's a venous sample, to be a respiratory acidosis. A PCO₂ of 52 millimeters of mercury is higher than average, however, it could be sampling error. With the degree of distention this cow was showing, it could also be that she had encroachment of the rumen or forestomach compartments on the diaphragm, therefore reducing the lung capacity. Rumen pH was a little higher than average. Her abdominal fluids were normal with the exception of total protein concentration of 6.1 grams per deciliter which is higher. The protein of the peritoneal fluid was high, yet the cell counts and the cellular morphology were not abnormal. We considered the following "rule-outs": traumatic reticuloperitonitis, parareticular abscess, liver abscess, and a type 3 abomasal ulcer. Type 3 abomasal ulcer is a localized peritonitis caused by a perforating ulcer. There certainly might be others. At this point we will call on Dr. Robert Whitlock.

Dr. Whitlock: Just reviewing the history, and to me, history is exceedingly important, we are looking at a 6-year-old cow weighing about 1,400 lbs. and the first evidence of illness was right after calving. It is suggested that she might have traumatic reticulitis, that she had some evidence of pain and fever and responded to antibiotics. My experience would suggest that traumatic reticulitis might be considered, but it is probably not the major diagnostic "rule-out." Why? Well, traumatic reticulitis can occur any time throughout the gestational period and it is not tightly associated with parturition or as a postpartum disease. I would not put that very high on the list. You can have exacerbations of reticulitis associated with a calving problem if she has a dystocia and straining and so on, an exacerbated abscess. I would be much more predisposed to think about an ulcer that had perforated and had local peritonitis. She had been treated for that, and she had inflammation in the anterior abdomen associated with a perforated ulcer at that time, based on probability of whatever, I'm not sure. I would put that higher on the list. So on the physical examination question, she had a recurrence of this twice. The heart rate was 48, and as Dr. Smith mentioned, I would certainly include vagal indigestion because of that. She has also marked distention of the abdomen, fluid-filled, and evidence of pain. Was there any question or any ability to discern whether this might be more localized on the right or left? It's in the central area, so therefore you can't differentiate between reticulitis and perforated ulcer with peritonitis. It could be an extension of either, so looking again at the laboratory results, I think the two most important things in the laboratory are easy to do, namely PCV and total protein. You can really learn a lot of information from that. Here's a cow that was slightly dehydrated and the PCV was up, as Dr. Smith suggested, and therefore she probably had a mild anemia of chronic disease. With that high protein, she had a chronic antigenic stimulation of weeks which would go along with the history and would reinforce my whole idea of a possibility of a perforated ulcer and local peritonitis occurring several months before. Do I
I recommend if you should "cut" or not and when? No more lab tests! But to me the critical question—I'm becoming more of an epidemiologist economist!—I would have to ask the owner the value of this cow. I mean here's a six-year-old cow that is now five months, well, what is she, four months into lactation? How much is she giving? Is she bred back? She's bred back. What was she giving before this last episode of illness? 90 lbs., 110. Good cow. How good is she?

Dr. Smith: This cow is classified 91. She is a good cow.

Dr. Whitlock: Classified 91. How much is it going to cost to replace this cow? I mean is it going to cost $1,500 or is it going to cost $10,000?

Dr. Smith: I want two things from you. What is this cow worth? But I also want you to talk about your experience appropriate to the last issue of Hoard's Dairyman.

Answer: Three issues ago. This is a 91 cow and she is milking 93 lbs. of milk when she got sick. She is 6 years old. She is probably worth between $8-$10,000. I might just say a few other things to be fair to Bob. I'd hate to be Bob up here, because I was caught in the same thing. My partner went out, operated on the cow the first time. I told him to be sure to check for an abscess in the bottom of the reticulum. He went on and he did try to do this when he did his exploratory rumenotomy. He inserted a long needle into the bottom where he thought was an abscess. He got nothing back. So I wasn't there that time and I got scared and I didn't want to do the operation. So I sent it up to let Don do it! I sent it up to the University. Do you want the whole answer now?

Dr. Smith: I want to comment on one of the things Bob talked about and that is the association of abomasal ulcer disease with early postpartum disease. I think that is a critical factor and certainly I would agree wholeheartedly with what he said. The point is now, and I will ask Bob to comment on this, do you "cut" this cow? And secondly, from what approach do you "cut" her? Do you go in on the left side or do you go into the right side? Obviously here we have two possibilities. We have a possibility that she has a recurrence of what was thought originally to be an abscess, or do we have a type 3 ulcer? Could it be, if we have an abscess, because of a type 3 ulcer? The feces were pretty sticky and of small volume, dark in color. Auscultation of the rumen showed it was very fluid and the rumen contractions were very weak, perhaps with the odd discernible. Dr. Whitlock, do we "cut" this cow, number one? Secondly, to rule out these two possibilities, which approach would be most appropriate?

