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Fifty Years at the Necropsy Table, 1955-2005: True Stories of a Different Kind

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C. L. Davis DVM Foundation

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This collection of stories is dedicated to my son,

Jon David King.

He liked hearing these stories and said you would also.

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PREFACE

These true stories are based on the thousands of necropsies performed on many species of animals during the fifty years from 1955 to 2005, mainly in the necropsy room at the Veterinary College, Cornell University, Ithaca, New York. Also, many were necropsied in various countries throughout the world, where the author spent months to years during sabbatical leaves or leaves of absence from Cornell at the request of the various countries and teaching faculties involved. The history for each case was obtained from the animal's owner, veterinary clinician, or during actual farm visits by the author and students assigned to necropsy room services during those years.

The majority of cases were subjected to a complete necropsy including the brain, and histological study of all tissues for each case as needed. Blood work or other clinical pathological studies, parasitological examination, and chemical studies were all done at the various ancillary laboratories by their personnel.

All conclusions and comments, including errors, are the author's alone if not mentioned otherwise.

It is estimated that thirty or more percent of final diagnoses can be significantly aided by an investigative farm or other involved type facility visit by the pathologist, who having performed the necropsy, can make a more definitive final diagnosis.

The necropsy would allow for the more specific direction of the visit for instance to find the partially buried, many stranded, wire cable causing the "hardware" disease outbreak; to learn that the calf was indeed pulled by extreme mechanical traction

rupturing the cow's small intestine in the pelvic canal at parturition; that the foal with fractures was exercised "outside" only at night for its eight months of life; that the suspect dogfeed was stored in a leaky roofed shed containing '1080' chemical for rat rodent control. Such histories may not be on the necropsy request form but only ascertained by these visits to the premises.

Most of the stories are grouped by species in which they are mainly seen. Several groups include other species in which a similar condition can also occur and is so stated.

Following each of the stories, WP (Web Page) number references are given from the Web site **http://w3.vet.cornell.edu/nst/** to illustrate at least some of the conditions, with pictures of the lesions as described.

I. Bovine

B-1

Double-Notched Bones

Again lucky to spend another sabbatical in Brazil and Argentina in 1995, I was able to pick up, in several veterinary facilities, four defleshed bones of about the same size and shape that had been obtained from the rumen or reticulum of cattle. All the bones measured about five to seven inches in length by two to three inches in width. They were suspected bovine metacarpal bones, each having a well-formed V-shaped notch at each end, an inch or so in depth. One of the four was a very shiny-surfaced bone while the other three were just dry bones.

The diagnosis made in all these cases from the various schools in Argentina and Brazil was botulism. The cases were thought to be related to a nutritional problem causing "pica," a craving of abnormal food such as bones, for the mineral value in them. In eating and chewing the bones, the animal incidentally releases the spores of *Clostridium botulinum* from the prolonged protection of the bone marrow. The organisms are still in the unopened bones even years after the owner of the bones has died. Sir Arnold Theiler worked on this clostridial entity problem in the late 1800s.

The diagnosis of botulism in cattle is a difficult diagnosis to make in most cases and very difficult to prove. In warm countries, it is made with more difficulty because of the often severe postmortem changes that occur quickly after death. The actual cause of death

in these cases may be in doubt, but the idea that the cattle chewed these bones to a similar size and shape and that they were formed by the cattle themselves is much more in doubt because cows do not have the bone-chewing teeth needed to make that shape of bone. The bones found did have other marks on them, probably made by rodents or other predators after the cattle's death. However, the partial answer was only discovered accidentally and recently (1996).

Several years after our return home with these odd-shaped bones, we visited a young Mexican woman, Dr. Ana Alcaraz, and her husband, Dr. Jose Peralta, at their home in Ithaca, NY. I was absolutely amazed to see four or five exact replicas of the bones lying on the rug and floor of their home, made by the chewing of their Labrador retriever. They had adopted the dog after his rejection from a Seeing Eye Dog program. The bones had been bought for the dog from the local grocery store. Originally they were slightly longer than their present size and shape, with flat, sawn ends which the dog, over a week or more, chewed down to the exact shape described above. Dr. Alcaraz put peanut butter in the cavity to encourage the dog to keep chewing on it. Amazing. The exact pathogenesis is still in question, but it seems obvious the bones found in the cattle's forestomachs were not chewed to their particular shape by the cattle themselves.

WP 264, 11258

(See Preface.)

I. Bovine

B-2

Lead Poisoning

Over the years and throughout the world, animals and man have been commonly poisoned by lead. Finding the source of the poisoning is often a major problem. My wife and I had the opportunity, during a sabbatical leave, to investigate several outbreaks of lead poisoning on different farms in Australia.

One involved calves weighing 100–300 lbs., which died suddenly, for no apparent reason. This is often part of the history. They seemed blind, but no paint was available (often a common source). The calves were bucket milk fed and kept in a small paddock. Just outside the fence were the remains of a small motor scooter battery. Inside the fence was a burned area with several broken lead battery plates that one of the farmer's sons had been melting down to make lead fishing sinkers.

This was no diagnostic problem in itself, but the lesions seen in the calf, of marked subperiosteal hemorrhages of the distal ends of the ribs, were diagnostic and new to this pathologist, being slightly different than usual.

On another Australian farm with a similar problem, the calves also had well-marked subperiosteal distal rib hemorrhages. The diagnosis was obvious, but on our visit to the farm the source was more obscure. The lady and her children had only been on the farm a short time.

We were shown the paddock. Only an empty shed was in it, plus the few remaining calves. My wife found a pile of dirt nearby and a small half-pint jar with the lid off, lying beside the jar. It turns out that the shed had been used for pottery making by the previous tenant and the jar contained ceramic glaze. It was the source for the lead when the calves licked some of the glaze from the jar. Only a tiny amount is needed, and only a small amount was missing from the jar. But more interesting is that the jar label, which I still have in my collection, has printed in large letters: SAFE FOR FOOD CONTAINERS, and directly underneath is printed CAUTION: HARMFUL IF SWALLOWED. SEE BACK PANEL, which says: CONTAINS FRITTED LEAD. How's that for confusion?

Incidentally but of significance, following several cattle cases of lead poisoning here in the States, we had an opportunity to measure the amount of lead in "nonleaded" paint and were shocked to see high levels of lead in the supposedly "lead-free" paint. In calling and speaking with Drs. H. Smythe and J. Carpenter, toxicologists at Carnegie Mellon University in Pittsburgh, I found out that paint manufacturers need lead in the paint formula for some reason, and in their legal efforts with the government, their lobbyists persuaded the government to allow the paint label to state that the product is LEAD FREE if it is below a certain level. However, it is still present and toxic.

WP 976, 979, 1025, 1050, 1112, 9001, 10561, 10562, 12055, 18818

I. Bovine

B-3

Cold Water Poisoning

In a northern state, a farmer's usual complaint is about this problem: The calves are urinating red urine (hemoglobinuria). Usually the farmer ignores the problem since it often affects the calves only mildly—although some animals die with this clinical sign.

The problem starts with the cold weather, when water pipes freeze and may not be noticed by the caretaker of the calves, especially when they have automatic waterers. The problem is noted only later, when the calves are really thirsty and bawling excessively. Then, when the frozen pipes are thawed and the calves are allowed to drink all the cold water they want, the excessive intake into the warm bowel allows absorption of the extremely cold, hypotonic water. This causes hemolysis (breakdown) of the red blood cells themselves within the blood vessels. This in turn allows the normally white tissues in the body to become icteric (jaundiced). If there is enough red blood cell breakdown, the calf may become anemic and the combination of both red cell pigment plus anemia can result in permanent damage to the renal tubular epithelium, and thus chronic nephrosis. Apparently, blood breakdown without anemia does not lead to toxic renal disease.

A similar condition may be seen in man, of red urine after a marathon, when racers who drink excessive amounts of cold water during the race have the same problem of hemoglobinuria.

WP 265, 1069

I. Bovine

B-4

Nitrofurazone Toxicity

For several years we had outbreaks of scattered, large hemorrhages and extremely pale tissues (anemia) in replacement and veal calves, and we could not determine the cause. Most did not have a fever, and many calves at a time were involved, with death as the end result.

We went to several affected farms to try to understand the problem. The calves were usually eight to ten weeks of age, in overall very good condition, but dying with severe anemia and petechial (pinpoint) or larger hemorrhages scattered throughout the body. Some of the calves had the condition externally in the gums, pale tissues of the eyes, and other external membranes including the pale skin of the ear canal, vulva, and other locations.

The farm visits were usually made on weekends, when the pathologist and residents in pathology or members of the family wanted to visit the countryside. For this reason we were able to visit one farm on a Sunday morning and talk with the farmer's wife. She was in charge of care and feeding of the 240 veal calves involved. Being invited in for coffee during the interview with the owner was great, but the only answers that helped were that the dying calves seemed to fade away, get weak, pass some blood in the feces (manure), and breathe harder and deeper when near death. Four or five were dying every day. The

only food given to the calves was milk replacer, a mineral and vitamin mix, and water. Pneumonia and diarrhea were not a factor. We could make no intelligent guesses from this first visit, but took several dead and dying calves back for necropsy purposes.

Similar lesions as described above were found in these calves. A toxic agent was still suspect. About this time, while reviewing Kodachrome transparencies given to us in the past by a former student, Dr. Frank Garry, an oversight was immediately noted. The slides were labeled erroneously as "cows" when in fact they were eight- to ten-week-old calves poisoned in Germany by nitrofurazone experimentally. They had the same changes in the tissues as the calves we were investigating in New York and Pennsylvania. Phone calls had been made to many other schools, diagnostic facilities in the U.S. and even Canada at this time, to see if anyone else had seen similar cases and to give us some help. None was forthcoming.

Dr. Garry, who donated the pictures to Cornell, had labeled them in the German fashion, whereas ours are labeled more akin to the age of the calves. Dr. Mary Smith from our Ambulatory Clinic was in Germany on sabbatical leave at the time, and I called her. She told us of Dr. Garry's work and helped us solve that problem.

The following weekend on the return visit to the farm, only the farmer husband was at the farm, while the wife was at church. The first answer given to us at his kitchen table as to what was fed the calves was: nitrofurazone for the first week, along with the other feeds. The farmer was questioned further since his wife, the week before, did not mention nitrofurazone. He added that he should know because he paid all the bills. It is illegal to use this product in the U.S. for calves, although it was (is) approved for use in chickens,

pigs, and mink. Calf diarrhea is a serious problem in veal calves. Since the product nitrofurazone is effective, it does get used.

We have not seen similar cases in the last six to eight years since then, but did try to prove its toxicity, as had been done in Germany. A local farmer allowed us to use his calves not being used for veal, and we fed a group at the dose level recommended in Germany, plus two different, higher levels for their first week of life. Those at 4X normal level had severe nervous signs after the first dose on the first day, so that level was discontinued. Those fed at 2X and 1X (normal) dose were fed for a week as recommended, and all lived with no problems, even months later.

These latter results have left some questions unanswered concerning nitrofurazone. Its exact method of action is only suggested as bone marrow suppression of megakaryocytes that produce the platelets. These are necessary for blood coagulation. At the end of the platelets' estimated half life of 56 days, this allows the calves to die at eight to ten weeks of age with severe anemia.

WP 329, 330, 10567, 19921, 20167

I. Bovine

B-5

Proliferative Pneumonia

Proliferative pneumonia, a relatively common disease in cattle and other animals, has been around for a number of years and is usually characterized by rapid, difficult breathing and death. It has a rapid onset associated in most cases with a change in feed, moving animals from a poor pasture to a new and better pasture, or in other cases by eating specific materials, such as moldy sweet potatoes with ipemeanol, and certain plants like Perilla mint or Crotalaria. The proliferation referred to is the proliferation of the type II alveolar lining cells that multiply rapidly in the affected animal. These cells cover the now naked alveolar walls, which the higher oxides of nitrogen and other inhaled chemicals have damaged following their inhalation from the eructated rumen gas. Proliferative pneumonia occurs in simple-stomach animals such as pigs and horses and even man, all of which do not have appreciable eructated gas to inhale. It is thought that they probably get the alveolar damage when the lungs eliminate the toxic materials as gases during respiration. A well-known veterinary pathologist, Dr. Charlie Barron, related to us that he suffered with this same lesion while being treated intravenously with an anti-leukemia drug like paraquat, much to his detriment.

Some cases were also seen in cattle stanchioned in barns near the bottom of silos (the usually tall, round structures holding silage near the dairy barns). Because of the silos' location, the disease was often confused with the human disease called silo filler's

disease (SFD), which is caused by inhalation of brown gas at the bottom of a newly filled silo.

In actuality, there is a close relationship between these two conditions. The acute exposure of cattle to the higher oxides of nitrogen, the brown gas at the bottom of recently filled silos, will then unite with the moisture in the pulmonary alveoli, forming weak nitric acid. This causes an acute lining cell necrosis and subsequent lining cell regeneration seen after 24 to 48 hours, giving the name of proliferative pneumonia in animals that live at least a few days. If the animals live two weeks or longer, they develop bronchiolitis obliterans fibrosis, which is the hallmark of silo filler's disease in man. In this latter condition of bronchiolitis obliterans, there is a unique proliferation of a connective tissue that bulges into the bronchioles from the weak acid damage to the bronchial mucosal surface, causing the more chronic obstructive pulmonary disease.

The first several recognized natural cases of this disease in New York State came from Spencer, NY, from a well-managed farm. On a Friday the owner, Zulo Huhta, had moved his dairy cows to a new pasture and by Sunday, five of these animals had died of respiratory distress. The necropsy on all of them, with the help of Dr. Per Andersson from Finland, revealed that almost all of the lung parenchyma was slightly firm, red to tan, with a few scattered, small, one-centimeter areas of more normal lung. The lung is best described overall as enlarged, firm, wet, and heavy. Emphysema is often seen throughout the lung and even subcutaneously, having leaked from the damaged lung as the result of the heavy breathing that occurs in these cases. However, terminal emphysema is so common in normal cattle that the underlying, firmer lung may be overlooked in these poisoning cases.

Many individual cases have occurred in cattle stanchioned at the opposite end of the barns, away from the silo. In a hallmark case here in New York, several cattle died in one barn without a standing silo, but had been fed silage that was hauled from a trench silo some distance from the barn. The hauling would have allowed any brown gas to dissipate during transit. This suggested that the material responsible was probably a product of fermentation from the flora and fauna in the rumen itself.

Around this time at Cornell, much research was being done on energy metabolism and the location of specific absorption sites of different substances from the bovine gastrointestinal tract. A large block of energy products could not be accounted for by various assays of blood flowing from the GI tract. It has also been noted that dwarf cattle commonly die of unexplained bloat and that a rumen fistula, an artificial window with a screwed-on cap put into its rumen wall in the flank, would protect the animal somewhat from the bloat proper. However, if the cap on the fistula was left off, the animal would lose weight and die of apparent starvation. Putting these bits and pieces together, it was shown that the key was the cow's process of rumination itself in which they eructate and inhale up to 80 percent of the rumen gas eructated. They use this gas as a source of energy and nutrients which would have bypassed the rumen venous return and not be measured in the experimental studies mentioned above.

The physiology department, after being told of this phenomenon and its suggested pathogenesis, proved this by putting tracheal cannulae with an inflatable ring into some sheep, whereby they breathed fresh air only through the cannula. They would not be able to inhale any eructated rumen gas since the pathway from the rumen was blocked by the cannula and inflatable ring. The idea worked perfectly. When normal sheep had an

anesthetic gas put into their rumen with a needle, the sheep went to sleep. Those sheep with the tracheal cannula which were therefore unable to breathe eructated rumen gas, did not go to sleep when the anesthetic gas was put into their rumen.

With the cause partially understood, we had to devise a treatment for the animals after they got the disease. On the suggestion by Dr. Robert Kenney (Oklahoma A&M, 1954) that atropine causes an almost absolute stoppage of fluid secretion in the mouth and probably in the lung, we treated new cases with large doses of atropine. We knew this also because a well-known clinician, Dr. Fox, suffered periodically when, as a joke, his students would often "salt" his snuff box with atropine to cause a dry mouth. It certainly worked to dry secretions. Dr. Ken McEntee and Dr. W. Hansel at Cornell had used doses up to 12 grams of atropine per cow to stop degranulation of the pituitary gland in numerous animals, without serious deleterious effects during their research. When Dr. Kenney advised its use for treatment at one gram per cow on the first and third day of the disease, it actually saved even downed cattle and many more since. The Zulo Huhta cows were the first animals treated successfully.

A delay of treatment is usually fatal, especially when the animal is down and secondary emphysema is too severe. Of course the atropine is the treatment of choice for the acute phase of the disease, with its alveolar lining cell proliferation, but for the more chronic phase, the bronchiolitis obliterans fibrosa, which is the connective tissue production phase in the smaller bronchioles, the use of corticosteroids would be the treatment of choice.

WP 345, 351, 361, 362, 2543

I. Bovine

B-6

Secondary Proliferative Pneumonia in Calves

As mentioned in the preceding story, we should note that proliferative pneumonia is a usually fatal disease of cattle and some other species. It is so named from the primary and major lesion of a proliferation of reparative lining cells of the pulmonary alveoli, the small gas exchange chambers of the lung. There are many chemicals, such as the higher oxides of nitrogen; 3-methylindole; ipomeanol, a mycotoxin produced by mold on sweet potatoes; paraquat; Perilla mint; and others that are inhaled and probably exhaled to cause the lining cell damage of the alveoli and thus stimulate their regeneration. The initial lining cells are simple-layered, flat cells allowing gas exchange with the capillaries, but the regenerating cells are more cuboidal and do not allow easy gas exchange. It has been shown that ruminants inhale up to 80 percent of eructated rumenal gas. This is somewhat logical since the lesion produced is a diffuse lesion of the lung that affects 85–95 percent of the entire lung.

In this secondary proliferative pneumonia of calves, however, the history always includes the fact that 21 days before—almost to the day—there was an outbreak of enzootic pneumonia in the calf population of the affected farm. In relation to the severity of the disease, methods and type of treatment, most calves are successfully treated with antibiotics for this initiating bacterial disease caused by *Mannheimia spp.* and other

organisms. However, in these outbreaks three weeks later a small percentage, probably 1–3 percent, show severe difficulty in breathing (dyspnea) for several hours before dying, unless treated with high doses of atropine. These calves, when they die, have the classical chronic Mannheimia pneumonia of calves in their cranial ventral lobes, but the remaining, more dorsal and caudal, are firm (meaty), wet and heavy. This is similar to what is seen in adult animals. The calves' age–breaking point of getting this entity is at about six months, when the adult form is more likely found. In these calves, which develop this proliferative entity 21 days after an outbreak of enzootic pneumonia, we call it secondary proliferative pneumonia, and do not know why it occurs since the young calves are usually not functional ruminants at the time and do not eructate. The histological lesion is that of the diffuse bilateral proliferation mentioned above in the previous story of the type II pneumocytes proliferating in the damaged pulmonary alveoli, best seen 24 hours or later after onset.

Even more bizarre is the fact that in a very low percentage of the secondary cases we see, the alveolar lining cell proliferation in only in the dorsal area, on one side of the lungs, with no lesion dorsally in the other side, except for the chronic enzootic pneumonia seen in all cases of secondary proliferative pneumonia. As with all the proliferative pneumonias, atropine is the drug of choice.

WP 358, 360, 362, 3641

I. Bovine

B-7

Toxic, Moldy Silage (Bovine Asthma)

One of our Cornell ambulatory clinicians, Dr. Susan Stehman, was the first veterinarian we know of to see these cases of dairy cows on several local farms between the Finger Lakes of New York, where there was a precipitous drop in milk production in late summer and autumn. It was seen primarily in Guernsey and Jersey milk cows, although a few Holsteins were also affected. Clinically the affected animals were coughing and had an increased respiratory rate. Fever or other signs were not apparent, only the drop in milk production.

Most of the affected animals were in stanchion barns and were manger fed in the barn. The farmer himself told us he coughed a lot when he fed silage and complained that he had to leave the barn as soon as he finished feeding the silage or green chop feed he was using. The cows were so seriously affected that several farmers were willing for us to kill these animals to establish the mechanism of damage caused by the suspect feed.

At necropsies, the lungs were enlarged with more air trapped in them than normal (emphysema), and the normally pale tissues of the airways and the lung proper were distinctly yellow to light orange, even more pronounced in Guernsey or Jersey tissues than they are normally. This yellow to orange color was more prominent in the few Holstein cows necropsied since their tissues are usually paler than the other two breeds.

Microscopically, the discolored lungs and airways were found to have a marked increase of edema and eosinophils in these tissues, and the usually small amount of smooth muscle around the airways that normally help lung function was greatly increased. The lung tissue had an increase of chronic inflammatory cells, mostly eosinophils in the stroma also. The eosinophils are one category of inflammatory white blood cells that have red-staining granules in them, giving the yellow-orange color grossly to the affected tissues. This color tends to be more green immediately after death and yellows more with time. The smooth muscle increase of airways is due to their increase in size in association with the apparent allergic response to the as yet unknown allergens in the silage that the cows and farmers inhaled. In almost all of these cases, the silage was wet—and probably moldy—red clover. There are many toxic substances associated with molds or fungi, which have been identified, but we have not identified the exact one in these cases of what is called bovine asthma. Cessation of feeding the causative feed is curative with time.

WP 340, 342, 344, 2765

I. Bovine

B-8

Dystocia-Related Intestinal Blowouts

Many cases in cows are presented for necropsy with a history of having been assisted by the owner during calving, when the cow appeared to be having trouble at this time, or the owner chose to help the cow if she was a bit slow in delivering her calf. No specific reason is usually given by the farmer as to why the cow needed their aid, except kindness on the farmer's part.

The help given in all these cases has been that the farmer would usually attach a chain or rope to the protruding calf head or legs and pull gently (?) with a tractor to help the cow expel the calf. In most instances the calf would be delivered with only minimal external help. If, to the farmer's thinking, a bigger problem existed, he would call the veterinarian for their more experienced help. The calf would usually come relatively easily, according to the farmer, by being pulled in this fashion with the tractor, and the cow would get up quickly to lick the calf and possibly get the calf to nurse. Everyone would feel more or less content, except the cow. It might then become seriously ill very quickly and die with severe abdominal discomfort and infection within the first 24 hours after the calf was pulled. Even the veterinarian would be unable to save the cow.

At the postmortem examination of the animal in these cases, nothing of diagnostic value is usually found in any other organ or tissue except in and around the pelvic birth

canal and abdominal cavity. Usually, when any cow dies around the time of calving (parturition), the cause of death is to be found associated with the uterus, birth canal itself, or the udder (mammary gland), such as with mastitis.

In the cases being described here, however, a severe septic and fecal-contaminated (bacteria-infected) pelvic canal is found, which has a loop of small intestine, often the jejunum, also present in the pelvic cavity at the time of the actual calf pulling. The whole abdominal area is severely congested and contaminated with intestinal content as a result of a ruptured segment of the entrapped loop of small intestine.

The calf itself can be pulled relatively easily during natural birth since it is inside the normal, moist, slippery birth canal of the uterus. With the loop of bowel also in the pelvic canal, though, it is absolutely in the wrong place at the wrong time. During natural parturition, portions of small intestine may be in this area when the cow is down or on its side, but when calving it would not be trapped in the pelvic cavity. In normal parturition, even if the loop of bowel was in the pelvic cavity at the start of delivery, the calf being normally expelled may cause compression of the looped bowel and elicit enough pain to stop parturition. But when being pulled by a tractor, the discomfort of the cow may be ignored so that the whole process can occur rapidly but disastrously.

Compression occurs of the two segments of the looped bowel lying in the pelvic cavity, by the calf being pulled through the birth canal (also in the pelvic cavity). The calf's bulk would push any intestinal content caudally along the two legs of the trapped intestine until it reached the loop center. At that time it would rupture the bowel and discharge the infective bowel content into the pelvic cavity, setting up the severe pelvic peritonitis seen.

The cow normally, if she happened to have a loop of bowel in the pelvic cavity, would most likely stop calving, stand up in discomfort, and move about, allowing the intestinal loop to fall forward out of the pelvic cavity and into the large abdominal cavity. Calving would then proceed without intestinal loop interference and its subsequent rupture.

This lesion is easily overlooked for the same reason in that after assisted calving and before the cow becomes critically ill, she may stand up, move around, and the affected ruptured bowel will usually fall forward into the peritoneal cavity. Then, during necropsy, if the ruptured bowel is in its relatively normal position in the abdominal cavity and if the pathologist or clinician doing the necropsy is not aware, it can easily be masked by the severe infectious reaction throughout the abdominal cavity, mixed with all the hemorrhage, inflammatory fibrin, and intestinal content. Finding fecal content in the pelvic canal in a recently freshened cow should also make the individual doing the necropsy suspicious.

WP 245, 2471, 7403

I. Bovine

B-9

Pasture-Related Arsenic Poisoning

We have seen sporadic cases of cattle that die for no obvious reason. They are often dry cows, cows that have been allowed to dry off (not milked for the last couple of months of gestation) and put out to pasture, or young stock not in milk production yet. At necropsy, sometimes no good lesions can be found to explain their deaths, and in others a very red, congested abomasum may be seen, suggesting gastritis. In others a white precipitate, suggesting a deposition of salt, may be found on the pharyngeal or nasal mucosa and larynx. This whitish precipitate was considered dietary, but now is thought to be elemental arsenic precipitated in the oral cavity from the saliva. Liver and kidney levels of arsenic are markedly elevated in these cases, allowing for the diagnosis of arsenic poisoning.

One of the major problems in these cases is to find the source of the arsenic itself. An illustrative case came from a farm pasture near New Paltz, NY, which we could actually see from the rocks of the nearby Shawangunk Mountains on climbing weekends. We could look far off into the valley below, toward the Hudson River, and imagine all the orchard properties that used to be there—of which only a few remained. Most have been abandoned now and are being made into residential communities or, in a few instances, maintained as pastures for grazing cattle.

When the orchards were there in the first half of the 1900s and earlier, the farmers often stored their lead or arsenic insecticides and other chemicals in wood and tarpaper huts or some such structure, to keep out the rain and as a place to mix them. I even remember, as a young man, mixing and spreading hundreds of pounds of arsenic for several crops—barehanded—and even mixing bales of dry asbestos powder with water by hand to make asbestos casts for hot water and steam pipes that we molded barehanded to these pipes. So much for toxic environmental hazards in the good old days. The arsenic and lead insecticides and other crop chemicals usually came in 40- to 80-lb. paper bags, and years after the orchard areas were defunct, one could often find these huts tumbled down with the roofs off and the bags dissolving, so to speak, into the ground. When the fruit trees were cut down to remake the fields into pasture or other types of farmland or housing properties, there would be patches where nothing might grow for years. Cattle and other animals would lick the spots for the salty taste of the toxic materials, which of course would be the source of the material killing the animals. It is often not easy to convince the owners of this without another farm visit. Sometimes, lead also can be diagnosed with these cases, since the combination of arsenic and lead was also used.

WP 1075, 2521, 2522, 9060, 12214

I. Bovine

B-10

Overeating Problems in Ruminants

Most large animal clinicians and pathologists recognize the common early problems associated with overeating in ruminants. These include laminitis, acute bloat, lactic acid indigestion with high blood lactate, and low rumen pH. Enterotoxemia is also included, with fluid and fibrin in the heart sac, splotchy gastrointestinal hemorrhage, and in some cases a bilateral, symmetrical focal necrosis in the brain (F.S.E.). Evidence for the overeating nature of these deaths may be found in the forestomachs, of excessive, easily digestible and absorbed concentrates, usually corn and/or other grains.

We have investigated cases of sheep eating only a handful of whole corn accidentally spilled by the farmer, which was twice what it usually ate. In other instances the cases were the result of animals eating the same, by weight, of ground corn, killing the animals when the farmer ran out of the usual whole corn. One case sent to us by Dr. William Rebhun involved over a dozen dead cows that had access to raw, mixed bread dough that could not be baked for sale because the bakery itself had just burned down. Even excess baked bread goods are capable of causing these overeating disease problems.

The explanation for such deaths is that when the easily fermentable feed is ingested, the rumen flora and fauna do the fermentation easily and more rapidly than the ruminant host can produce the highly basic pH saliva needed to neutralize the acidity of

fermentation in the rumen. This causes the various early lesions of overeating and the severe rumenitis often recognized with time after overeating occurs.

This rumenitis is easily recognized and can be seen to involve large areas of rumen mucosa, even 50 percent of the surface, especially the ventral sacs of the rumen, the omasal folds, and walls of the reticulum.

Some apparent chemical burns of the omasum and abomasum can be seen in a few cases. The burns in these forestomachs cause thickened walls with hemorrhage, necrosis, and edema that may extend to the serosa. The burn areas almost immediately become bacterial and fungal infected. The fungal infections are recognized commonly by their rounded shape, usually with several rings of discoloration suggesting "target" lesions.

The seriously affected animals may then show varying episodes of indigestion (anorexia, discomfort, lowered milk production, etc.) due to the now dysfunctional rumen.

These chronically infected forestomach lesions then may become the source of fungi and bacteria being embolic to the liver by way of the portal circulation, causing abscesses therein. Although the liver abscesses may be scattered throughout the liver, it is often noted that when a single abscess is found in the liver, it is often located near the liver hilus. When abscesses are seen in the liver, the forestomachs, especially the rumen walls, should be examined closely for healed foci as a predisposing cause for them.

