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PATHOLOGY OF FEEDLOT CATTLE

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ABOMASAL LYMPHOSARCOMA

Irregular thickenings of the mucosa with no or variable degrees of secondary ulceration. Abomasal lymph nodes should be enlarged and lesions in other tissues common. In young feedlot cattle, lymphosarcoma can be sporadic or due to bovine leukosis virus.

ABOMASAL PARASITIC HYPERPLASIA AND HYPERTROPHY

The fundic mucosa of chronic abomasal parasite infections can be thickened with a 'cerebriform' like appearance due to hyperplasia of gastric gland epithelium. Chronic ostertagiosis for eg, often does this. Abomasal lymphosarcoma also needs to be considered but lymphadenopathy or lesions in other tissues should also be found.

ABOMASAL ULCERATION

Commonly seen in various sizes, numbers and distribution in the abomasum in feedlot cattle. Often an incidental finding with no good explanation found. Can be very small, hemorrhagic and acute or can be large, irregular, pale and chronic. Most of the time BVDV is suspected but a workup on large numbers of cases often does not confirm this. The abomasal mucosa is thin with minimal support stroma and focal ulcers can occur easily, due to increased gastric acid output, which occurs in stress. These often hemorrhagic in acute stages, until granulation tissue production occurs in the depth of the ulcers. Do not perforate in feedlot cattle as they do in young beef calves on pasture.

ACTINOBACILLOSIS OR WOODY TONGUE

Various locations of the tongue firm and swollen. The cut surface shows multiple white to yellow, firm, often circular foci which represent granulomas. Adjacent granulomas can become confluent. Centers of granulomas can have small yellow foci which are the sulfur granules. Easy to diagnose histologically without cultures.

ACUTE ABOMASITIS

More common in younger calves on pasture but occasionally hemorrhagic, necrotizing and emphysematous abomasitis with varying degrees of edema seen in feedlot calves, often co-existent with other diseases, including enteritis or colitis. Clostridial agents often isolated but difficult to establish if these are primary due to invasion through previously eroded or ulcerated

areas, or are just postmortem invaders. Can be seen along with necrotic omasal leaves and rumenitis lesions secondary to chronic or subacute lactic acidosis. Often idiopathic and no cause established.

ACUTE RUMINAL TYMPANY (BLOAT)

A difficult gross diagnosis to make in many cases due to co-existence of other diseases, lack of all of the typical expected changes and varying degrees of postmortem autolysis. In my opinion, gas type bloat does not kill cattle, only frothy bloat does. Unfortunately, in many cases the ruminal foam has dissipated by the time the postmortem is done or only small amounts remain and it is then difficult to decide. Classic changes include: 1) cranial carcass congestion and caudal pallor. 2)varying degrees of flank fold fascial and subcutaneous edema 3) liver and kidneys very pale 4) right heart often dilated with little ventricular clotted blood 5) lungs bilaterally collapsed and congested 6) tracheal mucosal studded with petechial or diffuse hemorrhages but no blood clots 7) typical bloat line at the thoracic inlet but a common finding in many conditions and should not be used to make the final decision 8) intense congestion of meninges, brain, paranasal sinuses with hemorrhage 9) enlargement and congestion of prescapular lymph node and small, very pale prefemoral lymph node. I routinely will take histo sections of especially caudal lung lobes and look for intense hyperconstriction of pulmonary vascular smooth muscle. This is not pathognomonic for bloat but is consistently present even in cases with lots of autolysis. In lends support to my bloat diagnosis. This is not found in textbooks of pathology or taught in pathology classes or lectures.

ANATOMIC FEATURES AND PATHOPHYSIOLOGY OF BOVINE LUNG

Anatomically (especially cattle) marked compartmentalization means that different pathologic processes can be going on in adjacent lobes and lobules. Pulmonary hypertension occurs easily in cattle and this has serious implications for heart function (see under congestive heart failure and cor pulmonale). Feedlot cattle lungs are a cesspool for infectious processes and the fact that cattle are put together from many different farms, with different vaccination status and with different levels of immune system function and exposure to many new agents, means it is not surprising that respiratory disease is so common. Most cattle arriving at feedlots are under severe stress of various types. Vaccination at this time should not be expected to be of much benefit under these situations or at least not in all individuals. CPPS has become one of our most serious feedlot disease syndromes in recent years and the lungs are always involved to some extent. Chronic bronchiolitis due most often to viral infections is very common in most feedlot cattle and this has potentially serious implications for a variety of secondary lung diseases seen in feedlots, eg H. somnus bronchopneumonia, in feedlot interstitial pneumonias and in CPPS/Mycoplasma bovis lung infections. This is often seen grossly as a ‘bronchiolar pattern’ on cut surfaces of involved lung. This means tiny ‘miliary’ white or gray areas throughout especially the cranial-ventral areas are visible. Important when submitting fixed lung for histo that multiple dorsal, caudal, cranial and ventral pieces of right or left lung are submitted. These can all be put in one container for practical purposes. Ventilation/perfusion ratios likely are also important in the compartmentalized bovine lung and this plays a role in different processes occurring in adjacent lobules. This has not been studied in cattle thoroughly.

BOVINE PAPULAR STOMATITIS VIRUS
Usually when seen is of no clinical significance except to consider that the animal is likely immunosuppressed. Often seen in primary BVDV infections. Lesions most commonly on the hard and soft palate but if extensive, can be in the esophagus, on the tongue, and on the gingiva. In the esophagus are round or oval rather than typical linear BVDV lesions. Often appears as greyish or erosive circular areas with raised edges. Gray, outer raised rims common. Histo shows irregular, granular, basophilic inclusion bodies in squamous epithelial cell cytoplasm.

