The Pathology

of

Equine Gastrointestinal Disease

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I Declare that this dissertation has been composed by myself and the work described is entirely my own.

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ABSTRACT

To initiate the study a comprehensive review was made of the available literature relating to the equine gastrointestinal tract. This is presented in the literature review. It includes papers and texts concerning the anatomy and function of the equine gastrointestinal tract as well as causes of clinical disease and pathological lesions.

A total of 213 horses, ponies and donkeys underwent a full post mortem examination by Glasgow University Veterinary School Department of Veterinary Pathology in the the 5 year period 1987-1991 inclusive. The majority were mixed breed pleasure animals rather than pedigree competition horses. These findings are presented in Part I, the Necropsy Series. The lesions recorded were considered, on the basis of appearance and clinical history, to be either principal or incidental and were then grouped by the system in which they were found. Lesions were identified in all anatomical systems. The gastrointestinal system was found to be by far and away the most frequently affected by both principal lesions, ie those considered to have resulted in the animal's death or humane destruction, and incidental lesions with no reported history of associated clinical disease.

The Necropsy Series identified 92 animals with one or more lesions of the gastrointestinal system which were studied in more detail. These findings are presented in Part II, the Gastrointestinal Series. Here the general groupings used in the Necropsy Series were broken down by location and the actual lesion identified. In addition, other factors such as possible seasonal incidence and any concurrent, contributory lesions were also

studied. Grass sickness was the most frequently identified significant primary condition and parasitism featured heavily as a cause of incidental lesions. The Gastrointestinal Series in turn revealed a group of 20 animals with clinical evidence of malabsorption and a chronic enteropathy which were studied in depth. Histopathological examination of multiple tissues from these cases allowed them to be divided into 2 groups, one of recognised conditions where a definitive diagnosis was made and one where only a morphological diagnosis was made. This series is presented in Part III, the Enteropathy Series. The recognised conditions identified by the Enteropathy Series were alimentary lymphosarcoma, equine granulomatous enteritis, equine eosinophilic enteritis, cyathostomiasis, Phenylbutazone toxicity and coccidiosis. However, these 6 conditions only accounted for 11 of the 20 animals. Eight of the remaining 9 animals had gastrointestinal lesions which differed significantly from those previously described in the equine literature. These were the animals where only a morphological diagnosis was made. The remaining case had clinical evidence of malabsorption in the form of an abnormal oral glucose tolerance test result but apparently no significant gross or microscopic lesions of the gastrointestinal tract, just extremely abnormal dentition. The Enteropathy Series revealed that the recognised equine inflammatory bowel diseases do occur in the UK and are not restricted to Standardbreds and Thoroughbreds as the available literature would suggest.

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ABBREVIATIONS

G.I.	Gastrointestinal
N.S.A.I.D.	Nonsteroidal Anti-inflammatory Drug
P.B.Z.	Phenylbutazone
V.I.P.	Vacuum Impregnated Processor

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INTRODUCTION

There can be few students of veterinary anatomy who have not at some time wondered at the design of the equine alimentary tract with its sudden, sharp bends and constrictions. Indeed the complete lack of consistency from one portion to the next suggests the hand of a large committee at the initial design stage. Despite this, the average horse gut does appear to function remarkably well.

Nevertheless, problems do occur and equine alimentary tract disease has been recognised since the time of Hippocrates. The cold, clinical descriptions in todays' texts (**White 1990**) often augmented by reams of haematological or biochemical results are certainly informative but they lack much of the realism and emotion of reports from the 19th Century. One could not fail to comprehend the seriousness of "inflammation of the intestines" from the following description by **Captain Hayes (1889)** "The continuance of this torturing pain drives the animal to a state of extreme restlessness and distress; he is either pawing, or repeatedly lying down and rising again, or else he is walking around his box breathing hard, sighing and perhaps occasionally snorting..... The eye acquires a wild, haggard, unnatural stare; the pupil dilates; his heedless and dreadful throes render approach to him quite perilous..... when all at once, in the midst of agonising torments he stands quiet..... his body bedewed with a cold, clammy sweat; he is in a tremor from head to foot, and about the legs and ears has even a death-like feel..... death, and not recovery is at hand."