Not well described elsewhere, the deleterious effects of liver abscesses can be multiple. A small abscess at the liver hilus may rupture into the vena cava, showering the lung and resulting in the cow having minimal, short-term coughing as the only sign. The shower of pus to the lung may make tiny abscesses in the lung that will usually heal at

least temporarily and quickly in an adult, otherwise healthy, cow. The liver abscess may not have been treated since it was not clinically apparent. This single abscess pocket can enlarge again, producing more purulent debris in situ. The enlargement of this liver abscess may rupture again and shower the lung for a second time into an already sensitized lung, with the result of an anaphylaxis type, pulmonary, edematous response, with the cow dying suddenly with pulmonary edema. Careful palpation of the lung should be done, for the tiny nodules of initial healed necrotic foci that can be found, along with the rupture of the liver abscess, and even the chronic rumenitis that initiated the process by overeating.

Sometimes the liver abscess may be a massive one that ruptures suddenly into the lung, causing a voluminous septic shower. This also kills the animal suddenly with pulmonary edema. If the necropsy is delayed in either of these last two entities, cut sections of the lung will give off a definite odor of sepsis (pus) from the thousands of septic emboli released as liquid pus that may not be easily seen if the pus is fluid enough.

Another sequel to a liver abscess at the vena cava is its ability to allow the infection to stimulate a bacterial infection of the endothelium (septic thrombosis) of the vein itself, near the hilus of the liver. Pieces, large or small, of this infective thrombotic material can embolize to the lung as an embolic shower and cause multiple foci of septic pneumonia. Although these emboli go into the pulmonary arterial bed and block these end arteries, they do not usually cause lung infarction, as such material would likely cause in other end artery tissues such as the kidneys, heart, etc. This is mainly because the lung has a dual blood supply.

All these problems from overeating and the production of liver abscesses may also result in a thrombus large enough to completely block the posterior vena cava, causing passive congestion of the liver (nutmeg liver).

A third important sequel to the septic thrombus in the vena cava above the abscess is that a large septic piece of debris may become free as an embolus and travel to a major branch of the pulmonary artery, blocking the flow but not necessarily causing lung infarction because of the aforementioned dual blood supply to the lung. The thrombus, being septic, can stay in place in a bronchial artery branch and enlarge as any abscess might, and cause collapse of the softer surrounding parenchymal alveolar tissue, thickening the abscess wall. This continues until the enlarging abscess encounters a more solid structure such as a bronchus or bronchiole, with its firm cartilaginous wall. The septic process continues its erosive destruction of the airway wall and with the pulmonary arterial pressure behind it, the blood can rupture into the airway.

The animal then often dies of exsanguination through the airways and nostrils. A common clinical finding in these affected animals is the marked blood spraying of the walls and manger in front of the animal, or pools of blood from their nostrils with death, and swallowed blood in the forestomachs.

WP 255, 257, 309, 310, 311, 806, 807, 20194

I. Bovine

B-11

Chronic Inflammations and Lymphoid Neoplasia

Lymphoid neoplasia (lymphosarcoma) is a serious disease in animals, as it is in man. Its cause has been proven to be a virus in many species, but not in all animals. Its association with soft tissue calcification is well known, as are other relationships, but this story deals with its unique nature associated with chronic infectious processes.

Two common chronic infectious diseases are also found in calves. These are chronic enzootic pneumonia associated with *Mannheimia spp.* and white spotted kidneys associated, it is thought, with *Escherichia coli* infection, possibly as a parturient problem.

In the cases of chronic enzootic pneumonia, the lesions are usually located in the cranioventral lung lobes. When cut, they are characteristically firm, and ooze purulent debris from the affected lobes. Some connective tissue proliferation with chronic inflammatory cells (granulation tissue) may be found in these areas, usually around the abscesses. In less severe cases, the firm areas, even with purulent debris, can resolve completely with time. In the cases of white spotted kidneys, the necrotic foci usually resolve, also leaving merely pale foci of connective tissue and chronic inflammatory cells in situ.

In both these different diseases, it has been noted in a few cases the unique finding of the neoplastic lymphoid cell accumulations in these two different affected tissue areas

and nowhere else in the animal. The lung shows the differentiation best since the neoplastic lymphoid cells are only in the affected cranioventral lung lobes, with the remaining 75–80 percent of the total lung completely unaffected with pneumonia or lymphomatosis. The lymphoid neoplasia in white spotted kidney disease is less distinct but easily recognized on histologic study.

The suggestion is given that the chronic inflammatory cell foci in the tissues involved are "turned on" in some way to stimulate or allow neoplastic development in these bacterial-infected areas.

WP 321

I. Bovine

B-12

Fatal Abomasal or Gastric Hemorrhage

Fatal abomasal or gastric hemorrhage is not a major problem but is seen now and then in cats, cattle, and reported occasionally in humans. The hemorrhage is quite remarkable, but the underlying cause is easily overlooked, even by students who are told what to look for.

The affected animals are usually sent to the diagnostic facility because of severe anemia, although some animals have not been noticed to be anemic, but were found dead.

At necropsy the main findings have been limited to the stomach of cats and the abomasum of cows. This lesion is a massive amount of blood loss, exsanguination, into the lumen of these organs which may give off a distinct "apple cider" odor. This odor is noted far more commonly in pigs with bleeding gastric ulcers. After emptying the blood from the stomach or abomasum, the mucosa must be washed to see the causative lesion. This is usually a very small, 1–3 mm slight defect in the mucosa, occupied mainly by a dilated but ruptured aneurysmal submucosal arteriole. There is no evidence for prior ulceration in this region, as is so common in pigs. The edges of the lesion are free of fibrin, necrosis, and connective tissue, as may be seen if it were a chronic ulcer.

This lesion is considered an anomalous arteriole that has ruptured.

The finding of massive hemorrhage in the abomasum is sometimes seen without total body anemia, suggesting the animal died too quickly for blood revoluminization to occur. Most cases have lived long enough to have dark red, tarry stools in the distal bowel, and become anemic.

A genetic problem is suspected, according to Japanese workers, at least in man.

WP 2996, 3060, 17019

I. Bovine

B-13

Milk Allergy in Dairy Cattle

The entity of milk allergy is practically limited to Jersey and Guernsey dairy cattle.

Dr. Gordon Campbell, professor of microbiology at Cornell University, called one day to tell me that one of his Jersey cows had died suddenly, just as he was about to milk her. He had been delayed from doing so for a short time. Dr. Campbell had been collecting these known cases for some time into an experimental herd.

These animals have an odd problem associated with the "drying off' period, while the cow is preparing for parturition and a new milking season. The animals show some dyspnea, with edema of the head, eyes, and brisket at the time of daily milking, occurring in the environment of the many noises of preparing the milking machines and buckets, etc. It is especially noticed and more serious, even fatal, when there is a prolonged delay in milking for whatever reason.

I went to do the necropsy immediately at his experimental farm, since he said the edema was going away and we both wanted to see the internal lesions as fresh as possible. Surprisingly, only barely visible edema remained in this dead cow and without Dr. Campbell's presence and history, the author doubts if he could have made a definitive diagnosis of this being a case of "milk allergy" in the cow.

It should also be noted that the Cornell dairy herd has had several Jersey or Guernsey cows that they have not been able to "dry off' for years, because of this problem. Holstein cows are more resistant to this entity.

WP 256, 2680

I. Bovine

B-14

Emaciated Calves (or Pigs)

Often, calf or pig owners bring in emaciated animals in which, at the necropsy table and histologically, we are unable to find diagnostic lesions, except depleted fat stores. These fat stores, however, have no fat in the bone marrow cavities and, instead, have a watery replacement of the fat (serous atrophy). The liver may be darker than expected.

It is so common in calves and pigs that we are often at a loss to give an etiological diagnosis, especially when the owner is adamant that he or she is feeding the animals properly.

In many instances it is not the owners' fault, but probably the fault lies with the feed. How to prove it easily and relatively quickly, to most everyone's satisfaction, is difficult.

If possible, we ask for several animals they might give us and we will feed them Cornell's recommended feed without any other therapeutic treatment. This will show it is a feeding problem of not enough feed, under conditions of cold, wetness, and exposure.

Another way has been to use groups of young rats and feed them the same feed the farmer is using, and with another similar group of rats, feed a known commercial "good calf feed." Both groups of rats are then fed for three weeks and weight comparisons are made on a line graph. This will almost always indicate the difference in feeds although it will not tell why.

These results will indicate to the farmer, pathologist, or veterinarian where to look for the correction of the problem. It will also alert the feed producer that there may be a problem with the feed itself.

In a toxicology research laboratory where the author worked for a number of years on many animal species, each new batch of feed for any group was tested in these species. This was considered important because any study with unknown feed problems would have serious consequences, including financial or time loss.

WP 1262, 1792, 10577, 11283, 11346, 18938, 19934

I. Bovine

B-15

Fertilizer Poisoning

One year we were given several replacement Holstein heifer calves to necropsy. They were being raised on a well-cared-for, high-production dairy farm in upstate New York. In the middle of the week, several animals were sick in the group of 12 to 15 heifers. The several we received were in very good nutritional condition. Excess urination was the main clinical sign.

At necropsy, all the animals we worked on had enlarged, pale kidneys as the only significant lesion to be found. An elevated BUN (blood urea nitrogen) level was marked in all.

A farm visit was arranged, and the author, along with several pathology students, traveled to the farm. The heifers were in a separate paddock, at one end of a long, freestall dairy barn. Nothing suggesting being causative was noted. Because a renal toxin was the primary consideration, the feed storage area in a separate building was singled out to find the cause. This involved a separate, well-protected storage building in which were stored all the bagged animal feeds as well as chemicals, insecticides, cleaning agents, and fertilizers mixed among them. All were neatly piled separately, including the milk replacer.

The author had the opportunity to visit several similar outbreaks in Australia, in which their animals died with the identical type renal lesions of toxic tubular necrosis. On these Australian properties, the fault was found to be that, in pouring fertilizer into the tractor or truck-drawn spreaders to fertilize the pastures, some spilled out into little piles where the spreader had been loaded.

The cattle had access to and were actually seen eating from the little piles. My boss in Australia, Dr. Ian McCausland, had actually published on this several years prior to my sabbatical leave there.

In this New York outbreak, fertilizer was also involved in a roundabout fashion. An inebriated part-time farmhand had mixed white fertilizer content from brown bags instead of white milk replacer from similar brown bags during the weekend.

WP 276, 277, 278, 10692, 11800, 12024

I. Bovine

B-16

Aortic Branch Arterial Ruptures in Adult Cattle

Over the years, many individual anemic adult cattle, usually dairy cows, have been brought in for necropsy in a very anemic condition, for no obvious reason externally. One necropsy case, of many done by this pathologist, was a cow that had died in Wisconsin and was brought to Green Bay, WI. Here a demonstration was to be given during a state veterinary meeting. By just cutting through the skin and subcutis, but not the abdominal wall serosa itself, free blood was noted inside the peritoneum, which prompted a good discussion even before finding the cause.

As the necropsy progressed, other significant lesions were not found, until most of the viscera was removed. This left the mesentery and the aorta still in the carcass, to be examined last of the abdominal viscera, as is more easily done routinely. In this entity, one usually suspects a ruptured uterine branch of the abdominal aorta or a gastrointestinal artery within 30–60 cm from the aorta itself, as was found in a uterine branch in this case. The animal had recently calved, which is also a common associated finding, but not necessarily causative. With the uterine branches involved, trauma is often expected to be a cause.

In New York we were lucky to be involved in an outbreak of similar cases occurring, remarkably, almost entirely on the eastern side of Cayuga Lake, north of Ithaca, NY.

The lesions in the vessels were quite similar and consisted of discolored and aneurysmally dilated branches of the aorta, gastrointestinal or uterine arteries, any one of which could rupture and cause the bleed out. The affected vessels are often dark and dilated, with a sharp line of demarcation proximal to the aneurysm. Paired vessels may both show the aneurysmal enlargements and discoloration of apparent vascular degeneration, although only one may be ruptured. For this reason, it is advisable also to collect samples of vascular tissue for histological examination from the other non-ruptured, paired vessels.

A few scattered cases have come from the western side farms of Cayuga Lake, but these are well scattered in time and location. With the point source of so many cases on the east side of Cayuga Lake, a farm visit was arranged with the involved clinician at the farm. The results of this discussion were that recently a feed salesman had been asked about the odd hair coat that the farmer had questioned. The salesman suggested reducing the copper level in the commercial feed used, which they did before the outbreak. It is suspected that low copper may have been the proximate cause of the vascular lesions of muscular artery rupture, since it is well known to be the cause in young pigs getting almost a 100 percent milk diet. Sulphur excess was also considered, but because this excess sulphur had been a problem for many years without such vessel problems, it was not considered the problem. Adding some copper to the diet stopped the problem.

The aneurysmal dilatation of some of the larger ruptured vessels was measured at 10–12 cm. Other, smaller vessels, were also affected with brown discoloration and smooth muscle degeneration, without appreciable dilatation or rupture.

WP 404, 405, 407, 408, 409

I. Bovine

B-17

Dermatosparaxis (Easily-Torn-Skin Disease)

While on sabbaticals in Australia and Brazil, the author had the opportunity to necropsy several cases of newborn or aborted calves which had multiple bones that had healed with severely deformed fracture sites and calluses of the ribs and long bones. It had been seen in the United States in newborn calves having hyperextension of the distal joints of the limbs, with elongated, pale, abnormally gray-white tendons and ligaments. Several had easily torn skin, even to the extent that major portions of skin pulled away from the underlying subcutaneous tissue (degloving lesion).

I was in Switzerland on another leave several years later. A great case came to the necropsy service which consisted of aborted twin calves, both with easily torn skin and many ragged tags hanging from the carcass. One of the calves even had its head pulled off from its body due to the weakness of skin, ligaments, and tendons while the carcass was being put on the necropsy table. The necropsy assistant told the pathologist on duty, the author, that it was due to decomposition, but the author considered it a relatively fresh carcass and not decomposed. Most of the calves' bones had been fractured in utero, with massive distortion of fracture sites and irregular, bony proliferation and callous formations. The ligaments and tendons were not noted on the necropsy report.

The affected Australian sheep and the sheep in Brazil did not have the bone lesions as seen in the calves, but did have marked degloving lesions with massive skin patches up to 60–80 cm hanging almost free of the subcutis. Hemorrhage was minimal. Also, in Australia many of the sheep involved were clinically apparent, and by just looking at the frontal part of the skull one could see tears or scars in the skin from apparent "head butting during play." Dorset sheep on the same farms did not have this lesion. Flat finger pressure of both hands pulling apart on the skin anywhere on these affected sheep in Australia would easily tear the skin on the Merino sheep but not the Dorset sheep. It was seen on Merino-Dorset crosses, but not as well marked.

The Merino cases caused concern in the farmers primarily at shearing time and were the reason the animals were sent to the laboratory for diagnosis. It was later shown by researchers in Australia that it was a genetic problem affecting collagen.

WP 932, 933, 934, 973, 977, 10551

I. Bovine

B-18

Rumenal Drinkers

During their early growth phase, calves are considered to be simple-stomach animals, not functional ruminants. The abomasum (or true stomach), with its acid production, utilizes the milk mainly for growth and energy, but as the calves age and their feed changes, their forestomachs (rumen, reticulum, and omasum) also mature and change, allowing the calves to develop into true ruminants.

We in the necropsy room would be presented with one or two calves out of 50 to 100 calves that may develop problems of diarrhea, bloating, and poor weight gain.

The necropsy of these specific few calves showed that a major portion of the rumen mucosa had an ulcerated and necrotic surface, with irregular edges of epithelial proliferation in small focal clumps of fibrinous debris. No other lesions were found to account for their poor condition.

The reticulum, omasum, and abomasum are usually not involved.

The cause is thought to be brought about by feeding of excessive milk or milk replacer only one time, or even just two times a day, and not allowing time for the large quantity of milk to be digested normally. The milk tended to decompose more than ferment and caused the rumenitis. In such animals, multiple feedings of lesser quantity is preventative and would allow normal digestion.

This has not been seen in beef calves since they have free access to milk all day, with less quantity ingestion at any one time, which allows natural digestion.

WP 275, 18044

I. Bovine

B-19

Secondary Inhalation Pneumonia in Cattle

This is another disease of progress in the sense that it occurs as the result of the cow being placed on her back for abomasal replacement surgery when she already has an underlying disease problem. It is probably seen all over the world. My first case was seen and necropsied in Australia and since then, several cases in the U.S.A.

The displaced abomasum for which the surgery is done is the immediate cause, which itself results usually from heavy concentrate feeding. The cause for the displacement is often associated with pregnancy, with a large pregnant uterus helping to move and hold up the rumen. This allows the abomasum to slip to the left, under the rumen and omasum. Some digestive disturbance is recognized, often with a left-sided "ping."

Its secondary nature is recognized in the cow also having a relatively common chronic bronchopneumonia (chronic enzootic pneumonia), which is often a common sequel from calfhood enzootic pneumonia that has been present for months and even several years. The chronicity of the pneumonia is often recognized by the firm, mottled cranioventral lung lobes, primarily with pus and other debris in dilated airways. By itself the chronic bronchopneumonia, even with the purulent bronchiectasis, may not be fatal for many years. However, when the animal is placed on its back for the more acute abomasal displacement surgery, the more-or-less liquid pus in the now dorsally placed ventral lobes

and airways runs gravitationally down into the previously unaffected dorsal lung lobes. This causes acute bronchopneumonia in the dorsal lobes and severe emphysema throughout. This allows the name of secondary inhalation pneumonia to the scattered foci of pneumonia in the dorsal lobes which have much emphysema. Surprisingly, to me at least, is that while many believe the displacement is caused by the heavy feeding of concentrates, it never seems to occur in dairy cattle fed only on concentrates.

In Israel, a well-known veterinary pathologist, Dr. Ted Nobel, says it is rarely seen there, if at all. In several studies in New York it has been established in the past that up to 80 percent of dairy calves do have enzootic pneumonia early in life, but apparently most survive.

Some, when subjected to being placed on their backs years later for the surgery, may succumb to this entity of secondary inhalation pneumonia.

WP 376, 377, 2757

I. Bovine

B-20

"Apple Cider"

A rather nice surprise for a pathologist is to walk into the busy necropsy room and note a distinct, somewhat sweet, odor best described as one of slightly fermented "apple cider."

There are many odors that, with experience, we learn to recognize before or during a necropsy, and this is one of the most diagnostic. The fermentation odor itself is often recognized where there is sufficient substrate, such as milk for bacteria to metabolize, as in cases of mastitis in cattle and even in nursing bats or mink with mastitis, but usually one has to be close to those sources to appreciate them.

The slightly sweet apple cider odor is usually so pronounced that anywhere in the whole room, the odor is apparent.

This is ordinarily noted in pigs, which are usually anemic, from a bleed out of a gastric ulcer. The odor is caused by the gastric hydrochloric acid digestion of the blood lost into its lumen. Probably these bleeding ulcers are the most common source of this odor in animals, but another, less common, occurrence is the exsanguination hemorrhages into the abomasum of cattle, caused by a ruptured aneurysmal submucosal arteriolar vessel. These latter vessels are considered to be anomalous vessels and, in addition to cattle, have been described in cats and in man.

WP 17073, 18605, 625

I. Bovine

B-21

Multifocal Vascular-Related Hepatic Lipidosis

Multifocal vascular-related hepatic lipidosis is usually just an incidental finding at the necropsy table in older cattle, but must be differentiated from the common, somewhat similar condition called tension lipidosis.

The common foci of tension lipidosis, not this entity, are to be found along the dorsal edges of a normal liver and are usually recognized as discrete pale or distinctly yellow irregular foci, 2–5 cm, with sharp demarcation from the more tan color of the rest of the liver. They are almost always found in association with fibrous adhesions to the normal suspensory ligaments of the liver. They are most commonly seen in cattle but have been seen scattered in a few other species. The common ones at the dorsal edges of the liver are thought to be the result of the increased tension of the capsule of the liver locally, due to the increasing weight of the liver. This causes a vascular compromise in the local areas of parenchyma, leading to the fatty change (metamorphosis).

On the other hand, specifically, the multifocal vascular-related hepatic lipidosis lesions are recognized as a few to many discrete areas of fatty change on the visceral surface of the liver in its more ventral half and not along any edges and not related to thickened suspensory ligamentous tissue.

These differentiated, fatty, 1–5 cm foci are always associated with a surface accumulation of fine strands of connective tissue with a distinct increase of thin-walled venules, most of which have a central vessel connected to the fatty parenchyma underneath. The vessel is thought to be continuous with other soft connective tissue surface structures such as the omentum and mesentery.

The actual cause is not known, but some pathologists suspect tiny pinpoint trauma from a perforating reticular foreign body such as a fine nail or wire point. Since there is no infectious process associated with the surface of these multifocal fatty areas, some doubt is raised as to infection being the cause and, in the cases seen, no wires from the reticulum have been noted.

WP 298, 299, 300, 301

I. Bovine

B-22

Post Fogging Pneumonitis

Post fogging pneumonitis is another disease of progress and is recognized in Guernsey and Jersey dairy cattle, mainly and less commonly in Holsteins, by the clinician who sends the affected animals in for necropsy. The history usually is an early summer problem, with many animals in the herd having an acute onset of coughing and a marked drop in milk production almost overnight. Fever may be present but is not considered significant.

Several acutely affected animals sent to slaughter were observed by the author. From them, the lungs were collected for photography and further examination. No other significant lesions were found in other organs or tissues. The lungs showed moderate emphysema and many, probably several dozen to 100, green, 2–10 mm foci scattered in most lobes, but mainly in the dorsal diaphragmatic lobes, in all affected animals. Slight reddening and slight collapse were also noted around these greenish areas. Within the short period of two to three hours after death, the green nature of most of the lesions had a yellow tinge and loss of their green color. The green tinge was considered due to eosinophil invasion grossly. This color change is also to be noted in other eosinophilic-responsive lesions.

Histologically these foci were necrotic foci of lung with segments of nematodes in most of them, and some degranulated eosinophils, neutrophils, and lymphocytes. The nematodes found were identified as the common bovine lungworm, *Dictyocaulus viviparus* and larvae of the same.

Talking further with the farmer suggested this was a problem associated with lungworms. The farmer described of recently using an electric insecticide fogger to fog the face and neck region of the animals just before he let them out for pasture during the day. Apparently, in the first days of summer the animals picked up the larvae, which naturally migrated to the lungs, and the migrating larvae were killed in situ by the inhaled insecticide, to cause the problem. The Guernsey and Jersey cattle were mostly affected, with the Holsteins less so, which has been noted in several other disease problems of dairy cattle.

WP 2562, 2563, 2564

I. Bovine

B-23

Farmer's Lung of Cattle

Farmer's lung of cattle is seen mainly in dairy breeds, with Guernsey and Jersey cattle more than Holsteins. This disease is recognized clinically by chronic coughing, some loss of weight, and marked loss of milk production. It is also recognized in the farmers themselves by coughing at haying time or when dry, moldy hay or silage is being fed to stanchioned cattle. The entity in man is called farmer's lung. It is sometimes confused with miliary tuberculosis radiographically.

Most cattle are sent to slaughter. We were able to follow several to slaughter in order to collect the lungs for further examination and to rule out tuberculosis.

Fever was not a common sign. At necropsy no related lesions were found in other tissues, only in lungs. These consisted of slight to moderate emphysema in all lobes. Closer examination revealed small, 1–3 mm nodules scattered throughout the lung, by palpation, mainly. Some showed up as tiny elevations on the visceral surfaces, with use of a slanted light source.

Histologically, these tiny nodules consist of a few inflammatory cells within a connective tissue capsule, surrounded by bits and pieces of small segments of *Micropolysporon faeni*. These nodules are classical as multifocal mycotic granulomas, although all nodules may not show the organism.

WP 265, 1998

I. Bovine

B-24

Renal Vein Thrombosis

One of the first cases seen of renal vein thrombosis was in an adult cow treated heavily with electrolytes for diarrheic fluid loss. Subsequent cases have been seen in other cattle and also in pigs. They are also reported well in man and rats.

The necropsy on this animal was not remarkable except for an apparently new finding in the kidneys. Well-marked, pale thrombi, mostly fibrin, were present in many arcuate vessels from pinpoint to 2–4 mm in diameter, and a few larger, mainly at the cortex-medullary junction zone.

At the time of this case, Dr. Charles G. Rickard was the acting dean and a brilliant pathologist. It was a pleasure to ask questions of him. While the lesions were being carried to the dean's office on a tray, they were shown to him rather quickly and he was told the lesions were in the veins. After a short observation he asked where they embolized from. He immediately realized his error in asking and, like the rest of us, was astounded by this apparent new finding none of us had seen before.

Since then, the relatively few cases we've seen have been in cattle both young and adults and also in piglets, all of which, like those in man and rats, are associated with diarrhea or intensive fluid therapy.

The fact that they are found in the renal veins, which are vessels flowing from the kidneys, and not in the arterial vessels which flow to the kidneys, suggests they are probably propagated from an electrolyte imbalance beginning in the efferent vasculature near the glomeruli. In only a few of the cases have infarcts been found, again suggesting that blocked arterial vessels are not a major finding in such cases.

Probably many others have seen such cases, but when the veterinary profession was asked as a group, only Dr. Wayne Crowell at the University of Georgia, Athens, Georgia, sent me pictures of several cases he had had.

WP 293, 12023, 12065

I. Bovine

B-25

Incomplete Hepatic Infarction of Cattle, another Disease of Progress

Displacement of the abomasum (the true stomach) is a disease of cattle which is in dispute as to the actual cause. Some say it is a problem on modern farms where much concentrate is fed to the milking cows, but on the other hand it is practically unknown in Israel, according to Dr. Ted Nobel, a world-renowned veterinary pathologist. In his country, almost nothing but concentrate is fed as cattle feed.

Nevertheless, when the veterinarian encounters this problem in cattle and the abomasum is out of its normal position, it must be replaced to the normal position and surgically tied down so that it won't recur with the next pregnancy. The veterinarian usually puts the cow on its back to perform an abomasopexy, which is the tying down of the abomasum to the ventral abdominal wall with the use of toggles or other type restraining sutures.

At necropsy, in most of those that die after surgery, there may be found distinct, irregular, large, several-centimeter areas of discoloration scattered in the liver which histologically are areas of fatty change and degeneration.

Further examination will often show a few to many embolic blood clots and even thrombi in the portal veins leading to these distinctly discolored areas of liver

compromise. These areas should be considered incomplete infarcts in that they are areas of vascularly compromised but not dead (necrotic) liver.

It is the venous and not the arterial blood supply which has been compromised by the partially twisted (torsed) abomasum. When the abomasum was displaced, the abomasal vessels may have had blood clots form in them by stagnation. When surgery was performed and the organ replaced to its more normal position, the stagnation blood clots in the portal vessels were set free as an embolic shower to the liver and caused the scattered foci of liver degeneration. This is another lesion of progress.

These areas of compromised, damaged liver are not ones of actual necrotic tissue, so should not be called infarcts since the arterial blood supply to the areas is usually not affected as it would be in true infarction.

WP 290, 291

I. Bovine

B-26

The Bloat Bones

Years ago, 1951–1955, while I was a student at the then Oklahoma Agricultural and Mechanical College (now Oklahoma State University), we students went to a large beef operation in El Reno, Oklahoma, to radiograph the vertebral column of some beef calves. We were looking for the butterfly-shaped malformations of the lumbar vertebrae, indicative of dwarfism in Hereford and Angus calves. Under the direction of our radiology and surgery professor, Dr. N. B. Tennille, this was a lot of work for us students, yet so much like what real veterinarians enjoyed with their outdoor mode of earning a living. We all loved it. We had many calves to do, and it took weeks, off and on, to get them all done.

Several years later at Cornell, we had to necropsy animals that were rather short of stature and could have been stunted by malnutrition or dwarfism. Of course, no breeder would want to get back a diagnosis of genetic dwarfism, and if we said it was a dwarf we had better be able to back it up to the clinician who referred the animals to us. He would be the one to tell the owner.

At Cornell we would receive Guernsey and Jersey calves now and then in addition to the beef breeds that were smaller than normal. What was definitive for the diagnosis of

dwarfism in these animals was our problem because vertebral malformations were not observed.

While at Oklahoma I took several of the dwarf Angus or Hereford calves to the Roy Lindamood farm in Broken Arrow, Oklahoma, to raise. A common cause of death in these calves was acute rumenal bloat. Being a potentially famous surgeon, I would put in a rumen fistula to allow the gas to escape. This is a hole in the left flank that opened initially into the abdominal cavity, through which we would pull the rumen itself part way to the outside, then open the rumen and suture the rumen lining to the skin. This allowed the opening to be sealed at the edges so nothing would spill into the abdominal cavity to cause peritonitis.

This was easy to do and with proper care for aseptic technique, the calves did very well, except we soon learned that the calves seemed to lose weight rapidly. This turned out to be because the rumen is really a fermentation chamber for the rumen flora and fauna to break down, by their microorganismal digestion, the grass, hay, and grain the calves were fed. In essence, cattle do not digest the cellulose grass material they eat, but the flora and fauna in the rumen do the digestion of these feeds in the ruminant. These organisms in the rumen liquor then splash over into the reticulum and omasum during the rumen cycle, into the true stomach, the abomasum. Cattle, sheep, etc., the ruminants, then digest those billions of proteinaceous creatures produced in the rumen for nutritional use.

The rumen is an efficient fermentation chamber, and it also produces heat and volatile, highly digestible fatty acids and other gases and nutrients that are quickly lost with an open rumen fistula. We soon learned to keep them closed with a screw-type cap affair in the fistula opening.