BRONCHIECTASIS
Means a cranial ventral lung distribution of abscess – like structures with purulent exudate grossly surrounded by a thin to thick fibrous-like wall. This is not a cause of death, it only needs to be interpreted at postmortem as suggesting chronic, slow-progressive bacterial bronchitis by pyogenic type bacteria. Represents airways, mainly bronchi, which have a chronic, slow-progressive growth of pyogenic bacteria that eventually destroys the wall of the airway in a centrifugal manner. May project as nodules above the surface of the pleura and the lung parenchyma itself is dark red to purple and collapsed. Anything that initiates a bronchitis and bronchiolitis such as PI3 virus, BRSV, IBR virus and perhaps other respiratory viruses can lead to bronchiectasis. These viruses are the most common initiators of bronchiectasis. *Mycoplasma bovis* infection in CPPS cases often start out as a bronchiectasis but continue to spread and adjacent lesions coalesce to result in larger areas of coagulation necrosis and sequestration. The wall of the airway is not as easily seen in CPPS bronchiectasis like it is with other pyogenic bacteria involved, such as Staph, Arcanobacterium and E coli. M. bovis should not be thought of as a pyogenic organism. These nodular lesions often can grow to 1 cm diameter or more.

BRSV PNEUMONIA
We see this much more commonly in cow-calf operations than feedlot calves. Occurs in two forms and are considered stages or phases of the disease. Seldom do calves with phase I develop phase II disease at all and most recover completely. Phase I is a syncytial cell bronchiolitis and most cases recover or are not clinical. The morbidity rate is high but mortality low. Phase II often occurs up to a month later and calves die suddenly or after a few hours of severe respiratory distress with an expiratory grunt. Phase I lesions are mainly cranial ventral, dark firm lung tissue with varying amounts of subpleural, bullous and interlobular emphysema. Histopathology can easily make this diagnosis and IHC or DIF easily can identify virus in airways and alveoli. Multiple sections of lung from various sites are important. Only one lung or the other needs to be sampled. Phase II lesions are essentially the same as feedlot interstitial pneumonia and areas of subpleural emphysema are the best areas in which to still find syncytial cells to allow the pathologist to confirm the diagnosis. The mechanism of interstitial pneumonia due to this virus is unknown but experimentally a combination of 3 methyl indole and BRSV causes more severe pneumonia than BRSV alone. However, the typical feedlot AIP lung has still not been experimentally reproduced to my knowledge.

BVDV INFECTIONS
In our experience with almost 15 years of having IHC available for use on formalin fixed tissues, primary BVDV infections in the feedlot, with or without secondary infections, is far

more common than persistently infected (PI) cases, with or without mucosal disease. The PI animal however, is undoubtedly the source of infection for the primary BVD cases. We have been fortunate to NOT having experienced the acute primary BVDV infections experienced in Ont. And Que, in the early 90s. Grossly and histologically, these apparently cannot be distinguished from PI animals with mucosal disease. A hallmark feature of primary BVDV histologically is varying degrees of arteritis or proliferative arteriopathy in heart, lung,and sometimes other tissues and these are often the only sites where IHC for viral antigens is positive. We do not see arteritis in PI animals except rarely. Viral lymphocytic and necrotizing myocarditis is also common in these cases if it is looked for. Gross lesions give you no clue to this syndrome, unless there are obvious CPPS lesions, with *Mycoplasma bovis* in lung and/or joints. In addition to, or instead of CPPS, unusually extensive lesions of many commonly occurring bacterial and viral infections in feedlots should be considered to be predisposed to by primary BVDV infections, as a result of severe immunosuppression. For eg, severe mannheimiosis later in the feeding period than expected, extensive papular stomatitis or IBR lesions should be suspected of co-existing primary BVDV infection. Do not expect to find gross lesions of BVDV in these cases. Multiple sections of heart, lung and ileum are still the best tissues to examine in these cases.

BVDV LESIONS OF GASTROINTESTINAL TRACT

Typically the PI animal that dies of mucosal disease has necrosis/ulceration/hemorrhage over Peyer's patches plus lesions in other sites eg oral cavity and esophagus. Not all feedlot cattle that are PI however die of mucosal disease- these cases may have much milder to no typical MD lesions and die of other infectious diseases such as mannheimiosis, hemophilosis or even coccidiosis. Best way now to confirm or rule out a PI animal is submit a piece of skin from anywhere on the body for IHC for BVDV. Submit these in formalin only. Our IHC lab does five animals on one slide for $45. Another type of mucosal disease is called chronic mucosal disease and these will often show only mild esophageal and oral lesions if any at all and often chronic intestinal lesions only. The PP important to examine carefully in these cases. These will still be positive for PI on skin IHC. Primary BVD lesions in the intestinal tract are usually only severe atrophy of PP, a histo diagnosis only, or die of some other secondary bacterial, or fungal infection. Most primary BVDV cases are lung viral or bacterial infections with no intestinal lesions. Areas of depigmentation of ruminal or omasal mucosa is often due to chronic BVDV in PI individuals. Abomasal ulcers also occur in some PI cattle, but not all abomasal ulcers are due to BVDV.