Dr. Whitlock: The value of this cow may be $1,500 or even $2,500, and I think the farmer would be better off to let her go down the road and get $400 and $500. But if it's $10,000 we've got to make a decision to "cut" to see what the problem is. I'll make a decision on that in a moment. I would ask about the rectal examination of the rumen. Was the ventral sac pushed over to the right so there was an L-shaped rumen? What we're saying here is this cow had obvious ruminal or abdominal distention as seen from the back. The ventral sac was pushed over to the right indicating there was forestomach obstruction, and so on. The only other thing I would ask for would be a chloride test which we don't have. Was a chloride done? Or should we proceed without it? The heart rate is low and that kind of fits in. Remember that most perforated ulcers and bleeding ulcers occur within the first month to six weeks postpartum. The other important aspect here is that we have a six-year-old cow, and that's slightly disturbing for ulcers, because from an age point of view both perforating ulcers and bleeding ulcers occur more commonly in younger cows, 2-3-year-old cows. They can obviously occur in older ones. If you have a bleeding ulcer in an older cow, you almost always think about lymphosarcoma, associated with bleeding ulcer, especially cow 5 or 6 years old. This cow is 6 years old. It is extremely uncommon to have a perforated ulcer associated with lymphosarcoma. It does occur, but most of those ulcers bleed and do not perforate.

From a surgical point of view my only approach would be to go in on the left side, standing, and explore. You know you have adhesions there, and then make a decision at that point. She's got this massive distention. If you lay her down you greatly enhance the risk of inhalation and vomition. If you have to go in afterwards, based on what you find on exploration of the abdomen from the left and into the rumen, then you can empty it out and do something else with much less risk. So that would be my approach. I'll probably live to regret it!

Dr. Smith: No, Bob, I think you have made adjunct professor of the American College of Veterinary Surgeons with that answer! We did one other test, however, because it's an institution. Now it's something you cannot do in practice very easily, but because it's an institution we took a radiograph. The radiograph that I'm going to show is not the same cow, similar, but with one difference. This particular radiograph shows a wire. The wire was not evident on the radiograph of the cow which we had on this particular case. Perhaps, Sheila, could you point out the salient features here. We have a reticulum which is evident and you can see the magnet at the apex of the reticulum. You can see also a gas pocket up at the top with a clear line between the fluid and the gas. In this particular situation we have a wire. The cow that we're describing today had a very marked fluid/gas interface, which is classic for abscess. It did not have a wire, however, associated with it. Somehow it was not apparent at this time. In the lower left hand corner is the shadow of the elbow. At this point we know we have an abscess in the cranial ventral part of the abdomen, most likely, although not exclusively, associated with traumatic reticulitis, or traumatic pericarditis, secondary to it, not, however, totally discernible from liver abscess based on radiographic findings. Remember, of course, that this cow did not have that wire penetrating. We would find the abscess pushing the reticulum caudally and in most cases it is over there on the right side. On the right side of the reticulum we are now looking at the forestomach compartment of the abomasum on the right side. It is pushing the reticulum over to the left, so they are usually on the right side. The left surface pushes against the right surface of the reticulum, usually cranial to the omasum. We do a rumenotomy and we look down into the rumen. We're looking in the atrium of the rumen, up over the ruminal-reticular fold, at the reticulum, and we see the esophageal groove, the two lips of the esophageal groove, and we feel over to the right side and find a mass of approximately a small volleyball which is the abscess. It is a fairly fluctuant mass and it is very different from the feeling perhaps when this cow was first operated on because now two months have passed so it is much larger, perhaps more fluctuant at this point. We have an abscess which is in the perireticular area.

Is this abscess attached to the reticulum, omasum perhaps, omasal canal, or is it free of the GI tract? Is it therefore involved within the parenchyma of the liver? This particular abscess was closely adherent to the reticulum. Therefore, we have a perireticular abscess, probably associated with, although not completely distinct from, abomasal ulcer disease, probably associated with traumatic reticuloperitonitis. The method of choice in dealing with these is to go further along and put a large
bore needle through the wall of the reticulum into what you think is the abscess, attach it to a long hose, a long venous set, and aspirate. In most cases you have to use a fairly large bore needle, 14 gauge or so, in order to remove what usually is fairly thick pus. So we identified the fact that it was an abscess based on aspiration. The next step is even scarier. You go down there with a scalpel, and I can tell you the way to usually do this is to remove the scalpel blade, and that perireticular mass. At that sight with a scalpel you have to breathe deeply and just puncture right through! I won't say there is no other way, but there is no other economical way to do this particular approach. The idea is to make a hole that is large enough so the pus will drain back into the reticulum, so make a decent sized hole. If it is really thin, you can do it with your fingernails. If it is not quite so thin you can do it with a hemostat. But most times you will need a scalpel. Once you get in, make sure it is 2 or 3 fingers wide at least. In fact you can put your whole hand in some of these and drain them back. Lavage them if you will, or if it's very cheesy, you have to scoop the stuff back into the reticulum. The prognosis following this is in the neighborhood of 75-80%, provided that there are no other active lesions associated with peritonitis at the same time. This particular cow went home and did very well initially for about a week. She milked very well and they fed her excessively and she bloated and she came back into the clinic. She was decompressed, was sent home, and is doing well about two months or so after surgery. This particular cow has done well and, although we have not followed a large number of these, I would guess 75-80% of them, provided there is no other significant pathology, do all right. This condition was a failure of omasal transport, a condition that has been popularized by Dr. Whitlock, I think, or at least talked about by Dr. Whitlock for several years, failure of omasal transport due to peri-reticular abscess. Certainly the most appropriate way in my experience of handling them is by lancing through the reticulum into the abscess.