It is clear then that equine gastrointestinal disease has been recognised for a long time as a clinical entity and its potentially fatal course appreciated. However, few studies have been made into its

significance either as a cause of death or of subclinical lesions. To this end it was decided to review the pertinent literature and to study the lesions present at necropsy of all horses examined by the Glasgow University Veterinary School Department of Pathology over a 5 year period. The information gleaned from this Necropsy Series was used to establish the importance of diseases of the gastrointestinal system, compared to those of other anatomical systems, as a cause of death or of subclinical, presumably incidental, lesions.

One important group of gastrointestinal diseases, the enteropathies, has received little attention of a comparative nature although many individual case reports exist. It was decided that any such cases identified in the Necropsy Series would be selected to form an Enteropathy Series which would be investigated in depth to allow more detailed comparison of their pathological findings to be made.

The results of these studies are presented in three parts, first the Necropsy Series as a whole, secondly a more detailed account of lesions of the Gastrointestinal System, the Gastrointestinal Series, and thirdly the Enteropathy Series.

LITERATURE REVIEW

EQUINE GASTROINTESTINAL DISEASE

ANATOMY

The anatomy of the equine gastrointestinal system has been studied in great detail. Standard texts such as "The Viscera of the Domestic Mammals" (**Nickel, Schummer and Seiferle 1979**), "Sisson and Grossman's The Anatomy of the Domestic Animals" (**Getty 1975**), "Atlas of Equine Anatomy" (**Pasquini, Reddy and Ratzlaff 1978**) and "Rooney's Guide to the Dissection of the Horse" (**Sack 1992**) provide accessible and useful information on the position and size of each portion of the tract. "The Viscera of the Domestic Mammals" (**Nickel et al 1979**) also provides details of intestinal length and the normal microscopic appearance of some portions of the tract.

In addition **De Boom (1975)** gives a readable potted account of the development and anatomy of the equine alimentary tract. This description includes a number of interesting points - eg in the horse the ratio of intestinal length to body length is 10:1 compared to 5:1 for the dog and 25:1 for ruminants. Many of the "design faults" are highlighted not least the very wide mesentery of the small intestine and the large intestinal constrictions such as the pelvic flecture. The paper also includes a section on the innervation of the alimentary tract and points out the close proximity of the sympathetic ganglia to the cranial mesenteric artery and the possibility that a lesion in the latter may affect the function of the former.

The blood supply to the equine gastrointestinal system has been studied in some considerable detail. It plays an extremely important role in the production, exacerbation and outcome of a number of conditions eg

intestinal displacements (Snyder 1989) and thromboembolic infarction as a result of *Strongylus vulgaris* activity (Rous 1975).

The major vessels are fully described and illustrated by the standard texts (Getty 1975, Sack 1992). The microvascular circulation of the large intestine has received special attention from a number of authors. The first portion studied was the ascending (large) colon (Snyder et al 1989b). Various techniques were employed such as the use of radiopaque medium for microangiography, plastic material for the production of vascular replicas and light microscopy. The perfusion medium used for microangiography was a new mixture which did not require heat to maintain the gelatin in liquid form thus avoiding thermal tissue damage. The addition of potassium iodide meant that the gelatin solidified only on fixation by formalin. The two possible mechanisms to explain reperfusion injury were presented ie oxygen metabolite liberation and no reflow. The results of the study suggested that capillaries in the colon supply a relatively large area of the mucosa and are drained by sparsely distributed venules. Therefore obstruction of a submucosal arteriole supplying these capillaries would result in ischaemia of a larger lumenal surface area in the colonic mucosa compared to obstruction of a small intestinal villus arteriole.

Dart, Snyder, Julian and Hinds (1991) studied the microcirculation of the caecum. Similar techniques to those of Snyder and colleagues (Snyder et al 1989b) were employed. The authors showed that the medial and lateral aspects of the caecum were supplied by their respective arteries with minimal mixing of the two circulations. The primary supply to the apex was derived from the medial artery. At the

microvascular level many similarities and differences between the caecum and large colon were discovered.