CHRONIC INTERSTITIAL NEPHRITIS – "WHITE SPOTTED KIDNEYS"

Most often seen as an incidental finding in calves up to four months of age. Seldom manifest as clinical urinary disease; often an incidental finding at necropsy, except in younger calves on pasture or still in calving pens, where it can be seen so severe that chronic renal failure occurs. Lesions are due to a previous bacteremia, thought to be most often E. coli. Often randomly distributed, raised, firm white foci and often dramatic grossly. Lesions are often misinterpreted as neoplasia.

CHRONIC PASSIVE CONGESTION (CPC) OF LIVER

Stands for chronic passive congestion and suggests right-sided heart failure. The cut surface of a nutmeg shows a similar mottled picture, thus often described as 'nutmeg liver.' Mottled

appearance with a mixture of congested or red and paler areas. The red areas represent areas where hepatocytes have usually been lost due to necrosis and sinusoids are dilated and the paler areas represent viable liver tissue that shows varying degrees of lipidosis or just degeneration. If very chronic, some fibrosis can be demonstrated. In feedlot cattle, most often associated with cor pulmonale due to chronic lung disease and subsequent pulmonary hypertension. The lung should always be examined carefully, even if it is a normal color. A fibrotic lung is a common cause and can be a normal color for example.

CLOSTRIDIAL MYOSITIS - BLACKLEG

Most veterinarians recognize the characteristic odour at necropsy. If suspect it, and don't see typical gas bubbles and dry, dark muscle tissue anywhere, don't forget to carefully examine the heart, diaphragm and tongue muscles. See under heart lesions. Send in suspect muscle for histo and pieces of fresh muscle for Clostridial sp DIF. Autolysed carcasses will often show positive for Cl septicum by DIF, but not Cl chauvoei.

CONGENITAL PORPHYRIA

In feedlots most likely to be an incidental finding at necropsy but some cases will manifest clinically with cutaneous photosensitization. Bones and teeth are brown in color and urine may have a brown color as well. Woods lamp results in red fluorescence of teeth and bones – dramatic. Most common in shorthorns and crosses of. Rarely in Holstein cattle.

CONGESTIVE HEART FAILURE

Right sided heart failure in feedlot cattle often shows ascites and/or a CPC = 'nutmeg' liver. The right ventricle usually shows hypertrophy of the myocardium and is dilated as well. In feedlot cattle chronic and extensive lung disease is often responsible, which results in severe pulmonary hypertension. Cattle are particularly prone to pulmonary hypertension and right-sided heart failure due to chronic lung disease is known as cor pulmonale. Left sided heart failure in feedlot cattle is manifest as passive congestion of the lung. The lung is edematous, non-collapsed and mottled due to hemorrhage into alveoli. Interlobular lymphatics are often stuffed with blood. The most common cause is *Haemophilus somnus* myocarditis or sequestra/infarcts due to this organism. Vegetative endocarditis of the left A-V valves or aortic valves can also be responsible.

CPPS – CHRONIC PNEUMONIA AND POLYARTHRITIS SYNDROME

Mycoplasma bovis pneumonia and in about 10% of cases, single or multiple joint M. bovis arthritis/tenosynovitis as well. If looked for histologically and immunohistochemically, primary BVD infection is commonly found and likely makes the lesions more extensive via immunosuppression. Extensive antibiotic therapy may also promote M. bovis growth and make the lesions more extensive. The BVDV lesions may include vasculitis or chronic proliferative arterial lesions in arteries in heart and sometimes lung, severe atrophy of Peyer's patches of ileum and occasionally focal areas of lymphocytic myocarditis. Immunohistochemistry (IHC) will often but not always, confirm virus in these arterial and myocarditis lesions. The amount of viral antigen remaining in tissues is often very small and smooth muscle of vessel walls seems to be its last 'hangout.' The lung lesions vary tremendously in extent and severity, starting as growth of M. bovis in bronchioles and bronchi then coalescing into large areas of yellow caseous like necrosis with invasion and spread via interlobular lymphatics. Concurrent

fibrinous pleuritis also common. Lung lesions can be mixed with other bacterial or viral infections, especially *Mannheimia hemolytica* pneumonia, especially if recent antibiotic therapy was not carried out. M. bovis best confirmed in the suspect lesions by IHC on formalin-fixed tissue. M. bovis can be isolated from most feedlot lungs, so cultures alone do not confirm the diagnosis.

EOSINOPHIILIC MYOSITIS AND MYOCARDITIS

Caused in most if not all cases by sarcocystis spp. Multifocal areas of yellow-green discoloration in heart and or skeletal muscles, often an incidental finding at slaughter or during necropsy examination. Sarcocystosis cannot always be proven, even by histopathology. Presence of large numbers of eosinophils result in the green discoloration of the lesions. Appear not to interfere in skeletal or heart muscle function.

EOSINOPHILIC MYOSITIS

Usually an incidental finding and of no clinical significance. Either large patches or small, multifocal round areas of yellow to green discoloration in the muscle tissue. Often in heart as well (see myositis under heart slides). Due in most if not all cases to sarcosporidia sp but this cannot always be confirmed, even by histopathology. Of aesthetic importance only in slaughterhouse carcasses or farm-butchered animals. Green discoloration is due to eosinophils.