**Question:** What keeps them from reforming?

**Dr. Smith:** Once you drain them they will contract, even though an abscess is not supposed to have fibrous tissue in its wall so that it can contract. Once the distention is relieved, they seem to come down nicely in size. I have only had one experience of opening one of these cows at postmortem and seeing the actual cavity. In that case the cavity was very small, even though at the time of surgery it was very large, so I think they do inherently have enough contractility or pressure from outside to stay small. As long as the hole is big enough between the reticulum and the abscess cavity, they seem to stay empty, or if they fill up they drain back in. Beyond that, I have not had any experience to elaborate on that question.

**Question:** Have you had recurrences?

**Dr. Smith:** The chance of recurrence is much less than the chance of recurrence of liver abscesses, which seems to be significant. We'll talk about them a little later on today. So I would say the recurrence rate seems to be quite small.

**Question:** What about the value of the animal?

**Dr. Smith:** Not to belittle the point that Dr. Whitlock made regarding the value of the animal, obviously the value of the animal is extremely important, even if we're just dealing with something with a high prognosis for surgical repair like LDA. When you are dealing with a cow with a chronic disease, evidence of a condition which may or may not lend itself to surgical correction, especially vagal indigestion, then you have to consider that the prognosis and the value of the cow have to go together.

**Case No. Three**

**Dr. McGuirk:** The next case is a variation on a similar theme. This is a 7-year-old Holstein bull with vagal indigestion. On admission to our clinic, the bull weighed 2,560 lbs. He had a rather prolonged history. An excellent history was given to us of 9 months duration. The problem had mainly been one of recurring bloat, which had been dietary-related in this particular bull. That is what I mean when I say associated with indigestion, it had been associated with dietary problems. The signs, however, in the last couple of months had become more severe and they had been exhibited more frequently. The bull had lost weight. This weight is probably somewhat inflated by his abdominal distention, but he had lost weight and he had become progressively less enthusiastic about eating. During the periods prior to the time he was admitted to the clinic, he had been treated with penicillin at various intervals when he had a problem with bloat. Initially his bloat has been relieved in all these previous episodes by stomach tube quite effectively, but as time went on and his bloat was getting more frequent, passing the stomach tube was less and less effective in relieving his distention. The referring veterinarians had felt that perhaps the rumen contents were becoming more frothy.

He had numerous complete blood counts done prior to the time he was sent to us. He had, in his history, somewhat mild elevations in the white blood cell count, but always he had a neutrophil to leukocyte ratio shift. On all those previous CVCs the neutrophilia had always been about 80 percent of his total count. Rumen chlorides have been done prior to his admission to the clinic, and serum electrolytes, all of which had been normal on repeated tests. The other remarkable thing in his history was that the bull had an increased globulin fraction on a protein electrophoresis and on a regular profile. He was admitted to our clinic for evaluation based on a long history, the problem becoming more severe, and because this particular episode was relatively acute and severe. He had severe bloat the night before we saw him. The passage of a stomach tube had not relieved him. A rumenotomy or a trocharization had been done but did not relieve the pressure very well. When he was trocharized, the rumen contents did appear frothy but the rumen distention did not go away. On admission to our clinic, he had a packed cell volume of 30 percent and a total protein of 7.5 grams per deciliter. The white blood cell count was 9,300, of which 84 percent were segmented neutrophils. He had a relatively normal fibrinogen at 500 milligrams per deciliter. He had a venous pH elevation to 7.5 and as evidenced by the base excess, there was a metabolic alkalosis. Despite this fact, his electrolytes, sodium, potassium, and chloride were normal, and his rumen chloride concentration was normal. The amount of distention was quite severe. At this point, the laboratory data were really pretty typical for what we had in the bull's history. We continued to see a tendency for a mild inflammatory process, even though his total protein now was normal. He did have an elevated globulin ratio and a neutrophilia, 84 percent of his total white blood cell count. He had alkalosis. We would consider the most likely cause for this alkalosis to have been intraruminal administration of some sort of medication like carmalax boluses, mag oxide, or mag hydroxide. Generally, when we see a relatively mild alkalosis, and the electrolytes are normal, there is not a low chloride or a low potassium which might go along with forestomach sequestration of acid, then it has been administration of alkalinizing material, like carmalax boluses or magnesium, either hydroxide or
Abdominal distention was present. The ruminal contents were quite frothy and we doubted we could relieve them by stomach tube passage. The rule-outs for the condition we considered before. Normal electrolytes move us cranial to the abomasum so we are thinking of fore stomach obstruction. He had normal chloride, normal potassium, and we felt the most likely considerations were traumatic reticuloperitonitis, perhaps an abscess, like we saw in the other cow, liver abscesses is also a consideration in this particular condition. Again, we are looking at the cranial abdomen here because of the normal electrolytes, not just on this sample but on previous samples. Type 3 abomasal ulcer I have listed here but it was most likely a much lower consideration, based on our evaluation of the bull. Dr. Overly, would you be so kind as to comment on the case and how you might manage him at this point? If you would like to add any differentials to the list or move us one way or the other, we would like to decide whether to “cut” him or how to manage him in the meantime. The distention is inflating the weight, his tuber coxae are pretty prominent, his ribs are prominent, really tell us before you get to the abdominal distention.

