Chapter 1

Alimentary Tract

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Introduction and general comments

Diseases of the alimentary tract are common in ruminants, often resulting in either death or significant loss in productivity. In many cases, an accurate diagnosis will require postmortem examination of one or more recently dead or sacrificed animals. The alimentary tract should be given special attention in cases where diarrhoea and/or illthrift are characteristic features of the clinical syndrome, although a range of other presenting signs, including sudden death, may be associated with gastrointestinal diseases. Gross lesions are sometimes sufficiently specific to allow a definitive diagnosis, but even an experienced pathologist will often require ancillary tests. The challenge for practitioners is in knowing when to make a diagnosis on gross lesions alone, and how to make cost-effective use of laboratory services.

Examination of the lower alimentary tract, because of its accessibility on opening the abdominal cavity, is a routine part of most postmortem examinations, but the upper alimentary tract is often neglected. Lesions of diagnostic significance may be present in the oral cavity or oesophagus and could be overlooked if these regions are not checked.

At the risk of stating the obvious, it is important that practitioners performing postmortem examinations are aware of the normal structures and species variations in the different regions of the alimentary tract, as well as the range of artefactual changes that may accompany autolysis. Furthermore, the predilection of some diseases for certain regions of the alimentary tract, and other organ systems, must be recognised if appropriate samples are to be collected for microscopic or microbiological examination. These topics will be considered below in the relevant sections.

Autolysis proceeds rapidly in the lower alimentary tract because of its mixed bacterial flora and the presence of lytic, digestive enzymes. Subtle changes at the gross and microscopic level can soon become masked by autolysis. In general, the chance of making an accurate diagnosis of enteric disease is much greater if the necropsy is performed within a few hours of death. If the problem is occurring on a herd or flock basis then the best option may be to convince the farmer to sacrifice one or two animals for immediate examination rather than waste time and money on those that have been dead for 12-24 hours.

Special care must be taken when collecting samples for submission to a laboratory. The mucosa throughout much of the alimentary tract is readily damaged by rough handling and if histopathology is required then

it is important to select areas that have not been handled excessively. Ideally, the lumen should be opened to allow rapid penetration of fixative. Since infectious diseases are often regional, several segments of bowel should be submitted, even if the laboratory does not choose to examine them all. It makes sense to collect specimens for microbiology during the early stages of the necropsy in order to minimise the risk of contamination. Some laboratories prefer to receive swabs in culture media while other may be content with tied-off loops of bowel. In cases where worm counts are required, the abomasum and intestines should be opened into a tray or bucket to avoid loss of contents. Any specimens submitted to a laboratory should be accompanied by a description of the clinical syndrome and gross findings.

The intent of this paper is to review the gross pathology of selected diseases of the alimentary tract of ruminants, emphasising differential diagnoses for common lesions. Due to time constraints the list is not complete and the emphasis is on diseases of significance to Australia and New Zealand. For convenience, the diseases and lesions will be considered on a regional basis with brief, general comments at the start of each section. Although there are several important exotic diseases characterised by lesions in the alimentary tract, these will be discussed in a separate presentation at this seminar.

Oral cavity, Oesophagus and Forestomachs

General considerations

The oral cavity should be opened routinely to expose the hard palate, tongue, teeth and pharynx. In young animals the mandibular symphyses can be easily separated with a knife but in adults this will require the use of a saw or large bone-cutters. The mandibles can usually be disarticulated and the tongue reflected by cutting along either side, then through the hyoid apparatus. The oesophagus should be opened along its entire length after removal of the pluck, as lesions of diagnostic significance may be concentrated at one end or the other. When opening the forestomachs, make note of the nature of the contents and, if plant poisoning is a possibility, collect any leaf fragments for later identification. The green-stained mucosa of the rumen normally detaches readily within a few hours of death. Failure to do so suggests the possibility of chemical rumenitis or healed lesions of mycotic or bacterial rumenitis. The mucosa is best viewed after it is washed with running water.

Congenital and inherited anomalies

Congenital diseases such as brachygnathia inferior and cleft palate occur in all species, sometimes in association with other defects (or together). **Cleft palate** is more common in calves than in lambs and is probably inherited in some cases. The challenge is not in making the diagnosis, as the lesion is obvious, but in knowing what advice to give the client. A

genetic aetiology has been reported in the Charolais and Hereford breeds and should be suspected when several affected calves have been born to a particular sire in a season, or over several seasons. Most cases occur sporadically and the aetiology is never determined. Any exposure to potential teratogens would have occurred early in pregnancy and is unlikely to have been noticed.

Brachygnathia inferior (micrognathia) is also more common in calves than in lambs and may be inherited as an autosomal recessive trait. The defect may also be linked to other genetic abnormalities, including osteopetrosis in calves and osteogenesis imperfecta in lambs. Examination of the bones may therefore be warranted in affected animals.

Extensive loss of epithelium from the hard palate and tongue occurs in newborn Angus calves with **familial acantholysis** and Suffolk lambs with **epidermolysis bullosa**. Both disorders are inherited as autosomal recessive traits and are also characterised by loss of skin over pressure points and shedding of hooves. The oral lesions are no doubt related to detachment of the defective epithelium during sucking.

Tooth problems

The importance of abnormalities in **molar teeth** as a cause of ill thrift in sheep (and cattle) is probably underestimated. Such abnormalities include impaction of food in deep tooth sockets, loosening and/or loss of teeth, and recession of gums. In some cases, the maxilla or mandible is swollen due to chronic osteomyelitis. In a recent survey of ill thrift in sheep on a large property in New Zealand, lesions involving molar teeth were considered to be the primary cause of weight loss in several cases. In cattle, infection of the mandible or maxilla by *Actinomyces bovis,* an obligate parasite of the oral cavity, may lead to massive enlargement of the bone, (so-called **"lumpy jaw"**). The organism presumably gains access to the underlying bone by lymphatic drainage from ulcerative lesions in the oral mucosa or by tracking down tooth sockets.