FEEDLOT INTERSTITIAL PNEUMONIA OR "AIP"

Commonly seen in feedlots and often in animals that are nearly finished or at least late in feeding periods. Lots of cases seen in animals in earlier feeding periods as well. A poorly understood entity is an understatement. Should be thought of as a syndrome rather than a distinct disease entity since likely a variety of and mixture of causes can lead to the similar end-stage lesions. Important to realize that the meaty, brown colored, rubbery lobules seen most consistently in the caudal lung lobes is a combination of hyaline membranes and pneumocyte II hyperplasia and occurs because of acute necrosis of type I pneumocytes, the flat lining cells of the lung alveoli. Epithelial cells of terminal airways-bronchioles are involved in this process as well. It is cellular proliferation and the protein content of the hyaline membranes and alveolar edema fluids that makes the lung so heavy. Edema and emphysema are features of but varies in severity tremendously from case to case. Finding emphysema alone in a bovine lung should never be the criteria for calling a lung interstitial pneumonia since it can occur rapidly during terminal respiratory exertion even in normal lungs. This is true for interlobular and bullous emphysema, but not subpleural emphysema. Typically the lung is rubbery throughout most lobes but most consistently in caudal lobes. The lungs are mottled due to intermingled meaty, firm, lobules and yellow, aerated lobules which appear more normal. Airway exudates not seen on cut surface unless concurrent bacterial bronchopneumonia also present but a 'bronchiolar pattern' can be present and extending back into the caudal lobes, especially if BRSV is suspected as a predisposing or sole cause. This bronchiolar pattern is usually seen histologically as bronchiolitis obliterans. In my opinion, 3 methyl indole from rumen fermentation of tryptophan is partially responsible for the pneumocyte I necrosis but the lung is predisposed to this by some previous or concurrent lung disease, especially bronchiolitis causing entities, such as BRSV. The compartmentalization of the bovine lung and aeration/perfusion ratios likely also play a role but the disease has not been experimentally reproduced to be able to determine the exact mechanisms. Pulmonary hypertension with cor

pulmonale is often the cause of death but in acute onset cases, simple hypoxia from respiratory failure also is important. If you are going to submit fixed lung for histologic examination, important to take at least five sections from upper, lower, cranial and caudal parts of one or both lungs. If tiny bubbles of subpleural emphysema are visible, important to take sections from these areas, since they give us the best chance of finding BRSV on IHC.

FORESTOMACHS - HEMORRHAGE OF SEROSAL SURFACES

A 'popular' site for acute extensive hemorrhages of a thrombocytopenic BVDV is the serosa of forestomachs. The abomasal mucosa can be extensively involved as well. When suspected, fixed tissues should include ileum, heart, kidneys, for evidence of BVDV in blood vessels or Peyer's patches. Differential diagnosis is rupture of a serosal artery or vein of the rumen due to hardware penetration but more common in dairy cattle than feedlots. Other causes of hemorrhagic diathesis also need to be considered such as feeding sweet clover and Simmental thrombocytopathy. DIC due to some systemic toxemia or septicemia/bacteremia also needs to be considered.

HEMOGLOBINURIA

Results in port-wine type urine and kidneys are variably brown to black in color. Consistent with RBC breakdown, resulting in free hemoglobin in the blood and this easily passes through the glomeruli. Causes in feedlot cattle may include chronic internal bleeding due to a thrombocytopenic BVDV, Clostridium hemolyticum infection, leptospirosis and chronic copper toxicity. In Simmental cattle, a hereditary thrombocytopathy disorder can be responsible. Entities that result in intravascular hemolysis most often are involved.

HEPATIC FASCIOLIASIS

Not common in feedlot cattle but *Fascioloides magna* can cause various sized cystic areas containing a jet black pigment with trematodes being difficult to find. Lesions often seen at slaughter and more common in adult cattle or game farm elk.

HISTOPHILUS SOMNI MYOCARDITIS

Most consistently results in areas of acute to chronic necrosis of the left cranial and caudal papillary muscles but multifocal pale foci of necrosis may occur throughout especially the left myocardium as well. If chronic, the areas of necrosis may show a zone of purulent material between the necrotic zones and viable myocardium, and this process is called sequestration and the central free necrotic mass is then known as a sequestrum. Sometimes the sequestrum will be pulled off into the ventricle lumen by the chordae tendinae of the A-V valves. Histologically, the pathogenesis appears to be embolism, but the source is not always apparent, ie, there may be no pleuritis or ITME lesions. Often coexists with a chronic suppurative bronchopneumonia and H. somnus can be cultured from the lung and heart lesions if antibiotic therapy has not recently been extensive. IHC can be used to confirm the diagnosis and is done on formalin fixed tissue. Occasionally co-exists with Haemophilus pleuritis but not common. The lung may or may not show passive congestion, depending on whether or not the animal showed congestive heart failure. Sometimes the animal is found dead with no history of treatment or congestive heart failure signs. Acute lesions are hemorrhagic, as in blackleg, and careful examination of the papillary muscles are needed to make this diagnosis. Acute fibrinous pericarditis can also be present, but never without myocardial lesions also being

present. Sometimes there is vegetative mural or valvular endocarditis lesions as well as myocardial lesions. Debatable whether or not vaccination reduces the incidence of.

HISTOPHILUS SOMNI MYOSITIS

Occasionally and often along with ITME lesions, large areas of pale necrotic muscle tissue seen, most often in thigh muscles. The lesions are quite will defined and vary a lot in size. May or may not have any hyperemia zones around them, often do not. Confirm by histo only and cultures not necessary unless no histo service available. Important differential is a site of antibiotic injections.