**Question:** Was there any ruminal motility?

**Dr. McGuirk:** No. I'm sorry I did not dwell on that physical examination more. There is not much motility at all, easy to percuss fluid in the rumen, the ventral sac is large, pushed over to the right in the L-shaped rumen, which Dr. Whitlock described. No motility at all.

**Question:** What was the albumen level?

**Dr. McGuirk:** The albumen, I believe, was 3.1. My recollection of the direct ratio of albumen to globulin is hazy here. Actually I think maybe his globulins were over 5. I think all those things you said are valuable. I'll just digress here for a second since we have all mentioned heart rate and I am supposed to be saying something about heart rate. We've been puzzled on a number of cases with the relatively low heart rate associated with pretty severe conditions. Both in the amount of dehydration we find associated with cows that had pretty marked GI disturbances, cows that present with relatively distended abdomens or acute colic that Dr. Smith mentioned. Yet their heart rates are in the normal range or even in some cases low. We have read about these all along and find them associated quite frequently with vagal indigestion like we have here. We investigated this and we wanted to see the effects of simply fasting animals on the heart rate and when we fasted normal cows, the heart rate dropped about 25-30 percent in almost 90 percent of the animals. So perhaps with that drop in heart rate, we saw marked sinus arrhythmias and arrhythmias that we would associate with vagal nerve activity, increased vagal nerve activity. So I think, in retrospect, the association between slow heart rates and vagus indigestion may very well be meaningful. I think it probably broadens our approach to low heart rates and saying that cows can still be fairly sick and have low heart rates and perhaps the fact that they have just been off feed or acutely off feed for 24-48 hours is enough to drop a heart rate. I don't know why that occurs. The rumen is relatively less distended. You would think the vagus would be quieting down with less distention. But in fact that does occur and we've documented that in a number of animals, normal as well as sick. So I just throw that in there, the fact that this bull's heart rate was 84/min with his distention, probably, and with the vagal digestion you might have expected it to be lower but it probably tells you he had quite a bit of pain associated with his distention. Tim, we wrestled back and forth with the same problems you mentioned. Here is a bull with huge rumen distention. Dr. Smith was contracted to do the surgery and he did not want to operate on him with that amount of rumen distention either. So the one thing that we considered at this point was to pass a Kingman tube and try to relieve some of this frothy material in the rumen. In fact we were successful of relieving the bull of 430 lbs. of rumen content in a short period of time with a Kingman tube and that made him a much more likely candidate for surgical intervention. We also agree with you that given his long duration of problems, worsening prognosis as the problem went on, that his value as a breeding bull would probably be short without an exploratory in an attempt to correct the primary problem. Again we were about where you are. We thought the problem was in the cranial abdomen. We expected it was some inflammatory process, probably a perireticular abscess or a liver abscess were our most likely considerations at this point and we asked Dr. Smith to evaluate the bull as a surgical candidate.