The microvasculature of the descending (small) colon has also been studied (Dart, Snyder and Harmon 1992a). The work was undertaken because it had been suggested that postsurgical healing of this portion of the gut was poor partly because of a poor blood supply. Similar techniques to those used previously were employed (Snyder et al 1989b). The vascular arrangement of the small colon differs from other areas of the equine intestine probably to accommodate the thicker longitudinal muscle layer of the muscle band. The direct penetration of the short terminal arteries on either side of the mesenteric band may substantially weaken the intestinal wall predisposing these sites to tears. This is particularly interesting as many "rectal" tears in fact occur in the terminal colon and are usually located between 10 and 2 o'clock.

FUNCTION

The function of the large intestines and their interrelationship in disease were extensively reviewed by Argenzio (1975). The most critical function is the large intestines' capacity for storage and absorption of tremendous volumes of fluid, approximately equivalent to the total extra cellular fluid volume every 24 hour period. In addition much of the soluble and most of the insoluble dietary carbohydrate escapes the small intestines and is presented to the large intestines for microbial digestion. Retention of digesta by the large intestines is primarily controlled by barriers at the ileo-caecal, caecal-ventral colonic, ventral-dorsal colonic (pelvic flecture) and dorsal-small colonic

(transverse colonic) junctions. Motility, microbial digestion, transport of solute and water and functional diseases of the large intestine were discussed.

Sellers, Lowe, Rendano and Drost (1982) looked more specifically at the reservoir function of the equine caecum and ventral large colon and its relation to chronic non-surgical obstructive disease with colic. The Authors stated that the most frequent cause of colic is a functional abnormality of the large intestines and not a surgical emergency. No area of infarction or other gross lesion is found at necropsy, the abnormality being confined to an electrical "pacemaker" near the pelvic flecture. The Authors attempted to disrupt this "pacemaker" by the use of local anaesthetics, sudden change in diet, the acaracide Amitraz and infection with *Strongylus vulgaris*. All were successful to some degree but in the latter case, *S. vulgarus*, clinical signs did not always correlate with the lesions found at post mortem examination.

The effect of experimental vascular occlusion on small intestinal motility was studied by **Davies and Gerring (1985)**. Hydraulic vascular occluders were used to shut off the supply to a "thiry loop" of jejunum in conscious animals and the clinical response, mucosal appearance (using a flexible endoscope), and changes in motility (using strain gauges) were noted. Similar studies were made on an intact loop of ileum. A second experiment to simulate embolisation was performed under general anaesthesia. Strain gauges were placed in a portion of the terminal ileum and in portions of the ileum proximal and distal to it. The ileal and the most caudal intestinal artery were blocked by injecting a silicone material. Marked changes were observed in the

"thiry loop" but not in the intact loop of ileum. In the second experiment marked changes in motility were noted. The experiments showed that pain is a feature of small intestinal ischaemia before necrosis or peritonitis occurs and that although the affected portion of gut becomes hypomotile the portions on either side become hypermotile which could result in mechanical obstructions such as those encountered clinically.

Sellers and Lowe (1986) reviewed large intestinal motility and mechanisms of impaction in the horse. Under normal circumstances the caecum and ventral large colon serve a reservoir function and allow a 48 to 72 hour delay for the resident flora to get to work. Interruption of the extrinsic nerve modulation results in diminution of co-ordinated pressure peaks and neurectomy results in transient diarrhoea. The Authors described the normal rhythm and pattern of contractions in the caecal wall and their effect on ingesta. No clear connection has been found between *S. vulgaris* cranial mesenteric arteritis-thrombosis and non-surgical obstructive disease of the large colon, but derangement of the "pacemaker" at the pelvic flecture resulted in ileus, impaction and colic. This might come about as a result of a stressful environmental change but this would be difficult to assess. In another review article **Wingate (1986)** compared and contrasted colonic disease in the horse and in man. In the former the outcome is often fatal. This is not a consequence of delayed presentation because in man colonic disease is not a surgical emergency but may simply be a function of the organs' greater size in the horse. The Author also stated that studies on small bowel transit in human cases of inflammatory bowel disease suggest that the colon may be an innocent victim of small intestinal disfunction.

Post operative ileus is another condition rarely fatal in man but frequently so in the horse.