HONKER'S SYNDROME

Acute onset of respiratory distress with stertorous respirations or sudden death in respiratory distress. Severe submucosal edema and/or hemorrhage of the cervical trachea along with congestion, hemorrhage and edema of peri-tracheal tissues. Usually lesions not caudal to the thoracic inlet. Occurs in cattle nearing market weight and no predisposing causes known. Mechanisms of edema, hemorrhage unknown. Extensive histopathologic and immunohistochemical workup has not supplied any answers as to cause. Some cases have an inflammatory component and squamous metaplasia of the tracheal epithelium is occasionally seen. The significance of these findings is not known. Significant lung disease usually lacking.

HYDRONEPHROSIS

Urolithiasis the most common cause in feedlot cattle but usually not severe and not the cause of death. May be unilateral and not significant as a cause of death if the other kidney is normal. The contralateral kidney will often show compensatory hypertrophy if this situation occurs. A congenital form is often seen in feedlot cattle and can be so extensive that chronic renal failure occurs as the animal grows. Severity varies from dilatation of renal pelvic areas only to loss of medullary tissue to loss of all of medulla as well as much of cortical tissue. Lobules in cattle kidneys can be variably involved, especially in the congenital form. Concurrent ureter dilatation often seen.

INFECTIOUS BOVINE RHINOTRACHEITIS (IBR)

Most common is typical upper respiratory tract disease with rhinitis, conjunctivitis. Postmortem typical reveals fibrinous tracheitis, bronchitis and laryngitis. Severe cases and especially concurrently with primary BVDV infection, will see focal ulcerative stomatitis, esophagitis and even rumenitis. Lesions in the esophagus and rumen will often be white and raised rather then ulcerative or erosive. Typically a necrotizing bronchiolitis with secondary bacterial bronchopneumonia also present and viral inclusion bodies and positive DIF or IHC in the cranial aspects of the lung is present. We have seen outbreaks of pneumonia in beef calves on pasture and the diagnosis was initially missed because the pneumonia was more severe and striking than the laryngitis/tracheitis or the connection between the two was not made. Lung involvement needs to be differentiated from necrotizing laryngitis and aspiration pneumonia from other bacterial causes. Rarely IBR encephalitis seen without upper respiratory tract lesions.

INFECTIOUS THROMBOTIC MENINGOENCEPHALITIS – HISTOPHILUS SOMNI

ITME form of H. somnus infections seems to be on the increase again in western Canada and multiple acute deaths over a week or ten days time in a pen not uncommon. Do not always see tiny hemorrhagic or necrotic foci grossly and can be diagnosed only in leptomeninges by histopathology. In these acute septicemic cases, lesions of thrombophlebitis can also be seen in most other tissues submitted for histopathology. In the above situations however, there will always be a few joints with mild fibrin chunks or strands seen, and joint fluid mildly increased and sanguinous or cloudy. Most cases of H. somnus pleuritis do not have ITME lesions but a few cases of acute myocarditis cases do. Chronic myocarditis cases will not have ITME lesions. Whenever a case of ITME occurs, not uncommonly see bilateral laryngeal necrosis/ulcers/erosions and the typical thrombophlebitis can be seen in these lesions on histo. We use IHC to confirm H. somnus in formalin fixed tissue, especially in treated cases where cultures usually negative.

LARYNGEAL ULCERS, NECROSIS AND EROSIONS

Commonly see laryngeal lesions in feedlot cattle at postmortem and especially those with lung involvement. Usually secondary or develop concurrently with lung disease but once ulcerated and if laryngeal ulcers are secondarily infected, can seed the lung with a gangrenous pneumonia due to a mixture of gram negative and positive anaerobes, or an aspiration type *Arcanobacterium pyogenes* pneumonia. H.somnus septicemia cases often results in thrombophlebitis in the larynx with acute vocal fold necrotic lesions. These are always bilateral and can be quite small initially. Severe coughing can probably initiate laryngeal erosions just due to the vocal folds slapping together and these are called contact ulcers. *Mycoplasma bovis* likes to grow in laryngeal ulcers and these progress in size once so involved. This is because the organism will grow readily in tissue damaged by some other organism or mechanism, at a time when they are present in the bloodstream (bacteremia).

LIVER AUTOLYSIS

The liver may show autolysis without other tissues being similarly involved, especially on feedlot rations. These are variably sized putty-colored areas, often surrounding vessels but larger, diffuse areas may be present instead. These are due to postmortem overgrowth of Clostridium sp. The borders can be very distinct but note there are no surrounding zones of hyperemia, as seen in hepatic infarcts. If these areas are submitted for culture, Cl septicum will usually be isolated but this is not significant.

LIVER INFARCTS OR COAGULATION NECROSIS

Hepatic infarcts are often dry, putty colored or yellow areas of necrosis surrounded by narrow zones of hyperemia. Of various sizes and randomly scattered throughout the liver. These are either due Fusobacterium necrophorum and arise embolically from an ulcerative rumenitis or, and even more often in our experience, are due to fungal elements invading vessel walls and causing thrombosis of hepatic arteries and veins. These are originating from ulcerative lesions somewhere in the GIT and often no rumenitis lesions are found. The history often includes extensive antibiotic therapy and if one goes looking for the lesions, BVD vasculitis in heart and other tissues is often found. Immunosuppression therefore, often plays a role in feedlot cattle. Necrosis of leaves of the omasum is not an uncommon finding and can be the source of the fungal elements. Members of the Zygomycetes group Absidia, Rhizopus or Mucor are usually

involved. Histo alone can differentiate between these two etiologies of F. necrophorum and mycoses. Occasionally large single infarcts are seen which can be due to Cl. hemolyticum but hemolysis and hemoglobinuria are usually present as well, resulting in hemoglobinuric nephrosis.