**Dr. Smith:** This was a difficult case in the sense that we had a situation where we thought we could either go left side or right side and probably be right and probably be wrong at the same time. We decided to go left side. One of the factors involved in our decision to approach this from the left side is the fact that if you recall early on in the history, we did have a draining track, a draining fistula from the rumen to the left side. The importance of decompression should not be underestimated. Not only in terms of ease on the bull in terms of surgical stress, but more importantly at least, I think, especially in a bull that's standing, is the fact that you can do a reasonably good exploratory if the rumen is empty, even on a very large bull, which this one was. If in fact you went into this bull without Kingman tube dissolution of that fluid before hand, you would invariably have to do a rumenotomy before you could feel anything in the abdomen. So by virtue of this Kingman tube decompression, we were able to go into the abdomen and do a better exploratory. What we found, outside of the rumen, was a mass, an abscess in the cranial, right part of the abdomen, which in this case was not attached to the reticulum. It was involved with the left lobe of the liver. So we had a mass encompassing almost the entire left lobe of the liver. And so we are left with our decision. Basically all we did was an exploratory at that point and we made a decision to enter from a different approach the following day. That different approach, if we look at the right side of the abdomen now and envision an abscess involved within the liver in the cranial or right cranial part of the abdomen, we did a ventral approach. Now if we did a right-sided approach it would not have given us exposure to that cranial part of the abdomen. The only good exposure can be achieved through a ventral approach, and in fact we chose a ventral midline approach right up against the xiphoid. Again this is a bull which, even when he had decompressed, weighed almost a ton. In fact he weighed over a ton, 2100 lbs., I believe. He was under anesthesia for about 3½ hours in total, and yet despite the fact that we were very aware of the problems which Tim mentioned a few minutes ago with regard to muscle mass, this bull, because of good hydration during surgery, fortunately, and I say fortunately, did not have any significant problems in recovery. We were dealt with a pretty bad hand in this bull because we had a liver abscess and the way to treat those ideally, at least so far the best way we've found to treat them, is to pass a large bore tube through the right paracostal area and drain them directly to the outside, in other words form a fistula between the abscess and the body wall on the right side. Unfortunately, we were horrified to find this bull did not have any adhesions whatsoever between the liver abscess and any surface of the peritoneal cavity or the parietal
surface of the peritoneal cavity. Here we had this huge abscess, volleyball size abscess, filled with very thick pus, embedded within the liver, and embedded within the abdominal cavity. It was at least 8 inches away from any parietal peritoneal surface, obviously inaccessible for resection because you could not even envision resecting the entire liver on this bull or the abscess from the liver without obviously rupturing the abscess. Here we see the technique obviously of just making the diagnosis, that was the easy part. What we decided to do in this bull, and I'll tell you right now the outcome was not favorable, but we tried, to make an adhesion because we couldn't cause an adhesion between the abscess and the body wall. We didn't see that we could do that safely. We made a purse string adhesion and we put a purse string suture, in fact, two of them, large purse string sutures between the abscess and the reticulum. Therefore, we opposed, by virtue of a man-made adhesion, the serosal surface of the reticulum, which is the cranial surface, to the caudal surface of this abscess. Once we had those purse strings in place, we then made a small hole into the reticulum and reached down inside and cut into the abscess and let the fluid, the pus, drain back into the reticulum. Now before we did that, you might ask why we did not drain it by just suction? In fact, we spent the better part of an hour trying to do just that! We tried suction on the fluid, the pus, of this abscess by using very large, 24 French chest tubes, but the pus was just so thick that it would take too long. It would probably take 12 hours to drain all the pus. We in fact instilled saline at the same time and still it was not dissolving or becoming emulsified sufficiently to come out of the chest tubes quickly enough, so we aborted that situation and went to the anastomosis or the ostomy between the reticulum and the abscess. We ended up with a hole, which was probably our demise, which was in effect too small. As I told you in a previous case, you like to get them at least 2-3 fingers in size. We had this one about two fingers in size, but as it turned out, the bull did well for a few days and then over the ensuing week developed recurring signs of vagal indigestion and the bull expired! So the story didn't end very well.

Dr. Smith: The comment I believe here with reference to type one vagal indigestion you have a free gas bloat associated with the failure of eructation, where you have a lesion either in the caudal part of the pharyngeal area or down in the esophagus, certainly that is a valid comment. This bull had more of a frothy consistency and of course with the signs of inflammation and infection, even though we had to rule out what you're suggesting, it leans you the other direction, but certainly that's a good point. We did do a good oral examination on this bull and found no lesions.

Question: What about type one vagal indigestion?

Dr. Smith: The second common one is when you have the ones which are associated with the liver but do have adhesion to the right side of the abdominal wall. You can drain them percutaneously with a fair degree of success. The recurrence on these I think is much greater than the chance of recurrence after perireticular abscesses. And then of course this isolated case, and I hope it is an isolated case, where we have a bull with a liver abscess but with no adhesion to any parietal surface of the peritoneum. An alternative could have been perhaps, even though it wasn't attached to the parietal peritoneum, to put a tube into it and drain it into the abdomen anyway. That certainly was not a risk that we seriously considered worth taking. Are there any comments on this particular case before we go on to the next one?

Case No. Four

The next case is a calf with an infected umbilical mass. This is a one-month-old Holstein calf, 107 lbs. body weight. It was an unassisted calving. She consumed what was considered to be adequate amounts of colostrum and was normal until 36 hours before admission. She had a decreased appetite. Her temperature upon admission was 104°F, pulse rate elevated, 125, respiratory rate 38 percent with moderate degree of dehydration. She had abdominal distention, predominantly on the left side with a ping when palpated in the left side of the dorsal part of the paralumbar fossa, fairly small and not very sharp. She had diarrhea with mucosal strands which was positive for occult blood. She had a very noticeable, moist, painful, firm, swollen umbilicus. On deep palpation of the abdomen, you could feel that a stalk went from the umbilicus dorsa cranially towards the liver. The laboratory data showed that the PCV was 50 percent, which is obviously high; total protein concentration, 7.6 grams per deciliter; white blood cell count very...
high, 28,000 per microliter; 4 percent bands, so she had a marginal left shift, and a relatively high normal fibrinogen concentration, 600 milligrams per deciliter. The venous pH showed an acidemia with a high PCO₂ and a negative base excess. This calf had a metabolic acidosis and probably a respiratory acidosis as well, although we are looking at a venous sample. Her serum electrolytes were normal. We considered the following "ruleouts": diarrhea, an umbilical vein infection, which we were quite clear on from the palpation, liver abscess perhaps associated with the umbilical vein infection, and peritonitis.