Pitting, yellow/brown discolouration and irregular wear of incisor teeth is a characteristic feature of **fluorine toxicity** in cattle. Only those teeth exposed to excess fluorine during enamel formation are affected. Consequently, some incisors may be normal while others are severely affected, but the lesions are always symmetrical. Uniform red/brown pigmentation of all teeth occurs in cattle with **erythropoietic porphyria**, a rare inherited disease of certain breeds. Pigmentation of teeth does not occur in the erythropoietic protoporphyria of Limousin cattle. The molar teeth of ruminants are normally covered by a dark black/brown layer of tartar due to impregnation of mineral salts with chlorophyll and porphyrin pigments of dietary origin. This should not be misinterpreted as a lesion.

Enamel hypoplasia characterised by patchy absence of enamel and discolouration of the exposed dentine, in newborn calves is associated with intrauterine infection by bovine viral diarrhoea (BVD) virus.

Lesions caused by infectious agents

Bovine papular stomatitis, caused by a parapoxvirus, is a common disease of young calves characterised by the formation of papular to shallow erosive lesions on the muzzle, nares, gums, hard palate, ventral and lateral surfaces of the tongue, and occasionally in the oesophagus. The lesions are typically circular and often have a raised, pale margin. The disease is not clinically significant and the lesions heal rapidly, but several waves of new lesions may appear over a period of a few months.

Infection of sheep and goats with a closely related parapoxvirus results in **contagious ecthyma** (orf, scabby mouth). Although the lesions typically occur around the lips, face and feet, they may also extend into the oral cavity, involving the tongue, gums, dental pad and hard palate. Oral involvement is more common in goats than in sheep. The lesions are more proliferative than those of bovine papular stomatitis, and are more likely to be clinically significant, especially in suckling animals. Oral lesions are less likely to develop a scabby surface than those on the lips or skin and usually appear as raised, red coalescing areas on the tongue or around molar teeth. On rare occasions, similar lesions are found in the forestomachs of sheep, goats and deer. Deer may also develop lesions associated with parapoxvirus infection on their antler velvet.

Parapoxvirus infections in cattle, sheep or goats can usually be based on gross lesions, but histopathology may be useful in equivocal cases. Laboratory demonstration of typical parapoxviral particles using electron microscopy can also allow rapid confirmation.

Papillomatosis induced by bovine papillomavirus are very common on the skin of young cattle and occasionally occur in the oesophagus and/or forestomachs. The lesions may appear as smooth-surfaced nodules or consist of multiple closely packed fronds of squamous epithelium.

Mucosal disease, caused by superinfection of immunotolerant, seronegative cattle (persistently infected with non-cytopathic BVD virus) with a cytopathic strain of **BVD virus**, typically causes extensive erosive lesions throughout the oral cavity, oesophagus and forestomachs. Erosive lesions may also be present on the muzzle and nares. The severity and distribution of lesions vary markedly, depending on the stage of the disease at which the animal dies or is killed. In the acute stage, multiple, irregular-shaped erosions with a red base are usually present on the hard palate and tongue. Papillae on the buccal mucosa may be blunt and hyperaemic. Oesophageal erosions are typically linear, and may extend throughout the entire length but are more common in the proximal third. Fragments of necrotic epithelium are sometimes loosely adherent to oesophageal erosions. Plaque-like lesions or erosions are often present in the reticulorumen. Holes in omasal leaves may represent chronic lesions of mucosal disease. Mucosal surfaces heal rapidly and the oral or oesophageal lesions in animals that survive the acute stage of mucosal disease but die a week or so later are much less impressive. In such cases, the margin of the erosions will be slightly elevated due to

proliferating epithelial cells and the base will be pink or white rather than red due to the presence of a thin layer of new epithelium.

Lesions of mucosal disease may also be detected in the lower alimentary tract. In particular, there may be multiple, discrete, dark red ulcers on abomasal rugae. The small intestinal mucosa is often normal grossly but haemorrhage and/or necrosis of Peyer's patches may be present in acute cases. Peyer's patches may be visible from the serosal surface as dark red, elongated foci and fibrinonecrotic exudate may be adherent to the overlying mucosa. In chronic cases, the Peyer's patches may be either indistinct or even sunken below the adjacent mucosa. Colonic lesions of mucosal disease vary from fibrinohaemorrhagic inflammation in acute cases to multifocal cystic change in some chronic cases. The cysts represent markedly dilated glands that have herniated into necrotic submucosal lymphoid follicles and become filled with mucus.

BVD virus attacks rapidly dividing cells, including epithelial cells in the base of intestinal crypts and lymphoid cells in germinal centres. The most characteristic microscopic lesions of mucosal disease are therefore in the small and large intestine and in lymphoid tissues. Immunoperoxidase staining of sections of intestine for BVD antigen is now commonly used by veterinary pathologists to support a histological diagnosis.

BVD infection in immunocompetent cattle exposed to the virus for the first time results in a milder, non-fatal clinical syndrome characterised by lethargy, anorexia, oculonasal discharge and diarrhoea. Shallow erosions may be present in the oral cavity.

The severe gross lesions of mucosal disease are indistinguishable from those of **rinderpest**. This highly infectious disease is exotic to Australia and New Zealand and will be discussed in a separate paper.