Also in the Cornell necropsy room, we learned to check any bovine ruminant for dwarfism, a la Oklahoma, that came in for necropsy with a history of bloat, although we were not sure of what exactly we were to look for.

On an auspicious necropsy day, however, we had two beef calves from one farm. They were smaller than normal, had a history of intermittent bloat, and sudden death from bloat, according to the owner. As we took the brains out in all animals during a routine necropsy, we found that from the ventral lateral sides of the cranial vault encompassing the brain, two well-marked, curved, thin pieces of bone, not seen before in other animals or probably not noted before, appeared to bulge into the cerebral hemispheres, without any other lesions in the brain except for the indentations themselves. These were the wings of the orbitosphenoid (WOS) bones and the projection of the posterior intra-occipital synchondrosis (PIOS) bones.

Cornell at that time was blessed with great anatomists in Drs. Sack, Habel, Miller, and Evans. All veterinarians except Dr. H. Evans (who happens to be a fantastic biologist with an interest in everything biological) including anatomy. He had worked with people in California at one time, describing these anomalies of the WOS and PIOS.

Terrific! A real answer at last. The understanding of the problem almost ended there, since every dwarfed Angus, Hereford, Jersey, and Guernsey breed usually have these protruding bones. Initially we called these bones the bloat bones, but we were incorrect.

But what of the bloat? No one says it is directly related to these abnormally forming bones as cause and effect, but is there any relationship? A problem exists because I had never recognized a conformationally normal, dwarfed, Holstein calf as in the other breeds listed. We have seen many Holstein calves with bloat that die with a history of chronic

bloating for no apparent cause such as change of feed, esophageal warts, or mycotic ulcers. Amazingly these chronically bloating Holstein calves would usually not have these protruding WOS or PIOS bones, although a few had these anomalous bone structures. At last we had an answer to what was associated with bloat in our calves. The Jersey and Guernsey dwarf calves also had these anomalous bones and fatal bloat.

Thus we are forced to call the bones described above as "bloat" bones, not "dwarf" bones as used earlier, realizing also that most conformational dwarfs have these abnormally formed bones and that most dwarfs die with bloat.

After obtaining a copy of the original article, "Two Projections into the Cranial Cavity Associated with Achondroplastic Dwarfism in Cattle," by W. S. Tyler, L. M. Julian, L. S. McFarland, H. E. Evans, and P. W. Gregory, Am. J. Vet. Research vol. 20, No. 77, July 1959, pp. 702–707, the author called California and talked with the only living clinician of the group, Dr. W. S. Tyler, about the original cases there, asking if their calves bloated. He gave an affirmative answer that all did have bloat, but at that time it was not considered significant by itself and was not put into the published paper. How the bones are related, however, has not been answered.

WP 258, 259, 16421

I. Bovine

B-27

Bladder Emphysema (Emphysematosa Cystica)

Emphysema simply refers to gas or air in the tissues and may be due to many things, such as tissue infected with gas-forming bacteria. Keep in mind that all bacteria do not produce gas. Emphysema may be the result of a broken rib having punctured a lung, allowing air to escape into the pleural cavity (pneumothorax). The further escape of air into the soft tissue under the skin dorsally is also called subcutaneous emphysema. It is seen in the intestinal wall of pigs, rabbits, and even the lining of the rumen in cattle or cecal walls of the horse, but without appreciable inflammation. Gas gangrene, associated with infection in man and animals, is usually caused by the microorganism *Clostridium spp.* This is a common disease associated with gas in the affected tissues but it will have the odor of sepsis (infection).

A special type of emphysema is the gas seen in the epithelial lining surface of the urinary bladder of cattle, dogs, and other species. Shortly before death, these animals were given a large amount of glucose intravenously for therapeutic reasons. It is also seen in sheep that have died from an acute infectious/toxic disease called enterotoxemia, associated with overeating. This results in an intestinal flush of *Clostridium perfringens* type D organisms, which elevates the serum glucose levels rapidly, some of which is

eliminated into the urine as glucose. This can be easily checked for at the necropsy table as an aid in diagnosis.

In both the natural disease of enterotoxemia and following glucose therapy–type cases, the glucose in the urine is thought to be absorbed directly into the bladder mucosa and broken down to gaseous CO_2 and water by tissue enzymes. In man and dogs it is also seen in cases of diabetes mellitus, again diseases associated with sugar in the urine, glucosuria. In man, it has been thought that there is or has been a cystitis present, with bacteria at fault. But this is usually not so in animals since no inflammation, acute or chronic, is usually seen in the cattle or other animals given a glucose IV. They can die with gas present in the urinary bladder mucosa in a matter of hours, without either bacteria or inflammation present.

Of course, if gas is seen in the walls of any organ, bacterial agents may be involved if infection is suspected or even if autolysis is present, but this condition described herein, emphysematosa cystica, may be so named when no infection is present and glucose is involved.

WP 283, 284, 478, 479, 2380

I. Bovine

B-28

Capsular Cysts of the Liver

Over the years, we have seen numerous examples of capsular cysts of the liver primarily in newborn calves. One case only was found in a six-month calf and one only in a newborn lamb.

These cysts are limited to the ventral free edge of the liver in all these animals and most are only 2 to 10 cm clear, fluid-filled cysts. No green color is seen in any of them nor is blood usually present. Extra-large ones are often found up to 50 cm or more, which actually displace the normal visceral organs. They are attached to the ventral edge of the liver and do not appear to have any continuity to the biliary system. They are never seen to be associated with the umbilical fissure or its structures, the umbilical veins. Some appear as a single, lined-up group of sacs hanging from the liver edge.

Their actual origin is unknown, but it is postulated they are remnants of the ventral mesentery. They do not appear to hurt the calf except by visceral compression.

WP 286, 288, 289, 7130, 7131, 7132, 7143

I. Bovine

B-29

Grass and Winter Tetany

(Vitamin A/Carotene Is Antivitamin D)

The first related cases that we had at the Cornell necropsy table were in sheep that apparently died with vague nervous signs, which was subsequently diagnosed as grass tetany. The adult nursing sheep were on lush green grass for the first time in the spring. This was similar to the many cases of transport sickness from prolonged railroad or truck transport of western sheep when allowed to graze and drink only periodically on the way to market. This was also called grass sickness in the transported sheep, and many considered it a form of hypomagnesemia, since the disease could be prevented with magnesium therapy.

Hypomagnesemia was an acceptable, simple explanation, but why on grass diets? In the late 1950s a report came out titled "Hypomagnesemia: Some Inconsistencies," in which the authors had checked the blood of normal, sick, and dead sheep in the affected flocks. They found that many of the live and healthy animals had lower blood magnesium levels than the sick or dead animals.

From this it was considered that although the disease could be arrested with magnesium treatment, this was not the only factor involved.

Sometime later in the winter of 1965, when this pathologist was the "captive" pathologist at Mellon Institute, he was called for help with some beef cattle in Blairsville, Pennsylvania. The cattle were showing nervous signs, and another had died when the clinician was calling the veterinary pathologist for advice. Apparently, only adult nursing Angus dams with calves were affected out of a relatively large herd, and several had died.

On the author's visit to the farm that evening with a friend, Dr. Richard Crawford, the cattle did show distinct ataxia, and several went down unexpectedly as we checked the herd. The necropsies showed no significant lesions and the brain was normal, both grossly and histologically, as expected. In these animals the forthcoming diagnosis was winter tetany.

Magnesium oxide was spread immediately on the feed and, as a precaution, no single person on foot or dog was allowed near the animals to cause any more nervous collapse of the animals and possible deaths. This magnesium treatment stopped the problem, as it does in similar outbreaks, but it did not answer the question of why, especially since the cattle were not on green grass.

The relationship between these cases may be understood if one considers that this beef cattle outbreak, and other outbreaks of this disease in almost all cases as mentioned above, are in cattle on farms which feed their lactating beef animals good quality alfalfa hay. These animals also are usually on well-to-do owner farms, with not just timothy or other hay types being fed. It is to be noted that alfalfa has up to 40 times the level of vitamin A than most other grasses.

Males and non-lactating females on the same farm and feeds are not affected.

Certainly magnesium is involved, but it is considered secondary as to cause since vitamin A interferes with the function of vitamin D. Vitamin D functions in the active absorption of calcium from the diet, and the calcium will aid in the absorption of magnesium and phosphorus passively, along with the calcium. However, if calcium is not actively absorbed, then the other elements will not be passively absorbed unless excess magnesium is given, especially in some soil areas with low magnesium levels.

In one large outbreak of grass tetany of cattle, a major research effort was made to determine the cause, with exhaustive analyses of tissue fluids, blood, and forage from the many involved farms and animals. The final comment made on the case was that the results failed to show any specific changes except that lush green grass associated with heavy rainfall was at fault. No analyses for vitamin A or vitamin D were made.

WP 1058

I. Bovine

B-30

Soft Tissue Mineralization

These cases usually come to the necropsy table with several different diagnoses, most commonly in renal failure, hypervitaminosis D, and cases of hypercalcemia of malignancy. (See hypercalcemia of malignancy in the dog stories of this publication.)

We also see this entity of aortic mineralization of the abdominal aorta in a few cases of Johne's disease, for which we have no explanation. A unique, similar lesion is that of aortic arch mineralization seen in some but not all cases of hyperkeratosis (X disease of cattle).

In the latter entity of X disease of cattle seen at the necropsy table, it is diagnosed because of the scurfy hair coat of the neck and ears, along with hyperkeratosis of salivary gland ducts, and cysts of the liver and renal tubules, histologically. These have been proven to be caused by highly chlorinated naphthalene being added to grease to increase its resistance to heat and friction.

After World War II, grease was a plentiful war surplus item and when used on farm machinery and subsequently licked off by cattle, it resulted in severe toxicity worldwide. Dr. Peter Olafson and others even described it originating from bread cutting machines that caused hyperkeratosis in cattle in Israel, when contaminated bread crumbs were used as cattle feed.

In the late 1950s, one of my first cases of X disease had what Dr. Olafson called a "pipe stem" aorta and discussed its not uncommon occurrence in cattle. He said that it also occurs in sheep with X disease.

We also saw this disease in a group of six- to eight-month-old calves in western Pennsylvania, which had similar gross and histological findings. We were unable to find the source of the chlorinated naphthalene until we made a farm visit with several students. We were shown a small paddock where the calves were kept. It had several flimsy old chicken or turkey shelters still present. They had not been used for these birds for years, but the rolled tar-paper roofing was still present, in very ragged condition. This was the only "odd" material present in the paddock, even after we made a diligent search for other possible sources of the grease. The grease is commonly used for farm machinery and even highway machines, which provides a good source of poison for wildlife. However, no grease was found. With the advice of Dr. Charles G. Rickard, Dr. Ken McEntee, and Dr. William Hansel, we were able to ascertain that tar paper is usually made of used rags such as dirty "grease monkey" rags. These rags are laundered over and over by rag companies that supply them to many garages. They are sold in bulk and used because of their tough fabric (not paper) content that is used to make tar paper. The greasy rag content cannot be used efficiently for white paper production. Of course, the chlorinated naphthalene is still present due to its resistance to heat and friction. Even small residual amounts of chlorinated naphthalene in the rags are very toxic, especially to the less resistant cattle.

On this subject of tissue mineralization, it is of some interest that the deposition of minerals in cases of uremia from renal failure, is often seen specifically in many different

vessels and other tissues, such as the stomach mucosa, costal pleura, vocal cords, etc. For this reason, it is noted that experimental rabbits fed 4 percent cholesterol had cholesterol deposits in these same locations, attesting to its similar nature, at least in this respect.

The aortic mineralization (pipe stem aorta) in cattle reported by Dr. Olafson and reported herein with hyperketoses (X disease) from chlorinated naphthalene, is suspected to be related to the very diagnostic finding of low levels of vitamin A in serum and liver, used specifically to diagnose the condition of hyperkeratosis or X disease. This is another association that suggests vitamin A is antivitamin D.

WP 201, 202, 203, 204, 396, 463, 464, 466, 1303, 1420, 1650, 4231, 4233, 9518, 11339

I. Bovine

B-31

Postpartum Blackleg in Cattle

Most pathologists and clinicians recognize that young cattle lying on the necropsy table with a history of one-legged lameness, fever, and sudden death, usually about 6 to 12 months of age, have not been vaccinated for Clostridium chauvoei.

The disease is called blackleg because the gross findings are that of red to almost black, swollen (edematous) skeletal muscle of the affected leg or any muscle so affected. The external findings are usually fever, swollen limb, and a gaseous subcutaneous tissue feeling in the affected muscles (crepitus), noted by palpation and on auscultation.

When the muscle is incised, a characteristic odor of sour milk (the goat acids) can be identified in the black, edematous, gaseous muscles.

The cause is easily made in these cases and is usually attributed to external trauma of some sort to the area, which possibly activates dormant organisms in the affected muscle.

There is, however, a distinct entity called post parturient blackleg in adult females which affects only the muscles of the pelvic region, around the birth canal. It has the same characters of gas, edema, and sour milk odor. It is very different, however, since it occurs almost exactly thirty days after parturition.

Probably trauma from calving is the initiating factor, but it is hard to give the reason for the thirty-day delay, except the possible necessity of time to allow spore activation.

Another unique form of this disease is when it occurs only in the heart muscle. It is certainly blackleg in its being black, edematous muscle with fibrin on the endocardium or epicardium, on one or both surfaces of the heart, and with the odor of sour milk when incised. We have been unable to incriminate trauma in any fashion as being contributory with this cardiac location.

WP 243, 244, 402, 403, 1065, 1599, 1639, 2306, 2318, 2319, 20291

I. Bovine

B-32

Quiet Lung Disease

We have seen quiet lung disease in only one outbreak. It was seen in northern Vermont and sent to us by Dr. Milton Robinson. Because it affected many dairy cattle, a farm visit was arranged with a senior veterinary student, Jay Donovan, and the clinician, Dr. Robinson.

The affected farm was a well managed, clean dairy in which cows had a severe and sudden shortage of milk production, with the death of several cows. While we were at the farm, several of the worst cases were sent to slaughter for salvage and to help make a diagnosis.

These cows and others left in the stanchion barn for our visit were all examined by the veterinarian, the student, and the pathologist. The only abnormality noted in the six cows thought to be most affected and still stanchioned, was an increased respiratory rate of 80 to 100 plus, but almost no lung sounds, normal or otherwise. No fever was noted. Thus, the name quiet lung syndrome, given clinically.

The cattle sent to slaughter were essentially normal at the time of slaughter as regards to gross lesions, except for the slight pulmonary enlargement from emphysema. This emphysema was so minimal that this pathologist would have called them normal under different conditions, if not knowing their history.

We were allowed to take the affected cows' lungs from the slaughterhouse, back to the laboratory for further examination.

Although slightly emphysematous, the lungs all were considered normal grossly, with no increased firmness palpated.

Histologically, sections of lung showed slight to moderate interstitial fibrosis scattered throughout all lobes and lobules, but more concentrated in septal walls. These had to be looked for with more care than the cursory examination usually given a grossly normal lung section. It was actually missed on the first examination on most lung sections. Only a few chronic, inflammatory cells were scattered in the lungs.

This very diffuse but slight fibrosis was considered the cause of the failure of normal rales being heard, allowing the name, as stated above, of quiet lung syndrome.

Nothing was found in the environment to account for this severe fatal entity. One suggestive factor that may have been important was that the affected cattle had recently been imported from Canada. No explanation was forthcoming.

It is possible that chemicals such as paraquat may have been available prior to shipment of the cattle from Canada.

No WP

II. Canine

C-1

Enlarged Blood Cysts in the Liver (Peliosis Hepatis)

Two German shepherd puppies were donated to Dr. Jay Georgi at Cornell University, to investigate a problem with diarrhea in the puppies. They were raised by religious monks in eastern New York, as guide dogs for the blind. The puppies were in poor condition, with severe hip dysplasia, clinically, as well as diarrhea.

A necropsy was performed on one. The findings ruled out parasites, which were clinically suspected. The other dog was kept as an experimental house pet by this author's family, with the owner's permission, in order to investigate the problems further. The end results have demonstrated far more than could have possibly been expected.

The dog was examined by Dr. Jack Geary, radiologist, and by one of Cornell's senior clinicians, Dr. Bob Kirk. Both diagnosed a case of hip dysplasia. The radiographs were classic for the disease in this six-month-old dog, according to both clinicians. The diarrhea had cleared up several weeks after arrival, and the clinical signs of hip dysplasia had decreased, according to the evaluation of the pathologist / new owner, in just those few weeks.

The dog had been fed almost exclusively fresh meat for the first week to ten days in its new home. The diarrhea stopped without anyone knowing why, but started again every time after being fed a single meal of regular commercial dry dog food. Noticing this, the

pathologist's wife had changed the dog's diet to the easily available fresh meat, but after a short while the diet was changed to a soft-moist soybean diet, a common commercial preparation without wheat, corn, or other grains, since gluten sensitivity was suspected. The dog and family lived happily for the next 11 years, when the dog died from a ruptured large vascular cyst of the liver, but without diarrhea unless given a piece of bread. Also, pathological evidence for hip dysplasia was not seen.

At necropsy there were several 6 to 10 cm vascular cysts of the liver, one of which ruptured. They were diagnosed as peliosis hepatis, the name given to exceptionally large, dilated vessels found in the liver in animals and man. Often these cystic vessels are blamed on birth control pills in women. This dog had an almost constant 11-year diet of soft soybean meal along with fresh meat. Soybean is known to contain large amounts of the suspected causative estrogenic substances, genistein and gliadin.

In castrated young bulls, similar but much smaller vascular cysts called telangiectases develop, which cause the liver to be condemned at slaughter. This results in severe economic loss to the packinghouse and the rancher. It is thought that testosterone / hormonal loss from the castration is responsible by not being present to balance the estrogens from the plants. This is somewhat akin to the prisoners of war in Germany that developed gynecomastia from their being fed so much cabbage on prison diets. In some non-continental North American locations, 80 percent of the cattle being slaughtered in their regional slaughterhouses have this lesion of telangiectasis.

Often, big cats (lions, tigers) coming to the necropsy room from zoos, especially, but even from private caretakers, have these large vascular cysts of peliosis hepatis. All may not rupture. They are degenerate lesions, not neoplastic, and they may not have been

clinically important, yet their presence may indicate an increased estrogenic content. This may also be responsible for some breeding problems in these captured wild animals. This certainly should be investigated further, since soybean is a common source of cheaper protein than the foul-smelling meat and bones left in cages to become flyblown and even more odiferous.

To partially explain the disappearance of severe clinical hip dysplasia in this dog, Duchess (the name we gave her in the family), Dr. Lennart Krook suggested that young dogs of large breeds that are fed too much high-quality feed can make osteoid, the precursor of bone, faster than the animal can mineralize the osteoid to make mature bone. This is likely to happen to dogs on a high meat diet, with its high phosphorus to calcium ratio of 40:1, causing bone weakness and deformity, as seen with hip dysplasia.

WP 9145, 9146, 7370, 7563

II. Canine

C-2

Malignancy-Caused Fibrosis of the Gastrosplenic Omentum

This relatively new finding is easy to overlook in recognizing it for what it really is. This is surprising since it is commonly seen in both dogs and cats. It is often seen as a seemingly incidental finding in the anatomical area between the stomach and the spleen, thus its name as part of the omentum. The lesion is noted as distinct 1 to 10 mm masses of soft, white, depot fat bulging from the omental surface, with minimal but definite pink, young connective tissue strands around most of the many nodules of fat in this affected portion of omentum. The maturation of the involved connective tissue contracts to help form the small 10 mm bulging discrete masses of fat.

The main omental sling itself has not been found to be involved in any of the cases, which is also not explained, but does differentiate this lesion from carcinomatosis, which appears to spread to most peritoneal surfaces and does not cause prominent bulging of depot fat as that seen in this condition.

Following up with histology in these cases of gastrosplenic fibrosis associated with malignancies, the connective tissue is seen to be very reactive in the presence of a relatively few bizarre epithelial cells of the tumor. With careful observation one can usually, but not always, find the primary source of malignancy, which is usually pancreatic or small intestinal carcinoma.

It is not considered carcinomatosis mentioned above, which is a peritoneal spread of abdominal cavity tumors, usually epithelial, from any visceral organ such as the ovary, uterus, or gastrointestinal tract. The carcinomatosis-causing tumors are a major portion of the metastatic implants, and they are definitely on the surface more often than mixed in the connective tissue, as different from the tumor cells seen in malignant fibrosis–associated metastases.

A second odd relationship between mesentery- and omental-related metastatic tumors is the relatively rare finding of some melanosarcomatous metastases that are located *only* in the very thin, almost clear, portion of the omentum (often called fenestrations, although they are not truly open windows) and mesentery. They are usually *not* in the stromal portions of the mesentery or omentum which hold the blood vessels, fat, nerves, and lymphatics. Some say the omentum, also called the "watchdog of the abdomen," has small, smooth muscle elements present that may be involved. It is still an enigma.

WP 469, 471, 500

II. Canine

C-3

Gastric Eversion into the Esophagus of Dogs

This classical lesion is a common one easily missed if the prosector is careless in gastrointestinal tract removal at necropsy.

The routine method used to remove the GI tract is to pull gingerly on the intestine, cutting the mesentery at its attachment to the bowel. When the bowel has been almost completely removed, the prosector is advised to reach in with one hand to pinch off the stomach at the cardia, to prevent content spillage, before transecting the esophagus at the esophageal hiatus of the diaphragm. The stomach's cardia is normally soft and no more dilated than the esophagus itself. In this condition of eversion, however, the act of pinching off the cardia will reveal a firm, enlarged junction between the stomach and the esophagus, which will be evidence for eversion of the stomach partway into the esophagus. This may be reduced prior to transection of the esophagus at the cardia if handled too roughly. The eversion can be recognized when the cardia is finally opened. The gastric mucosa shows a definite congestion of this everted tissue.

Vomition was noted in several of the dogs prior to necropsy, which may explain some of the cases, and was noted in one dog diagnosed with fatal parvoviral infection. Several of the cases, however, had no history of vomition. Thus, its importance is not understood.

WP 461, 462, 463, 17733, 18712

II. Canine

C-4

Prostatic Cysts in Dogs

Certainly, cysts of lymphatic origin are commonly seen on the serosal surface of the prostate, especially in cases of prostatic glandular hyperplasia. These are considered to be caused by the enlarging gland obstructing the surface lymphatics outflow from the area.

The prostate cysts herein discussed are the small, 1 to 10 ml, or larger cysts originating in the prostate itself, which bulge out into the pelvic cavity. The fluid present in them can often be squeezed from one side of the gland, to increase the pressure and bulging of the cysts on the other side of this gland.

Sometimes the cysts bulge caudally or cranially for some distance, enlarging to 50 ml or more, and even protrude from the perineum, alongside the anus. Many have a bony structure to the cyst wall, attesting to association with the acid phosphatase, of prostate secretions.

There is discussion as to their cause. It is well known that many small, 2 to 5 mm cysts develop throughout the gland as a hormonal response with clear, cystic hyperplasia. It is also seen that small cysts are often mixed with larger cystic cavities, with varying amounts of connective tissue mixed in. It is thought that these cystic glands, with time, may become infected, which causes the prostatitis often diagnosed.

It is also known that many dogs with this cystic hyperplasia of the gland are large farm dogs chained to the doghouse, often with limited access to water. These dogs tend to have diets that result in hard-formed stools which, when passing in the limited confines of the pelvic canal, will by necessity squeeze out their prostatic fluid content into the surrounding prostatic stroma and set up the chemical prostatitis seen with the greenish-black discoloration areas of these enlarged prostate glands. Many will enlarge to cause the protruding cysts with their continuous tracts from one part of the gland to another. Of course some may get secondarily infected, but far more cases at necropsy are just the irritating glandular secretions causing the inflammation seen in the enlarged glands.

WP 522, 523, 524, 525, 528, 529

II. Canine

C-5

Soft Bones (Osteoporosis) in Older Animals

In many of our aged animals, their rib bones seem to bend and break a little easier than expected without there being a specific cause found, such as renal disease or a specific causative metabolic problem. This is noted during the usual routine necropsy technique, which involves the breaking of a midthoracic rib bone after being cleaned of meat, against its curvature, to test for the general breaking strength of bones.

Not much is thought of this easy-to-bend condition since it is considered an aging change—as long as it is not an excessively easily broken bone.

Something is different, however, when excessively soft rib bones are found. This was not explained until a report came out and was reported to me by Dr. Lennart Krook in his research with Dr. Eagle and others. Young foals with an immobilized limb, for whatever reason, can lose up to 10 percent or more of the actual bone weight of the immobilized limb in about six weeks.

This information confirms (along with knowledge I gained as a research student at Oklahoma State University while going to veterinary college) that bone fracture healing takes place much faster under appositional pressure than just simple apposition alone, without pressure. This has suggested that in non-active dogs who sit or lie down most of the time, there is less stimulation for calcium deposition in bone remodeling and thus,

less dense bone is maintained in non-active dogs (and people). Serum calcium is not affected and what excess calcium is eaten is eliminated and not deposited in bone, there being no compensatory need for it. This makes the bones weaker by osteopenia (osteoporosis) and easier to bend without the fracture of older animals' rib bones.

WP 193, 317, 16615

II. Canine

C-6

Gastric Inversion into the Omental Cavity

Most pathologists are familiar with simple gastric torsion (volvulus) in usually the larger breeds, but it can occur in smaller breeds. The dogs come in acutely ill, with an enlarged, firm abdomen and acute colic. Most have a history of this occurring shortly after eating a very moist meal and jumping up to and down from a raised surface from excitement. At necropsy the spleen is often enlarged with congestion on the right side of the abdomen, whereas in the normal dog it is on the left side. The stomach is also noted to be torsed 180 degrees to either the right or left. In most instances, the clinician makes the correct diagnosis of simple gastric torsion, followed by emergency surgery with tubing or laparotomy or both. The stomach, with spleen attached, is turned by hand in the direction opposite the actual twist. The stomach is often then tacked to the abdominal wall to prevent recurrence.

We have had, however, several cases with a relatively similar history, and the animal dies relatively quickly. At necropsy the stomach and spleen are twisted out of position and partially covered by omentum and mesentery. These soft tissues cannot be replaced to normal, and the stomach cannot be returned to its normal position by simple untwisting, as noted above. In one early case, we even had the doubtful surgeon and two

anatomists try to return the displaced stomach unsuccessfully in a dead animal with this condition.

Further study has shown that the stomach, with its extremely fluid content, has actually "inverted" through the epiploic opening into the omental bursa. In this position it could not be removed without emptying the fluid from the stomach and pulling it back through the epiploic opening. The spleen in many of these cases, both the simple twist and the inversion type, has its venous vasculature compromised, which helps cause the severe passive congestion and swelling.

The pathogenesis is thought to be that a small portion of the stomach gets into the epiploic sac, which then tends to pull the rest of the stomach through the hernial ring so made, and the animal's position allows the moist ingesta to slip in gravitationally. This mechanism has not been proven, but the absolute inability to untwist and remove the stomach without first partially emptying it makes gastric inversion a serious surgical problem for correction.

WP 287, 485, 486, 11492, 18716

II. Canine

C-7

Incomplete Subaortic Stenotic Rings (All Species)

These common anomalies of the heart, usually of no significance, are seen in almost all species. In fact, they are the most common heart anomalies in all domestic animals put together, but certainly they are not often fatal. They are considered to be the result of an early congenital defect which has healed during continued in utero development.

These rings appear as transverse strands or bands of connective tissue that incompletely cross from either the left aortic cusp flap toward the right or from the right aortic cusp flap toward the left. In neither case do they complete the connection or they would then correctly be called complete subaortic stenotic rings.

Sometimes they appear as a patch of connective tissue just under the cusps, either right side or left, and in a few cases, both sides, but they do not extend all the way across.

Another attribute to these common anomalies is that they are usually small and rarely are associated with secondary vegetative endocarditis. This is quite a different sequel from the less common complete subaortic septal defect that is often involved with vegetative endocarditis, an infective process.

We also recognize that 90 percent of the cases of vegetative endocarditis are usually associated with one of three specific conditions noted as follows.

One is the presence of any type of heart anomaly that causes turbulent blood flow, allowing endocardial roughening to attract platelets and possible macrophage with viable organisms within. This condition is seen in about 30 percent of the cases. The second condition, also associated with about 30 percent of vegetative endocarditis cases, is chronic infection anywhere in the body. Chronic joint infections are another rather specific category associated with about 30 percent of vegetative endocarditis cases.

This leaves roughly 10 percent to be accounted for by specific infectious agents such as *Erysipelothrix rhusiopathiae* in pigs, *Aspergillus terreus* in German shepherd dogs, and *Streptococcus viridens* in captured opossums.

WP 279, 280, 541, 19812, 20679

II. Canine

C-8

Gastric Rugal Hyperplasia (Hypertrophy)

The first examples and most cases of this condition were noticed in dogs and subsequently have been seen in most other species, such as foals, pigs, and woodchucks (*Marmota sp.*). No known cause is apparent but some, not all, animals have had a history of vomition before death.

The lesion is not seen on the serosal surface of the stomach at the time of necropsy. It is not easily seen even when the stomach is first opened, but will be easily seen when the stomach is fully opened and the fundus fully exposed. The affected area varies from 5 to 15 cm in medium-sized dogs and may be bigger or smaller in other animals and species. The central area of the gastric fundus will show a greatly thickened mucosa with wide sulci and gyri in a round or semilunar 10 to 20 cm patch of the fundus.