**Dr. McGuirk:** Diarrhea was not noted before the calf was admitted to the clinic, but it was obvious immediately on admission. With the acidemia we have here, the acute onset of the diarrhea with mucosal shreds and blood we felt that salmonellosis was also a possibility. The calf was treated for the diarrhea and put in isolation for I believe two days prior to anything else being done. She was given antibiotics, which at that time were gentamycin and penicillin. The diarrhea resolved. It did become obvious after the second day of hospitalization that there was a pneumonic problem as well. The calf stabilized nicely but with the continuing problem of the umbilicus. We asked Dr. Smith to re-examine the calf and to consider surgery at that time because the condition had stabilized. We didn't isolate a salmonella. The feces were cultured, and on the basis of the rapid stabilization of the calf in terms of the GI problem, we ruled out a consideration of clostridium infection.

**Dr. Smith:** Those are all good comments. We are talking now about calf that has an enlarged umbilicus and which has a palpable structure; a painful, swollen, warm structure, which does exude material, which extends dorso-cranial in the abdomen towards the liver. Contrast that with a similar type structure which goes dorsal caudally into the abdomen through the urachus to the bladder. If the stalk goes caudally it is most likely to be the urachus although occasionally we get umbilical arteries. If it goes cranially, obviously it is umbilical vein. Umbilical vein infections are much less common than that. Let's talk about how to surgically manage umbilical vein stalk infection. All you have to do with these is cut them off. If you want to be safe, ligate them first. These stalks might be half a centimeter in diameter. They can be fairly large. They always have a lumen. They may have a little clot of blood. Perfectly normal. Even a calf that's 6 months old or even two years old might in fact still have a lumen and a vestige of the umbilical vein. In fact there is the fossa-formed ligament which goes from the umbilical vein up towards the liver. In some cows it is still present. So that's normal. What we are talking about however are the abnormalities. The calves that have a tract that goes from the skin towards the hiatus of the liver, at which point if we open up the liver, we find an abscess. These are the ones we are concerned about, because we cannot get along that tract and cut it off at any one point and isolate the infected from the noninfected material. In other words, you can't get above the level of the infection. The way to deal with these surgically, if in fact the infection does go to the liver, as it did in this particular calf, is to use a midline approach. You circumferentially resect the umbilicus, which has been oversewn, so that it does not exude material into the incision, and go around outside the umbilical area. You enter the abdomen and trace the umbilical vein with its associated adhesions down towards the liver. After freeing the umbilicus, if you notice that the umbilical vein does not come to abrupt end, you can't get above it, the next thing to do is marsupialize it. If you can get to the infection, obviously just ligate it and remove it. If you can't, marsupialize it by making an incision to the right side of the midline and incision and then take the whole stalk and bring it out through that small stab incision to the right side of your midline incision. Firstly, you are removing a great portion of the umbilical vein and bringing it to the outside so that you don't have as long a distance for the pus to travel. Secondly, you are removing the stenosis caused by the body wall. You have now removed about three or four inches from that umbilical vein. Next suture the umbilical vein to the stab incision, inside and out. You now have about 4 or 5 inches of umbilical vein which has been exteriorized through the right paramedian area. At this point you can then cut across and find a lumen which is filled with pus. You can flush this right down to and including the liver abscess. At this point there is good drainage of the liver abscess directly to the exterior. This is a technique that was used in this calf. The idea is, after several days or perhaps a couple of weeks of flushing, you get to a point when the skin sutures are removed and the small fistula is now ready to close over. This particular calf progressed reasonably well along this stage, although the fistula did not close over in three weeks. Despite antibiotic therapy consisting of gentamycin with penicillin or one of the penicillin-like drugs (we changed it on a couple of occasions), this calf did not respond as well as we felt she should have. She also had a recurring low grade fever. Dr. McGuirk suggested the use of a different antibiotic, which she is now going to discuss.

**Dr. McGuirk:** This calf nursed well. The diarrhea was never another problem. The lungs, over a course of about two weeks, we thought were improved dramatically. However, a fever remained at over 103°F, mainly around 103.2, for a week or so after surgery. We had cultured C. pyogenes from the umbilical vein abscess and it was suggested that we were redeveloping an abscess right under the skin. We changed at that point to rifamycin, an antibiotic that has good distribution, is able to go into body cavities well, and cross blood-brain barrier milk membranes very well. Our selection of the antibiotic was really based on two or three points. The persistence of the temperature; we still had an abscess. This particular drug penetrates PMNs and macrophages very well, and therefore you may have some increased efficacy in the abscesses. I hasten to insert at this point that its pharmacokinetics is relatively untested or poorly described in the bovine species. It is available in an oral form most easily and we put the calf on this dose twice a day, orally. In combination there is a problem with rapid development of resistance to the drug and so commonly in humans this drug is combined with another antibiotic—erythromycin, penicillin, gentamycin. We initially put her on rifampin by itself and then combined it at a later time when we sent her home with penicillin. I would also insert here that rifampin is absorbed and follows the enterohepatic route at least initially with high dose levels, and it does concentrate in the liver, where it is metabolized. An additional benefit of trying this particular drug in this calf was perhaps concentrations of high levels in the liver. Within two days of therapy I think the temperature was down to 101°F and the calf made an uneventful recovery. At that time she was sent home. Three months later the calf was doing fine.