The principal differential diagnosis for mucosal disease in countries without rinderpest is **malignant catarrhal fever (MCF)**. Similar lesions may be present on the tongue, hard palate and oesophagus, but are usually deeper and more haemorrhagic in MCF due to the underlying vasculitis. Haemorrhagic and/or ulcerative lesions may also be present in the abomasum, small intestine and colon. Lesions in other tissues may assist in differentiating MCF from mucosal disease at the time of necropsy. In MCF, the kidneys often contain multiple small (2-4 mm) yellow/grey foci caused by interstitial aggregates of lymphocytes. Similar nodules may be present in the mucosa of the urinary bladder. Other common gross lesions in MCF include enlargement of lymph nodes throughout the body and keratoconjunctivitis with marked corneal opacity.

In peracute cases of MCF in cattle and deer the gross lesions may be mild or non-existent. In chronic cases, a characteristic gross lesion is the presence of prominent arcuate arteries in the kidneys. These vessels are clearly apparent on cut surface of the kidney due to marked thickening and inflammation of their muscular walls.

MCF can be readily differentiated from mucosal disease by demonstrating the typical vascular lesions histologically. Although the vasculitis may be found in a wide range of tissues, preferred sites for examination by pathologists include the brain, kidney, urinary bladder, liver, small intestine, lung and lymph nodes.

In cattle and deer with severe **photosensitisation**, for example in sporidesmin toxicity, there may be extensive ulceration of the muzzle and undersurface of the tongue. The tongue lesions are due to exposure to sunlight during persistent licking of the irritated muzzle.

Oesophageal lesions similar to those of BVD and MCF may occur in **acorn** or **oak bud toxicity**, probably secondary to uraemia. Diseases associated with reflux of abomasal contents into the oesophagus or forestomachs also cause ulceration since the squamous mucosa lining these regions of the alimentary tract is not protected by a layer of mucus.

Actinobacillosis (wooden tongue) caused by *Actinobacillus lignieresii* infection, is a relatively common infection of the oral cavity in cattle. The organism presumably gains access to soft tissues following mucosal trauma and initiates a chronic, pyogranulomatous inflammatory response. When the tongue is affected it becomes extremely firm and may have multiple, yellow/tan nodules on cut surface or protruding through ulcers in the overlying mucosa. Lesions may also develop in other soft tissues of the head and neck, including the lips and pharynx, and are occasionally found in the wall of the forestomachs. The infection often extends to regional lymph nodes and may resemble those of tuberculosis grossly. Affected tissues become markedly swollen and firm and involvement of the pharynx or associated lymph nodes may result in dysphagia or severe dyspnoea. The diagnosis is best confirmed by histopathology, as the microscopic changes are classical.

Oral necrobacillosis (necrotic stomatitis) is a severe, necrotising disease caused by invasion of soft tissues by *Fusobacterium necrophorum*, usually following traumatic or viral disruption of the mucosal barrier. Pharyngeal infection in calves is well known as **calf diphtheria**. Grossly, the lesions are characterised by locally extensive areas of ulceration covered by a thick layer of grey/green or yellow material consisting of necrotic tissue and inflammatory exudate. Death may occur rapidly due to asphyxiation. Similar oral lesions caused by either *Fusobacterium necrophorum* or other bacteria are sometimes associated with **drenching gun injuries** in sheep, goats and deer. In some cases the lesion extends deep into surrounding tissues causing extensive cellulitis, massive swelling and death due to either toxaemia or asphyxiation. *Fusobacterium necrophorum* occasionally causes necrotising lesions in the forestomachs of cattle usually as a sequel to ruminal acidosis.

Mycotic rumenitis and **omasitis** caused by opportunistic zygomycetes (e.g. species of *Rhizopus, Mucor, Absidia)* may also follow ruminal acidosis, particularly in cattle and deer, but sometimes occur in animals

without a history of grain feeding. The lesions are typically multiple, discrete, dark red, transmural infarcts, reflecting fungal invasion of submucosal blood vessels and the associated thrombosis. Similar lesions are rarely seen in calves in association with **adenoviral infection**, which also causes vascular damage and thrombosis.

Parasites

Sarcocystis gigantea causes grossly visible, ovoid white nodules up to 1cm in length in the oesophagus of sheep. In spite of the abundance of such nodules in some sheep, they incite little or no host reaction and are not clinically significant. Other species of *Sarcocystis* commonly produce microscopic cysts in the striated muscles of the tongue and oesophagus of sheep.

Adult stages of the fluke ***Paramphistomum*** are found in the rumen of sheep, goats and cattle. These small, pale red, droplet-shaped parasites are not much larger than ruminal papillae and do not cause any problem in the rumen. Immature stages inhabit the duodenum and heavy infestations may cause severe enteritis.

Chemical rumenitis

Ruminal acidosis secondary to carbohydrate overload is an important and relatively common disease of cattle, sheep, goats and deer. Some animals die acutely of metabolic acidosis while others survive the acute stage but develop secondary lesions, such as mycotic or fusobacterial rumenitis or hepatic abscessation. Ingestion of excess carbohydrate in the form of grain, root crop or fruit, leads to a marked change in the rumen micro flora and increased production of lactic acid. The pH of ruminal fluid may fall from a normal level of between 5.5 and 7.5 to as low as 4.0 to 4.5. Most normal ruminal microorganisms die once the pH falls below 5.0. Body fluid is attracted into the rumen due to the increased osmotic pressure created by the accumulating lactic acid. This results in severe dehydration and circulatory collapse. Concurrent absorption of lactic acid into the systemic circulation may lead to metabolic acidosis.