Histologically, the mucosa appears slightly swollen (hypertrophy) but otherwise normal, and there may be more epithelial cells than usual (hyperplasia). There is no appreciable inflammatory response except for the area having a slight reddish zone of congestion at the edges.

There is no known cause, and a cursory attempt to prove their cause, by special stains for possible causative organisms, has been negative.

WP 452, 453, 457, 611, 2948

II. Canine

C-9

Nutmeg Liver

This name for a lesion is to suggest a liver lesion roughly having the appearance of the cut surface of the common spice nut called nutmeg. The liver will have a very mottled appearance of pale parenchyma interspersed with darker areas, often with a tree-like arborization. It can be seen on the liver's capsular surface and will more often be recognized on the liver's cut surface.

This nutmeg pattern is seen in chronic cases of obstructive hepatic outflow problems and prevents normal outflow of blood for whatever reason, usually heart problems. Even lung problems can cause blood backup through the heart, which result in the darker central vein regions of congestion in the liver.

While the lesion seen centrally usually affects the entire liver, it can also be noted that small or large regions of the liver may not be severely nutmeg even when adjacent areas do show the nutmeg pattern. This is thought to be that some areas, for instance those nearer the outflow veins, have an easier outflow pattern to follow than areas farther away from the major return vessels and thus will have more of the nutmeg appearance. With time, however, the entire liver is more uniformly and grossly affected.

The causes of the nutmeg liver are often with the right heart itself, in instances of defective AV valve function allowing blood to regurgitate back to the liver and not

forward to the lung. Chronic lung disease, diffusely affecting the lung by preventing normal blood flow through it, can also be a cause. Mucoid bronchiolitis (acute heaves) and heaves (chronic heaves) in the horse can certainly cause such a backup, as can heartworms in dogs. Vegetative endocarditis of the right heart, or even the left heart, may also be causative.

We do have cases of left heart malfunction that allows backup into the lungs, which will also prevent right heart outflow and thus a nutmeg liver.

There are certainly many other potential causes, but a major problem has been to explain a nutmeg liver in cases of having a well-defined left heart outflow problem and also having a chronic nutmeg liver *without* a heart failure lung.

One explanation is that the lungs are more resistant or the liver is less resistant than the other organ, to this passive congestion. It is thought that with time, both lung and liver would be affected, as is usually seen.

WP 105, 106, 107, 383, 1246

II. Canine

C-10

Brain-Heart Syndrome

(Neurogenic Cardiomyopathy)

Late in the 1950s, we did a necropsy of an adult sheep that had been circling for seven days and which was diagnosed correctly as listeriosis. We noted multiple, small (several mm), white, irregular foci scattered in the heart muscle of this adult sheep, but no other specific lesions (changes in the tissue due to disease) in the whole carcass. Even the brain was normal grossly. With listeriosis, a specific brain infection caused by bacteria, and even rabies (a specific viral brain infection), we may not see gross changes (lesions) in the brain. Incidentally, the listeria organism was named many years ago in honor of Lord Lister in England, who discovered the beneficial effect of phenol solutions to prevent childbed fever in humans. Phenol has been used right up to the present time to sterilize instruments and in other uses where sterility is necessary.

A common disease in lambs (not usually adults), it is associated with heart muscle damage and looks somewhat similar. It is more commonly found in the right pulmonary outflow tract of the heart and is called white muscle disease. It is a vitamin E and/or selenium responsive disease. It should not have been present (and wasn't) in this adult sheep due to both the animal's age and its overall nutritional status.

Close to this time, a mature dog came in for necropsy after being euthanized by a local clinician. The dog had a severe infection of the head, mostly in the nose area. The animal had been shot several times and disposed of as dead in the local dump. It was not dead and in some fashion made it home in such poor condition that the owner considered it best to humanely put it to sleep and have Cornell perform a necropsy. In addition to the nasal trauma and maggot lesions, we saw the heart lesion as described, somewhat similar to that of the lamb. Both showed histologically multifocal areas of myocardial muscle fiber degeneration, with minimal cell response and scattered fibers with mineralization. I was told that the bullets hitting the head was trauma enough to affect the heart, but that was considered unlikely by this pathologist.

In the early 1960s a veterans hospital patient died in Pittsburgh, and this author, while studying with the local human pathologists, had the opportunity to help autopsy the veteran, who had been in World War II. He had just died from some nonwar-related disease. When the heart was opened, many small, scattered pale foci were noted, somewhat like that in the dog and the sheep. These in this soldier were far more chronic, however, and mainly had connective tissue and chronic inflammatory cells present when looked at histologically. The human pathologist was asked to look at the heart lesions in the gross at autopsy and he told me to open the heart vessels. That had already been done, and no emboli or thrombi (blood clots, free or attached) were found. It was then suggested that the clots and thrombi had resolved. I am sure that all of us would have liked to have access to that diet. No answer was forthcoming from the autopsy so far, but as the autopsy continued, it was discovered that the soldier had a large stainless steel plate in his skull from a shrapnel wound during the war.

Putting these three cases together with many dozens since, it has been shown that in a low percentage of cases in which either the brain, spinal cord, or major nerve plexi were severely traumatized 5–10 days previously, there was a neurogenic effect on the heart, causing a vasospasm or fibrillation that resulted in damage to the muscle fibers in those areas of pale heart muscle. Since these first few cases, the degenerative changes in the heart muscle have been seen in many cases during the author's world travels. These changes were the result of trauma, infections, tumors, or other insults to the brain, spinal cord, and major nerve plexi, or even associated with acute severe pancreatitis or peritonitis. Gastric torsion in dogs, both natural and experimental, has also been a cause for neurogenic cardiomyopathy.

This syndrome was initially called the brain-heart syndrome. It is now more properly called neurogenic cardiomyopathy, to distinguish it from many other heart muscle diseases known. At first and even today, it may be likened initially to actual trauma to the heart itself by severe trauma such as automobile trauma, for instance, but since it is seen far more often with cases of prolapsed discs, cord tumors, or infections (all without trauma), one should consider neurogenic cardiomyopathy first. It occurs in just about all species, and in most instances it is not a fatal condition itself.

WP 513, 514, 227, 228, 1727

II. Canine

C-11

Linear Foreign Bodies

Most of us who see cases of animals with septic peritonitis are familiar with their being caused by string or similar long foreign bodies being swallowed. Usually this occurs only when the upper, more proximal, ends are anchored in some way higher up in the gastrointestinal tract, such as around the tongue at the frenum or even on a tooth. Sometimes a bulky foreign body that cannot pass the pyloric sphincter but has a string-like tag going down the bowel can be causative. Normal peristalsis may then continue allowing friction rubs and subsequent erosive, linear, mechanical ulcers of the intestinal wall. Some may erode deep enough to perforate the bowel and cause septic peritonitis. There have been cases of an intussusceptum itself being involved with this portion of intestine, acting as the linear foreign body eroding the intussusceptiens due to peristaltic bowel action.

The involved intestine often shows an accordion-type pleating, which the string may erode through the wall on its lesser curvature, the mesenteric attachment side of the pleated bowel.

This type of linear foreign and accordion pleating is more common in the dog and the cat than most other species.

A special linear foreign body in the colt is the adult equine ascarid, *Parascaris equorum.* Several of the cases had only a few of these parasites. In one case only a single ascarid was present, but the small intestine wall had a single linear ulcer along the lesser curvature, as did most of the cases with only a few ascarids or even many ascarids. The pathogenesis suggested is that a single parasite positions itself in the bowel longitudinally to the bowel wall, with its head at one end and its tail at the other, along the antimesenteric border side of the bowel. But the ascarid's body mass is against the lesser curvature of the bowel, where it will wear away the linear mucosal lining, to finally erode the entire wall and cause septic peritonitis. The ulcers formed in most of these have a longer defect in the mucosa itself and a shorter one through the muscle coats, and may only be a small perforation through the serosa. This mechanism suggests that the parasite maintains its position in the bowel in this fashion, allowing peristalsis to cause the mechanical damage. Some have explained the action as similar to "belly dancer" gyrations.

WP 171, 172, 173, 1043, 2916, 3105

II. Canine

C-12

Hepatic Fibrosis, Cirrhosis, and Regeneration

The common, probable misconceptions about hepatic fibrosis, cirrhosis, and regeneration are so ingrained everywhere that it almost makes the author want to shy away from putting these ideas on paper. I happen to believe in them and hope I can get others to at least consider them.

Let's begin with a dog getting hit by a car and having a severely traumatized liver, or a horse with the same type trauma, but adding that both animals miraculously lived. At one time I used to ask a student to lie on the floor of the classroom in front of the class and mimicked crushing the liver with my foot and then said that they lived. Next I asked the class what the liver would look like six months later. (This demonstration would apparently be politically incorrect today.) Both examples of liver trauma would most likely have mixed large and small, irregular areas of shrunken liver with fibrosis.

Another similar example is related to the fact that only a certain percentage of vessels flowing to the liver under normal conditions are open at any one time. These vessels open and shut periodically. Now think of an animal swallowing a small amount of a strong hepatotoxin, which is absorbed. It would reach the liver in a few minutes to do its damage quickly to only those areas that are supplied by the 60 percent of the vessels which are open at that time. German researchers proved this periodicity effect years ago, that all

portal vessels are not open all the time. Thus if they are not open at this one time and a limited amount of toxic material is absorbed quickly, only those limited areas supplied by the opened vessels will be damaged. Most will recognize this as post necrotic scarring months later, just like the irregular scarring that would have occurred in the automobile traumatized livers. Actually, both traumatic damage and the scattered locally extensive areas of toxic damage would be considered as post necrotic scarring. The fibrosis resulting in these areas should be recognized as fibrosis, a rather helter-skelter effect, since it does not affect each and every lobule of the liver to the relatively same degree as is seen with cirrhosis.

On the other hand, if someone inhales chloroform every day, thinking he's killing infectious agents in his lungs—as has been reported in the literature—this liver toxin, or other liver toxins, such as carbon tetrachloride or aflatoxin, given in small doses over a long time, will affect each and every lobule to the relatively same degree of the entire liver. The toxin would end up later as central or periportal fibrosis called cirrhosis, according to where such agents cause their damage. The same lesion in a dog or a cow can be produced by chronic heart disease, with the anoxic change of chronic heart disease causing central damage. When each and every lobule is damaged more or less centrally or peripherally, according to the chemical effect, and is replaced by connective tissue, the result should be called cirrhosis, not fibrosis, since the connective tissue would be seen rather uniformly throughout the entire liver.

Of special note here is that in none of these examples would hepatic regeneration necessarily be involved if the hepatic damage was not enough at any one time to reach the threshold of regeneration. In man and rats, regeneration has been estimated in

literature reports at about 20 percent. Of course, severe trauma or serious poisoning could occur, and with more than 20 percent damage the threshold for the man and the rat could be reached to get the stimulation for regeneration—providing the animal lived.

Regarding fibrosis of the liver and cirrhosis, I would think it best to review these terms, knowing that with post necrotic scarring (no matter what the cause—trauma or acute partial toxicity), the resulting scars of connective tissue will be scattered throughout the remaining organ, with the scarring as evidence of prior damage in those scattered areas only. Following hepatic trauma or single-dose effect of a large amount of rapidly absorbed liver toxin, the scarring will be "helter-skelter," leaving large areas of hundreds to thousands of hepatic lobules essentially normal and without any evidence of connective tissue production. The only connective tissue will be in the actual area of prior damage caused by the trauma or hepatotoxin.

Cirrhosis, on the other hand, is the increase of connective tissue in the same location of each and every lobule to the relatively same degree and may be either in the central or peripheral portion of the lobule, depending on the insulting agent. For instance, chronic heart failure in the animals as explained above, will cause primarily central degeneration and subsequent central cirrhosis, also called cardiac cirrhosis, while aflatoxin will cause peripheral damage in dogs and subsequent portal cirrhosis. Of course, both types of fibrosis would be evidence for absolute fibrosis since it is newly formed connective tissue. This is to be differentiated from the fibrosis seen in Doberman pinscher dogs with liver atrophy, which is a loss of parenchymal liver cord cells, leaving only the original, essentially normal stroma in place, and is called relative fibrosis for obvious reasons.

There is no absolute increase of connective tissue, but only the original connective tissue is more condensed.

In both of these types of fibrosis, fibrosis and cirrhosis with chronic continuous liver damage, the threshold for liver regeneration may not be reached in a single, relatively short time, so nodules of regeneration may not be seen. If enough damage is caused to reach threshold and thus stimulate regeneration, then nodules of regeneration may be seen in the damaged liver. Thus, regenerative nodules should not be a criterion for either type of fibrosis without consideration of the threshold factor. The term "fibrosis" is generally thought to be associated with connective tissue anywhere in the body that is stimulated to be produced by fibroblasts and is also the generic term causing concern with the use of cirrhosis and fibrosis as described above.

WP 107, 170, 701, 2019, 7042

II. Canine

C-13

Anesthetic Gas Machine Lung

This entity can occur in goats, horses, and other species, but it is most commonly seen in dogs. Often, the case we see is a dog that has been ovariectomized (spayed) shortly before death by a local veterinary surgeon using gas anesthesia, with no apparent problems encountered. The animal would be allowed to waken after cessation of gas use, and the animal may actually start to stand up apparently normal, only to be found dead a short time later.

Certainly, a suture may have slipped or a hemophiliac state was not noted prior to surgery or the animal inhaled some ingesta. These and other problems must be ruled out before this titled diagnosis can be used properly.

In cases as this one turned out to be, the major finding usually is a soft but markedly reduced size of lung bilaterally, with no negative pleural pressure. At first it may suggest pneumothorax with a collapsed lung, but no tears or other lesions suggestive of such were found and, clinically, respiratory distress was not seen. At first glance at necropsy, some even thought the tongue and heart were too big for the size of the animal.

All the cases we have seen have been anesthetized with gas and not with barbiturates. Following surgery the animals are often tightly bound around the abdomen or have been given muscle relaxants. With these conditions that are usually seen, we believe that the

common, fully metabolizable gas anesthetics used today may completely replace the 79 percent nonmetabolizable nitrogen in the residual air space of the lung proper. Then, following the shutting down of the metabolizable anesthetic gas machines at the end of surgery, the residual air spaces shrink with the normal elastic recoil of the lung if the animal is not bagged with normal air immediately (with its 79 percent nitrogen). The continued uptake and metabolism of the gas from the alveoli will allow the lungs to collapse further, causing suffocation and death without filling the alveoli with normal air.

WP 419, 2627, 2630

II. Canine

C-14

So-called Nodular Hyperplasia

(Regeneration vs. Neoplasia)

In cases of thyroid damage in young dogs, for instance from accidental choking or partial strangulation (another too graphic demonstration for today's young students), the thyroids may be traumatically damaged. And what would one see in them six months later if the animals lived? One would see scars (connective tissue) in the thyroid and probably large nodules of compensatory regeneration. But make note that there is evidence in almost all cases, of prior damage to the organ by the presence of connective tissue (fibrosis). Masses in the thyroid in tumor-aged animals without connective tissue present should be considered more likely tumor, and not the hyperplasia of regeneration.

The same holds true for the liver. If you get nodules of compensatory regeneration (hyperplasia), there is usually evidence of prior damage in the form of local connective tissue scarring of the area involved. So what does one call the individual or even multiple nodules in tumor-aged animals without any evidence of prior damage? They should be called neoplastic (hepatic adenomas or hepatomas), but absolutely not nodular hyperplasia. These tumors are usually benign in the dog and the cat, but can attain great size and rupture to kill the animal by exsanguination. However, they still represent neoplasia, not regeneration. Of course you can have malignant hepatocellular carcinomas,

the malignant variation of benign tumors, but these are quite rare in the dog and the cat. During a sabbatical year in Argentina and Brazil, I did many old dog necropsies there and was surprised to find not a single case of hepatoma, as compared to 30–40 percent incidence or higher in older dogs in New York.

Those masses in a young or old animal's thyroid with lots of scar tissue also represent nodular regeneration, but those in older animals without evidence of prior damage are tumors such as liver masses. In the thyroid or other paired organs, one can certainly find tumors (neoplasms), but in most instances there will be no extra connective tissue present. It is certainly possible to find both types of masses and connective tissue in animals with previous organ trauma, and caution is advised in interpretation.

In regards to regeneration, one can take out the thyroid of one side in rats or dogs, as we did experimentally, and the gland on the opposite side will enlarge. When students were asked if it would be a nodular or diffuse regeneration, it was a problem for them initially, but they got the point and answered correctly: diffuse in the remaining gland. The same is what happens with other paired glands. Even in the liver of the woodchuck, another good example, which can have a 50 percent hepatectomy done by a two-second transection of its tiny hilar attachment (half the volume of its liver), the other half enlarges diffusely, never nodularly, if it did not have prior damage to the one side left in the body.

WP 107, 7612, 7613

II. Canine

C-15

Soft Foreign Bodies of the Small Intestine

Certainly, veterinary pathologists and clinicians see cases where animals eat indigestible things that are not normal components of their diets. These we call "foreign bodies." Most of them are usually of the hard types, such as stones, bones, nails, bolts, pieces of wood, and even parts of machinery which they can swallow. Even whole apples and hard nuts like hickory nuts or peach pits can be causes for animal deaths when the "foreign body" size or shape, or both, are not compatible to passage down the gastrointestinal tract, after leaving the stomach or even the throat.

The cases described in this story are different in that these "foreign bodies" are unique by their being soft and elastic.

The dog first seen with this entity was a mature, small, mixed-breed dog with a history of not wanting to eat, vomition, losing weight, attempting to drink but not doing more than licking at the water, and showing abdominal pain for a few days before it was taken to a clinic. It died shortly later, before any workup could be performed. Similar findings can be seen in cats and foals with this disease.

At necropsy the first portion of small intestine, the duodenum, was dilated without proper tone, being more flaccid than expected, and having a few small steaks of

hemorrhages in the wall. The remainder of bowel was, as is usual, considered normal grossly.

The first foal case we recognized had similar signs (animals show signs, humans have symptoms) of not eating and a painful abdomen. The bowel of the foal was similar to the dog described above when the necropsy was performed.

When the affected flaccid bowel was exposed, the first impression was that the bowel was not remarkable except that the content of the bowel at the junction between the affected and the remaining more normal bowel contained an elastic type of material in a loose mass. In the foal, it consisted of hair and plant fiber only. In affected dogs, "playdough," plastic window pane putty, or wood excelsior could be the foreign material.

All of these foreign substances have the character of spongy elasticity which, when in the bowel, distend the wall and cause the bowel to contract, trying to move the material along in the intestine as is the normal function of the intestinal muscle. The foreign material, being soft and spongy, is compressed by the intestinal muscle but also may be elongated due to its distensible, elastic nature. The bowel is thus unable to efficiently move the material along. When the rhythmic contractions of bowel over time cannot move the soft foreign material caudally, except slowly, the material stays in one place. This process of continued contraction and expansion without content movement causes the muscle of the affected portion of bowel, in effect, to exhaust itself, resulting in degeneration and atrophy of the smooth muscle involved. This leads to upper intestinal obstruction and finally death from perforation or electrolyte imbalances. Of note is that even with severe muscle degeneration and atrophy seen histologically, there is often not a

remarkable gross lesion except the flaccid segment of bowel itself and possibly feeling the soft foreign body in place.

Of course, when surgery is done—if done in time—the material will still be present in the intestine at the junction of diseased bowel and normal bowel. Resectional surgery of the bowel should remove the entire damaged portion of initial duodenum and enough of the transected lower limb of bowel, and then be sure that what bowel is left is functional.

This small intestinal soft foreign body problem is generally markedly different than hard foreign body disease, as with pieces of corn cob, hard stones, nuts, or even apples, as mentioned in the first paragraph.

These hard materials allow the intestine to clamp down on the foreign body in trying to move it distally. If the foreign body is too big, hard, or sharp to be moved, it may damage the local soft tissues and interfere with the local blood supply, causing death (by infarction) of the immediate area of bowel, with subsequent perforation and fatal septic peritonitis.

WP 439, 440, 441, 20949

II. Canine

C-16

Multifocal Renal Tubular Ectasia and Fibrosis

Dr. Donald Meuten at North Carolina Veterinary College was one of the first to work up cases of hypercalcemia of malignancy, a condition of soft tissue mineralization of many tissues associated with some malignant tumors in dogs. It is commonly seen with malignant adenocarcinomas of the anal sac glands. These are not to be confused with the anal sacs themselves, which often have benign neoplastic tumors that may be multicentric but rarely fatal.

The normal anal sac glands are to be recognized as discrete, yellowish, 1–2 mm nodules in the wall of the anal sacs. When they become neoplastic they are often malignant and tend to spread locally and metastatically, and have a functional parathyroid hormone effect of soft tissue mineralization throughout the body.

Another lesion associated with this condition is very diagnostic and even considered pathognomonic. It consists of multifocal, 3–5 mm, pale, round foci scattered on the kidney. Often they have an indented, central, dark area and a white periphery of connective tissue and mineralization histologically. The darker central areas are dilated tubules in the cortex. These are the foci of renal ectasia and fibrosis, and to this pathologist, are considered parthenogenetic.

The exact cause is unknown.

WP 492, 493, 494, 1222

II. Canine

C-17

Unequal Blood Expulsion from the Spleen

Often one notices this most interesting finding unexpectedly in dogs, especially in dogs that are euthanized for humane reasons.

These lesions were first seen in a group of dogs euthanized during a toxicological study in treated, as well as control, young dogs of less than three years of age.

The lesions consisted of very irregular, red, slightly flattened, round or plaque-like swollen areas from 10 mm up to 7–8 cm scattered in the spleen, with no explanation at hand. The animals had been put to sleep humanely, usually with a barbiturate overdose and necropsied immediately.

Most of the spleens were well contracted and relatively bloodless—except for these discrete one or two nodules of irregular, raised bloody areas or nodules, usually along an edge or scattered in the spleen. Some involved over half the spleen.

The blood in these areas may be slightly less fluid and, even when incised, may not flow out. The difference of these affected areas is marked with the surrounding contracted areas being almost bloodless, having had the blood squeezed out of them, as is commonly seen. Thrombotic vessels are not present in most instances, which helps to then distinguish them, especially those along the splenic edges, from infarctions. Infarcts

would also tend to be pale as the result of necrotic tissue presence and no extra blood itself.

The areas of these incomplete expulsion areas, although firm from the contained blood, are not otherwise discolored.

Further differentiation must also be made from small or large, round, tan or pale, discrete nodules in older dogs, with the usually discrete round nodules, often pale, centered, or with a mixture of pale lymphoid tissue and blood. They must be identified by histology and are often considered as nodular hyperplasia of the lymphoid tissue present. The term "hyperplasia," however, is fraught with ambiguity since it suggests compensation. But from what? These latter nodules are mainly seen in older animals, but the histological orientation is that of multiple lymphoid follicles, relatively normal in the appearance of dozens to hundreds of essentially normal germinal centers and normal peripheral areas about each center of the lymphoid tissue. The cells of both areas are considered normal in large, round masses, and closely adjacent to each other.

The exact cause for these foci of unequal blood expulsion are thought to be due to the blood being squeezed physiologically from one area of the spleen faster than from others. This results in some areas not contracting at all, or too late, at the time of death, with no blood able to pass the constricted areas. There is some suggestion that these areas at the edge of the spleen are the early forms of siderotic plaques also common in dogs.

WP 503, 506, 4610, 13208, 13231, 13233, 13234

II. Canine

C-18

Chewing vs. Gulping Certain Whole Chunks of Foods

This story starts with Mr. John Oset, my father-in-law, who asked me why his dog gulped all the meat he was given, yet deliberately chewed any bread given. Then in Australia, a discussion came up as to why cattle "chew the cud," a bit of regurgitated rumen content it has ingested.

If one asks almost anyone with some knowledge of animals, the answer to the last question is: To chew what is eaten into smaller bits, for better digestion. There is some truth to that, but another answer is also held that by "chewing its cud" it produces that much more saliva with its powerful neutralizing effect of the bicarbonate content. This helps neutralize the lactic acid and other acidic materials produced by the fermentation process in the rumen proper. The fermentation process in the rumen, by the mass of bacteria and other organisms, is the actual cause for plant digestion in the bowel. Without constant neutralization, the acidity would increase to toxic levels, especially with easily fermentable, finely ground carbohydrate feeds such as corn and other grains.

The probable answer to my father-in-law's question answers the reverse reaction. A carnivorous dog's bicarbonate-rich saliva that is produced by continued chewing would diminish the function of the stomach's acidic secretions. This would inhibit normal acidic gastric digestion of the swallowed chunks of meat.

This pathologist is aware of the common admonition to chew one's food many times before swallowing, but one should also consider the type of food being eaten by the herbivore or carnivore, as partially explained above.

No WP

III. Feline

F-1

Acquired Unilateral Renal Shutdown with Atrophy

As general diagnostic veterinary pathologists, many of us do necropsies on animals with a specific finding of one large kidney and one small kidney. We usually describe the small kidney as having unilateral atrophy (a = not or without; troph = growth), and the larger kidney as being normal, or maybe enlarged if the problem started in the growing young animal. Of course, it is possible that the animal may have been born with a small kidney, called hypoplasia (hypo = less than; plasia = development). Of the many cases we see of one small and one large kidney, most are in an adult animal and rarely do we see it in newborn animals. This is probably an example of the condition called acquired unilateral renal shutdown. as described below, and it is not congenital (con = with; genital = birth) hypoplasia or aplasia.

Interfering with the mechanical function of one kidney, for instance, can certainly prevent its normal development, as with infection of one kidney in the newborn animal which, if treated successfully, may end up with one small kidney. Likewise in the adult: a tumor, infection, or calculus in one kidney may cause one kidney to be reduced in size because of damage, and this would be called unilateral atrophy also, with the other kidney not being affected, or at least not as severely.

In order for the condition to be called acquired unilateral renal shutdown with atrophy, however, it must have the initial insult damage to both kidneys at the same time and to the relatively same degree. We had seen many cases of one small and one large kidney, but had no explanation that was satisfactory until a veterinarian, Dr. Harlow Cameron, sent in a partially necropsied cat for further necropsy. He had operated on it many months before because of urinary blockage from sand calculi in the urinary bladder. He had opened the cat surgically, and successfully, to remove the calculi. He mentioned in giving us the animal's history that the cat now had one large and one small kidney, whereas at surgery months before, they were of equal size and he wondered why the difference.

Now we recognize the problem, seen mainly in cats, but also in most other species. In almost every case that we suspect of having this problem, we can find gross and microscopic, similar chronic lesions to "both" kidneys. Because of this, it is suggested that the problem is neurologically caused when both kidneys are damaged enough at the same time. For instance, with pigmentary nephrosis in which it is sometimes found, and in injury from other toxic agents that affect both kidneys equally, or as in this first suggestive case, when calculi block the urethra and not the ureters, we would have no way of knowing which ureter was affected first, causing back pressure to both kidneys. Further damage is prevented by surgical removal of the blockages. The exact mechanism is unknown, but increased blood flow neurologically through one kidney and not the other, may be involved.

WP 106, 464, 530, 557, 9167

III. Feline

F-2

Dead Cats in the Garage

This is a rather unique finding and as bad as it was in killing these cats, it could have been worse. The true story is that a three-generation family who owned two cats was going to have a driveway barbeque with hamburgers, etc. The grandparents, father and mother, and two young children all were preparing for the event on the driveway when it started to rain, so the event continued in the garage. Apparently the garage door was shut, and after a while the grandparents thought they would rather go into the house because they weren't feeling well. A bit later the children also went into the house for the same reason. Mom and Dad finished the cooking, but both said they had slight headaches.

All went into the house, not realizing the two cats were left in the garage with the incompletely extinguished charcoal grill.

The following morning the two dead cats were brought to the necropsy room. Immediately noticed was the bright pink color of their blood (cyanomethemoglobin) and that most of their normally white tissues were discolored (also pink), which is diagnostic of carbon monoxide poisoning.

In this case the oxygen in the garage was not enough to allow the still-burning charcoal to burn completely to carbon dioxide and water, but only to the incomplete

oxidation stage of the toxic carbon monoxide. This is a great lesson to never have a fire burning in a space restricted of air.

WP 596, 11508

III. Feline

F-3

Suffusion Hemorrhages of the Diaphragm

The first appearance of this entity was found in a cat sent to Cornell for a necropsy after being found dead shortly after an ovariohysterectomy. The animal's peritoneal cavity was opened by the clinician after death, and it was reported to be blood filled. The necropsy revealed that a slipped ovarian ligature was found hanging on regional soft tissues but not around the vessels, as intended.

Also revealed was a large, diffusely spread out hemorrhage (suffusion) on the peritoneal surface of the diaphragm, which was considered unique and unexplained.

Since this first case, several cases have been seen in cattle dead from spontaneous ruptures of the abdominal aorta or other large vessels, probably related to copper deficiency. It has been seen in several cases of lymphosarcomatous enlargements of the spleen, with rupture on the surface of the spleen and subsequent exsanguination. All of these have been associated with fatal exsanguination. Other cases of this entity have been seen in other animals dying from exsanguination for whatever reason.

We have not seen the lesion in slaughtered cattle, for which we have no explanation unless it is prevented by head trauma during slaughter.

WP 418, 431, 1756

III. Feline

F-4

Slow-Forming Hepatic Hematomas

This lesion, seen mainly in cats, is recognized as scattered areas of severe lobular congestion in extensive areas of the liver, usually in one or just a few locations. The congestion tends to be only within lobules and appears to separate the cords themselves from each other within the lobules. In some areas, the congestion extends directly to adjacent lobules and still does not form lakes of blood as seen in true hematomas.