MANNHEIMIOSIS – SHIPPING FEVER PNEUMONIA

Pasteurella hemolytica now called *Mannheimia hemolytica* and typically causes a fibrino-necrotizing pleuropneumonia. Cases which occur after about one week on feed often have concurrent primary BVDV infection resulting in severe immunosuppression. Begins as a bronchopneumonia and passes through stages of acute bronchopneumonia with dark red, swollen consolidated lung tissue and mottled with pale areas of acute coagulation necrosis on the cut surfaces. More swollen, consolidated lung that is dark red and hemorrhagic on cut and pleural surface with venous thrombi visible is just a more severe and peracute form. Later stages and with antibiotic use, many areas will become paler and necrotic, not dissimilar to the lesions of necrosis caused by Mycoplasma bovis and the two can be mixed together as well. The amounts of fibrin on the pleural surface will vary tremendously but usually is abundant and this fibrin is throughout the interlobular spaces as well on the cut surfaces. More chronic and treated cases will have whole lung lobes with gray consolidation and early stages of pleural fibrin organization. We can confirm the presence of the organism in fixed tissue by IHC and differentiate it from Haemophilus somnus, Mycoplasmosis and other bacteria. Past multocida causes only a suppurative bronchopneumonia, especially in cases where there is previous bronchiolar damage. I have yet to see what I would call a primary Past. Multocida bronchopneumonia and even if I cultured this organism, I would not consider it as a cause of death.

MENINGITIS – BACTERIAL

In feedlot postmortems the meninges can only be evaluated properly if the brain is removed by using an axe or meat saw and cutting the head down the center longitudinally and taking out the brain in two intact halves. The cerebellum surface often the easiest to evaluate since the surface of the folia will have an opaque appearance in cases of mild fibrinous meningitis. The ventral region along the brainstem is another popular place to visualize mild meningitis grossly. Sulci between gyri also need to be examined for white exudate. Make sure an abscess in the pituitary region has not ruptured as a cause of the meningitis. H. somnus/ITME can cause a mild meningitis but it often is not visible grossly. Cut transversely across the caudal third of cerebrum and see if any purulent exudate in the lateral ventricles. If you suspect meningitis, submit for histo if nothing seen grossly-often it is present on histo only.

MYOCARDIAL BLACKLEG LESIONS

A high percentage of blackleg cases may have heart involvement and varying degrees of fibrinous pericarditis/epicarditis is often the first clue. Cut surfaces of the left ventricle will often reveal dark red hemorrhagic areas of necrosis, very similar to the acute lesions of *Haemophilis somnus* myocarditis. Involvement of the papillary muscles is common, similar to hemophilosis. Easily differentiated by direct immunofluorescence for Clostridium spp and/or by histopathology from hemophilosis. The odour of the opened carcass usually characteristic of blackleg but this is sometimes missed when a necropsy is done outdoors.

NECROTIZING ENTERITIS AND ENTEROCOLITIS

Gross evaluation of the intestinal tract is difficult, fraught with artifacts, postmortem change, and state of hydration of the carcass. Coloration changes depending on cause of death also important and can vary along parts of the intestinal tract. Postmortem breakdown of RBCs, release of hemoglobin and hemorrhage into the gut lumen also occurs postmortem quite easily. Most infectious enteritides involve the ileum and colon especially in feedlot cattle. Dehydrated animals often have thick mucus over the mucosa which can look like necrosis. Important to evaluate serosal surfaces of especially ileum and lower jejunum to see if the Peyer's patches stand out either as being dark, pale, raised, ulcerated or perhaps hyperplastic. Remember the PP are along the anti-mesenteric side of the gut wall and they are continuous in the ileum and discontinuous in the distal jejunum so often easiest to evaluate in the latter area. Tie of loops of bowel for culture, but never when putting into formalin. Necrosis of gut mucosa can occur by the normal gut flora if antibiotic therapy or some other disease process such as a virus infection, has breached the mucosa or altered the normal gut flora. When submitting for histo, make sure multiple sections submitted to include spiral colon, ileum, jejunum proximal and jejunum distal. Do not need to identify these separately in different containers if not convenient to do so. Histopathologic evaluation of the intestinal tract and especially small intestine, is difficult if animal has been dead for a few hours due to villus epithelial sloughing, but this is more important in young calves than feedlot cattle. The large intestine does not autolyse near as readily.

OMASAL NECROSIS

Extensive necrosis of omasal leaves is not uncommon in feedlots and a cause is not always found. The fact the leaves have a free border means they are sensitive to ischemic damage, such as fungal invasion and vascular thrombosis, cardiac insufficiency, BVDV damage to epithelium and/or blood vessels and secondary to ruminal acidosis. Disseminated intravascular coagulation = DIC can be responsible as well by blocking small blood vessels. If a history of extensive antibiotic use, mycotic vasculitis would be my first differential. Unfortunately, the omasum is seldom opened at postmortem. The omasum is often not enlarged at all in such cases.