The gross lesions in animals that die of acute ruminal acidosis are not specific and must be interpreted in conjunction with an appropriate history. The carcass is dehydrated and dark due to haemoconcentration. The rumen content typically resembles thin porridge, may have a fermentative odour and its pH is usually less than 5.0, although the pH starts to rise within an hour of death. The absence or death of protozoa in the rumen content of a freshly dead animal is supportive. The ruminal and omasal mucosa may show patchy or diffuse reddening on removal of the green-stained cornified layer. Since characteristic microscopic changes are present in the ruminal epithelium, histopathology is the best means of confirming the diagnosis.

Rumenitis may also be associated with the ingestion of certain **toxic plants**, including rhododendron and oleander. In such cases, history of access to the plant (or prunings) is important, as is the demonstration of leaf fragments in the rumen contents.

Miscellaneous

Bloat (ruminal tympany) is a common cause of sudden death in cattle and may be either primary or secondary. It occurs only rarely in goats, sheep and deer. The primary form is the most common and is due to excessive formation of foam in the rumen of animals on pastures containing high concentrations of legumes or in feedlot cattle fed predominantly on grain-based rations. Affected animals are typically found dead. Secondary bloat is usually chronic and intermittent, and occurs when there is a physical or functional defect in eructation of ruminal gas.

Gross lesions in a cow that has died of bloat are not specific and may be confused with clostridial diseases accompanied by acute death and rapid autolysis, or with anthrax. The abdomen is usually so distended that the animal rolls partly onto its back with its legs in the air. Blood may be exuding from body orifices and the eyes and tongue may be protruding. The blood is dark and poorly clotted due to anoxia and there is usually oedema, congestion and haemorrhage in the subcutaneous tissues, musculature and lymph nodes of the head and neck. Blood clots are often present in paranasal and frontal sinuses of the skull. In general, the anterior regions of the carcass are congested due to pressure on the thoracic cavity impaired venous return. This is reflected in some cases by the so-called "bloat line" in the oesophagus, characterised by congestion of the oesophagus anterior to, and blanching caudal to, the thoracic inlet. The lungs are compressed and subpleural, epicardial and endocardial ecchymoses are common (but non-specific). Abdominal viscera, particularly the liver, are usually pale due to ischaemia, as are the muscles of the hind limb. In primary bloat, the rumen is filled with frothy ingesta, but the foam disappears gradually after death and may be gone if the necropsy is delayed for 12 hours. Laboratory tests are of little value in confirming a diagnosis of bloat, other than to rule out differentials.

Traumatic reticuloperitonitis following ingestion of a nail or short length of wire is well recognised in cattle. There is a tendency for sharp metallic foreign bodies that have lodged in the reticulum to perforate the reticular wall in an anteroventral direction. The object may be walled off at that location by a granulomatous inflammatory response, but some objects progress further, penetrating the diaphragm and causing traumatic pericarditis. The accompanying infection results in a severe, fibrinopurulent pericarditis and epicarditis. In such cases, the pericardium may be massively thickened and the pericardial sac filled with purulent exudate. The reticulum will be firmly adherent to the diaphragm. An extensive search may be required in order to find the offending nail or piece of wire.

Newborn ruminants fed on rations high in concentrates and with inadequate roughage have shortened, club-shaped villi, which tend to clump together and have a thickened, parakeratotic epithelium. The syndrome is referred to as **ruminal parakeratosis**. Affected calves often have large hairballs in their rumen.

Abomasum

General considerations

Examination of the abomasum and its contents is an important part of any postmortem examination in ruminants, particularly in cases where gastrointestinal parasitism is suspected. Collect the contents into a bucket or tray in case a worm count is required and rinse the mucosa gently in running water. The mucosal surface should be smooth, glistening and pale pink. If the content is dark red or brown/black, search carefully for the presence of ulcers. Abomasal folds should be thin and lie flat against the mucosa.

Most diseases involving the abomasum will also induce lesions in other parts of the alimentary tract or in other organ systems.

Abomasitis

Inflammation of the abomasum can be caused by several infectious or non-infectious agents, only some of which are discussed in this paper.

Diffuse, intense, reddening of the abomasal mucosa is a feature of **salmonellosis** in sheep caused by either *S. hindmarsh* or *S. typhimurium* infection. Multiple shallow ulcers may also be present. Histopathology and culture of abomasal or intestinal contents should allow confirmation of the diagnosis. Similar gross lesions also occur in some cattle with **MCF**, and in ruminants that have ingested caustic chemicals, such as **arsenic**.

Braxy, caused by *Clostridium septicum* is an acute bacterial abomasitis characterised by marked oedema, hyperaemia and necrosis of the abomasal mucosa. Although typically a sporadic disease of young lambs, a similar syndrome is reported occasionally in calves and goat kids. In this disease the abomasal lesions may be either diffuse or focal. The folds in affected areas are thickened, oedematous and reddened, and may contain dry, yellow foci of necrosis. The lesions usually extend deep into the submucosa or musculature and may be visible from the serosal surface. Diagnosis is confirmed by histopathology and demonstration of *Cl. septicum* in typical lesions by either immunochemistry or fluorescence technology.

Shallow abomasal erosions and moderate reddening of the mucosa may be present in cattle with mucosal disease but, as discussed above, more remarkable lesions are likely to be present elsewhere in the alimentary tract.

Mycotic abomasitis is occasionally seen in calves and is most likely secondary to mucosal damage caused by other agents (usually bacterial or viral). The lesions are typically discrete, dark red foci (infarcts), 1-2 cm in diameter, and may be transmural.