This lesion in the cat is very similar to that seen in chickens that are obese, caged, and primarily layers. In the chicken the cause is thought to be related to increased abdominal pressure on the liver, possibly related to egg laying. However, it has been seen in a single rooster.

In all of the cat cases there is a known cause, established by follow-up histories consisting of increased abdominal pressure. It is known to be related, in each cat, to playing with children who had lain on them under a pillow or blanket or such.

WP 583, 585, 15186, 15281

III. Feline

F-5

Pigmentary Calculi and Nephrosis

These cats are usually sick a few days, but icteric (jaundiced) for a much longer period. When necropsied, the major lesions are located in the kidneys.

These unexpected lesions usually, but not always, consist of dark kidneys and the constant finding of a few to many soft but very dark, friable calculi in the renal pelvis and calyces. They are usually accompanied by slight to moderate hydronephrosis, with dilated pelvis and calyces.

The combination of these lesions of slight hydronephrosis, pigment calculi, and dark kidneys suggests a pigmentary nephrosis which is not usually severe but is found histologically.

The cause of the pigmentary nephrosis is considered to be a blood-related problem such as from chronic mycoplasma or frank hemorrhage, but this is yet to be proven.

WP 565, 566, 567, 956, 11851, 11853, 11854

IV. Equine

E-1

His Sister's Dead Horses

One winter morning, a veterinary student came to the author on a weekend to tell him his sister's two horses were found dead in their stalls at a boarding stable near New York City.

His sister had called, greatly upset that of the many horses in the barn, only her two horses were dead, although they were in two different and widely separated stalls. No explanation was forthcoming for these deaths (electrocution, angry boyfriend, etc.), so our only recourse was to suggest that they bring the dead animals to the university as soon as possible for a complete necropsy. Even in winter, the internal heat produced by the large number of bacteria in a herbivorous animal's bowels will increase the rate of decomposition, necessitating a timely necropsy.

Two well-conditioned, mature, dead horses were submitted, and absolutely no external or internal lesions of diagnostic importance were found in any tissue or organ. After questioning, the young owner said it had been snowing in the area of the stables, and she had not been able to take the horses out for riding exercise in several weeks. The evening before they were found dead, she had gone to the boarding stable and let both horses out for exercise in a nearby paddock while she watched them. After an hour or so she put them back in the separate stalls, where they died overnight, without observation.

Although no diagnostic lesions were found in the two horses, both horses did have several identifiable twigs of the ornamental tree, *Taxus spp.*, in their stomach content. These were submitted to plant experts for proper identification. This poisoning does not cause lesions in any animal.

Why this occurred at this time is the mystery. Possibly the trees had grown tall enough to be eaten from over the fence or possibly the snow covering the ground made these trees attractive. In similar cases, horses, cattle, and sheep (though not deer) which commonly eat such trees, may also die as rapidly as these horses had. Taxine is the suspected toxic agent. In some of our experimental cases, some horses have been force-fed large quantities of the evergreen without deleterious effect, while other horses eating only a small mouthful from the same shrub may die within hours. Taxus branch trimmings thrown over the fence to sheep and cattle may be similarly toxic and if not looked for specifically, this diagnosis can be easily missed. The leaves or twigs may sometimes be found in the stomach or farther down in the gastrointestinal tract.

WP 190

IV. Equine

E-2

Tied-Up, Haltered Horses

Numerous horses have been brought in for necropsy, without a decent history for trauma except for being tied low to a tree or post for a period of time shortly before being found with nervous signs or dead.

We all know about the horse that rears up and falls over backwards, hitting the back of its head on the ground or even on the ceiling or an overhead supporting beam. Depending on the strength of the impact, this may in effect cause severe flexion of the head. We often have the subsequent history of nervous signs and even bleeding from an ear or ears, and then finding the fractures of the basisphenoid and other nearby bones in the floor of the cranial vault.

However, another group of cases, somewhat related by having fractures of the same bones with hemorrhage and nervous signs, has helped us clarify the problem. These initial examples were described to us by my late professor of veterinary pathology, Dr. Charles G. Rickard. If you had ever shaken hands with him, you'd realize his strong, large hands suited him perfectly for one of his jobs as a young man in the West—breaking horses on his family's ranch.

Many horses are tied to the hitching post or rack and no problems seem to develop from that, even when the horses are scared and pull on their halter or bridle to escape. In

other instances of horses that are tied to low posts or when the bridle falls to ground level and is stepped on, some horses would suffer by going down rapidly or be severe enough for the need to euthanize the animal. Fractured bones in the floor of the skull would be found at autopsy. Horses with heads tied low to the ground can be made to walk to the end of such a low rope and suffer these fractures.

On the other hand, according to Dr. Rickard, if the horse in halter was tied high off the ground with a longer shank on the halter, the horse could be forced to run to the end of the rope and be pulled sideways or up and off its feet to crash on its side. This may possibly break a rib or other bones, but would not cause neck flexion nor a ventral skull fracture, as above. This led to the idea that they do not fatally fracture skull bones if on a lead rope tied high in the air, but are far more likely to if their lead rope is tied near the ground. This is one probable reason horses will naturally stand when the bridle reins are dropped.

This has some relevance to bulldogging steers in cowboy rodeos and also as a crude but effective method to see if some animals have an increase of cerebrospinal fluid. One can flex their necks suddenly to increase the intracranial spinal fluid pressure by this sudden decrease of the cerebrospinal space, and the animal will be spastic for just a second or two and often give the cowboy more "still" time to tie their feet. This would also suggest increased cerebrospinal fluid pressure during central nervous system (CNS) evaluations clinically, if done gently.

WP 8, 9, 2583

IV. Equine

E-3

Muscle Ruptures

We have seen horses with the distal musculature of the esophagus as it enters the stomach and even in the stomach muscles themselves, or in the distal ileum as it enters the cecum, which are markedly thickened. The thickened muscles in these cases is called hypertrophy. Usually when muscles enlarge, they do so by an increase of muscle fiber size and *not* by an increase in the number of muscle cells. In these two locations of smooth muscle hypertrophy, it is called idiopathic hypertrophy of the distal esophagus or idiopathic hypertrophy of the distal ileum, since we do not know the cause in either instance. The esophageal idiopathic hypertrophy is usually not clinically important, but the ileal type is often the cause for the clinical signs of colic. Sometimes when we see these cases there may be a one-sided, outpocketing diverticulum in these thickened areas, and in a few cases there may be a perforation of the entire wall, with fatal pleuritis or peritonitis resulting.

Another related condition potentially may be seen in any animal that has a chronic obstructive respiratory condition, such as chronic alveolar emphysema (heaves) in the horse, and as a result may develop a diaphragmatic hernia, with varying portions of abdominal viscera dislocated into the thoracic cavity. It again is related to a much thicker

diaphragmatic muscle than normal, caused by the increase in diaphragm workload even though it is skeletal muscle and not smooth muscle, as is the bowel.

Horses and other species also get thickened bowel walls as the result of increased peristalsis (muscular contractions), with a compromised lower bowel lumen caused by foreign bodies or even adhesions. Although there are many instances in which the distal ileum of the horse is noted to be quite thick at necropsy, it is considered to be normal by many pathologists and even by anatomists. However, newborn and young foals have no appreciable thickening of this portion of bowel. For this reason, we consider that the increased thickness of the ileum, just like that of the esophagus, is a lesion, and not normal.

In all these cases, the suggested cause is increased contraction of the affected muscles, even in the uterus. An example can be given that in a normal, not pregnant, mare, the uterine muscle coat is not hypertrophied and is quite tough when handled, and hard to tear on purpose, but when pregnant and thickened it is then physiologically hypertrophied and the smooth muscle wall is much easier to perforate with mishandling, as in doing a caesarian in these animals.

These examples all tend to show that smooth or skeletal muscles which have been hypertrophied from an increased workload will certainly increase the size of the muscle fibers themselves and their contractive strength. There is no increase of the cellular side-to-side fiber connection, however, and thus it is much easier to separate the fibers and perforate the muscle wall between the fibers. It is usually not by fiber rupture itself. This is the actual cause of these hernias mentioned in the title. Of course, hypertrophied

muscle fibers can rupture, and do, under stress, but the actual opening is more often between adjacent fibers.

Many muscle-building people, weightlifters, etc., are very careful of balance when lifting weights, since they are more prone to torn muscles or other fibers with their thicker muscle bundles, probably for the same reason.

Mention here is needed concerning ruptures of the prepubic tendon in animals. In the numerous cases we have had in different species, it was the abdominal muscle fibers themselves that had been ruptured and not the prepubic tendon, as the lesion is often called.

A practical way to show the amount of hypertrophy in each case is to weigh a portion of a normal animal's similar muscle and compare it with a similar-sized piece of the affected animal's muscle. Weigh a square piece of muscle such as from the diaphragm or colon wall, or in the case of a linear organ, the ileum. Then weigh a similar length of ileum or the same muscle from the affected animal, and a normal control to at least get an objective indication of increased muscle volume.

WP 3, 72, 74, 2351, 2387

IV. Equine

E-4

A Severe Case of Probiotic Toxicity

In the mid 1980s, foals were found sick or dead in all parts of the United States, but only a few were submitted for necropsies. One very sharp veterinarian in Oregon, Dr. Claire Lodahl, sent some samples of liver that looked "odd" to her, from a recently dead, several-days-old foal. She also sent a tube of a commercially available medicinal probiotic paste material from which she had given one dose, as recommended by the label, to a foal that died suddenly a day or two after dosing. The label said it was a microbiological mix of vitamin and mineral paste, a probiotic, to be used to treat the mare and foal soon after birth in an attempt to establish more quickly the normal flora for the foal's gastrointestinal tract.

The foal died rapidly with a severe liver lesion. The liver lesion produced was also unique in that large, locally extensive areas of liver were completely necrotic and undergoing severe atrophy, without appreciable regeneration. The volume of liver affected in the several cases, including this one, was about 33 percent of the entire liver, with the remaining 2/3 being essentially normal grossly and histologically. The grossly irregular scattered areas, 1/3 of the affected liver, consisted of irregular, slightly depressed, pale red areas of atrophied liver without much residual necrotic liver or true fibrosis in these affected areas.

One local clinician, after he showed us a case he had received in the mail before the Oregon case came in, was asked if the foal had been treated with anything since birth and the answer was no. This made us think of a colostrally transmitted toxin, but which one? We immediately changed our opinion when the lady veterinarian sent us her case *and* the toxic paste.

The Oregon paste material was then fed experimentally, at the recommended dosage of one ml per foal, to a locally owned, newborn foal with congenitally deformed limbs. The foal was considered normal otherwise, but was to be put to sleep shortly since the leg deformities were not amenable to surgery or other treatment. The liver lesion was remarkably similar to the previous cases, demonstrating that the toxic substance in the paste was the problem.

It was suggested by some that the iron fumarate was the principal toxin involved, and others suggested that it could be a mycotoxin produced by a mold during the mixing and fermentation process used in making up large batches of the paste at one time for commercial use. Certainly, all probiotics are not toxic.

The pathogenesis is suspected of being one of a *single-dose toxic effect* on the liver in which a powerful toxic material is absorbed from the stomach or gastrointestinal tract over a short period of time. Because not all vessels to the liver are functioning at the same time, the whole liver is not affected. Only affected are the scattered areas vascularly open at the time of transport to the liver, and the small amount of toxin is absorbed and causes the damage. A similar single-dose effect can be seen in kidneys with renal toxins, affecting discrete areas of kidney tissue, such as from hemoglobinuria in anemic animals, but not in normal animals.

Regeneration was not observed. The animal died only a few days after dosing, which did not allow the time needed for regeneration.

The material was removed from the market immediately.

WP 1, 2, 7012, 7015

IV. Equine

E-5

Pyothorax

(Empyema of the Chest Cavity, Purulent Pleuritis)

In a busy necropsy room, we commonly find in the pleural cavity, either on one side or more commonly bilaterally, a large amount of purulent fluid, debris, and fibrin attached to surfaces of lung lobes. The chest wall itself and the thoracic surface of the diaphragm are also involved. In the horse, it is often only in the lower half of the pleural cavity with the involved viscera, probably due to the horse's upright stance most of the time.

Most pathologists would culture this infected material from the animals in which they find it, and a mixed population of organisms can usually be isolated, unless the animal has been heavily treated with antibiotics. In some species, like the dog and the cat, *Nocardia spp.* is a common isolate but it probably is not the primary cause, as you will read below. *Rhodococcus equi* is a common organism isolated from the horse.

Over the years, numerous cases have been examined in all species and at least 90 percent have been associated with a *ruptured lung abscess*. Of course, the important thing here is to establish the cause of the abscess in the first place, and in most species it is an inhaled foreign body of almost any type. In the cat it is sometimes a barbed-type plant, awn. Arborvitae tree twigs have been seen more commonly than others in New York cats.

However, any foreign object that is small enough to get down the bronchial tree can elicit this response, since almost any object accidentally inhaled will probably be contaminated with bacterial agents of many different types and, if sterile itself, it certainly can bring organisms picked up on the way from the mouth and the saliva. These can act in concert with the foreign body, which the lungs will try to eliminate by a localized purulent reaction, forming the lung abscess or abscesses. This may rupture into the pleural cavity. The major type of organism or the primary agent involved may be replaced secondarily by other organisms as the environmental factors change and the host's response changes, which probably allows the predominance of *Nocardia spp.* as in the dog or the cat. This organism is known for its microaerophilic propensity, which would be the typical environment found in such a thorax.

The ruptured abscess is often difficult to find, especially in the cat, because the abscess ruptures, and the debris and the foreign body are expelled into the larger pleural cavity. The original abscess pocket may not be found. The abscess may heal over and only its surface scar covered with debris may remain. The students are told to look carefully, but they may still miss the initiating abscess lesion. One cow sent in by Dr. Kenneth Gumaer, of Stuyvesant, New York, had a five-gallon-sized abscess pocket in the lung, with the abscess wall at the time of necropsy continuous with the chest wall. All of the debris was sifted through a kitchen colander, after much washing with a hose to break up the purulent debris, and a single oat kernel with its attached hull was found as the inhaled foreign body.

A strong index of suspicion and perseverance will usually determine the cause. In the cat and other small animals, careful palpation may demonstrate the healing scar in the

lung and histological evaluation will show the characteristic purulent nature of the lesion. At the necropsy table, a rounded chest in a cat is certainly suspicious for this condition.

WP 112, 113, 114, 115, 2576, 11602

IV. Equine

E-6

Guttural Pouch Hemorrhage

Many horses come to the necropsy room with the history of bleeding from the nose. Most clinicians recognize that probably the hemorrhage seen (epistaxis) is coming from a unique lesion of a progressive ethmoid hematoma or possibly from a one-sided, dorsal, usually mycotic, infection of a branch of the internal carotid artery which allows bleeding into the guttural pouch. This pouch being continuous with the nasal passages, or even into the oral cavity, may allow the blood to be swallowed and actually, in some cases, may prevent the telltale epistaxis.

The first cases seen by this pathologist were in 1957–1958, when the horse came in, having bled out from such a lesion or about to bleed out, with the guttural pouch completely filled with clotted blood. Both pouches were cleaned out and carefully examined, and the involved damaged vessel was found in association with a mycotic infection. Mycotic infections anywhere in the body generally affect vessels, and in this case the branch of the internal carotid was affected. All were happy with that explanation, when considering the cause and effect.

The guttural pouch is a natural dilatation of the Eustachian tube in the horse. It is open to external air. The air a horse breathes varies under the many conditions of husbandry, such as during dusty trailer travel or at haying time. Horses are likely to inhale mold

spores such as from *Aspergillus spp*. These can inhabit the pouch itself since it is not a sterile chamber and is open to the outside. Most of the time no infection occurs. So the question is, why do they get this guttural pouch mycosis?

One possible answer is that we have seen many cases of the disease, and cursory examination of the area of infection will demonstrate the mycotic inflammation. But unless the affected head, guttural pouch, and hyoid bones are cleaned by cooking (as for a skeleton preparation), the underlying cause will be missed. Most of the cases will have arthritis of the involved hyoid joint, as evidenced by exostoses in the region of this bone and joint, with the subsequent mycotic infection.

During one summer season, a retrospective necropsy study by a group of students, of about 100 horse heads from normal and mycotic guttural pouch–infected horses, had the heads cleaned by cooking in an autoclave for soft flesh removal. Eleven of the 12 cases of mycosis had evidence of arthritis only on the side affected with arthritis. Six cases with arthritis alone did not have infection. As most of the mycotic-affected animals did have arthritis, it suggested that the arthritis with its inflammation comes first. Trauma by halter or bridle is the suspected cause for the joint trauma. This arthritic joint becomes secondarily infected by mold spores that are normally found in the airway of the pouch itself. The organisms themselves do not cause the problem unless hyoid joint arthritis occurs with its congestion and edema.

WP 97, 100, 1104, 2762, 11341, 16589

IV. Equine

E-7

Dimethyl Sulfoxide (DMSO) / Mercury Toxicity

This case involved a mature horse being treated for lameness in one front lower limb, but the problem has been seen in many other horses. The treatment consisted of the use of what is called a "blister," a chemical salve that is relatively irritating and was a common treatment for horse lameness in years past, but only used infrequently at the present time. Part of the rationale for its use has been to increase circulation to the area treated, by the rubbing which in itself will sometimes help. Some say the blister material may irritate the limb enough so that if applied to the opposite, normal leg, it will force the animal to use the opposite leg and thus accomplish the desired effect. Today, ultrasound and other treatments are used more commonly and effectively.

The "blister" material is often a mercury-containing salve and is quite toxic if taken internally, but not if merely applied to the skin. Thus, it is important to prevent the horse from licking the material from the treated area, and for this purpose a "cradle" is employed. This is a cone-shaped collar about 24 inches long, of multiple, loosely held together plastic or wooden sticks, placed around the neck, with the larger end opening at the chest and smaller opening near the head. This effectively prevents the horse from turning or bending its head enough to lick the "blister" salve from its legs or the rest of its body. The collar was put on this animal before the "blister paste," or salve, was used, so

that it could not lick at this mercury blistering agent. Approximately 10 to 14 days later, the animal became ill with renal failure, showing the classical signs of polyuria, polydipsia, and weight loss.

At necropsy, two unique findings were apparent. One was the finding of swollen, pale kidneys, the result of mercury damage to the kidney tubules proper, and the other was the finding of large scattered areas of dark red patches of edema and congestion on the serosal surface of the colonic wall and larger patches on the mucosal surfaces. The colon content had a definite odor of ammonia. Normal animal urine and colon do not have this odor when fresh. Bacterial contamination in the stall floors, bedding, and in the colon will allow the breakdown of the urea in urine.

Three questions now must be asked: (1) Why is only the large bowel affected? (2) Why the smell of ammonia? and (3) How did the mercury get into the body?

The partial answer to the first question is that in many (not all) cases of renal failure in some species, the body tends to eliminate waste products that normally are eliminated by the kidney. The waste products are now eliminated in part through the large intestine mucosa, but not successfully. The waste materials are irritant substances themselves, and they cause inflammation of the bowel wall, called uremic colitis, as in this case.

The partial answer to the second question is that normal urine does not have an ammoniacal odor since the urease enzyme is not present in the urine. However, if urine is exposed to bacteria having the urease enzyme that is needed to break down the nitrogen products, including urea, and the urine is now being eliminated into the bacteria-filled colon, then the ammoniacal odor will be noticed in the colon.

The third question is answered by following up with more questions to the owner and caretakers. What was done after or prior to putting on the mercury-containing "blistering" agent? In this case, they said they cleaned the leg first with DMSO (dimethyl sulfoxide), a product of the lumber mill industry. This is a great organic solvent which by itself is not very toxic, but being one of the best organic solvents, it greatly enhances the skin absorption of almost any substance dissolved in it—in this case, the toxic mercury from the blistering salve.

WP 18, 20, 1035

IV. Equine

E-8

After the Storm

The necropsy service was sometimes presented with tissues from horses that had died within a few days of a severe windstorm in the vicinity of various farms, usually along the East Coast of the United States. Initially the histories given us on these cases did not include the mention of a windstorm in the area, or it was ignored, and the pertinent history given was that the horses had died with apparent renal failure, with dark urine, for some unknown reason.

Because the initial evaluations made were from formalin-fixed tissues for biopsy evaluation, the diagnosis was often based on the findings of a pigmentary nephrosis, a degeneration of the kidneys associated with pigment in the renal tubules, and the gross observation by the referring veterinarian that the kidneys were darker than normal at the time of the necropsy. The clinicians would often comment that the horse had renal failure when they died.

The most common cause of such a change (lesion) in kidneys would then be reported out that a plant toxin should be considered, but which one? We were not able to be definitive from the biopsy samples submitted.

The problem remained unsolved, until one day when one owner had several horses die following a windstorm which had blown down several red maple (*Acer rubrum*) trees in

the paddocks and the horses had been seen eating the wilted leaves. The horses were jaundiced, with distinct, dark yellow mucous membranes of the eyes, gingiva, and vulva clinically and diffusely throughout the body, at necropsy at Cornell. The kidneys were also very dark from the methemoglobin involved.

This owner was also very astute and brought in many branches with their leaves that the horses had been eating. These leaves were initially just identified and more or less ignored since no one suspected that wilted red maple leaves were toxic.

The owner was adamant that the leaves were the culprit, so the clinicians fed the branches, with wilted leaves, to a research pony. The pony promptly died in the same manner as the owner's horses had.

This of course solved a long-unidentified problem, and after further research the information was published by the clinicians to make everyone aware that red maple tree leaves, under certain conditions, namely wilting, can be toxic and fatal. Hydrogen cyanide poisoning from blown-down leaves or hedge trimmings of the several types of cherry trees (*Prunus spp.*) can also be very toxic and fatal, with the production of bright cherry-red blood and tissues from cyanide, which are different from the red maple cases.

WP 22, 23, 24, 25, 11790

IV. Equine

E-9

Aortic Rupture in the Horse

In this case, the horse dies rapidly as the result of a rupture of the aorta, the main vessel leaving the heart, that supplies blood to most of the body except the lungs. The rupture is often a wide, V-shaped opening at the site of the aortic valve leaflets' attachment to the inside of the aorta itself. Because this catastrophically sudden death is seen in stallions at time of copulation, or in horses during a horse race, many people attribute it to the extreme exercise period with its rise in blood pressure. There may be another, better explanation to encompass these cases and in other, less strenuous, times when it occurs.

Not too long in the past, a flurry of deaths occurred at several New York racetracks, where the horses were racing. When on a single weekend, four horses died during the races at two different tracks, foul play was suspected. The horses were seen to go down and die during the race. Several were sent to our laboratory for necropsies. Two of the four had ruptured aortas, as described above.

Years earlier at Cornell, a group of aged Army horses had to be put down for economical reasons. They were shot in the head one morning after being walked over to the parking lot adjacent to the necropsy room. Two of these docile, not excited horses, also had similar aortic ruptures when necropsied. Many other cases with similar findings have been seen when horses were killed for humanitarian reasons by gunshot, and in

several others that were killed by chemical injections. All had acute traumatic aortic ruptures of a similar appearance and died with cardiac tamponade (blood-filled heart sac) as a result. The chemically killed horses had to be held up when given the chemical; otherwise they would tend to fall sideways and possibly hurt the attending clinicians.

It is easy to rationalize that high blood pressure may have been a factor with high-strung breeding animals or animals during a race, but most do not fit that scenario. What does fit them all is that of probable severe chest trauma from falling onto the sternum while breeding, tripping during a race and falling onto the sternum first, and collapsing at the time of euthanasia. It is suggested that the chest trauma causes a sudden forward displacement of the heart that in effect kinks the aorta when most heart valves are closed, and then something must give. In this case it's the attachment site of the aortic valve cusps at the base of the aorta itself. It apparently does not happen in all such trauma cases since all valves and outflow tracts from the aorta may not be closed at the exact same time during the cardiac cycle, allowing enough bypass to prevent the rupture.

I am told by my human pathology colleagues that this is a common finding in humans killed in automobile accidents with steering wheel trauma, even at slow speeds.

A similar aortic rupture can be seen in piglets on an all-milk diet. A copper deficiency, not trauma, is suspected in them. This also suggests that copper may play a role in these cases in horses and even in cattle with other vascular ruptures, since copper is important in the formation of elastic tissue in large arterial walls.

WP 41, 42, 43, 45, 1758

IV. Equine

E-10

Shipping Fever Pneumonia of Horses

(Inhalation Pneumonia in Horses)

Horse caretakers have been seeing pneumonia in horses after their being shipped for long distances, as from racetrack to racetrack or to and from breeding farms. No satisfactory answers were obtained from clinicians or veterinary pathologists, except that the horses had scattered areas of pneumonia recognized by finding firm areas of diseased lung, usually in the anterior and ventral regions of the lungs. Often these areas were only mildly affected, with the animal having a fever or being off feed. Sometimes, large confluent (locally extensive) areas were involved, making the animals severely ill or even killing them.

One of the first cases submitted to this pathologist involved several experimental ponies which on necropsy were diagnosed as mild inhalation pneumonia. They had been treated by clinicians and students for parasites. After finding the lesions at necropsy, it was suggested that maybe they should let the clinicians deworm the horses since apparently the students were putting the medicines down the trachea and not the esophagus. Their major clinician, Dr. Mary Smith, spoke up quickly in the students' defense, saying the horses were not tubed with the medicines. The students only used a paste wormer applied to the horses' palate, which the horses had to lick off. This gave us

the clue that while inhalation is one cause, it was not from drenching or stomach tubing in all cases.

Because the horses usually had been recently shipped, for whatever reason, it was called shipping fever of horses, but the actual cause was still not ascertained.

To understand its widespread presence in numerous horses killed daily for human consumption in one slaughterhouse in Italy, the author went there one day to see approximately 80 percent of these horses with these same lesions of shipping fever. The horses had been transported to Italy primarily from Eastern Europe. An interesting side note of that time is that the slaughterhouse floors in Italy, Argentina, and Brazil were so clean that one could almost eat off them. Amazing!

Practitioners have attempted to determine the cause of this type of pneumonia by culturing the affected areas of lung for bacteria, viruses, and other potential causative agents. Many different organisms were found, but the clinicians were unsuccessful in reproducing the disease as seen clinically. Bacteria were found in most instances, unless the animals had been heavily treated with antibiotics which prevented the bacteria's cultural isolation in the laboratory.

No answer was forthcoming until recently, when a group of Australians reproduced similar lesions by tying the individual horse's head high with a halter during transport. This had the effect of not allowing the horse to "snort" or shake its head when necessary to eliminate nasal mucus and dust, especially during a long, dusty ride. The posterior nasal opening would then, in effect, allow the mucus with or without bacteria and debris, to drain down the trachea and bronchi, into the anterior ventral lobes primarily, causing the cranioventral inhalation pneumonia.

Since nasal mucus contains many natural defensive chemicals, antibacterial agents, and cells, the various foci of pneumonia were often not highly infective. However, if not treated correctly, the pneumonia could certainly become worse. Prevention of such cases is the best route to follow, by not feeding dusty hay during transport and not tying the head high, thus preventing inhalation into the trachea from the posterior nares.

One wonders about the many cases of pneumonia that occurred in shipped horses from all parts of the world during wartime, that developed pneumonia and died from this entity unknowingly.

WP 9, 11, 4310

IV. Equine

E-11

Right Dorsal Colitis and Stretch Ulcers

The original cases of this entity in the horse, and even current cases, were presented to the hospital clinics alive, but in a few cases came in for necropsy directly after being found dead or dying following an acute episode of colic (abdominal pain). With either presentation, the horse usually has had a chronic history of weight loss and episodes of clinically evident colic with the weight loss. These signs are certainly not specific for this disease entity, but in retrospect are often noted in the history of such cases.

When the abdominal cavity is opened, the pathologist may sometimes find evidence of peritonitis with fibrin, excess fluid, and partially digested plant material, supporting the diagnosis of a bowel perforation directly into the peritoneal cavity.

Most of the time, however, this plant- / feces-caused peritonitis is not found, since the right dorsal colon usually does not get perforated. On further examination, the wall of the right dorsal colon may seen to be thickened or shrunken somewhat in lumen size, or even dilated, with only slight or no involvement of the small colon.

When the entire bowel (gastrointestinal tract) is opened it is usually only the mucosa of the right dorsal colon that is involved, thus the name of the condition. The initial small colon and the cranial portions of the large colon are not involved.

The bowel in the affected area is most often completely ulcerated and devoid of any mucosal epithelium. Most of these cases also have hypertrophy of the muscle coats of the affected bowel. This is due to the loss of the protective mucosa (the epithelial lining of the bowel), which exposes the now naked submucosa to be exposed to the irritating intestinal content. This in turn causes the bowel to contract continuously, resulting in smooth muscle enlargement (hypertrophy). In a few of the hypertrophied muscle areas, opaque white areas of muscle may be seen that histologically are characteristic of Zenker's degeneration, with some mineralization.

With time, if the animal lives, a few various-sized patches of remnant mucosa may form islands of smooth-edged, rounded, regenerating epithelium. These islands of regeneration are usually too small to replace the massive loss of surface epithelium. The submucosal connective tissue, stimulated by the ulcerated bowel, will mature and often contract, to cause stenosis (constrictive blockage) of the bowel and subsequent death.