**Question:** What about tissue residues?

**Dr. McGuirk:** I don't know any hard facts on tissue residues. Based on other species, it probably has a short half life because of its ease in movement across membranes, probably not a problem. But I don't know that in calves.

**Question:** What about oral therapy in cows?

**Dr. McGuirk:** We have given it orally to cows and again we have given it orally to cows and again
was a corynebacterium abscess in a cow with persistent mastitis. I'm not really sure of the response. Compared to humans and to rats, the availability by the oral route in horses is relatively poor, being less than 50 percent. But at that dose level, 10 milligrams per kilo, the plasma levels are adequate for most gram positive infections in horses.

**Question:** What about withdrawal time?

**Dr. McGuirk:** We did not recommend a withdrawal time. I think based on the half life a 7-day withdrawal is adequate.

**Question:** Were you surprised by the normal electrolyte when the calf came in?

**Dr. McGuirk:** Yes. The acute onset of the diarrhea may have had something to do with the normal electrolytes at the time. The calf did have some hypocalcemia the next day in spite of fluid therapy. We adjusted the postassium dose in the fluids.

**Case No. Five**

The final case for the afternoon is a 4-year-old Holstein cow, 1,550 lbs., with a history of 15 days ago having twins (we seem to have a high prevalence of twins today). The cow did have a retained placenta. Five days after calving she had metritis and an LDA. The diagnosis of LDA was made one afternoon and she was repaired the following day by a right paramedian abomasopexy. Her condition improved for three days following the surgical procedure, and then gradually her condition deteriorated. In the past week or the week prior to our seeing her, she had had almost complete anorexia or she had eaten very little. She was drinking very little water, and she was becoming progressively weaker. When she was presented to us, she was a downer. She had a temperature of 101.8°F, a pulse rate of 78/min. and a slightly elevated respiratory rate. She was unable to stand, and she was extremely depressed with moderate to marked dehydration. It is difficult to evaluate abdominal conditions with an animal down but she did have very poor rumen motility. The contractions were weak and infrequent. By rectal examination we felt the rumen was possibly slightly enlarged, but we were not really impressed with the size of the rumen at that point. The uterus was enlarged with a purulent discharge but not a very large amount. The laboratory values on admission were: a packed cell volume of 47 and a total protein of 10. We felt that again that was largely attributed to the degree of dehydration. She had a very high white cell count of 28,000, 70 percent of which were mature, segmented neutrophils. There was no bands present. There was an elevated fibrinogen level. The sodium concentration was below normal. Postassium was very low at 2.3. The chloride was 75. The cow has an acid urine. The peritoneal fluid had an elevated white count of 10,000, 76 percent of which were segmented neutrophils, no eosinophils were present, and a total protein of 4.6 grams per deciliter.

Our considerations for this cow's problem were vagal indigestion as a primary consideration, probably, perhaps related to the surgery, perhaps related to peritonitis. We have evidence of an inflammatory process going on with the white blood cell count and peritoneal fluid problems. The cow had been down and a perforating abomasal ulcer could be contributing to the peritonitis and perhaps vagal indigestion. Perhaps she had a hypocalcemia. Following surgery, she has not eaten. This hypocalcemia may be the reason she is unable to rise, for the depression and her failure to eat and drink. Metritis is a continuing problem, but we did not think it was severe enough to be causing all the metabolic disturbances we had seen and the fact that the cow was down. I should point out one thing in the history that did sort of come back to haunt us, so to speak, or something that we focused on in the history was that we did not know the cow was displaced at the time of surgery. The surgery was a difficult one according to the referring veterinarian, and there was some question about the location of the surgical abomasopexy. That was something that we kept referring back to. On admission we were not able to get any rumen contents back. We did pass a stomach tube but we did not relieve any gas or any fluid. The neutrophils were relatively well preserved. There was no evidence of sepsis in the peritoneal cavity, at least in the fluid that we obtained. That's exactly how we proceeded. We thought the electrolytes were certainly, if not the primary reason, she was down. We were worried about outflow obstruction but the fact the cow was down was not clear to us. We were not able to obtain a rumen fluid sample and the rumen at this point we didn't feel was very distended and the outflow obstruction may only be functional so that repair of the abnormal electrolyte concentrations and the acid-base status may improve her condition significantly.