OSTEOCHONDROSIS – OCD

Cattle most often involving distal condyles of femur with chronic ulcerative lesions or flaps of cartilage lifting off. Usually chronic lameness part of history. Often an incidental finding if routinely open stifle joints and don't confuse with a chronic septic arthritis which has resulted in ulceration of articular cartilage due to chronic bacterial infection. In young game farm bison, severe copper deficiency results in a severe form, especially in the stifles. Usually bilateral lesions present. Don't confuse chronic OCD lesions with normal articular fossas, which are normal not-weight bearing areas of several joints.

PARASITIC PNEUMONIA

Not seen commonly in western Canada, due to our drier climate and is caused by *Dictyocaulus viviparus*. When opening lungs, should always be looked for by thoroughly opening the right dorsal bronchus which extends to the caudal aspect of the caudal lung lobe. Grossly is suspect when a lung shows discoloration of especially the caudal aspect of both caudal lung lobes. Lungworms can result in an AIP type lung as well and the mechanism of this interstitial

pneumonia remains undetermined. Sometimes diagnosed histologically by seeing peri-airway eosinophils or even sections of Dictyocaulus larvae in histo sections from caudal lung sections.

PI3 PNEUMONIA
In my opinion, does not cause significant disease in feedlot cattle except for some chronic bronchiolitis obliterans lesions which can then play a role in cranial-ventral collapse and perhaps then predispose to AIP/interstitial pneumonia or bronchiectasis. More important in some cases of enzootic pneumonia of dairy calves.

POLIOENCEPHALOMALACIA
Two forms seen: 1) on high grain rations with cerebral cortices only involved-most feedlot cases. 2) on high sulfate water (alkali) in adult cows, usually on pasture. The #2 type often shows hemorrhagic areas of the thalamus on cut surfaces of the brain, as well as having some usual cortical involvement. Polio now determined to be due high sulfur/sulfide intake, not thiamine deficiency. No other condition results in positive Wood's lamp fluorescence on the cut surface of involved cortices and this works on formalin fixed tissue as well. Gross lesions often only subtle yellow discoloration of especially sides and depths of sulci, more-so than tops of gyri. Using a hand-held magnifying lens can help.

POSTERIOR VENA CAVA SYNDROME - LIVER LESIONS
Feedlot cattle should be opened with the left side down and hepatic abscesses and voluminous, mottled lungs should give a clue. The vena cava over the dorsum of the liver should be opened before the viscera are removed to confirm the diagnosis. This is best done by hand, without a knife. Septic emboli, abscesses, thrombi and pulmonary ruptured aneurysms are often visible with septic areas throughout all lung lobes. If an aneurysm has ruptured in the lung, the large airways and trachea should contain large amounts of blood clots and the rumen often contains clots that have been swallowed. If the vena cava is thrombosed, ascites may also be present. Often no ruminal lesions of rumenitis at the time this syndrome occurs. May be found dead or show respiratory signs for several days prior to death.

POSTERIOR VENA CAVA SYNDROME - LUNG LESIONS
Lung lesions are randomly distributed throughout most lobes but especially caudal lung lobes. Lesions vary from mixed gas-filled and purulent areas, septic thrombi, large areas of hemorrhage due to ruptured aneurysms and in these latter cases, large blood clots are in the large airways. The lungs are pale if the animal bled out and swallowed large amounts of blood. Vegetative endocarditis of the right side of the heart resulting in showering into the lungs may look similar but gas producing organisms are not involved and aneurysms rare. Abscesses and thrombi are more likely and are often seen only histologically. In cases with aneurysms, aspirated blood into lung lobes occurs resulting in hemorrhagic areas which are often visible on the pleural surface. Don't forget to carefully examine the liver for abscesses and especially the vena cava (pvc) where it passes dorsally over the liver, passes through the diaphragm, and dumps into the right atrium of the heart. This is easiest to do before removing the abdominal viscera. Simply pull the dorsal aspect of the liver down or ventrally with your hands and this can only be done if the carcass is lying with its left side down. This exposes the vena cava as it traverses over the liver. The lung lesions can be severe and the liver lesions relatively minor if

one abscess that has eroded into the PVC happens to continually shower septic emboli to the lung over a long period of time.

PULMONARY EMPHYSEMA

Three types seen grossly- bullous, an exaggerated form of interlobular emphysema, interlobular and subpleural. A fourth type, alveolar emphysema is seen in chronic obstructive pulmonary disease but is mainly a histologic diagnosis. Finding interlobular or bullous emphysema do not constitute a diagnosis of interstitial pneumonia by itself, since it can occur in any case where animals die with terminal respiratory distress. Subpleural emphysema occurs where bronchioles are partially obstructed by exudate or bronchiolitis obliterans, a chronic form of necrotizing bronchiolitis. BRSV commonly shows subpleural emphysema, both in phase I and localized areas of phase II lesions.

PULMONARY PASSIVE CONGESTION – LEFT-SIDED HEART FAILURE

Lungs typically voluminous, edematous (especially interlobular but mixed with blood), sanguinous froth in large airways and the lungs are heavy throughout. Pleural surface shows bloody mottled appearance of especially caudal lobes, mixed with edema. On cut surface, bleeding that has occurred into alveoli may have entered interlobular lymphatics and this is going to result in large round foci of blood only visible in interlobular septae. This blood will be very dark because it is non-oxygenated blood. This is especially readily visible in the caudal lung lobes. In feedlot cattle, most commonly seen in Haemophilus somnus myocarditis/infarction cases but vegetative endocarditis of aortic or left A-V valves may look similar.