Another unique new lesion is seen with this disease and in these affected areas only. It consists of linear streaks of edematous wall without any epithelium, that has been stretched and actually ripped apart a centimeter or more and also linearly for 10–20 cm by the internal gut pressure. These linear streaks with some reaction are more acute and are called stretch ulcers. Those that are acute and without much, if any, tissue response probably occurred almost at death or as artifacts caused by the prosector's handling of the bowel.

Right dorsal colitis has usually been noted in horses given nonsteroidal anti-inflammatory drugs (NSAIDS) when the treated animal was dehydrated. It is also very easy to miss this lesion at necropsy. We have pulled several from the discard containers,

"dumpster diving" after the necropsy. It is thought to be related to prostaglandin since the lesions seen are related to the highest production sites of prostaglandin in the adult horse. Severe ulcerated lesions, often with perforation of the stomach and duodenum, can be seen in foals when they are treated with NSAIDS. Right dorsal colitis is not seen in foals, nor is renal papillary necrosis seen in foals like it is in older horses. One should also consider that dehydration increases the toxicity of NSAIDS. The textbooks speak of the action of prostaglandin in foals as being opposite that in adults, and this may explain the different responses.

In the adult horse with the lesion of renal papillary necrosis associated with the use of NSAIDS, the necrotic renal tissue may slough, leaving a cavity in the renal pelvis of the horse. This is called postnecrotic cavitation and is another disease of progress. The necrotic debris may partially remain and become the nidus for calculi formation.

WP 144, 146, 148, 149, 17919, 17933

IV. Equine

E-12

Visceral Torsions (Twisted Bowels)

Many times a veterinarian is called out to a farm to look at a horse that is ill with what is called colic (acute abdominal pain). The horse may be uneasy, looking at its abdomen constantly, pawing the ground, stretching out its body like a toy rocking horse, or even rolling on the ground. Many things may cause this, but one of the more common is a twisted bowel segment within the abdominal cavity.

The common explanation is that the horse in these cases twisted its bowel. Probably the horse did not actually twist its bowel, but instead the bowel stayed in place while the body of the horse did the twisting.

Other examples of possible causes are when a horse accidentally and suddenly tumbles head over heels, or is caught in its harness in an awkward position, or is rolling on its back and suddenly pulls on a chronic adhesion, or has another source of sudden abdominal pain. In these odd positions, when it cannot exert the normal "abdominal press" necessary to hold the viscera in place, the horse may suffer a displaced viscus, often the colon. Under these conditions, the horse may continue to turn but the viscera (intestines) remain in position, resulting in a twisted bowel. It's somewhat analogous to the law of physics in which a body at rest tends to remain at rest, and similar to an olive in a martini. When one turns the glass quickly, the olive remains stationary. My drinking friends tell me this.

Of course, other findings such as adhesions of various types may allow the moving bowel to actually move into compromising positions and allow the bowel to stay twisted. Since bowel twists are so common in horses that are trapped in harness, stumble and fall, that the twisting horse, not the bowel twisting explanation should be considered in any case where it's possible. Also it should be considered that it does not happen very often to horses even though many of them roll on their backs quite commonly, even completely from one side to the other without ill effect. It does not happen to these animals when they are prepared for the rolling action with proper abdominal press. Abdominal press can be used to explain it not happening in active sporting events under similar conditions with other animals and man, when they are prepared mentally for the exercise.

Large male dogs especially, but almost any dog, even small females, can be affected by what is called gastric torsion. While the actual cause is often not proved, it has been noted by some that the dog usually has a platform-type structure such as a chair, couch, board shelf, or some similar available structure they can jump *up* to. The dog, when it gets excited, especially after a very fluid meal such as dry dog food and water, may jump up to and down from the platform and if, for any reason, the antics are interrupted in flight, the stomach turning may not stop when the dog does. The stomach torsion occurs at that time, as in the horse, and somersaults around its fluid-filled abdominal viscera. The viscera itself stays in place when abdominal press is too slow to hold all the viscera in place during the somersault or other quick-acting body movement. Of course most horses that roll on their backs are capable of natural abdominal press to hold their movable viscera in place.

WP 236, 3110

IV. Equine

E-13

Core Temperature–Related Distal Limb Gangrene

Affected foals come to us for necropsy with a history of lameness in the distal limbs, often associated with sloughing of one or more hoof walls.

Further evaluation of the foal limbs will often reveal that one or all of its distal portions of limbs are cold to the touch, suggesting that although one foot is sloughing its hoof wall, the other feet may also be involved. The foals usually will have a recent history of a high fever for any one of a number of reasons in the recent past, such as an upper respiratory tract infection apparently treated, successfully or not, with antibiotics.

The owner will usually bring the animal with the sloughing hoof in for treatment, but because of the usually poor prognosis in these cases, the animal may be left for necropsy to identify the problem more fully.

The necropsy may not reveal the initial cause of the high fever since the animal may have responded well to initial high fever treatment by the clinician or owner, prior to presentation. The high fever itself, however, was the initiating cause by an apparently neurogenic shutdown of peripheral blood vessels in the distal portions of the limbs. This occurs in order to maintain the normal temperature in the core of the body, the brain, heart, and lungs.

At the time of necropsy of such affected animals, one notes a sharp line of demarcation between the normal upper part of the affected limbs and the darker, dull-appearing distal portion of affected limbs, caused by the apparent neurogenic shutdown of blood to the distal areas. A similar picture can be seen in pigs' ears and tail when they have a high temperature. This is often the result in pigs with a bacterial infection on their heart valves called vegetative endocarditis, which results in fever and neurogenic shutdown of the peripheral blood vessels to their ears and tail. The result is gangrene, for the same hypothesized reason. It occurs in baby rats and is called ringtail, even to the extent of the baby rats' losing their tails, digits, and legs, or even all four legs, and not just from one animal but all or most in the entire litter. In these baby rats it is almost always due to the mother's bad habit of pushing nesting material out of the nesting box, piece by piece, and allowing the nesting area to remain wet and too cold for the baby rats.

The modes of maintaining normal core temperature in animals vary somewhat among species, as indicated above. Rats lose their tails while horses lose the distal parts of their limbs, and pigs lose their ears and tail when their bodies must respond to heat loss by peripheral vasoconstriction of the more peripheral blood vessels. With pigs, many practitioners believe that emboli in the arterial supply to the ears from the heart is the cause, but often the pigs may not have vegetative endocarditis in either side of the heart to be a source. In man, those with a fever feel so cold that more blankets may not help. Emboli from the heart certainly would not explain why the specific vessels of the ears are involved bilaterally, but this neurogenic mechanism may be involved.

WP 117, 188, 644, 960, 3267

IV. Equine

E-14

Prunus (Wild Cherry) Toxicity

On a farm in Ithaca, NY, the owners had four adult donkeys for years without a problem, only to have one die suddenly for no known reason. Several days later all of them had died, and they were brought in for necropsy.

The first animal was necropsied and had no diagnostic lesions grossly or histologically. A farm visit was arranged, with veterinary students, to visit the pastures involved and the general environment.

The fence rows around the pasture did have numerous *Prunus spp.* trees, but their leaves had fallen weeks before, probably ruling that out as a cause because of the long lag period involved.

Two of the last three donkeys to die, however, did have pale to bright-red, discolored tissues and blood, allowing a diagnosis of hydrogen cyanide (HCN) poisoning.

To establish the cause, the pasture walk-around was very informative in that the owner told us he had brush-hogged (large tractor–mowed) the fence rows four to five weeks before the first frost of the season in order to destroy the weeds and young field cherry shoots.

The adult cherry trees indeed did have much of their bark chewed by the donkeys, but relatively speaking, the bark did not contain appreciable HCN present. Closer

examination of the fence rows showed a great number of newly sprouting *Prunus* suckers growing close to the ground, with many wilted leaves from an even more recent frost. It is well known that new growth of many plants is considerably more toxic than more mature growth.

The findings of no bright-red blood at necropsy is explained by the fact that animals can have a high enough blood level of HCN to kill by cyanomethemoglobin production, without having the bright-red color. One can often enhance the color by shaking collected blood in air.

WP 26, 27, 28

IV. Equine

E-15

Gastric Rupture in Horses

Often we are asked to do a necropsy on a horse which died after a short period of acute, severe abdominal pain (colic) followed by a brief episode of apparent relief and then death. With some cases, vague abdominal signs may have been noted prior to the severe colic.

At necropsy, the abdominal cavity may be found with varying amounts of relatively fresh, undigested, gastric content throughout the mesentery and viscera, or only a local accumulation may be found relatively limited to the mesenteric omental sling. So far it is all you would expect to see in cases of classical stomach rupture in the horse. Looking at the stomach itself, you will usually find the serosa torn along the greater curvature of this organ, along with the muscle coat, but with the mucosa and submucosa only partially torn and maybe slightly everted. This rupture mechanism is explained by the muscle coat's ability to contract forcefully on the stomach contents, whether it is gas, fluid, or solid ingesta. In most cases it is gas or fluid. The stomach can handle a very full load of dry ingesta without problems. In fact, the normal horse's stomach can be actually so filled with dry ingesta that the neophyte pathologist will even consider them abnormally distended (impacted).

The problem in these cases is to determine why the gas or fluid accumulated.

In the majority of such cases, one will find a lower bowel obstruction such as an intussusception, which is one segment of bowel inverted into another, causing the lower obstruction. Tumors, abscesses, and granulomas in the lower bowel wall can also cause the obstruction. Even a chronic adhesion may be involved.

Many believe that the horse's overeating of a concentrate such as grain can cause gastric ruptures. It may do so in a few instances, but a mechanical lower obstruction is usually the proximate cause and must be looked for. Again, it is easily overlooked if the animal lived long enough after the rupture to completely mix ingesta with the viscera and mask the obstruction—unless a complete necropsy is performed.

WP 3, 5, 2945

IV. Equine

E-16

Blister Beetle Poisoning, a Disease of Progress

Certainly the world knows about cantharidin toxicity associated with Spanish fly, Russian fly, and several other blistering beetles. The two flies named are really types of beetles, and all of them are known for their ability to produce a toxic blistering agent, cantharidin, a yellow fluid associated with their joints that makes them unappetizing and toxic to potential predators.

This blistering agent has been used commercially by grinding up the insects' bodies and making a salve or paste, or even a tincture, that can be rubbed on the skin to cause irritation. Its use in veterinary medicine has been as an irritant over horses' or other animals' joints, to stimulate increased vasculature with the thought it would help heal a sore joint.

It has also been used as a counterirritant by its use on the opposite good leg or joint, making it sore enough so that the horse would use the opposite, actually involved joint, in forced exercise. The substance has been used erroneously as an aphrodisiac. It is a severe epithelial irritant when put on the skin and is absorbed, and is subsequently eliminated in the saliva and burns the mouth. When eliminated in the urine it irritates the urinary tract.

One of the main outbreaks in animals has been described by Dr. Roger Panciera and Dr. Lois Roth in Oklahoma, with massive stomach and heart lesions included. My first encounter was with severe tubular necrosis of the kidney of sheep caused by this agent.

The massive outbreak that occurred in nature, as described by Panciera, et al., can be considered a disease of progress in the following description. It seems that these insects tend to swarm seasonally, often in fields of alfalfa. In the old days of haying, the farm mower, pulled by the tractor or team of horses, left the hay in rows to dry naturally for a few days. The farmer hoped it would not rain before baling or other handling efforts. Since the beetles might be in the cut alfalfa, they would leave the grass being allowed to dry before it was baled. However, in modern fashion, the crimper was invented. Its double opposing rolls of hard-ridged rubber grooves roll all the cut alfalfa between the rolls and crush the stems of the grass so that the alfalfa will dry faster. It also crushes most of the beetles in place, leaving them in the hay. The result is this disease of progress when this alfalfa is fed to the animals, horses included.

WP 1035, 1632, 6883

IV. Equine

E-17

Colitis X in Horses (Exhaustion Colitis)

We have seen many cases of this disease in horses only, for which we still do not have a definitive answer.

The histories usually include the information that the affected animal or animals have been shipped over a long period of time (for more than one or several days), often in cold weather. Several of the original cases, first identified by Dr. Peter Olafson at Cornell, were seen in horses that were known to have pulled a load of coal from the bottom of the State Street hill in Ithaca up to Cornell University. Sometimes, within hours of this very strenuous effort, the horse would have severe diarrhea and die within hours. A necropsy performed immediately would show no lesions except for a slight to severe edematous colon wall with minimal feces in the tract.

This has been seen numerous times here since those early cases, and although the hauling of coal uphill is not involved, the cases are related to shipping horses several hundred miles and back again with severe diarrhea resulting, often described as explosive diarrhea and death. The lesions are usually limited to edema of the wall and an empty bowel. There is no gross or microscopic evidence for inflammation except the edema.

Salmonellosis should be considered also, but no septic tank odor is noted and no surface necrosis or causative organisms are found. Potomac horse fever was not known to

occur that long ago, and the seasonality of PHF is not similar, nor are biting vectors considered a problem in this entity, colitis X. Sometimes a partially twisted bowel can be suspect, especially if it has replaced itself by body movement or even shipping activity, leaving only slight edema and maybe congestion.

The disease of exhaustion colitis in horses is not seen commonly in this area now, possibly because more care is taken with horses.

WP 180, 182, 185

IV. Equine

E-18

Osteodystrophic Lines

For years, people have been talking about rickets in animals and man, and most believe that it is caused by a deficiency of vitamin D. It is probably true in some instances, but other factors are commonly involved and should be considered. For example, animals deficient in vitamin A (carotene) as well as vitamin D will not get rickets. Other examples suggest that vitamin D deficiency is not the only answer, and in fact the more correct answer is that vitamin A (carotene) is antivitamin D. "Vitamin A Antagonizes the Action of Vitamin D in Rats," J. Nut. 129:2246–2250, 1999, by C. M. Rhode, et al.

Many foals and young horses have been brought in for a fracture or fractures of various bones, and a common finding in these animals has been, in addition to broken bones, a distinct enlargement of the costochondral junction (the junction zone of bone to cartilage in the sternal ends of the ribs). This enlargement is often called the rachitic rosary. When the rib is cut at this junction, at right angles to the curvature, a distinct zone of pale bony tissue extends several millimeters to over a centimeter up the bony portions of the sternal end of the rib. This is the osteodystrophic line (osteo = bone, dys = abnormal, trophic = to grow). Most of these foals are still nursing, or have been weaned recently or even some time ago, but they are still growing rapidly.

In New York many foals are born around the first of the year, and fractures may be noted between three to eight months of age. Often the history given to the pathologist by the owners or veterinarians is that the foals were kicked by the dam, or in one of our most serious cases, that the animal ran into a brick wall. Certainly, horses kick foals and young animals may have accidents, but it appears in our necropsy experience at Cornell that young animals with fractures usually have soft bones with osteodystrophic lines in their ribs, attesting to the fact that the animals have a vitamin/mineral problem also. This suggests that the fractures may be due to something in addition to being kicked or traumatized.

We have had several outstanding cases in which, for instance, an eight-month-old foal came in with multiple fractures, and the foal was euthanized since the fractures were not thought to be clinically amenable to being repaired. The clinicians were advised of the suspected nutritional problems and that other foals could be involved. The clinicians, however, were adamant that the foals and their dams were allowed out of their stalls daily for exercise and sunshine, so it could not be a metabolic problem with vitamin D. This was accepted initially, but when the owner called for a diagnostic answer some time later, we had to say we could not give one since apparently the foals were allowed out daily to exercise and we could not say it was due to the lack of sunshine activating the precursors of vitamin D. The owner replied, "What sunshine? We only let them exercise in an enclosed pavilion."

The next great case was somewhat similar in that the veterinarian said the owner told him the affected young horses and dams were allowed outside every day for at least eight hours. Again, the owner called weeks later because we did not have a specific diagnosis

after the necropsy. We had to explain that we usually only report the diagnosis to the veterinarian, and that we could not explain why the foals had the rachitic rosary with osteodystrophic lines since the history said the foals should have had enough sunshine. Immediately the owner's reply was, "Who said they had sunshine? We only let them out at night because of the severe problem with flies pestering the animals during the day."

In these cases it probably was primarily a vitamin D–related problem, by not having sunshine to activate the vitamin D precursors. Of course both cases, and a great many since, have educated us to more fully question the veterinarians as well as the owners in such cases, especially when the osteodystrophic line is seen in the affected farm animals.

Histologically the osteodystrophic lines contain damaged and necrotic bony tissue.

While involved in research at the Mellon Institute in Pittsburgh, Pennsylvania, I had need of vitamin D for a rat's experimental diet, but I was unable to find concentrated vitamin D without significant amounts mixed with vitamin A. This suggests the possibility that this mixture could be a factor for vitamin D–resistant animals when it could have been the antivitamin D action of excessive vitamin A (carotene).

WP 191, 194, 318, 16634

IV. Equine

E-19

Small Strongyle Colitis

Every now and then some owner brings in a very diarrhetic, thin, even emaciated, adult horse for necropsy that they have no idea as to the cause. Their veterinarian has wormed the animal and examined the teeth to no avail, and the owner is adamant that they are well fed. In some cases the animal is brought in for necropsy by an officer of the Society for the Prevention of Cruelty to Animals or the police. This is also the type animal and case the author has necropsied in several foreign countries.

The necropsy is usually quite instructive in regards to the many lesions of emaciation, with serous atrophy of fat in most fat depots, including the bone marrow with its marked jelly-like content. Many tissues, including the liver, are much darker than normal due to the chronic loss of fat.

There are no other specific lesions in these cases except in the large colon, which may be edematous from hypoproteinuria and contains only minimal moist, even watery, feces. Initially, a diagnosis is often given as emaciation, cause unknown, following a parasite evaluation of the remnant feces.

In the horse, especially, with these findings, the large bowel must be washed clean with water to examine the mucosa closely. With a clean mucosa, one will often see tiny, not elevated, dark 1–3 mm foci often with a darker periphery of 1 mm. Further

examination of the foci will usually demonstrate the presence of the causative parasites, small strongyles, cyathostomes, cyclostomes, and other small strongyles.

These parasites have embedded themselves in the mucosa of the colon, and they are resistant to many medicines used for treatment of most parasitic diseases. The treatment has often been given, but the animal still does not respond and the problem continues. There is often a definite legal problem with these animals, because often the owners are charged with cruelty when in fact the resistant parasites embedded in the bowel wall are at fault.

WP 126, 128, 17926

IV. Equine

E-20

Pneumocystosis in Horses

This disease is primarily one of horses, but can be found in other animals. The clinical signs are usually related to the serious infections of *Rhodococcus equi* in young horses' lungs or lymph nodes or anywhere else in these animals. The primary sites of bacterial infection are in the gastrointestinal lymph nodes.

The lungs, however, are the major organs affected with this protozoal infection caused by pneumocystis carinii.

The large areas of lung tissue with abscesses up to 15–20 cm may be located deep in the lung. They are usually filled with yellowish, almost fluid, pus in any lobe or bronchial nodes. These abscesses are easily seen and are mostly well defined even though deep in the lung. Affected lymph nodes are also seen in the head and neck regions and along the GI tract.

While the abscesses of *Rhodococcus* are easily seen and palpated, lesions of *pneumocystis* are only palpable as slightly firm, 6–20 cm areas scattered in the lung also, but more often near the bacterial abscesses.

Histological findings would of course easily show a purulent, infectious process in the areas of the bacterial pneumonia, but the surrounding protozoal areas have a variable, chronic, inflammatory cell response with the additional characteristic cellular response of

relatively clear, swollen mononuclear phagocytes filling and even distending the alveoli. This lesion may be easily overlooked unless one stains the slides with silver stains specifically to show the very characteristic positive-stained organisms in the previously clear macrophages filling the alveoli. It should be noted, however, that the same organisms may be seen in the normal lung of horses without the disease, and some areas of affected lung may not be heavily laden with them even when the phagocytes are present. The firm lung tissue around the abscesses are most commonly the best tissues to examine for this disease of pneumocystosis. It is thought that the chronic bacterial disease involved causes exhaustion of the immune system, allowing the protozoa to proliferate.

This disease is seen in other immune-related problems in foals, such as in CID (combined immunodeficiency disease) of Arab foals. It is also seen in rats and people treated with immunosuppressive drugs for cancer therapy and in HIV patients.

WP 214, 215, 216, 2694

IV. Equine

E-21

Gastrosplenic Herniation

The affected horse usually comes to necropsy with clinical signs of abdominal pain (colic) and is euthanized *in extremis* or dies. Our first case came to us with a history of sudden death.

At necropsy, the lesions are found related to a gas-filled small bowel with moderate fluid ingesta. The distal small bowel (ileum) is fairly normal, but at varying distances from the colon the small bowel is seen to be constricted at the splenic attachment area of the stomach. With careful dissection, the small bowel is noted to have penetrated completely through a 6–10 cm opening, the hernial ring, of the gastrosplenic ligament. This hernial ring opening usually has a slight, 1–3 mm firm, white rim of connective tissue through which the hernial content, a loop of small bowel, has penetrated.

Our first cases were thought to represent an initial herniation by the ileum, with incarceration. However, subsequent cases demonstrated that almost any portion of the small bowel can move through the ring, both in and out of the hernial sac, until the distal ileum is reached. The distal ileum cannot pull the colon itself toward the ring. It is at this time that the small bowel and vessels become strangulated.

Our biggest problem not solved yet is to give a plausible reason for the hernial ring formation itself in the gastrosplenic ligament.

The necropsy, if not done in a careful manner, will often allow destruction of the ring, making for a difficult diagnosis.

WP 50, 51, 863

IV. Equine

E-22

Aortic Valve Kissing Lesions

This is a lesion seen only in the horse and is not an important lesion since there are no clinical signs or other problems in a carcass associated with it.

The lesion is only recognized on the aortic valve leaflets and appears as round, roughened surfaces from 4–10 mm in diameter just to the side of the central edge of each leaflet, beside the normally present nodules of Arantius.

These "kissing lesions" are usually oval or round excrescences having a depressed center and are about 1–3 mm thick. They are on the opposing endocardial surfaces of each two leaflets that would come together when the valve leaflets close. They are not on the sinus of Valsalva surfaces, which do not contact each other.

It is suggested that they form by cusp apposition and pulling apart as the valve closes and opens.

The nodules of Arantius are to be differentiated from these kissing lesions. The nodules of Arantius are normal, small nodules in the center of these leaflets and are more prominent in pigs than in other species.

These "kissing lesion" nodules are seen in older horses and are considered to be due to an ageing change. Usually there is only a single pair of these lesions involving only two of the three cusps, but two or three pairs have been seen.

Another common lesion seen in many species, yet best noted in the aortic valve of horses and to a lesser extent on the free edges of the atrioventricular valves, are multiple, irregular, elongated openings (fenestrations) up to 1–5 mm.

Again, these fenestrations are considered an ageing change usually of no clinical significance.

WP 84, 85, 1762

IV. Equine

E-23

Fundic Necrosis of the Urinary Bladder

Most of these cases have come to the necropsy room following several days or longer of vague urinary signs of obstruction. Some of the animals may show remarkable spontaneous relief just before they die. Some of the signs may include posterior paresis or paralysis. It is seen in all species, but seems to be more common in the horse, sheep, and dog.

At necropsy, excess, usually clear fluid is present in the peritoneal cavity, without any appreciable odor when no other infection is involved, but there may be an ammoniacal odor if at least a slight infection is also present. The clear fluid may be heated to enhance the odor of urine (ammonia).

The bladder is usually smaller than normal, and its wall may be thinner or thicker depending on how long the condition has existed. In many cases, the bladder ruptures in the fundic portion, allowing urine escape. This is not always present.

The involved fundic area may show a definite green or yellowish discoloration, with or without mineralization of the entire fundic portion of the bladder. In other cases a major portion of the bladder may be affected similarly and have only a small tear in the center of the necrotic area.

This bladder lesion is considered to be an example of fundic necrosis as the result of bladder obstruction, for whatever reason, and subsequent increased pressure. Urethral calculi are a common proximate cause, as are trauma or a tumor affecting the lower vertebral column and spinal cord. This may cause a neurologically induced lack of bladder function, resulting in a functional bladder obstruction.

In small animals such as the cat or even the mink, the affected bladder may rupture at this necrotic fundus site when palpation is even slightly applied, with the animal showing almost instant relief, only to die hours later.

The pathogenesis for such a lesion in the fundus is that with obstruction, mechanical or functional, the subsequent distension causes the end arteries in the bladder wall to be compressed, depriving the fundus of a blood supply.

WP 130, 131, 475

IV. Equine

E-24

Medial Cecal Base Rupture

This is seen only in horses subjected to elective surgery for minor problems such as minimal eye surgery or such, in which the horse has been anesthetized and strapped on the surgery table.

The onset of the problem is recognized by acute abdominal signs and sweating following the elective surgery procedure. The animal dies or is euthanized. On opening the peritoneal cavity, an acute septic peritonitis is observed, with cecal content free in the peritoneal cavity mixed with the intestinal viscera, mainly at the medial side of the base of the cecum and primarily with a roughly 6–12 cm, irregular, acute ruptured cecal wall.

The actual cause has not been established, but a suspected "strap down" procedure during the elective procedure is involved. At the time of the author's sabbatical year in Australia, we had had 11 cases at Cornell in the necropsy room with the acute abdominal signs following surgery. Also while in Australia, when visiting the various schools, I was lucky (unlucky) to see a horse that had just hours before come off the elective surgery table and was being walked as part treatment for an acute abdomen. It was their 26th case of this same entity.

Of note, we have not seen it at Cornell for the last 15 years, 1980–1995.

WP 855, 856

IV. Equine

E-25

Abdominal Fat Necrosis of Horses

Numerous cases of this specific type lesion have been found in the horse but not in other species, and they consist of marked, firm, mottled, 1–3 cm thick, rounded 10–30 cm plaques of fat and connective tissue on the ventral abdominal wall, mainly bilaterally on both sides of the midline. Several have encroached on the midline and have been continuous there without any separation. A few of these affected areas have also extended up to the tuber coxae as an ear-shaped extension to either one or both coxae.

The overall area of the peritoneum is often 10–30 cm in diameter, which would sometimes be larger when including the ear-like extensions to the tuber coxae.

Thick bands of connective tissue are present throughout these usually flat masses of firm, even hard, fat in various stages of degeneration and necrosis. Most have an irregular but smooth surface as they bulge from the abdominal wall under the peritoneal lining.

The cause of these plaques of granulomatous fat necrosis is not known, but saddle belly bands or other bits of harness have been suggested. The location, however, is not really compatible with saddle or harness straps. The lesion is behind the belly bands of the saddle. It is quite different from the abdominal fat necrosis in cattle since this entity is in the ventral abdominal wall of the horse, not scattered in the abdominal cavity as in the cow.

The lesion is almost always only an incidental finding at necropsy.

WP 186, 187, 188, 189

IV. Equine

E-26

Incomplete Nutmeg

Actually this is not truly incomplete, except in the sense of time. At the necropsy table, in the horse and the dog where it is seen commonly, the lesion is best described as scattered, irregular areas of dark liver interspersed in areas of more pale liver, often in a tree-like pattern. It is not a diffuse change in the entire liver initially. It is considered locally extensive and may involve large areas of liver, but it will be diffused with time from the continuing causative passive congestion. The paler areas often are more discrete than the darker areas of congestion, which are redder than the less congested areas.

Its time dependency is probably brought about by the severity of the passive congestion in the more peripherally located portions away from the larger venous return vessels from the liver proper, to the posterior vena cava and lungs. It is usually more noticeable in the periphery of the entire liver, being darker than more central areas of liver not necessarily related to lobular architecture.

It is thought that with time, if the animal lives long enough, the entire liver will be affected with the "nutmeg" appearance of an almost tree-like (arborization) distribution.

WP 105, 106, 384, 538, 1952

IV. Equine

E-27

Vertebral Fractures

Using the swift, electrical method of euthanasia in horses with instant death, we sometimes see the animal go into the stiff "rocking horse" position of instantaneous, total body spasm within a second or two, with arching of the back. One may hear at this time, a loud crack which indicates a vertebral fracture. It being an instant death, there is still blood pressure for a time that will bleed into the fracture sites internally, and blood may be seen at the time of necropsy associated with the vertebral fracture site.

This same set of lesions can be seen in horses accidentally electrocuted, in rabbits when physically restrained, or even in rabbits picked up by the nape of the neck, as is commonly done. Their strong abdominal muscular contractions can fracture the lumbar and thoracic vertebrae.

In some rabbits housed in usually quiet surroundings, a sudden loud or unexpected noise may elicit the same rapid muscle contraction that may rupture the psoas muscles and also cause vertebral fracture.

Accidental electrocution in other farm animals can occur, but usually the abdominal musculature is not strong enough in these animals to cause vertebral fractures like in the horse or the rabbit.

WP 14, 15, 242

V. Miscellaneous

M-1

The Catastrophic Decline of Caribou in Newfoundland

Around the beginning of the 1900s, an estimated 500,000 barren ground caribou were in the island of Newfoundland, Canada, but over the years their numbers continued to decline so that in 1950, less than 20,000 were present. A major research effort was made at the time to determine the cause. This was arranged and paid for by the Wildlife Service of Newfoundland, headed by Dr. Stuart Peters and aided by this author.

Bits and pieces of information and even tissues from the five distinct herds of caribou on the island province had been sent to Cornell to be investigated, but no answers were found. Research efforts were enlarged by using a team of scientists to try and find the answer. Small Beaver aircraft, a type of fixed-wing airplane, and helicopter aircraft were engaged to trap animals, survey, and count the animals in the five various migratory herds. The scientists also conducted extensive research to evaluate the exact problem accounting for the loss of the caribou.