VASCULAR OCCLUSION STUDIES

The effects of damage to the gastrointestinal blood vessels and interruption of their supply has also been investigated by a number of Authors. **White, Moore and Trim (1980)** looked at mucosal alterations in experimentally induced small intestinal strangulation obstruction which was defined as interruption of the intestinal blood supply with simultaneous blockage of the intestinal lumen. Intestinal strangulation obstruction is a frequent cause of acute abdominal crisis in the horse and usually results from small intestinal volvulus or incarceration through a mesenteric rent or natural opening. A system of grades was used to assess mucosal degeneration with I corresponding to normal through to V corresponding to loss of villous architecture and early necrosis of crypt cells. Each grade is both described and illustrated. In all cases the grade chosen increased in proportion to the length of time the gut had been ischaemic. The mucosal degeneration was found to continue after the ligatures were removed even with gross evidence of perfusion and muscular activity. This may explain the temporary recovery of horses with an eventually fatal outcome following such a lesion. This demonstrated the usefulness of a microscopic assessment of the mucosal lesion after intestinal strangulation obstruction.

Sullins, Stashak and Mero (1985) used procedures similar to those of **White and colleagues (1980)** to assess the pathologic changes associated with induced small intestinal strangulation obstruction (ischaemic strangulation obstruction) and nonstrangulation infarction (haemorrhagic

strangulation obstruction). In the former both the artery and vein were occluded, in the latter only the vein was occluded. Again the I-V grading system was used. The ischaemic strangulation obstruction grades correlated to some degree with the length of time the vessels were occluded but the grades assigned to the haemorrhagic strangulation obstructions correlated completely.

Early mucosal healing and chronic changes in the jejunum after various types of strangulation obstruction have also been described **(Freeman et al 1988)**. The Authors used techniques similar to those of **Sullins and colleagues (1985)** clamping just the vein or both the artery and vein and graded samples I-V. This study was more long term. The gross changes were noted and samples of compromised and normal gut were taken both during the procedure and after the clamps had been removed. The animals were euthanised and examined post mortem either 12 or 42 hours postoperatively. The changes were not confined to the compromised segments. The mucosa continued to deteriorate, presumably due to reperfusion injury, after the clamps were removed. Repair was subsequently rapid and complete. The only lesion encountered at 42 days was fibrosis at the clamp sites.

Snyder and colleagues (1988) studied the morphologic alterations during experimental ischaemia of the large colon. Again the Authors clamped the colonic vein or both the colonic artery and vein or compressed the colonic wall plus venous or venous and arterial occlusion. Tissue samples were graded for a number of specific changes, the extent of the haemorrhage and oedema, the interstitium to crypt ratio and alterations in lumenal and crypt epithelium. Cellular changes appeared 30 minutes

earlier in the group where the arterial supply had been occluded suggesting that some oxygenation is achieved even with capillary stasis. The epithelial changes differed from those previously recorded in the small intestines following similar periods of compromise. In the colon small groups of epithelial cells became necrotic and broke away from the basement membrane and from other epithelial cells. In the small intestines sheets of epithelial cells lifted off *en mass*. Another interesting observation was the lack of lamina proprial cell infiltration when compared to infarction by thromboembolic disease. Changes continued to increase in severity for at least 30 minutes after release of vascular perfusion, as has been described in the small intestine due to reperfusion injury.

Snyder (1989) tied together the available information on the pathophysiology of intestinal damage re effects of lumenal distention and ischaemia. The Author explained the different types of strangulation obstructions and the significance of the various induced changes such as oedema and lumenal distention. The pattern of injury in the small intestinal villus was explained. For example, lifting of the epithelium at the tip which occurs in response to hypoxia produced by the countercurrent mechanism allowing oxygen to short circuit the villus which under normal circumstances, never mind general hypoxic states, means the villus tip is relatively hypoxic compared to the base. Other factors are also involved such as the presence of pancreatic proteases within the intestinal lumen. It has also been shown that the colonic mucosa is more resistant to ischaemic change than that of the small intestines requiring the ischaemia to be present for 25% longer to produce a severe lesion. The most likely explanation of reperfusion

injury, ie a rise in reactive oxygen metabolite/oxygen radical concentrations, was also presented at some length as was the role of the neutrophil.