Abomasal parasitism

The most important parasites of the abomasum in cattle, sheep and goats are *Ostertagia spp* and *Haemonchus spp* either on their own or in combination. *Ostertagia spp* are small, brown, thread-like nematodes up to 1.5 cm in length and are difficult to see grossly unless present in large numbers. *Haemonchus spp* are larger (2-2.5 cm in length) and the females have characteristic red and white striped (barber's pole) appearance.

Ostertagiasis (caused by *O. ostertagi*) is one of the most important production-limiting diseases of young cattle. In cattle that die of the disease, the carcass is dehydrated and wasted. The abomasal mucosa is often thickened due to oedema and to the presence of multiple small (1-2mm) nodules, which in severe cases are confluent. The pH of the abomasal contents may be increased above 4.5 due to extensive replacement of parietal cells by proliferating mucus cells. Worm counts on abomasal contents are not always high and digestion of the mucosa to release pre-emergent stages may be required in order to achieve an accurate count. In sheep and goats, *Ostertagia (Teladorsagia) circumcincta* produces similar mucosal nodules to those caused by *O. ostertagi* in cattle, but they are seldom as numerous or as significant clinically. Concurrent infestation with *Trichostrongylus axei* and/or *T. colubriformis* (the latter in the small intestine) is common and the combined effect may result in illthrift or death.

Haemonchosis is an important cause of blood-loss anaemia and sudden death in sheep and goats, usually following summer rainfall. The entire carcass is very pale and there may be oedema in the submandibular region or elsewhere due to severe hypoproteinaemia. In heavy infestations, the parasites can be readily detected in the abomasal contents, sometimes forming tangles masses between mucosal folds. A total worm count will add support to a diagnosis but is often not necessary.

Miscellaneous

Abomasal bloat occurs sporadically in calves, lambs and goat kids, usually after engorgement on milk replacer. Bacterial fermentation of the milk results in excessive gas production, marked abomasal distention and death, presumably due to asphyxia. At necropsy, the abomasum is usually distended with gas and milk and may show patchy mucosal reddening. In some cases, the abomasum ruptures after death due to continued gas production, spilling its contents into the abdominal cavity. The absence of peritonitis or of haemorrhage along the margins of the rupture indicates that the rupture has occurred post mortem.

Abomasal volvulus is much less common that intestinal volvulus (see later) but may occur in young milk-fed ruminants. As in abomasal bloat, affected animals are usually found dead with a distended abdomen, but in this case the distention is due to a gas-filled, markedly congested

abomasum. The twist can easily be detected at the time of necropsy. Laboratory tests are neither required nor useful.

Abomasal ulceration is common in cattle, including calves and adult dairy cows, and is considered to be linked to stressful events such as weaning and parturition. No doubt many abomasal ulcers go unnoticed, but some will perforate and cause septic peritonitis while others will result in exsanguination. Abomasal ulceration should always be considered a likely option in an adult dairy cow with blood loss anaemia, particularly if there is evidence of melaena. The ulcers are usually multiple and may have a red/brown base, indicating that they are active. In such cases, the abomasal content may be dark brown or have a "coffee ground" appearance due to the action of gastric acid on haemoglobin. Previous ulceration may be indicated by a scalloped margin to rugae. Ulceration often accompanies infiltration of the abomasal mucosa with neoplastic lymphocytes in cattle with enzootic bovine leucosis.

Multiple 1-2 mm, dark brown **microhaemorrhages** are sometimes found at necropsy on the abomasal mucosa of young calves, lambs or kids that die acutely. These appear to represent the earliest stage of stress-related abomasal ulceration perhaps due to gastric hyperacidity. Although the foci may be so numerous as to result in significant discolouration of abomasal or intestinal contents, the cause of death is likely to be found elsewhere.

The abomasum is a predilection site for infiltration with neoplastic lymphocytes in adult cattle with **enzootic bovine leucosis**. The submucosa is massively thickened with homogenous, pale cream tissue and the overlying mucosa inevitably contains several variably sized ulcers. Other predilection sites for neoplastic involvement in this disease are the heart and uterus. The tumour may also infiltrate a range of other tissues, including lymph nodes, liver, kidneys and muscle, but the abomasum is always involved in this form of lymphosarcoma. In contrast, the abomasum is not a predilection site in cattle with sporadic forms of lymphosarcoma.

Small Intestine and Colon

General considerations

Autolysis proceeds most rapidly in this part of the alimentary tract because of its mixed microbial content and the presence of digestive enzymes. As a result, diseases producing subtle enteric lesions may be difficult or impossible to diagnose unless the postmortem examination is performed, and samples collected for laboratory testing, within the first few hours of death. This should not however be used as an excuse for not performing a necropsy or taking specimens for histopathology in an animal that has been dead for a longer period as diagnostic lesions may still be present histologically if not grossly.

Because of the regional nature of many enteric diseases, it is important to open (and sample) several segments of both small intestine and colon. If a worm count is likely to be required then the contents should be collected as each segment is opened. Make sure that the gut wall is handled gently during this process or the delicate mucosa will be rendered useless for histopathology. Also pay attention to mesenteric lymph nodes as they will often be involved in animals with infectious or neoplastic diseases of the lower alimentary tract.

As with the abomasum, the mucosa of the small intestine and colon is normally smooth and glistening. Adherent material that cannot be easily removed by gentle scraping is likely to be abnormal, perhaps indicating mucosal necrosis or fibrinous exudate. The nature and volume of the contents varies markedly both in normal and diseased animals but may provide useful information (see below). Similarly, the colour and thickness of the gut wall may provide valuable information and should be noted in descriptions accompanying specimens submitted to a laboratory for histopathology. But they can also be misleading. Patchy or diffuse reddening of the small intestine may indicate inflammation or volvulus, but it may also reflect splanchnic pooling in an animal with terminal circulatory collapse, rather than a primary disease of the alimentary tract. Thickening of the gut wall is a feature of certain diseases, such as Johne's disease, but in freshly dead animals contraction of smooth muscle in the outer coat can produce a thickened, corrugated appearance to the jejunum closely resembling the gross change associated with Johne's disease.