Initial herd surveys over the years showed that researchers on foot could count the animals, adult females and potential calves, using binocular field glass observations. The researchers stood downwind to actually observe the enlarged udders (mammary glands) on the pregnant and milking females. Sterile females would not have a well-developed udder. Body size was a good indicator for adults and calves, and by using both

parameters and counting the young caribou in late spring or early summer by the on-foot observation, downwind study, combined with aerial photographs, it was determined that 10–80 percent of the calves that had been born in any one year often did not survive through the first few months of life. Most of the adult females should have become pregnant and had calves with them through the first year.

These migration-type herd animals, which tend to migrate along specific areas from winter to summer and back again, follow the approximately same trails. After the breeding season, the males separate from the females, and the females tend to calve alone in certain particular regions along the same migratory routes each year.

Expanded field research efforts included full necropsies of more than 100 animals, including a few normal male and female caribou, a few immature adults, and many dead or affected calves. This research was done specifically during the calving season, which is late May to late June in Newfoundland. Adult caribou were found to harbor many parasites, one of which, the pharyngeal worm, *Cephanomyia nasalis*, is a fly maggot. The maggots were found in varying numbers (9 in one female, up to 106 in another) in a parasite-induced pharyngeal diverticulum (pouch) in most of the caribou. Another historically interesting fact is that eight of the many species of parasites found in these animals are normally found only in the reindeer of northern Europe. This suggests that the last land bridge to Newfoundland may have been attached to northern Europe and not North America, according to the late professor of parasitology, Dr. John Whitlock at Cornell University.

This research seemed to show that the severe drop in caribou numbers was indeed through the loss of calves in the first few weeks to months of life. The necropsies

demonstrated that the major calf lesions consisted of abscesses located in the neck region and were associated with draining tracks from holes in the skin of the neck. The calves often died from other related causes such as starvation from not having been able to nurse, or from rupture of an abscess into surrounding tissues including the brain, spinal cord, or trachea. Fatal septicemia was a common cause of death. One of the early calves had fractures in the cartilage of the trachea and larynx, associated with infection, and considered, erroneously, to be pathological fractures (fractures of hard tissue associated with infection to the hard tissues).

During the calving season, the pregnant females would be migrating with the herd of other females, but at actual calving time a pregnant female would usually wander to one side to have her calf by herself. This would have made it difficult to find sick calves except with the aid of the helicopter. This helicopter made it relatively easy to find the aborted or sick calves, which we needed in order to continue the research. The helicopter was the only way, except by accident, to find these sick or dead calves. The mother would stay with the downed calf for days or even weeks, walking toward the calf and grunting, then walking away and trying to induce the calf to follow. From the helicopter in the calving areas, researchers looked for a single female to the side or in back of the migrating herd. Actually, we looked for a "moving white rock," since females were pale with their white winter hair coat, and from far away we could not tell them from rocks if they were not moving.

Some affected calves were found by looking for (from the helicopter that was moving with the herd) and seeing a calf with obvious draining abscesses in the neck region, often lagging behind the herd.

Experiments were designed to find the possible causes, such as isolating calves on platforms or on the ground to see which would develop similar infected skin lesions (abscesses) in the neck, or which parasites, such as the throat (pharyngeal) bot or maggot, could possibly penetrate through the skin to cause the lesions. None of the experimental animals showed up positive for parasites. All we could establish early on was that the calves were dying of draining abscesses of the neck, with holes of various sizes (from pinpoint to several centimeters in diameter), mainly in the skin of the neck. *M. hemolytica* and *P. multocida*) were the common bacterial organisms isolated, but they did not cause the problem in any of the experimental calves.

Many suggestions were made as to possible causes, such as predation by bears, insects, infections, poisonous plants, and others, but none were thought to fit until a large four- to six-week-old male calf was found ten days after the second major project began in the field. This calf had many skin wounds around the neck, but no infection. It also had fractures of the tracheal cartilage and larynx, with traumatic hemorrhages of the region, but again had no obvious infection. This first one with no infection was also the largest calf found affected. More males than females were affected.

None of the animals had been partially eaten. Several more calves that were examined, as well as adult moose and caribou, had no similar lesions. The neck lesions found in the fresh-killed calf suggested a big cat, probably a Canadian lynx.

Biologists, when told of the suspected causative agent, the Canadian lynx, would not believe this. They had never seen lynx attack calves, even when biologists followed the herds during the conduct of other studies.

Of course, lynx do most of their hunting at night when humans are sleeping, and if humans are approaching the herd by day or night from downwind, with the lynx also downwind of the herd, the lynx would more likely be aware of the humans than vice versa.

It is well known that big cats and even domestic cats often play with their prey while not intending to eat what they kill. With more experience, one learns that predator cats often kill by biting necks or noses, as wolves are known to do with moose or other similar large prey. To some observers this behavior suggests the predator is suffocating its prey.

The biologists in these studies were well aware that the male calves were usually the first to wander and wander the farthest from their protective dams. This would explain males being the predominant victims. The calves would probably make enough noise when attacked, to bring the dam too late to its rescue, but the dam would chase the lynx away. She can kill a lynx with a hoof strike since she usually weighs close to 350 lbs. and a lynx weighs only 30–40 lbs.

The affected calf, after the initial trauma to the skin of the neck, may then develop infection and abscesses with time, which would be the actual cause of death. After much hair pulling by biologists to expose and study the wounds, the noninfected skin wounds were often perfect fits for the dental outlines of the lynx. Cultures from subsequent captured lynx often revealed pure bacterial cultures of *Pasteurella multocida.*

Trapping of lynx from the calving ground area and other methods of lynx control have been effective in stopping these die offs of caribou calves that had been increasing yearly up to that time.

The answer to why these losses started is not so easy. Other causes include the possible lack of human predators on the lynx themselves because of the fall in value of animal furs. A cyclic phenomenon between predator and prey is common and may be suspected.

WP 816, 818, 1228

V. Miscellaneous

M-2

Fat Embolism

Probably one of the most perplexing problems extant for man is the finding in the lung, brain, or elsewhere, of globules of embolic fat and even bone marrow in phagocytic cells in blood vessels, with or without causing vascular compromise to these areas. Most of these emboli are explained as coming from patients with traumatized fat or broken bones. However, in late 1950, a group of dogs and rabbits muddied those waters for us. Dr. Asdell, a well-known nutritional researcher at Cornell University, sent four or five dead rabbits that had been savaged by a pack of dogs, for necropsy.

Dr. Asdell and one of his graduate students, Dr. Gary Rumsey, had 35 rabbits on a 4 percent cholesterol diet. They lived in scattered huts raised on wire above the ground. All of these pens were enclosed by a large chain-link fence and gates, one of which, unfortunately, had been left open. Several German shepherd dogs got into the area on a Saturday. They apparently ran under, around, or against the cages and grabbed any part of any rabbit they could get through the cage wire. Ears, legs, skin, and viscera were pulled off while the dogs "played." When the damage was found, Dr. Gary Rumsey brought the dead and dying rabbits to us for necropsy on Sunday. Ten rabbits were received dead or in extremis between Sunday and the following Wednesday.

All had large plaque deposits of cholesterol scattered in the tissues, where it is commonly seen in cases of hypercholesterolemia in many species, including man. It was seen externally in the iris of the eyes and grossly also in the aorta and descending aorta and other scattered vessels at necropsy. Of additional interest to the veterinary pathologist was that it was also pronounced in the pleura of the cranial intercostal spaces as opaque, white, thin plaques and in very pronounced, round, white nodules in the gastric cardia. These are similar to the locations of calcium deposition in cases of uremia in animals and some cases of hypervitaminosis D. No pneumonia or significant lung lesions were noted in these early cases in the savaged rabbits from the first few days. This turned out to be significant.

Actually, these lesions of cholesterol deposition were rather unique but not really unexpected, with the hypercholesterolemia from the diet they were on. The traumatic lesions were as expected from dog attacks on any species. They often tear the prey apart in such gang attacks, even from gangs of only two dogs. On Monday and Tuesday, several more traumatized rabbits died from the initial trauma, also with lesions of chronic hypercholesterolemia, but again no lung lesions were observed. Then, between Wednesday and Friday they brought in 12 more dead or dying animals without trauma, but the rabbits now had severe, firm, lung disease in the cranioventral lung lobes. About 50 percent of the ventral portions of all lobes had firm, dark-red and pale speckled lungs, which certainly looked like inhalation pneumonia, only not as septic as expected when seen in most inhalation pneumonias in animals.

Dr. Rumsey was called on Wednesday and told "for goodness sakes, to stop dosing these rabbits with the stomach tube or he would lose them all." He was amazed that I

blamed him. He went on to explain that the rabbits were not stomach-tubed at all, but that he mixed 40 lbs. of pure cholesterol dissolved in acetone with 960 lbs. of dry rabbit pellets, by using a big mixer until each pellet was covered with cholesterol. The pellets were then laid out to dry and to evaporate the acetone, thus yielding a 4 percent cholesterol diet.

Two more interesting findings were made. In order to take proper pictures of all the great lesions that come into a necropsy room including these, we clean off the blood and other extraneous materials from the tissues to make it a clean, neat picture. In doing this we often put the smaller specimens of lung on brown paper towels to absorb the blood, etc. We had put the cut surfaces of these lung sections on the towels and then pulled them off the paper. This left a classic translucent lung imprint of oil on the brown paper. Histologically the lungs were shown to be examples of lipid pneumonia but not caused by inhalation. Thanks to serendipity, similar pieces of lung put on white paper towels did not easily show the oil translucency. Do not use white paper for oil identification.

Dr. C. A. Rickard, my major professor, came on the scene and said that this type of pneumonia would be a great one for a PhD project. I should have followed his advice, but luckily the current project, of aflatoxicosis, worked out very well.

I knew about fat embolization and its relationship to fat and bone marrow trauma. Also about this time, a related article came out from human pathologists in New York City, in which they had performed a series of retrospective studies on a large number of autopsy cases. This was done including use of the not so very routine fat stains on the heart, lung, and brain tissues from groups of individuals who had died for these three specific, different reasons.

It should be remembered that making frozen sections on tissues in order to use fat stains on the various tissues is time consuming and labor intensive. It is not usually done unless it is needed to show or prove the presence of fat emboli in cases of auto accidents or other traumatic deaths, often for insurance purposes.

The New York City researchers made these stains on lung tissue and other tissues from 100 people killed in auto trauma cases, 100 people who died quickly without trauma (heart attacks, etc.), and 100 people who had severe stress with a premonition of death, such as those who lived days or weeks after a heart attack and knew they might die.

To reiterate, the rabbits dying in the first few days had severe trauma without the lung lesions of lipid pneumonia, yet after three to four days a larger number died with no obvious trauma but did have severe lipid pneumonia. Where did the lipid pneumonia come from?

In the three groups of human examples, using fat stains on the hundred that were traumatized, about 30 percent had positive fat droplets in blood vessels in various tissues. In the sudden-death group without trauma, only about 3–4 percent had positive fat globules, but in the 100 people without trauma and with a premonition of death (not acutely fatal heart attacks), again about 30 percent had the positive fat droplets similar to those dying with trauma. These last two groups in routine hospital autopsies probably would not have been examined routinely with lipid stains to see if they did or did not have fat emboli. There have been several textbooks that suggest severe non-traumatic stress may be associated with fat emboli, as may be surmised from these rabbits.

One suggestion as to the cause of the lipid pneumonia and the fat embolization seen in these rabbits, may be that the severe stress itself can allow precipitation of circulating

lipids, which may also be a factor in lipid deposition in coronary vessels and elsewhere. The deposition may follow into the cranioventral vascular bed of the lungs, causing the endogenous lipid pneumonia but not inhalation pneumonia. The exact pathogenesis is not known at this time, but stress of any kind, as in sports, sex, or emotional aspects shortly after eating, when blood lipids may be high but still in solution, should be prevented if possible.

With four years in the Army from 1945–1948, I was often encouraged to eat lots of eggs, bacon, and home fries, etc., on the days I was going out for boxing practice or matches. It is also known that wildlife, like the male moose, can lose up to 30 percent of body weight during the rutting season. Polar bears, which do not get appreciable atherosclerosis but still eat predominately fat-filled diets, only exercise in order to eat and not vice versa, as with most humans.

Another experiment was done to cause severe stress in rabbits. After being put on a 4 percent cholesterol diet, a group of rabbits was put alive into a deep freeze (as occurs naturally in the polar regions) for a week. We knew that cold stress was probably not a stress for furry bunny rabbits, and it wasn't. At the end of the week they immediately jumped out, fully active, and none showed any ill effects. We suspected they would not be affected, and when this story was presented to a group of students they complained a bit about cruelty being involved. However, one of their classmates, Bridget Bloom (C.U. '98) spoke up. She told her story of using rats from an undergraduate research project, where recently weaned rats were gassed to death and then put in the deep freeze over the summer, to be held for feeding to traumatized birds of prey during rehabilitation. When the deep freezer was opened several months later, all of the dead rats had been

cannibalized except a now fully-grown, single, remaining rat which had a cute, white fur hut to live in. He survived to old age as a class and teacher's pet, apparently none the worse for wear. Of course it was also deprived of water for those months, but still survived.

WP 13001, 13060, 13062, 19101, 19116, 19133

V. Miscellaneous

M-3

Drastic Culling

This story is a bit different from most of the stories in this series. It does not deal with a single disease or lesion, but rather just gives a single answer for many problems seen at the necropsy table.

Dr. John Whitlock, Cornell University, Ithaca, NY, inspired this story. It is based on his work and the numbers he used with his hundreds of sheep and lambs over the years in his research efforts and the many animals he sent to necropsy for a diagnosis.

Most of the animals sent to necropsy were from his breeding efforts with his large experimental flock before, during, or after lambing season. The flock's problems were those commonly seen and included congenital malformations, lack of newborn vigor, prematurity, stillbirths, umbilical infections, septicemias, and other conditions with the lambs, and abortions, dystocia, metritis, mastitis, agalactia, mis-mothering (abandoning the lamb) involving the dams. A diagnosis usually could be made for most of the problems and treated as needed and possible, but with such a large flock it was a time-consuming effort and expensive, even with more help.

Dr. Whitlock then proceeded with a well-recognized form of control which is followed in nature by the survival of the fittest technique. His method was to drastically cull, or

certainly to remove any of the problem ewes or offspring, from his future breeding programs.

This worked well, and over a short number of years he used as replacement breeding animals, only those that never had any of the problems commonly seen in the past, as mentioned above. The actual number involved was about a 50 percent reduction in the flock each year.

The real reason for this story is that he proved the general idea of the use of drastic culling and that it worked in a short period of time. For the pathologists and others involved, although we were able to give a diagnosis for most diseases, we were unable to give a cause in most instances, except to suggest that genetics may play a significant part.

However, this method may not be applicable to animal pets and the like, such as Bessie, the farmer's best milker, which recovers almost completely from a case of severe mastitis except that she has lost one quarter and now has only three functional teats. He may breed her again and maybe will get a fine heifer calf again. Maybe.

When he had Bessie bred this last time, he also had a less-than-Bessie cow bred and, let us say, she delivered a fine heifer calf. But if the farmer has room for only one more cow in his herd, which animal will he keep, with everything else being equal? We are sure he would keep Bessie's baby, which in many instances would be wrong according to the discussion above. This is not to say that some cases of mastitis are a genetic problem, but can it be proven that it is not?

No WP

VI. Porcine

P-1

Ruptured Intestines of the Piglet

It is common for most people to think that large sows step on their babies and cause all sorts of trauma to their piglets, but there is some doubt in this assumption. One in many cases in point was in a piglet that had a complete separation of a section of bowel from the other end, with both ends being partially turned inside out. Certainly it is probably traumatic, but how does it occur?

I would ask the students if sows actually do commonly step on any of their many piglets. Most would answer yes, since that is what they have heard so often due to crowded sow/piglet housing, but I would advise caution with this answer for all cases. In most of the instances I've been able to follow up by farm visits, it usually turned out that piglets and even foals may be traumatized by their dams, but not usually, and only in exceptional circumstances.

Even under crowded conditions with the sow and her piglets, the sow, when walking around, may step on a sleeping piglet, but if it is over the firm head, chest, limbs, or hindquarters areas, the sow will know instinctively that it is probably a piglet and she'll not step down on that firmness. It is extremely rare for us to see much, or any, trauma to hard tissue as noted above. However if a sow steps into the triangular soft space, the paralumbar fossa, behind the piglet's rib cage, in front of the hip and pelvis, she may step

down, thinking it is bedding or feces. Stepping down on this soft area, the sow can crush the segment of bowel against the abdominal wall and hard (cement floor), transecting the bowel segment in this fashion but not necessarily causing gross external lesions. One may often find, by careful search, subcutaneous edema and hemorrhages in this soft area on either side of the body.

One could think that the sow might consider the thing she is stepping on is her own feces and thus cause the trauma, but this again can possibly be a mistake in our thinking. Pigs commonly defecate in one area of their pens naturally and probably would "know," again instinctively, if it was not feces. Could this be one reason pigs are so clean in their "toilet"?

The failure to see gross lesions in the flank skin is similar to using the Burdizzo emasculator for calf castration, which cuts the spermatic cord internally but does not puncture the skin in this closed technique for castration.

In practicing for rodeo work as a student at Oklahoma, the author often attempted to switch horses in midstream while bareback riding on the multiple horses in the corral and often fell off, under the feet of the other horses in the group, and never once was stepped on. Apparently, four-footed animals are more cognizant of holes or soft footing in their movements than two-footed animals.

WP 958

VI. Porcine

P-2

Ear Infarction of Swine

One can ask almost any student anywhere in veterinary medicine, "What is the usual cause of ear tip infarction in young pigs?" The common, erroneous answer is: "Embolism of septic debris, usually from a heart-associated lesion with *Erysipelas rhusiopathiae*–infected vegetative endocarditis, a bacterial infection of the heart valves." This is another worldwide misconception. There is some truth as to the cause, but the pathogenesis is incorrect.

Admittedly, in some areas of hog farming, *Erysipelas* is a common pathogen. It does cause significant losses, with heart valve vegetative endocarditis in pigs that have ear necrosis and tail loss for the same reason, but it is not likely due to emboli to the ears. If this were so, one would have to invoke, in the pathogenesis, that a small piece of debris from the left heart broke into two equal pieces very rapidly just before the embolus got to the common carotid arteries, with one piece going into each of the two arteries and both then following the same vascular path on each side to the auricular vessels bilaterally, to cause the bilateral ischemic necrosis of the ears. Very unlikely!

Instead we should probably consider what is known in some young animals, certainly baby rats and probably piglets and others, that the tails, ears, and limbs are important temperature control surfaces for the body. Rats, for instance, often develop "ringtail" so

severely that they will completely lose their entire tail and even more astoundingly, digits from all their feet and even all or parts of their legs. Their cage flooring is usually devoid of shavings and the wood flooring is soaked with urine and water and, thus, is cold. In our research laboratory, when we raised hundreds of rats a year, we would find many instances of complete litters affected at several weeks of age, when not a single baby rat in the litter had all four legs or tail and most of the wounds may have healed. Thus it is suggested that "ringtail" is a classic example for regulation of core temperature. The tail in baby rats is important in control of body temperature. Many cases will show affected rats to just have dried partial digits or limbs, with rings of dried, dead skin along the tail.

In order to maintain their normal body core temperature, baby rats with constricted peripheral vessels accomplish this so efficiently that they cause distal gangrene of their extremities.

Thus it is thought that in foals with core temperature gangrene and in hogs with tip of the ear and tail necrosis, it is more likely due to the peripheral vasoconstriction enabled by a bacterial infection elsewhere that causes the core temperature reflex to shut down the vessels neurogenically to the ears and tail. With the necrosis, there can be secondary infected thrombi that develop in the affected skin and vessels, causing some confusion as to what came first. There are other causes of ear necrosis not discussed in this story.

WP 20121, 20122

VI. Porcine

P-3

Gastric Ulcers in Swine

Having had the honor of spending all my sabbatical leaves from Cornell being invited to a foreign university or research facility, much was learned and shared with others, so that I often heard the same explanation for many of the common disease entities for which I have a different explanation. This is one of my favorites from all over the world.

Many young pigs and some adults have come to the necropsy room after a cursory external examination suggested that the pigs were anemic and may have passed some blood, as noted from the dark, tarry feces smeared around the perineum.

Such findings have several suspected causes, but one of the first should be that when the animal is necropsied in a prescribed routine fashion, the first thing noted besides the anemia is that the intestinal tract, both small and large, may contain very dark content. Frank red blood may be present even in the rectum or anywhere along the intestinal tract when examined. Another early lesion to be found is when the prosector reaches for the esophagus-stomach junction: The stomach wall in this area only is firm and thicker than normal, and often has a significant increase of blood vessels on the serosal surface (medusa head vascularization). The stomach is the source of the gastrointestinal blood and in almost all of these cases, shows a distinctly square or rectangular area of complete

ulceration of the normally stratified squamous epithelium from this pars esophagogastric portion, the nonglandular area.

In some very small pigs, the ulcerated area may be only partially healed, or so completely healed and scarred over that the lost epithelial surface is hidden completely. It may be severe enough to partially obstruct the esophageal opening into the piglet's stomach, causing problems when the young piglets are switched from the dam's milk to solid foods. Regurgitation may then be noted.

These hemorrhagic ulcers with a blood-filled stomach, or even abomasal bleeding ulcers in cattle, often have a very strong diagnostic and characteristic odor of apple cider when opened, and allow the diagnosis. Again, a Taiwan sabbatical allowed some investigation into this common problem. Contrary to common thought even today that the disease is associated with a particular size of the feed, it is suggested here that the particle size of food for the pigs, either small or large, is not the cause. Dr. Peter Olafson, at Cornell for many years, taught that any unsolved problem around for many years with many different stated causes, probably has not had the definitive cause established yet.

The pigs involved with anemia in Taiwan were often related. Many experiments all over the world have been conducted to prove this condition was due to long, coarse fiber feeds, but others indicated it was due to the shorter, fine plant fibers. With this dichotomy it was wise to consider another cause. In the different countries where we have seen the disease and where we have been able to leave the diets alone for a period, changing the breeding boars successfully stopped the problem.

WP 623, 624, 627, 629

VI. Porcine

P-4

Porcine Stress Syndrome (PSS)

(Malignant Hyperthermia of Swine)

When invited to Taiwan, I worked at the Taiwan Sugar Corporation's swine facility under the direct control of Dr. Robert C.T. Lee, a scientist and veterinary pathologist whose brother had won a Nobel Prize several years earlier. This swine research facility was directed in part by Dr. Lee. I was hired to teach pathology and necropsy techniques to many of the young pathologists at that facility and National Taiwan University (NTU).

One of the first problems encountered was the sudden death of large, 400–600 lb. sows immediately after farrowing, and even in larger boars that died after some, even slight, exertion. A greater economic loss was the sudden death of 250–300 lb. slaughter pigs on the way to market. The relationship between the two groups was not known.

It was noted that the large sows or boars that died and that we were able to necropsy within three to four hours, were already surprisingly well on their way to gas-filled decomposition. The market pigs, on the other hand, were first noticed to have seizures or fits, and the skin would be red and often they felt very warm. A young man was usually hired to ride on the trucks to market with the pigs, and he would bang on the roof of the truck cab to indicate affected pigs were being seen. The driver would immediately pull

over to a ubiquitous roadside ditch, and the young man would splash water on the affected pigs with a handy bucket.

When the slaughter pigs were driven off the trucks at the slaughterhouse, the affected animals would come off with a stilted gait or have to be pulled off. If any died, they were noted to have gone into almost instant rigor mortis in association with high fever, tremors, or convulsions. Adult sows and boars were also noted to have high fevers and early rigor mortis.

The initial necropsies of the sows and boars often demonstrated severe gaseous and hemorrhagic edema of the thoracic inlet area and axilla. These areas, when cultured, often revealed pure cultures of various *Clostridium spp.*

These tissues and cultures were considered a postmortem artifact, so another cause had to be looked for. With many pigs on the facility, possibly 10,000 being at any one facility and with several being found dead on any one day, we found the answer based on the author's experience with acute stress and hyperthermia-related disease in lions, tigers, bears, etc., that had been necropsied elsewhere. An adrenal lesion was suspected and looked for.

The normal adult pig, at 400–600 lbs., has a total (combined) adrenal weight of about 40–50 gm, with the adrenals being 3–4 cm and long and pale. The affected pigs' adrenals, when weighed, were often in the range of 10–15 gm total, and the adrenals were tan to dark brown, firm, and sometimes slightly smaller than normal. The adrenals of affected pigs of the same total body weight were only one-half or less of normal weight. Roughly similar findings were seen in the slaughter hogs, but on a smaller scale. In many instances the gross appearance of the adrenals was essentially normal in size, but the weights were

reduced, which caused some problems in recognition of a lesion in the gross. Surprisingly again, histologic evaluation with regular hematoxylin and eosin staining failed to be helpful in differentiation.

Further work with many porcine stress syndrome (PSS) dead pigs demonstrated that as the pigs grew in size and weight, their adrenal glands failed to keep up with the progressive growth and total body weight.

Most of the pigs dying with these diseases also have distinct groups of muscles, such as the long back muscles and the hind leg muscle groups with a pronounced pale appearance, almost a parboiled appearance (pale, exudative pork).

Several zoo polar bears, lions, and a tiger that at Cornell I've had to necropsy, had the same problem. They were found dead after a thunder and lightning storm or truck backfire, with people on hand, luckily, to see several animals die in convulsions. At necropsy, very small adrenals were found, and the carcasses of several that were necropsied soon after death still had high body temperatures. The pathogenesis is not quite the same as in the pigs, although some of the signs were similar and helpful. In this case with wildlife, it is considered a disuse atrophy of the adrenals, whereas in the pigs it is considered a genetic-related hypoplasia.

While on sabbatical leave in Europe, I encountered a similar experience in three large sows from one farm that died for no apparent reason over a period of a month and were submitted for necropsy. The postmortem stomach-bloated carcasses were almost normal, except for their adrenal glands. The adrenals of these three had to be rescued from the discard barrels (dumpster diving), before the carcasses were discarded. The first pair of adrenals weighed 15 gm total when they should weigh 35–45 gm. The diagnosis went out

erroneously as bloat, but no reason was given and no mention of the adrenal glands was noted. A second sow died and again no weight of adrenals was allowed, although the adrenals weighed only a total of 12 gm after being rescued from the discard barrels. Again, bloat was the diagnosis, with no cause given and no mention of the adrenal glands. The reason they weren't weighed was that the prosector had not heard of this adrenal relationship and did not know the normal weights and ranges expected. A third sow with a similar history came in from the same farm, and for the third time the rescued adrenals were of light weight, at 18 gm, when 35–45 gm were expected. In the meantime, other "normal" pig adrenals were collected at a slaughterhouse to help establish the range of 35–45 gm for that Swiss area's pig adrenal gland weights at slaughter.

It is a genetic problem, since simply eliminating that strain of pig in Taiwan and elsewhere by drastic culling has stopped that problem in the meat-type hogs. Of course, good breeding records are needed to eliminate the responsible boars or sows, but drastic culling also works.

Stress certainly plays a part in the appearance of this clinical hyperthermia, but it is not the initial cause and can be controlled by selective breeding. It is also seen as a genetic problem in South Africa, in some human females related genetically to the Royal House of Orange dynasty, with fatal hyperthermia with the use of barbiturates for surgery years ago, and used in swine testing today for PSS.

WP 604, 605, 2411, 2412

VI. Porcine

P-5

Atresia Coli of Pigs

Most of these affected young pigs, weeks to several months old, are sent to necropsy for an explanation of their emaciated condition with a greatly enlarged abdomen. They usually are heavier than same-size littermates. An anal opening is always present, which rules out a common initial diagnosis of atresia ani. The enlarged abdomen is due to a greatly distended bowel filled with doughy-type feces, often noticed through the abdominal wall as the distended spiral colon with their curves of the spiral itself imposed on the outside wall of the distended abdomen.

At necropsy, the enlargement is seen to be the distention of the spiral colon and most of the remaining colon is distended with feces and some gas, all the way distally to about 4–6 cm from the anus. The anal opening is normal, as is this last 4–6 cm of rectum, with a normal mucosal surface and muscle coat. Forward of this normal section of rectum, there is no mucosa on the ulcerated, thickened wall for several to 20–30 cm, depending on the age of the piglet. The ulcerated wall is markedly dilated and feces filled. The small intestine is usually relatively normal. There is always a small opening for gas passage from the distally dilated rectum to the terminal 3–5 cm of the rectum and anal opening. Without this gas escape opening, the animal would die shortly after birth from intestinal bloat.

Histologically, the gut is normal except for the denuded mucosa of the distal colon and rectum, which is thickened with granulomatous connective tissue and smooth muscle disruption irregularly in the wall. The serosal surface of the distended colon usually has many tracts of apparent lymphatic vessel obstruction with moderate fibrosis. This is a result of the enlarged bowel pressure under the serosa compressing the lymphatic vessels themselves. This lymphatic fibrotic reaction is often thought erroneously to be evidence for chronic peritonitis.

This entity of atresia coli is considered to be a congenital anomaly caused by a congenital epithelial defect of the colonic mucosa, which allows inflammation, dilatation, and partial obstruction.

Some have considered that this atresia coli entity is caused by salmonella, but this organism is rarely recovered by culture. When seen in Australia, salmonella also was not cultured even after many attempts.

WP 631, 636, 637, 20726

VI. Porcine

P-6

Intestinal Knotting

The unique fatal lesion called intestinal knotting has been seen numerous times in pigs, but more commonly in woodchucks.