The vascular injury associated with naturally occurring strangulating obstructions of the equine large colon has also been studied (Snyder et al 1990). The Authors stated that volvulus of the large colon is the most severe form of large intestinal disorder and occurs in 7% to 11% of horses undergoing surgery for colic. The ascending colon is predisposed to volvulus because of its size and single mesenteric attachment to the body wall. Most commonly a 360° volvulus is present but only a 180° twist is required to cause strangulation of the vasculature. Ten animals with volvulus of the terminal colon were studied. Biopsy specimens were taken from the pelvic flecture and the morphologic damage to colonic tissue assessed. Studies of the colonic vasculature were made by perfusing the vessels with a gelatin based substance and microangiography. In all horses the colonic volvulus was at the mesenteric attachment of the colon. The rotation was 360° in 9 horses and 720° in one, in a medial direction in 7 and dorsolateral in 3. The arterial pulse did not return in 3 animals and the mucosa remained black in 2 post correction. The serosal colour improved in only 7 animals. Haemorrhage was identified in the mucosa and submucosa and almost all the surface epithelium was lost in each case. A decrease in the number of perfused vessels was seen in microangiographs of the serosa and muscularis in 9 animals. The extent of the vascular lesion correlated with the serosal colour noted. There was little vascular perfusion of the mucosa. The perfusion studies revealed vascular obstruction at the muscularis mucosa with frequent thrombi in the submucosa. Post mortem

examination of the case which survived 8 days revealed complete reepithelialisation and more than normal mucosal perfusion. The Authors concluded that loss of 50% or more of the cells within the colonic glands suggests a grave prognosis.

More recently **Meschter, Craig and Hackett (1991)** looked at the histopathological and ultrastructural changes in simulated large colon torsion and reperfusion. The samples taken while the vessels were occluded degenerated sequentially as previously described **(White et al 1980)**. The lesions were slightly more severe in the haemorrhagically infarcted bowel, ie where only the vein had been occluded, compared to the ischaemically infarcted bowel where both the artery and vein were occluded. Total necrosis of the colonic mucosa occurred during the reperfusion period with marked neutrophil accumulation. The Authors emphasised the importance of reperfusion injury in allowing the absorption of toxins and loss of fluids leading to the animals death. An interesting point to note was the progressive obstruction of the microvasculature with large masses of platelets during reperfusion, thus further blocking blood flow to the mucosa.

DISEASE SURVEYS

All published surveys of equine disease include references to conditions of the alimentary system. The British Equine Veterinary Association Survey of Equine Disease 1962-63 (BEVA 1965) places the alimentary system second only to the musculoskeletal system in the league table of reported incidences of disease, accounting for 10% of the horses surveyed. Even when the cases of upper alimentary tract and liver disease are excluded the numbers are still significant with parasites and colic topping the list.

One survey of post-mortem findings in 480 horses found the alimentary system to be the most frequently involved in the cause of death accounting for 33% of the cases (**Baker and Ellis 1981a**). The alimentary system was also the most common source of incidental lesions, supplying 88.1% of the total, over 90% of these being parasitic in nature (**Baker and Ellis 1981b**). Lesions of the alimentary system may even be responsible for the unexpected if not sudden death of an animal (**Brown, Taylor and Slanker 1987**). Although almost all potentially fatal conditions cause some degree of abdominal pain the course may be extremely short, a matter of hours, so the signs may not be observed. A survey of 69 sudden and unexpected deaths in horses pin-pointed lesions of the alimentary tract as the cause in a number of cases (**Platt 1982**). In the group found dead in the morning with no history of illness the previous day, alimentary disorders were the most common cause of death, being responsible in 16 of the 28 animals. In another group where the animals were apparently well on the morning when last seen alive but were later found dead, lesions of the alimentary tract were responsible

for 4 of the 17 deaths. This included one apparently instantaneous death in a brood mare. Post-mortem examination revealed boluses of food in the oesophagus and a ruptured caecum but no associated haemorrhage. The findings were consistent with cardiac arrest due to vagal inhibition, stimulated by collapse of the distended caecum. Lesions of the alimentary tract were not detected in the group of 24 animals which were witnessed to die suddenly or within minutes of the onset of signs.