Absence of gross lesions in the small intestine or colon does not exclude the possibility of significant enteric disease. In some cases the lesions will only be apparent histologically.

Congenital anomalies

Segmental aplasia or **atresia** of the ileum or colon is relatively common in newborn calves, lambs and kids. A genetic aetiology has been suggested, but not proven, in certain cattle breeds (e.g. Holstein, Jersey, Swedish Highland). An association has been made between rectal palpation for pregnancy diagnosis prior to 42 days gestation and the occurrence of atresia coli in calves, possibly linked to pressure on the amnionic vesicle. In sheep, occasional "mini-outbreaks" on individual properties have led to suggestions of a genetic aetiology, but there appears to be no convincing data to support this. Affected animals are normal at birth, but do not pass faeces and develop gradual abdominal enlargement over the first few days of life. Some animals survive for a week or more, but death is inevitable. The gross lesions at necropsy are classical and no laboratory support is required to confirm the diagnosis. The bowel anterior to the atretic segment is markedly dilated and filled with ingesta and gas. The distal portion is considerably smaller and is empty.

Atresia ani is the most common congenital defect of the lower alimentary tract. Clinical signs resemble those of ileal or colonic atresia but at necropsy, both small and large intestines are distended. The absence of a perforate anus makes the diagnosis obvious and a necropsy is usually not required, but the defect is sometimes associated with other anomalies of the spinal column or urogenital tract, including rectovaginal fistula.

Lesions caused by infectious agents

Infectious enteritis is a common and important cause of diarrhoea and death in neonatal ruminants and may involve ***Escherichia coli*, rotavirus, coronavirus and *Cryptosporidia sp*** alone or in combination. In all of these infections, the gross lesions are usually unremarkable and non-specific. In addition to faecal soiling of the perineum, the eyes are sunken due to dehydration. The small intestines may show mild, patchy reddening or may be distended with gas, the gas formation presumably due to bacterial fermentation of poorly digested or unabsorbed milk. The contents are often watery and yellow/white. An aetiological diagnosis is best achieved by a combination of histopathology and microbiology. The presence of cryptosporidia, and sometimes *E. coli*, can be confirmed histologically, but intestinal contents should be tested (by ELISA) for the presence of rotavirus and cultured for enteropathogenic types of *E. coli*. The histological lesions of coronavirus infection in calves are more specific but confirmation would require culture of the virus.

Age is a useful factor when considering the likely aetiology of neonatal diarrhoea. *E. coli* is unlikely to be a problem in calves, lambs or kids older than 1 week of age, rotavirus infections usually within the first 2 weeks of life, while coronaviral and cryptosporidial infections typically occur between 1 and 4 weeks of age.

Salmonellosis is an important cause of enteritis in cattle and sheep but is rare in goats and deer. *S. typhimurium* is the most common cause in **cattle**, particularly calves, but *S. dublin* is responsible for some cases both in adult cattle and calves. Salmonellosis in calves seldom occurs less than a week of age and is more common from 3 weeks to 6 months. In the acute septicaemic form, which usually occurs in younger calves, there are few gross lesions other than engorgement of intestinal blood vessels and, in some cases, subserosal petechial haemorrhages. The lesions of acute enteritis in older calves and adult cattle are more characteristic. Segments of the jejunum, ileum and colon may be fluid filled, reddened and thick walled. The mucosal surface is often markedly inflamed and may be partly covered by a loosely adherent layer of fibrinous or fibrinohaemorrhagic exudate. A cast of fibrinohaemorrhagic exudate may be present in the lumen of severely affected regions, typically in the ileum. Mesenteric lymph nodes are enlarged and oedematous. The diagnosis is best confirmed by a combination of histopathology and culture.

In **sheep**, salmonellosis tends to be a disease of adults rather than lambs, and is usually stress-related (e.g. overcrowding, transport). In New Zealand it occurs most often in the summer, often in outbreak form, and may be caused by either *S. typhimurium* or *S. hindmarsh*. Affected sheep are usually found dead with evidence of recent khaki-coloured diarrhoea. Grossly, there is an acute haemorrhagic abomasitis and enteritis.

Yersiniosis has long been recognised as a cause of haemorrhagic enteritis and death in young farmed **deer**, but has now become a significant entity in cattle, goats and sheep in Australia and New Zealand. In Australia, *Y. pseudotuberculosis* infection in **cattle** is reported in adults on river flat properties following winter flooding or persistent rain, whereas in New Zealand it occurs in yearlings during autumn or winter. The disease in **sheep** and **goats** is usually caused by *Y. enterocolitica* and also occurs in young animals entering their first winter. The gross lesions of yersiniosis in deer are typically much more remarkable than those in other ruminants. The small intestine may be diffusely reddened and the contents haemorrhagic. In cattle, goats and sheep, the intestinal reddening is only mild. The contents are usually watery, but there is seldom any evidence of either haemorrhage or of fibrinous exudate attached to the mucosa. The diagnosis is best confirmed histologically as the microscopic lesions are virtually pathognomonic. Culture of the organism from intestinal contents provides additional support, but *Yersinia spp* can also be cultured from the faeces of clinically normal animals.