It is not an easy lesion to recognize since, at first glance, most would be considered an example of simple intestinal volvulus (torsion). By careful dissection, without cutting any mesentery and untwisting of the involved small intestine, one can recognize the three components that make up the entire lesion.

Overall, this three-part lesion consists of two simple loops of the freer parts of the small intestine, duodenum, and jejunum, that wrap on themselves as a single overhand knot, like that of the first tie of shoelaces. There is no tear or opening in the mesentery itself. With time, the two loops become congested and edematous, with early vascular compromise leading to bowel gangrene. However, between the two loops of intestine there is always a single portion of bowel that retains its functional blood supply because it will not have a total vascular obstruction. This protects this small section from vascular compromise, but the two loops that are now trapped in the knot become gangrenous.

The actual cause appears to be happenstance; there is no logical reason or other apparent cause for it.

WP 616, 617, 618, 619, 620, 2060, 2193, 17753, 17754, 17755

VI. Porcine

P-7

Atrophic Rhinitis of Swine

The author had a great opportunity to necropsy pigs while in Taiwan, under the auspices of Dr. Robert C.T. Lee, at several of their large pig production facilities there.

This disease, atrophic rhinitis, was prevalent there and easily recognized at the time of necropsy by using a saw to cut across the face and nose at the level of the commissure of the lips. Otherwise it may not be seen easily in the gross exam.

During the year, hundreds of pigs were necropsied, but during one week a specific survey was made of 88 mostly young, susceptible-aged pigs. They had died from a number of other causes, since this disease is not considered fatal.

The primary gross lesion of this entity is the relatively obvious loss of the turbinate scroll tissue that should, in normal pigs, practically fill the nasal passageway in this region. The frontal saw cut makes comparison of the lesions more uniform. It is apparent that both dorsal and ventral scrolls are involved in most cases, and in most animals, BOTH scrolls are affected BILATERALLY. The ventral scrolls are usually the most severely affected.

This bilateral affect helps differentiate it from "bull nose," which is most commonly unilateral, with pus and obviously necrotic tissue seen in the deviation of the snout in these bull-nose pigs. In many with bull nose, the turbinates not infected are considered

normal. This deviation is usually not seen in the atrophic rhinitis animals, although both conditions may be seen in any one animal.

These two entities, bull nose and atrophic rhinitis, are differentiated based on these findings of the primarily unilateral nature of bull nose and the bilaterally similar turbinate lesion of atrophic rhinitis.

Dr. Lennart Krook, at Cornell University, studied the disease and considers it a metabolic disease of calcium deficiency or imbalance, such as the disease in the Cornell herds. The disease was eliminated just by adding calcium to the diet. The author also accomplished the same good results with Dr. Krook's recommendation on the high-production farms in Taiwan.

Histologically, in atrophic rhinitis cases there is minimum inflammatory reaction in the atrophic bones, while the necrosis and suppuration in the bull-nose pigs is the major lesion.

WP 661, 662, 663, 2537, 2888

VII. Ovine and Caprine (Sheep and Goat)

O&Cap-1

White Muscle Disease (and Hepatosis Dietetica of Swine)

The first disease, called white muscle disease (WMD), is usually seen grossly and is characterized by opaque white streaks and patches of affected cardiac and skeletal muscles, usually in the more active muscle groups. It can be seen in many other very active muscles as the muscles of deglutition, including the tongue in really young animals and in the neck muscles of foals holding the head up for nursing. The white muscle lesion is histologically seen as swollen muscle cells with fragmentation, degeneration, and loss of striations, and is called Zenker's necrosis. Mineralization of the fibers may also be seen and, with time, nuclear proliferation (muscle regeneration) may occur.

The disease is also called stiff lamb disease, and in most species it is recognized as nutritional muscular dystrophy. The gross lesion is not seen in all cases, so histology is necessary.

In lambs, sometimes the lesion of white streaks of muscle is only in the pulmonary outflow tract, just under the pulmonary valve in the heart. It is often seen in beef calves after they have been let out in early spring with their dams. They exercise freely and excessively, only to die suddenly. Their severe heart lesions may affect up to 50 percent of the heart muscle itself in being opaque white. They may also have severe leg muscle

degeneration from the sudden exercise. The disease is rarely, if at all, seen in dairy calves unless their dams have been fed and housed as are beef cattle.

The cause for this disease, called nutritional muscular dystrophy, is considered by most to be a vitamin E/selenium deficiency disease. This is probably correct at the cellular level, but it is considered also that there is an antivitamin E factor that makes the vitamin deficient at the cell level. This antivitamin E factor is probably a product of mold or other spoilage factors that develop in spoiled hay or grain, since by far most cases seen in lambs and beef calves are from dams being fed moldy or poor-quality hay. One of the most remarkable cases was associated with beautiful, fat Hereford calves from a farm at Alpine Junction, south of Ithaca, NY. The calves had the most severe heart lesions seen by this pathologist. A farm visit was arranged. Bales of supposedly good hay that was being fed to the adults released clouds of gray mold spores when dropped on the floor.

In this remarkable case as well as in exotic deer lambs in a Swiss zoo, 50 percent of the heart muscle was degenerate grossly, making it difficult to explain why other calves, etc., die with significantly less heart muscle affected grossly.

A note should be considered here about farms which have dairy cattle, beef cattle, horses, and sheep in any combination. Usually it is the animals in the reverse order that are fed, which get the worst quality feed and are thus the ones first affected. In addition, it is the money-producing animals, the dairy cattle, on these farms that are fed grain concentrate, with its higher content of vitamin E in the seed than is fed to the other animals. Beef calves have it most commonly when dams are fed poor quality or even moldy hay, but white muscle disease is rarely seen in dairy calves.

The second related disease listed in the title is hepatosis dietetica (HD), which occurs in pigs. It is a known vitamin E/selenium deficiency disease at the cell level, like that in ruminants. Likewise, however, a great case of this disease consisted of many pigs submitted to the necropsy room in Pullman, Washington, when the author was teaching there. The case came from Idaho, just across the border. A farm visit by several veterinary students (who needed a necropsy case for a class report) was made with the author. The farmer grew all of his own feed ingredients and stored them outside in separate, upright storage bins. He gave us a hundred pounds of each ingredient, including several bales of alfalfa. The ingredients were mixed back at the university and fed to donated, experimental piglets from the university herds that were known to never have had the disease. The ingredients included oats, wheat, peas, barley, and alfalfa. We mixed the feed at the same ratio of ingredients as the farmer had done, except leaving out one of their suspect ingredients and making up for it with the same ingredient from the university farm. Two piglets were put on each mixed diet, and the piglets were killed 30 days later when we ran out of diet. All the piglets that had a share of the barley, had the classical lesions of hepatosis dietetica, but those that did not receive the barley, DID NOT get the disease.

The farmer was called and was told the results after he told us the stored barley had been moldy because of a hole in the storage bin's roof.

The characteristic lesion of hepatosis dietetica in the pig is complete necrosis of adjacent liver lobules, or scattered pie-shaped wedges of part of a single lobule, or complete hemorrhage of scattered individual lobules.

Both entities have been seen in many other species and again usually are associated with moldy feed. A serious suggestion as to cause is that they are usually treated with selenium and vitamin E with success. This of course allows everyone to think it is the result of a deficiency of these items when, as it is thought by others, this treatment works because the additional vitamin E just overrides the level of detrimental amount of antivitamin E factor that has developed in the spoiled feed.

WP 323, 324, 885, 2327, 2406, 3307

VII. Ovine and Caprine (Sheep and Goat)

O&Cap-2

Fatal Lumps in the Throat

Sheep, goat, horse, dog, cat, and probably all species at times may come to a clinic because of a history of difficulty in breathing. In sheep and goats it may be due to a large retropharyngeal or laryngeal caseous lymphadenitis abscess (bacterial disease) in a lymph node. In young brachiocephalic dogs, bulldogs and boxers, an elongated soft palate may be involved. The clinician/surgeon may be caught off guard with these since they usually have anesthetized many animals for all types of surgery, but maybe not those with lumps in the throat. A necropsy service anywhere usually gets to see these cases in animals which have died soon after the clinician has put the animal under anesthesia, but before having the endotracheal tube sized and ready for placement.

This is best explained that even with a potential mass capable of causing suffocation, such a mass usually does not cause suffocation as long as the animal is conscious, such as resting or sleeping. However, as soon as the animal is rendered unconscious by the anesthetic agents, the gag reflex is lost and suffocation begins without coughing or gagging by the animal if there is any delay in finding or placing the proper endotracheal tube.

WP 814, 858, 1577, 13595

VII. Ovine and Caprine (Sheep and Goat)

O&Cap-3

White Liver Disease

During a great year in Australia, I did necropsies almost daily on animals from usually large flocks of sheep, cattle, and other domestic animals, as well as many different wild animals. I was lucky to work with some of the unique diseases in the animals in one of the many competent diagnostic facilities in Bairnsdale, Victoria, Australia. One such disease is called white liver disease. This affects young sheep that do not grow well and can be easily picked out of the flock by their appearance alone. They have a hunched back, with tucked-up rear quarters, and are obviously much lighter in weight than pasture mates. They often have a rougher-appearing hair coat than the rest of the flock.

At necropsy, the livers are paler than normal on both the serosal and cut surfaces. Histologically the liver lobules are slightly smaller overall, with varying numbers of small, round cells, lymphocytes, plasma cells, and mononuclear cells in the connective tissue about the portal triads making up this diagnostic lesion. Many of the animals also had lesions of Zenker's degeneration in their heart muscle, but this was considered incidental in these cases.

Most of these sheep farmers rotate their crops regularly and have four pastures or fields about the same size in which potatoes are raised in one field and alfalfa (lucerne) in the other three. They rotate the potato fields every year. Potatoes have an increased

requirement for cobalt, which has to be supplemented in the fertilizer every year, and it is this cobalt deficiency, apparently, which is the cause of the white liver disease of the lambs.

Within several months of my return from the Australian sabbatical leave, a Mr. Muhlenbacker from western New York brought in several lambs with this same white liver disease problem. On a field trip to this affected farm, it turned out that he did raise quite a few sheep, but guess what? He was one of the largest producers of potatoes in New York and New England.

WP 876, 878, 881, 882

VII. Ovine and Caprine (Sheep and Goat)

O&Cap-4

Perforated Balanitis

This most interesting disease has been reported only a few times in the world. My first encounter with this disease occurred in Australia during a sabbatical visit in 1984. It was found in a flock of several hundred ewes using nine to ten merino rams on a rolling hill pasture at breeding time. The farmer brought in a merino ram for necropsy because it apparently died of urinary obstruction. All seven other rams were also affected though they were not yet obstructed.

This ram did have an obstructed urethra in the distal penis, caused by an inflammation of the glans penis, associated with two perforations completely through the glans, one about 1 cm in diameter near the sheath fold and the other, slightly more distal on the penile shaft, about 1½ cm in diameter. Much congestion, infection, edema, and fibrin were in the prepuce and on the penile shaft, causing the obstruction with urinary bladder distention and death.

The pathologist and his wife visited the farm to check the other affected rams. With the obligatory help of three herding sheepdogs (probably voluntary on their part, one with only three legs), these semi-wild sheep were rounded up, making the impossible possible in order for us to catch the sheep. Each ram was examined using water from a nearby creek (carried by the pathologist's wife in the farmer's hat) to wash the rams' abdomen,

prepuce, and penis for examination. This procedure revealed that all the rams were similarly affected and most had some degree of secondary inflammation. All seven had two perforating, quite similar holes completely through the end of the penis only.

No other lesions were observed in any other tissue of the body. The cause is not known, but in trying to find some type of answer the owner allowed the sacrifice of these very expensive rams with the above results. He put another group of 10 Dorset rams in with the flock to insure a lamb crop for the season and not one of these became affected over several weeks. He then changed back to merino rams, thinking the problem was solved, but once more the new merino rams became affected in the same manner, at which time the breeding season was over. The actual cause was not determined in this case or the other cases found in the literature.

WP 676, 679, 680

VII. Ovine and Caprine (Sheep and Goat)

O&Cap-5

Hard Bone Disease of Rams

In spite of all the research done on nutrition in the world, there are still many unanswered questions. One is the complicated diets of domestic animals, especially where there is a failure to recognize the important interrelationship between vitamins and minerals.

This case in point is a four-year-old adult ram that was presented to necropsy from the Catskill Mountains in eastern New York. After the necropsy, a visit was made to the farm with several students. It was a beautiful sheep farm making money on special wool and wool products. Everything indicated an efficient, good husbandry sheep farm. The animals looked great, but the best ram had died, and they wanted help. They had five or six rams on the property, with 100 ewes.

The necropsy on the ram had reflected the prosperity of the farm, with plenty of fat on the carcass, but the ribs failed to break as expected. The inexperienced student who had taken off the right rib cage during the routine necropsy procedure failed to notice how hard they were to remove, but the pathologist cleaned off a rib and tried to break it in its middle, a routine technique. But no way would it break by hand. Something was amiss.

On continuing the necropsy, the only other lesion seen was a thyroid mass (tumor) in one thyroid gland. That also was very odd for such a young animal. Thus the farm visit road trip was arranged.

It turned out neatly and roughly as expected. The rams were getting the same feed as the breeding, milking ewes, which was very high in calcium and proper for the milking ewes. However, the feed contained too much calcium for the rams, which had no way to eliminate the excess calcium that the milking ewes were able to secrete in the milk.

It is well known by the work of Drs. Krook, McEntee, and Hillman, with bull studs in Ithaca, that bulls have a more than normal number of vertebral fractures from apparent increased bone hardness, as well as serious discospondylosis and C-cell tumors of the thyroids in young bulls. all associated with calcitonin stimulation by the dairy milk production cattle diets also erroneously fed to the bulls, although considered normal by NRC recommendations.

The vertebral fractures were associated with the hardening and bridging bony exostoses (spondylitis) between fused vertebrae, resulting in vertebral fractures during mounting and sperm collection from the bulls.

A change in diet was an effective cure for the sheep and the bull stud mentioned.

On a recent trip to teach in Korea (2008), more very hard ribs were found that the author could not break by hand, but no history was available for explanation.

WP 929, 930

VII. Ovine and Caprine (Sheep and Goat)

O&Cap-6

Pulpy Kidneys

It has been in the books and literature for years that pulpy kidney disease (enterotoxemia) in sheep and other animals is due to *Clostridium perfringens* type D, but only one aspect is true in that connection.

Glucogenesis, associated with carbohydrate overeating, causes intestinal organisms' rapid proliferation in the bowel and produces the glucose involved. When it is absorbed, it increases the glucose level in the blood. When the sheep dies of the disease, usually suddenly, the high glucose level in the renal tubules is immediately available as an energy source. Enzymatic enhancement with rapid autolysis of the renal cortex, called pulpy kidney, occurs soon after death.

We see a similar condition commonly in foal necropsies, in which the entire cortical portions of both kidneys may be soft and pulpy, and are greatly reduced in size, while the medulla is more or less normal. This is also considered a pulpy kidney, caused in most instances by the rapid autolysis of the cortex in association with glucose therapy just prior to death. The glucose in high levels has acted as the energy source for the enhanced autolysis.

An added suggestive proof for this is the finding of severe, acute infarcts of cattle kidneys which are considered to be at least several hours or days older than the time they

were given any glucose (sugar) therapy. These infarcted areas with red peripheral zones of inflammation had histologically better cellular detail hours after death than the remaining entire autolytic cortices because these renal cortices had access to the glucose therapy, while the vascular, blocked, infarcted areas did not receive the glucose.

It should also be noted that the tentative diagnosis of enterotoxemia (pulpy kidney disease) in sheep or cattle is often made by positive testing of urine for sugar.

WP 223, 866, 971, 972, 11786

VII. Ovine and Caprine (Sheep and Goat)

O&Cap-7

A Short Story regarding Sheep Stomach Worms

Hemonchus contortus, the barber pole worm, is the largest nematode in the abomasum of sheep. Many sheep harbor a few of these worms without clinical disease, but they can certainly kill the sheep when too many are present. Most of the time you may find them in the abomasum at the time of death. It is also possible that like "rats leaving a sinking ship," the prosector may not find any worms in the abomasum. There may be few, if any, present in the intestine because the adverse pH environment in the intestine will destroy them. The story above is a well-known one, but another common way the animal becomes involved is quite unique.

In the winter seasons at Cornell when their flocks were housed only part of this time, we were given one or more of their most robust sheep, which were severely anemic from hemonchosis. The affected sheep were the largest and the more aggressive ones of the flock, according to the shepherd. The animals were found dead usually following weekends in which temporary student help had been their caretakers. There was no easy answer, until a "farm visit" was organized to actually visit the barns involved. It was considered that maybe the straw bedding was moist enough to allow larval forms to crawl up and be eaten, but this did not explain, among other things, why the most aggressive animals were being affected. Further investigation showed that most pens with affected

sheep had the drinking water source from tubs in which the tub tops were at, or just above, the level of the bedding and thus easily contaminated with feces. This allowed eggs and larvae to develop in the tubs. These tubs were supposed to be dumped periodically and filled daily with fresh water, which was not being done by the temporary help. Thus the larvae concentrated at the bottoms of the tubs. With limited water, the dregs of water had concentrated numbers of larvae available to the more aggressive sheep.

Strict preventative care stopped these problems, by raising the water tubs higher and keeping them cleaned out and refilled periodically.

A similar problem has been recognized elsewhere, such as in pastures where drying up of water sources allowed the increase of parasites in these sources. When water was plentiful, only a few parasites may be ingested at one time, but with concentration, many more infective larvae would be ingested with each swallow.

WP 696, 697, 2089

VII. Ovine and Caprine (Sheep and Goat)

O&Cap-8

Goiter (Thyroid Hyperplasia, Hypertrophy)

Several cases of congenital goiter in aborted lambs have come to the necropsy room. They have swollen upper neck regions with firm, often massively enlarged thyroid glands. Many, not all, have hair-like wool instead of the normal curly wool usually seen. Some are almost hairless.

This region of Ithaca, at the south end of Cayuga Lake, one of the Finger Lakes of New York, is in the so-called goiter belt of the U.S.A. It is common that horses from this Ithaca area also have enlarged thyroids, as incidental findings in many equine necropsies.

The dams of these goiterous lambs have all been fed rape and/or kale, which are the more likely cause since these plants are known goiterogens. The reason for this story is bound to the fact that for most of my training period, I was told, learned, etc., that goiter was of two types; one hyperplastic, with many more cells than normal, and the other was hypertrophic. with each thyroid cell being larger than normal and that the cause(s) were different for both. However, we have had several cases of aborted twin lambs in which one had hypertrophic and the other hyperplastic goiter. We cannot explain this in twin aborted lambs and are still without an answer.

WP 843, 844, 845, 3326, 6119, 6194, 11056, 11058, 15519, 18055, 20862

VII. Ovine and Caprine (Sheep and Goat)

O&Cap-9

Functional Hydronephrosis

Dilated ureters and dilation of the renal pelvis in both kidneys, called hydronephrosis, without explanation is often seen in these young animals from a few days to a few weeks of age, all over the world.

It has been seen mostly in lambs and fawns, especially when orphaned early. The usual causes, such as bacterial renal or bladder diseases, urethral calculi, or other obstructive causes, are not present.

This type of hydronephrosis without obstruction is also seen in Finn sheep lambs, with the common entity in them of congenital immunoglomerulopathy commonly seen in this breed. Certainly these lambs have bilateral glomerular disease, but why the associated hydronephrosis? Also, these animals are not usually orphaned. The partial explanation for the presence of the mild hydronephrosis seen in these Finn lambs is that the urine must taste abnormal to the dam and she will not stimulate the genitalia by licking. The cause in the orphaned young is also obvious, there being no dam to stimulate urination.

A similar problem is seen in other species, such as the fawns that are orphaned early and are raised by hand by good Samaritans. The animals do not die from this obstruction, and the hydronephrosis will resolve spontaneously.

An explanation that best explains this entity is suggested by pouch-related species, such as the opossum and marsupials in general.

The young in the pouches may be attached to the individual mammary glands or teats in the pouches for the early part of their lives, and they are in a relatively clean environment since the dams must stimulate both urination and defecation by use of their more or less warm, wet tongue which in effect keeps the pouch clean. The same mechanism is apparently served by the deer doe that lick the genitalia and anus to stimulate both functions in their fawns. This prevents predators from following the fawns' trail.

An additional use of this reflex is employed by many veterinarians to stimulate cows to urinate, in order to collect a urine sample for ketone testing. They accomplish this by rubbing the vulva with a warm, wet cloth.

WP 855, 856, 9206

VII. Ovine and Caprine (Sheep and Goat)

O&Cap-10

Abdominal Wall Hernias in Lambs

Cornell has a strong research endeavor in sheep husbandry under several brilliant researchers including Dr. Douglas Hogue. It has been a very good cooperative effort working with him. He brings animals to our necropsy service from his large flocks, both experimental and natural, and can answer many of our questions concerning sheep.

This case is one of several perinatal lambs brought in for necropsy. It had swollen, dark, soft masses in the inguinal region on one or the other side. Most masses were 10–20 cm in diameter or larger. The mass was easily palpated and considered to be in the subcutis of the affected region.

Most of the lambs were dry and clean, having been cleaned by the dam after parturition. At necropsy the dark discoloration of the mass was found to be due to the massively enlarged, meconium-filled, displaced blind end of small intestine, but not any of the large intestine. The inguinal area and inguinal ring were distended and torn, allowing the greatly distended bowel to herniate through the abdominal wall into the subcutis.

Continuing, the necropsy showed the distal bowel, colon, and rectum to be very undeveloped compared to normal and were completely empty from disuse, all the way forward to the level of the cecum and the small 8–10 cm of the distal blind segment of

ileum. The forestomachs were essentially normal, but the bowel became increasingly distended with meconium (fetal feces), ending in a pouch of enlarged blind small bowel (about 8 cm in diameter) not attached continuously to the remaining bowel as described above.

This affected bowel was diagnosed as an example of segmental aplasia, a common malformation attributed by some to a genetic problem. The herdsman had noted several more cases from a specific sire.

The pathogenesis of the hernias that followed the development of the aplastic bowel is that at the time of parturition, progressive compression of the fetus, with its abnormally distended bowel, was forced through the inguinal ring of the fetus during birth canal passage.

WP 941, 942

VII. Ovine and Caprine (Sheep and Goat)

O&Cap-11

"Meow"

The author spent a major portion of one sabbatical year (for professional growth) in the beautiful country of Argentina, doing many autopsies and also teaching pathology using Kodachrome slides, with discussions of the lesions shown.

One of the more interesting cases came through the university service, following a phone call in lambing season. The Pathology Department receptionist was told that a farm had many dead lambs and some affected but live ewes, and they needed advice. The history continued from the owner, and the veterinary practitioners involved said that it looked like an allergy to plants or a toxic problem because of the swollen lips and faces in both lambs and adult ewes. Because it is not a common finding in animals to have both the dams and young affected and dying of the same allergic condition—or any common condition—we were not able to help over the phone. We advised them to bring in the dead or affected animals for a necropsy. Affected but live animals are, of course, more valuable for diagnosis than only dead animals with the same disease, since we would have fresh blood and other samples available for ancillary diagnostic testing. Initially, they were reluctant to bring any animals in for necropsy examination, but as animals continued to die they finally brought in four dead lambs and three ewes with swollen faces and heads.

In speaking with the owner and the veterinarians on their arrival and with their original diagnoses still in mind, our cursory examination suggested the problem was more traumatic in nature. Necropsies were started on the dead lambs. (Necropsy = dissection examination of the animal, while autopsy usually refers to a dissectional examination of a human.) A few hemorrhages were noted scattered in the subcutaneous tissues of the head and face, with irregular puncture-type openings in the skin. Necrotic and infected debris was mostly associated with ragged 1–3 mm tears in the skin. Further dissection revealed fractures of the various bones of the skull and jaw.

The first lamb necropsied had several fracture holes in the facial bones and one in the jaw. The severity increased incidentally in the remaining lambs, to more severely fractured bones and even both jaws being fractured in several places. The entire skull was fractured in the last one, so that the entire head was bent in the middle, with the brain exposed subcutaneously. Hind limbs on most showed subcutaneous hemorrhages and some with small, 2–3 mm skin perforations. Only a few front limbs had similar lesions.

The owner refused to accept that it was trauma with the first dead lamb findings, but one of the clinicians said it could be. After the necropsy of all the lambs, two of the veterinary clinicians agreed that it was probably caused by a large cat that the veterinary pathologist doing the necropsies had asked about even before starting the necropsies. The owner was still adamant that it was not possible since he and his farm personnel had not seen any such animals in years, anywhere in the neighborhood.

Pictures were taken during the necropsies and cultures made when considered necessary. The three ewes were alive but unable to eat properly. They were euthanized and necropsied the next day.

The owner went home dissatisfied with the diagnosis given for the lambs. The necropsies performed on the adult ewes the next day demonstrated traumatically torn lips, faces, tongues, and ears, along with more subcutaneous hemorrhages in limbs than were found in the lambs.

After the completion of the necropsies of all the animals, the report to the owner included such elucidating information that wolves and big cats often help incapacitate their prey by biting and holding the noses and mouths of their prey, together causing crippling suffocation that helps weaken the prey. It is also known that domestic cats often play with their prey before killing it, and apparently they do that at times even without the intention of eating it. Their "gifts" are often found whole near the door to the back porch.

Over the next few weeks, several phone calls indicated that the owner was still a nonbeliever. Radio news and newspaper reports mentioned several pumas having been seen and killed at sites away from the affected farm, but the owner was still not convinced. Weeks later, several of the involved veterinary practitioners came to the university to take a continuing pathology course, and they said that during the several weeks of the outbreak the owner lost over 150 lambs (killed), and over 15 adults had to be killed because of similar injuries. They also told us that much later the farmer and his friends had a roundup hunt, or drive, to kill the pumas that were in the vicinity. To everyone's surprise, probably the owner's mostly, they killed 16 adult pumas. When the investigation was over, it was concluded that the farm probably had a mother puma teaching her young how to hunt.

After this outbreak the owner actually called the university and said "Meow" over the phone. The Canadian lynx has the same effect with caribou herds, which this pathologist had worked on 20 years earlier.

WP 816, 818, 890, 891, 892, 894, 13628, 16836, 16837

VII. Ovine and Caprine (Sheep and Goat)

O&Cap-12

Chronic Copper Toxicity

Chronic copper toxicity is a common problem that has been seen in sheep and in other animals. It has been a difficult problem to solve. Many individual sheep have severe icterus while alive and, at necropsy, normally white tissues are distinctly yellow. A yellow, fatty liver and dark "gun metal" (dark brown) kidneys are usually seen at necropsy.

At one time, before going to South Africa, the author was asked to bring the latest information we might have of the above problem. I was not able to do so. Similarly to Cornell, they had many animals with the problem, but with no increase of copper in the liver or kidneys, neither in the cortex nor in the medulla, as is present in many cases of known copper poisoning.

Common sources for copper poisoning are copper sulfate drenches for parasites and exposure to footbaths by sheep eating the grass near such footbath treatment areas. In sporadic cases, usually in individual pet sheep or small flocks fed horse or even cattle grains, the commercially higher copper content than found in sheep feeds may be at fault. These poisoning cases were usually easily diagnosed by the toxic copper levels in the liver and in the kidney cortices.

No suggested cause for the classical clinical signs and necropsy findings without elevated copper levels had been available until a graduate student, Dr. Catharine Wilhelmsen, at Cornell, fed a group of sheep a very low level of copper for an extended period. After this time, the sheep showed a great increase of a copper protein complex, by way of Coombs positive blood test soon after an episode of stress with fatal icterus developing, but without evidence of a significant increase of liver or kidney copper. The stress reaction, along with chronic poisoning with low levels of copper, may be a partial answer to these cases without excessive liver or kidney copper levels.

WP 1377, 6131, 10362, 11812 , 12211, 12412, 12812

VII. Ovine and Caprine (Sheep and Goat)

O&Cap-13

Pulmonary Hypoplasia

Travel certainly helps round out experience. This pathologist had another sabbatical leave from the university, to visit the Union of South Africa, the Theiler diagnostic lab and University of Pretoria, Onderstepoort, South Africa. During these months Drs. Pienaar and Koos Coetzer showed and discussed with me their beautiful Kodachrome collection of cases, and I did the same with my travel collection. We had the opportunity to do necropsies in the afternoons.

One of the great lesions seen was in lambs that had died at birth with a very small, underdeveloped lung. All other tissues, except for the brain, were normal size by comparison. It was of special interest to me, since we had seen small lungs in the Cornell lab without an acceptable explanation. It turned out that the Onderstepoort lambs died from Wesselsbron disease, which is endemic in South Africa. Liver lesions of necrosis and also brain lesions of cerebral and cerebellar hypoplasia are seen. These are apparently part of the complex of CNS hypoplasia, resulting in hypoplasia of the lungs, as reported in the literature.

Overall it was great working there at Onderstepoort. Faculty in the primary facility were willing to freely share their information of diagnostics in their whole country. It was a fantastic experience to see such an abundance of pathology all in one place, with all the

necessary scientists and their well-shared facilities available as needed. A similar educational experience was also shared in Bairnsdale, Australia, during my year there.

WP 10123, 13544

BLURB

Mistakes and misinterpretations are the author's only, working professionally over fifty years with undergraduate and graduate veterinary students both grossly and microscopically. Most have not been publicized except as class instructions and formal presentations.