PYELONEPHRITIS

In feedlot cattle, may or may not be co-existent with urolithiasis. Often some degree of cystitis present and is the source of infection which has ascended the ureters. Very large variation in degrees of severity in terms of amount of kidney parenchyma involved, especially in feedlot cattle. Usually pale foci visible first on the cortical surfaces and important to open the kidney longitudinally in order to see all regions, including medulla and pelvic regions of the kidney. Elongated pale streaks of necrosis and sepsis seen extending from the pelvis out to the cortical surfaces. Little value in spending money on cultures and Corynebacterium or E. coli most often involved. May be cause of death if severe enough, or concurrent with other disease processes.

RENAL CORTICAL NECROSIS

Varying degrees of acute to chronic necrosis of kidney cortex associated with a wide variety of diseases which result in hypotension or hypovolemia and associated shock. Cases of multiple infarcts can actually often be this entity since with the bovine kidney being lobulated and thus compartmentalized, cortical necrosis can be multifocal rather than diffuse. With cortical necrosis however, no conical shaped areas of necrosis are seen on cut surface like in true renal infarcts due to vascular obstruction. Usually lines of demarcation between viable and necrotic kidney tissue are obvious and may involved an entire kidney or large local areas only, similar to renal infarcts. The cortical capsular surfaces can be very coarsely mottled and it is important to open the kidneys transversely or longitudinally to make this diagnosis. Cases have been seen in severe bacterial toxemias, white muscle disease, clostridial disease, necrotizing enteritis, hardware disease and grain overload, to name a few. Severe anemia and dehydration can result

in acute renal cortical necrosis. Sometimes histopathology on the kidney needed to confirm but seldom identifies a cause. 40% of arterial blood passes through the kidney cortex and makes them particularly susceptible to.

RENAL INFARCTS

Represent areas of coagulation necrosis due to vascular obstruction, most often of arteries. Can vary a lot in size and distribution and a cause cannot always be found. Most often involve the cortex only and conical shaped infarcts often seen on cut surface extending from corticomedullary junctions to the subcapsular surface. Most common causes include vegetative endocarditis, bacteremia or emboli such as neoplastic cells. Pyelonephritis cases can sometimes result in infarcts due to obstruction of vessels secondary to inflammation and necrosis extending from the renal pelvis area. Infarcts can be pale or hemorrhagic depending on size and acuteness and mixtures of both types can be seen in the same kidney. In cattle it is important to look at heart valves carefully when infarcts are seen, for lesions of endocarditis. Seldom can thrombi be seen grossly unless large arteries are involved.

RUMENITIS – ACUTE LACTIC ACIDOSIS

A difficult diagnosis to make in my opinion, unless the carcass is very fresh and a low rumen pH is present, along with increased fluid contents in the rumen, and dilated fluid-filled upper small intestines. Normally the ruminal epithelium peels off easily and this is normal within a few hours after death. If it does not, this suggests a rumenitis may be present and sections should be take for histo examination. Unless chronic, ulcers or rumen wall necrosis should not be expected. The rumen content is important but does not help with the diagnosis on feedlot rations. Chronic cases are much easier once mycotic infarcts and resulting areas of hemorrhage develop ventrally in the rumen, reticulum and sometimes omasum. Invasion of vessel walls and thrombosis by Zygomycetes group of fungi are responsible for these lesions.

SALMONELLOSIS

In feedlots, organ involvement usually confined to the intestinal tract, with varying degrees of necrotizing enterocolitis. Outbreaks often occur with several animals involved in one or more pens. Lesions are not at all specific and altered in most cases by antibiotic therapy. Lesions can be severe with necrotic casts of mucosa and fibrin, especially in the ileum, or may be confined to the cecum and colon with necrosis of the mucosal surface. Gross lesions can be similar to coccidiosis, BVDV, and nonspecific necrosis by normal intestinal flora of a mucosa damaged by some other disease process such as enterotoxemia or similar sudden alterations in feed. Isolation of salmonella sp can be difficult with antibiotic therapy and including mesenteric lymph nodes and gall bladder as well as tied off intestine can increase the chances. Make sure the lab knows about the antibiotic therapy. When outbreaks of salmonellosis occur, make sure you keep in mind that primary BVDV infection may be making a group of calves susceptible to this organism, so heart, ileum, and a variety of other tissues should be examined histologically and by IHC to see if this virus may be present. We have run into this situation several times and remember no gross lesions of BVDV will be seen. “if you don’t look, you won’t find” Because of primary BVDV infection, other concurrent lesions may also be present, such as pneumonia. Rarely are septicemic forms seen in feedlot situations unless in calves that have just arrived. We often use IHC to identify Salmonella sp in formalin fixed tissues and this works well where antibiotic therapy has been extensive.

UROLITHIASIS – "WATERBELLY"

In cattle the site of obstruction is most often the sigmoid flexure. Not in all cases are uroliths found; sometimes only masses of soft necrotic debris and fibrin are found and these may represent mucosa that has been damaged and sloughed by a passing urolith or areas of necrotizing cystitis. To examine the entire urethra properly, requires opening the pelvis. To easily do this, split the symphysis then cut the shaft of the ilium on one side and pull the pelvis open with your hands. Associated with ventral rupture of urethra or ruptured bladder. A local or diffuse peritonitis may be present if the bladder is ruptured and bacterial contamination is present. Often accompanied by varying degrees of hydronephrosis within renal lobules.