Enteric listeriosis has been recognised with increased frequency in sheep in New Zealand over the last 5 years, possibly due to increased reliance on baleage or silage (sometimes of poor quality) as winter feed. Affected animals usually develop diarrhoea and depression prior to death, or may just be found dead. Gross lesions include marked, patchy reddening of the alimentary tract, including the abomasum and caecum, and could be confused with salmonellosis or toxic enteritis (e.g. following ingestion of fertiliser). Histologically there is an acute suppurative and/or ulcerative abomasitis, enteritis, colitis and typhlitis. Confirmation requires culture of the organism from intestinal contents as the gross and microscopic lesions are not specific.

Johne's disease caused by *Mycobacterium avium paratuberclosis* is a chronic wasting disease of ruminants and has received more publicity in recent years than any other enteric disease. In most species, it seldom occurs in animals less than 2 years of age and is considered a disease of adults, but in deer it typically occurs in the first year of life. In many animals with advanced Johne's disease, the gross lesions are sufficiently characteristic to allow a definitive diagnosis, at least in the hands of an experienced pathologist. But because of the significance of a positive diagnosis, it is dangerous to rely on gross lesions alone. Similar changes may occur in some other disease syndromes and lesions are sometimes detected histologically in animals with equivocal or inapparent gross changes.

Animals that die of Johne's disease, or are euthanased in the advanced stages, are emaciated and usually have minimal body fat reserves. Some animals also have oedema in the submandibular region, intestinal serosa and mesenteric lymph nodes. The most characteristic lesion is diffuse thickening of the wall of the intestine, particularly the ileum and distal jejunum, but sometimes extending into the proximal colon. Affected areas of bowel appear enlarged and flabby. The mucosal surface is often corrugated and may be pale tan. Similar corrugation of the small intestine, particularly the jejunum, occurs in freshly dead animals due to contraction of smooth muscle in the wall. Serosal lymphatics are sometimes prominent and contain small, nodular thickenings corresponding to foci of granulomatous lymphangitis. Although this lesion is sometimes considered to be pathognomonic for Johne's disease, it occurs occasionally in association with gastrointestinal parasitism in goats and sheep due to granulomatous lymphangitis surrounding degenerate nematode larvae.

In deer, the lesions are spread more uniformly throughout the small intestine. Mesenteric lymph nodes are usually enlarged, oedematous and on cut surface the cortex is often expanded with large, contiguous pale cream nodules. In deer, goats and occasionally sheep, the mesenteric lymph nodes may contain foci of caseation and mineralisation, similar to the lesions of tuberculosis.

Unless the gross lesions of Johne's disease are unequivocal, samples of ileum, ileocaecal valve and mesenteric lymph node should be submitted for histopathology. Alternatively, scrapings of ileal mucosa stained by an acid-fast method (ZN or Kinyoun's) may allow confirmation of the gross diagnosis.

As mentioned earlier in this paper, intestinal lesions are often present in cattle with **BVD** and cattle and deer with **MCF**. Haemorrhagic or necrotising lesions in the small intestine and colon also occur sporadically in cattle and deer in association with **adenovirus** infections.

Parasites

Coccidiosis occurs in intensively reared calves, lambs and kids, often causing clinical disease and sometimes death. In **calves**, the disease may occur anywhere between 1 month and 1 year of age, depending on the time and magnitude of exposure to infectious oocysts. Clinical signs include diarrhoea, dysentery, tenesmus and dehydration. In severe, acute cases, affected calves may die before oocysts have had a chance to appear in the faeces and diagnosis is best achieved by postmortem examination. Gross lesions are most marked in the large intestine and consist of a fibrinohaemorrhagic typhlocolitis, sometimes extending as far as the rectum. The terminal ileum may also be involved. The mucosa of the bowel may be oedematous, reddened or contain multiple petechial haemorrhages and may be partly covered by a diphtheritic membrane. A "tiger-stripe" pattern created by exaggerated, congested longitudinal or transverse mucosal folds is sometimes present, perhaps reflecting the tenesmus.

The gross lesions of coccidiosis in **lambs** and **kids** differ from those in calves and tend to be less haemorrhagic. Some species of *Eimeria* induce discrete yellow/white nodules (0.5-1.5 mm diameter) in the mucosa of the small intestine. These consist of hyperplastic enteric epithelial cells containing various stages of coccidia and are often detected incidentally during postmortem examination of kids and lambs. In animals that have died of coccidiosis the nodules are often abundant and may be virtually contiguous. Not all *Eimeria spp* in lambs and kids induce nodule formation. Others may infect epithelial cells in the colon and between nodules in the small intestine and their significance may be underestimated grossly. The mucosa may contain petechiae but is seldom as reddened or the intestinal contents as haemorrhagic as it is in heavily infected calves.

A diagnosis of coccidiosis can be supported by the demonstration of large numbers of protozoal stages in mucosal scrapings or tissue sections, but attributing significance to a coccidial burden is often difficult.

Nematode infestations of the small and large intestine are extremely important in grazing ruminants, but the gross changes are seldom specific. *Trichostrongylus spp, Nematodirus spp* and *Cooperia spp* reside in the proximal third to half of the small intestine but are too small to be easily seen grossly in the gut contents. In heavy infestations, the content is watery and there may be oedema of the mesentery and mesenteric lymph nodes. Diagnosis should be based on a combination of drenching history, clinical signs, faecal egg counts in live animals and total worm counts in animals that have died or been sacrificed. *Oesophagostomum columbianum* infestation in sheep is characterised by the formation of large nodules (0.5-1.0 cm diameter), with a caseous or mineralised centre, in the submucosa of the small and large intestine. The nodules are caused by invasion of the mucosa by larval stages. Adult worms reside in the lumen of the colon where they damage the mucosa and induce catarrhal inflammation. *O. venulosum* also infects sheep but is much less pathogenic and does not form nodules. In calves, *O. radiatum* is more pathogenic than *O. venulosum* and causes lesions similar to those of *O. columbianum* in sheep.