In the absence of any evidence for septic peritonitis, we decided to proceed with local treatment of the uterus and to put her on amoxycillin. She was given a rich salt solution, sodium chloride, potassium chloride, with some dextrose. She received 60 liters of fluid in the next 24 hours, at which point her acid-base and her electrolytes were virtually back to normal and the cow looked much brighter. She was assisted to her feet and was able to stand. She was very weak at first but she stood for a good hour and a half and looked good at the time. She was anxious to eat some hay. She still had a low potassium, and the urine was still acid. We offered her some oral electrolyte solution, which she drank readily and we decided to see if we could manage her on oral electrolyte solution since the improvement was marked. She had not passed a large amount of feces but there were feces in the rectum and we decided to let her go over the following night with oral electrolyte solution and we would assist her to her feet in the morning if need be, but we thought she may be able to stand. She did have some peroneal nerve damage that was noticed when she stood, but with time she was able to manage well enough. The next morning the cow was down again and dehydrated. The acid-base picture had returned to the pre-admission sample. We were able to obtain rumen fluid at that point. The rumen was distended with fluid. The chloride content in the rumen at that point was over 100 millieqivalents per liter. That probably did reflect obstruction. It also did reflect the intake of oral electrolyte solution, but it certainly indicates that she didn't have normal emptying at this particular time. With her return to almost the admitting electrolytes and the worsening of the acid base status, we felt that she did have outflow obstruction with the surgery prior to when she was admitted. The cow, because of her inability to stand, at least only stand with assistance, was approached through the old incision site and in fact because of adhesions there was pyloric obstruction. That was relieved. Abomasopexy was performed again. The cow was medicated intravenously with the same electrolyte solution as when she was admitted. Twenty-four hours after surgery she was assisted to her feet and from that time on she could stand without assistance and she made a relatively uneventful recovery. She was continued on antibiotics and the uterus was treated 2 or 3 more times prior to the time she was sent home. I think she stayed in the hospital for approximately two weeks. We do have a 3 month, maybe even longer than that, follow-up on the cow, and she is doing well at home.

**Dr. Smith:** To prevent or minimize the chance of recurrence of those adhesions, there are two things that we would like to do. One
is the use of anti-inflammatories, predominantly banamine. I think it has at least a reasonable chance of helping minimize the recurrence. The other thing, probably more important, is good lavage of the abdomen or most certainly at least of the area in question. Lavage can minimize the chance of recurrence of infection within that area or if there is no infection, at least to take away the blood clots and the fibrin which have formed in that area. A good lavage with warm saline, not cold saline, and then the use of banamine for 2 or 3 days I think is certainly advisable in trying to minimize adhesions. We have used some heparine therapy in our hospital but I do not have any good personal clinical evidence to support its continued use, although I think there are people in our hospital who feel that it has some usefulness.

**Dr. McGuirk:** We did consider metritis a problem in this cow but not the whole answer. We felt that the metabolic disturbance was too severe based on what was palpated. There was tone in the uterus and we could express some purulent material, but to my way of thinking, this would have had to have been a toxic metritis and the condition of the uterus didn't really support that.

**Question:** What was the calcium level?

**Dr. McGuirk:** Calcium levels were run and they were normal. I've just given you selected data. Most of these were obtained with a complete electrolyte profile and her calcium and phosphorus were normal.

**Question:** How do you measure rumen chloride?

**Dr. McGuirk:** It is measured the same way serum chloride is measured. The important thing is to filter the sample and get all the vegetable matter spun down in the laboratory before it is run. The sample preparation is just to ensure that it is a clean sample. We consider anything over 25 miliequivalents per liter as elevated, and to indicate that there is reflux of hydrochloric acid from the abomasum.

**Question:** How did you treat the cow that had the extensive abomasal resection that had the very severe type 3 ulcer?

**Dr. Smith:** Specifically in that particular cow we obviously could not resect a good part of the abomasum without getting a lot of local contamination. We have a technique which I think works about as well as anything, at least that I've been exposed to for lavage of the abdomen, be it a cow, horse, standing, or when the animal is down. What we do is warm as much fluid as we can and we happen to have 20 liter aliquots of fluid. In this particular cow, if I remember correctly, we used 80 liters of fluid because she was dirty. No question about it, she needed extensive lavage. What we do is warm it to as close to body temperature as possible, we put it in a sterile pail and essentially pump it into the abdomen. If the cow is standing we do it by holding the incision apart. If the cow is down we just dump it in. We can usually get between 8 and 16 liters of fluid into the abdomen before it starts to overflow. Then we just let it overflow, which is not a very good technique. What happens is you just get the top coming back off. We do have a fair amount of success. We use a fairly large bore stomach tube, one half inch minimum inside diameter. We fill it up with the same sterile fluid and just start a siphon, put the deep end down below the fluid line and pump it with your hand so as to let the fibrin come into the tube through your fingertips, but you don't let the omentum or anything like that get in. You'll get a good siphon going. Try to remove as much of that fluid as possible at any one time. When you've removed 10-12 liters, then pour in another 10 or 12 liters and just repeat it somewhere between 4 and 8 times, whatever is needed, until the fluid comes out fairly clear. What you will do is remove blood clots and fibrin. You will remove a lot of blood so the fluid will initially be bloody but later on it will become much more clear. I prefer not to instill antibiotics at the end of this. I like just to use saline without antibiotics in it, and then have the cow on high level of systemic antibiotics which I feel get good peritoneal fluid levels.

**Question:** Which antibiotic do you use?

**Dr. Smith:** Amoxycillin is what we generally use. It is a payout between efficacy and cost. Five miligrams per pound. I.V. usually. Probably in six months it will be something different.