In papers where observations were confined to conditions of the stomach and intestines it becomes apparent that a great many lesions exist. These papers also allow the assessment of their relative importance. **Rollins and Clement (1979)** reported on the incidence of colic in their private practice in Arizona confining their observations to the type of colic seen, most commonly symptomatic undiagnosed and sand colic, and the time of year it occurred, usually May to October. However, an idea of the incidence is given by the fact that 1042 occurrences of colic were identified in 10,541 case records examined.

More recently **Proudman (1991)** published a 2 year prospective survey of equine colic in general practice in England. The bulk of the cases fell into the spasmodic undiagnosed category (72%) with the remainder being flatulent (5.5%), pelvic flecture impaction (5%), other impactions (9.5%), surgical cases (7%) and colitis (1%). The incidence of surgical colic in horses over 15 years of age was significantly higher, and the incidence of colic over all in stallions significantly lower, than expected in comparison to the control group. No definitive explanation could be given. No pathological details were included.

GENERAL ARTICLES

Conditions of the stomach and intestines exist which do not cause colic. These and many potential causes of colic have been reviewed by a number of Authors. Robertson (1982) reviewed conditions of the stomach and small intestines with the emphasis on differential diagnosis and surgical management. Nonetheless this is an extremely informative paper giving a brief description and explanation of the majority of recognised causes of acute gastric and small intestinal disease in the horse, including relevant references. Chronic inflammatory conditions were generally ignored. Diseases of the large intestines were dealt with in a similar fashion by Foerner (1982). The Author describes surgical conditions of the caecum, large colon and small colon in turn and makes the interesting observation that the caecum seems to be particularly susceptible to thromboembolic infarction which may be progressive. This is probably due to the fact that the caecum is only supplied by a single vessel, the right branch of the cranial mesenteric artery.

Dart and colleagues (1992b) focused their attention on conditions of the descending (small) colon, reviewing the findings in 102 cases. The Authors regarded the incidence of lesions in this portion of the gut, and consequently their level of knowledge, to be low. A number of general facts emerged, certain breeds and females were over represented. The incidence was reduced in horses less than 5 years old and increased in horses greater than 15 years old. The most frequently encountered conditions were enteroliths (35.29%) and impactions (34.30%). The remaining conditions identified were strangulating lipomas, faecoliths, foreign body obstructions, volvulus and nephrosplenic entrapment.

Breed, age and sex predilections for the individual conditions also emerged.

Roberts (1990) took a different approach and reviewed a condition, acute equine colitis, rather than an anatomical region. The many known causative agents were listed all of which induce similar pathophysiological changes possibly leading to death in 6-24 hours. The microbiological and clinical features and the therapeutic management were described and explained. Experimental colitis was also reviewed and the previously described models listed. The Author believed that the administration of castor oil induced a colitis indistinguishable from the naturally occurring syndrome and was the first reproducible, reversible non-infectious model.

In general the above papers, with the exception of **Roberts (1990)**, confine their observations to the gross findings and even then they are presented from a surgical, rather than pathological, point of view. The histopathological findings in the gastrointestinal tract of horses with colic were described by **Meschter, Tyler, White and Moore (1986)**. The Authors took samples from the lesion sites and from specific sites throughout the gastrointestinal tract. A number of conditions were studied, some lesions were common to all and others were more specific. The Authors graded both the small and large intestinal lesions in a numerical fashion for case comparison. Sloughing of small intestinal epithelial cells was prominent and common in the strangulation obstruction, thromboembolic and inflammatory groups, most marked in the former. The change was observed not only at the lesion site but proximal and distal to it as well. The Authors also assessed eosinophil

and mast cell numbers, which were significantly related. It was proposed that the former were present in response to chemoattractants produced by the latter. Also, eosinophil numbers were not related to evidence of parasitism and were significantly elevated in horses which survived. They may be important in the suppression of inflammatory reactions triggered by mast cell degranulation and thereby increase the chance of survival.