Acute duodenitis may occur in cattle, sheep and goats following the ingestion of herbage contaminated with massive numbers of **Paramphistomum** metacercariae. Immature paramphistomes invade the duodenal mucosa and may be detected grossly and/or histologically in large numbers in animals that die of intestinal paramphistomosis. As mentioned above, adult stages of the fluke are found in the forestomachs where they do not cause significant damage.

The **tapeworm** *Moniezia expansa* is a common incidental finding in the small intestine of lambs and kids. Heavy tapeworm burdens have been incriminated as a cause of diarrhoea and illthrift in lambs but such claims are not supported by convincing evidence. *Cysticercus tenuicollis*, the cystic form of the canine tapeworm *Taenia hydatigena* is found incidentally in the peritoneal cavity of sheep. While not clinically significant, the presence of these cysts indicates that the farmer has

been feeding raw offal to his dogs. This creates the risk of infecting his sheep with *Cysticercus ovis*, which has important implications for meat quality.

Miscellaneous

Mesenteric torsion is a relatively common cause of acute death in young ruminants, but can also occur in adults. Affected animals are usually found dead with a markedly distended abdomen. On opening the abdominal cavity, the dark red, gas-filled loops of bowel bulge from the incision. In most cases the diagnosis can be readily confirmed by palpation of the root of the mesentery and demonstration of a twist. It is important to check the mesenteric root before the bowel is removed, otherwise the twist may no longer be apparent. In some cases the lesion involves only a short section of small or large intestine, while in others the entire small intestine and/or colon may be involved. Animals that survive for a few days before dying of torsion usually have an acute fibrinous peritonitis due to leakage of toxins or bacteria through the devitalised wall of the twisted bowel. A syndrome referred to as "red gut" is recognised in sheep and occasionally calves grazing lucerne and other lush, leguminous pastures. Although the gross lesions are consistent with mesenteric torsion, a twist about the mesenteric root is not always apparent at necropsy and the pathogenesis may be more complex, at least in some cases. Histopathology is of little value in confirming a diagnosis of mesenteric torsion.

It is debateable whether **enterotoxaemia** should be included amongst diseases of the alimentary tract as the gross and microscopic lesions occur elsewhere. The disease is well recognised as a cause of sudden death in lambs and occasionally adult sheep. It also occurs in calves and kids but its importance in these species is probably over-rated. Excessive production of epsilon toxin by *Clostridium perfringens* type D in the small intestine is the basis of the disease. If sufficient epsilon toxin is absorbed, and if there is insufficient protection by anti-epsilon antibodies, widespread endothelial damage occurs. The lungs are typically congested and oedematous and there is usually an excess of clear fluid containing fibrin in the pericardial sac. The enteric changes are unremarkable even though some animals show profuse, terminal diarrhoea. Some loops of small intestine may be distended with gas and contain a modest amount of creamy "mayonnaise" content. The "pulpy kidneys" commonly associated with enterotoxaemia are not always present and can be misleading even when they are. Glycosuria is also an unreliable diagnostic criterion as it may also be present in other CNS diseases of sheep, including polioencephalomalacia and listeriosis. Confirmation of a diagnosis of enterotoxaemia is best achieved by histological examination of the brain. Endothelial cells in the brain are particularly susceptible to epsilon toxin and small blood vessels in some areas become surrounded by lakes of protein-rich fluid.

Intestinal carcinoma is a relatively common tumour of adult sheep in New Zealand, causing a chronic wasting condition resembling Johne's disease clinically. It also occurs in other countries but the prevalence is much lower. The gross lesions are sufficiently characteristic to allow a definitive diagnosis in most cases. In the advanced stages, multiple firm, white plaques varying from a few millimetres to several centimetres in diameter are present on the serosal surface of the jejunum or occasionally the ileum. A polypoid projection into the gut lumen is often present and is likely to be the site of origin of the tumour. In some cases, a scirrhous band at the site of origin causes partial obstruction of the jejunum. The tumour metastasises to mesenteric lymph nodes and by coelomic spread to the serosal surfaces of other abdominal viscera. Distended lymphatics caused by blockage of lymphatic drainage may be confused with the lymphangitis associated with Johne's disease in some cases, but the lesions in intestinal carcinoma generally involve the jejunum rather than the ileum. The diagnosis can easily be confirmed by histopathology.

Lymphosarcoma of the intestine occurs occasionally in sheep and may be confused with intestinal carcinoma. In lymphosarcoma, the involved segment of bowel (perhaps up to 10 cm) is uniformly thickened and pale cream due to infiltration of the mucosa and submucosa with tumour cells. The corresponding mesenteric lymph node is also likely to be enlarged and uniformly pale cream.

Mesothelioma of the peritoneal cavity is rare but a congenital form of the tumour is reported in cattle. Multiple, firm, red or yellow nodules or plaques, varying from a few millimetres to several centimetres in size are scattered throughout the peritoneal cavity.

Abdominal fat necrosis is an unusual but not uncommon disorder of adult cattle, particularly Channel Island breeds. The pathogenesis is not known but a dietary origin is suspected. Extensive areas of necrotic omental or retroperitoneal fat may surround intestinal loops or other viscera, sometimes causing obstruction of the intestine or ureters. Necrotic fat in the pelvic canal may cause dystocia. The necrotic fat is firm, dry and may be surrounded by a zone of hyperaemia.