Not all sequelae to gastrointestinal lesions become apparent immediately and are therefore not identified at the time of surgery. Indeed, lesions may arise at some later date as a consequence of surgery. A retrospective evaluation of 53 horses which underwent repeat laparotomy was made by **Parker, Fubini and Todhunter (1989)**. This group represented only 8.2% of the horses which underwent a laparotomy in the time period. Forty six of the group underwent one additional procedure but 7 animals had multiple additional laparotomies. Twenty three horses had a lesion at the second surgery which had occurred as a sequela of the initial lesion or procedure, 19 horses had a progression of the initial lesion, 8 horses had recurrence of the same condition and 3 had developed an unrelated lesion. The most common finding at the second laparotomy was intestinal ischaemic necrosis without concurrent mechanical intestinal obstruction. This occurred in the small intestines of 7, large colon of 5 and small colon of one animal. The second most common lesion was small intestinal obstruction by adhesions which occurred in 10 animals all of which had a small intestinal lesion at first surgery. The interval between laparotomies in this group varied from 8 days to one year.

Baxter, Broome and Moore (1989) limited their observations to adhesions after small intestinal surgery. The Authors pointed out that initially adhesions are beneficial providing an additional blood supply to compromised serosa and intestine but when they are converted to permanent fibrous adhesions problems may be encountered. This conversion begins 3-4 days after the initial injury and depends on the functional ability of the fibrinolytic system within the peritoneal cavity and bowel serosa. The study included 113 horses, 25 of which (22.1%) developed adhesions which resulted in clinical problems. This problem was 3 times more likely to develop in males. The most common consequence was ileal impaction which occurred in 12 horses. Strangulated inguinal hernias and small intestinal volvulus each occurred in 2 animals, proximal enteritis in 2 and ileocaecal intussusception, jejunal impaction and mesenteric rent with small intestinal incarceration each occurred in one animal. Only 4 animals survived repeat surgery and in each case ileal impaction had been identified.

CONGENITAL DISORDERS

A number of congenital disorders of the gastrointestinal tract have been recognised. Some become apparent almost immediately after birth **(Estes and Lyall 1979)** some cause problems in later life **(Hooper 1989)** and some may never become apparent clinically **(Freeman, Koch and Boles 1979)**

Two reviews of equine congenital defects have been published. The first lists all the conditions reported to that date **(Austen, Saperstein and Leipold 1977)** including atresia of the colon, of the rectum and of the

anus. The second describes the findings in a series of foals necropsied because of a congenital defect but does not include any involving the gastrointestinal tract (**Crow and Swerczek 1985**).

Becht and Semrad (1986) reviewed gastrointestinal diseases of foals and included a section on congenital defects. The Authors stated that atresia ani is a common anomaly and varies from a well developed anus and short blind rectum to a barely perceptible depression. Atresia coli and ileocaecal aganglionosis were mentioned along with inguinal and diaphragmatic hernias.

Case reports of atresia ani in the foal are sparse but **Furie (1983)** did report one case which also had a persistent cloaca. The filly was able to urinate but was not observed to defecate. Post-mortem examination revealed that the rectum terminated in the pelvic region, by inserting into the caudodorsal vagina, thus forming a cloaca. The urethra inserted normally into the floor of the vagina cranial to the rectal opening.

There are two repeatedly described conditions which exert their effects on the neonate. The first, atresia of the colon has no breed restriction but is frequently seen in the white male progeny of Overo Spotted Horses (**Estes and Lyall 1979**) and the second, ileocolonic aganglionosis is confined to the white progeny of Overo Spotted Horses (**McCabe et al 1990**).

Estes and Lyall (1979) reviewed the literature on atresia of the colon and contributed an additional 4 cases, all Thoroughbreds,2 with a large

colon defect and 2 with a small colon defect. The two opposing mechanisms were explained, the largely discredited recanalisation theory and the broadly accepted vascular accident theory. The anatomical classification into Types I to III, as used in man, was also given.

A more recent paper (**Cho and Taylor 1986**) described blind-end atresia coli in 2 foals, one Appaloosa and one Quarter Horse. The Appaloosa lacked a dorsal colon and the proximal small colon and the Quarter Horse lacked the left ventral and dorsal colons and colonic mesentery. Again the anatomical classification was given and type III blind end atresia deemed appropriate for the 2 cases described. Although the condition was considered to be rare in the horse the literature as reviewed pointed to a hereditary basis.

Van Der Gaag and Tibboel (1980) reviewed 34 cases of intestinal atresia and stenosis in animals. Only 2 equine cases were included, one 5 month female with duodenal stenosis and one 2 day foal of unknown sex with colonic atresia. The various mechanisms were discussed and the German literature reviewed.

There is a single report of congenital pyloric stenosis in a 2 month old filly (**Barth, Barber and McKenzie 1980**). At surgery the stomach was markedly dilated and the intestines were almost empty. The pylorus was firm on palpation, small in circumference and contained a narrow canal, the patency of which could not be determined externally. Surgical correction was successful. The Authors listed the 3 possible causes of pyloric stenosis namely scarring post gastritis, neurogenic pylorospasm

or congenital hypertrophy. The latter was considered responsible in this case.

The largest study of ileocaecal aganglionosis (lethal white foal syndrome) in the white progeny of Overo Spotted Horses was carried out by **Hultgren (1982)**. He recorded the findings in 19 foals, 10 males, 6 females and 3 of unrecorded sex. Only 2 foals had any pigmentation and this was extremely limited. In all cases there was retention of the meconium, dilatation of the gut by gas was variable and the small colon was usually tightly contracted. Histopathological examination revealed the presence of myenteric ganglia in the stomach, duodenum, jejunum and proximal ileum but their complete absence in the terminal ileum, caecum and colon. The correlation between lack of pigment and lack of myenteric ganglia was explained by their common neural crest origin and some abnormality in embryonic migration of these cells. The comparison with Hirschsprung disease of man and similar conditions in mice was made.

The most recent study **(McCabe et al 1990)** considered the condition to be an equine model of aganglionic megacolon (Hirschprung disease). The findings in 2 foals were described, both had a constricted area in the large intestine described as "pseudotransition zones" with aganglionosis extending proximally for some distance. The histopathological findings were similar to those of **Hultgren (1982)**. The genetics of the condition were described as compatible with autosomal inheritance and the incidence given as 6% of all Overo X Overo matings. The embryological connection between pigment and myenteric ganglia is explained and mechanisms for the development of the syndrome proposed.

Murray, Parker and White (1988) described a single case of myenteric hypoganglionosis in a 6 month old Clydesdale foal. The submucosal ganglion cell population was normal throughout the small intestine but was greatly reduced in the caecum and right dorsal colon, moderately reduced in the transverse colon and mildly reduced in the left ventral colon. The animal's external pigmentation was normal with brown hair on the body. The Authors proposed that there had been degeneration or abiotrophy of the myenteric ganglion cells.

There is an isolated report of a congenital jejunal diverticulum in a 3 week old Thoroughbred filly **(Yovich and Horney 1983)**. It was discovered incidentally during correction of a small intestinal volvulus and was considered uninvolved. The surgery was unsuccessful and the foal was euthanised 12 days later. At necropsy the diverticulum was still considered to be an incidental finding. It was 25cm long, 2cm in diameter, blind ended, arose 60cm from the stomach and was closely attached to the jejunum. Other previously recorded gastrointestinal tract anomalies were listed.

Another condition which became apparent early in life was caecocolic fold hypoplasia **(Harrison 1989)**. It had resulted in caecal torsion in an 8 month old Standardbred filly. At necropsy a 360° clockwise caecal torsion was identified at the level of the caecocolic junction. This had apparently come about because the caecocolic fold was virtually absent allowing free rotation about the longitudinal axis of the caecum. In addition the ileocaecal fold was thick and did not attach the ileum to the caecum.

Problems related to both Meckel's diverticulum (**Hooper 1989**) and the related structures the mesodiverticular bands (**Freeman et al 1979**) have been described. Meckel's diverticulum results from the incomplete closure and disappearance of the omphalomesenteric duct (**Hooper 1989**). It persists as anything from a small fibrous projection on the antimesenteric border of the ileum to an ileal-umbilical fistula (**Grant and Tennant 1973**).

Grant and Tennant (1973) described 2 cases. In the first, a 3 year old Quarter Horse mare, small intestinal volvulus had occurred around a diverticulum attaching the ileum to the umbilicus. The lumen of the diverticulum communicated with that of the ileum for 4cm then continued as a fibrous band for 6cm. In the second case, a 5 year old Tennesee Walking Horse, with small intestinal obstruction, a similar diverticulum was found. It was not clear from the description what had caused the obstruction or whether the diverticulum was involved. Neither animal had a history of recurrent colic.

Sprinkle, Swerczek amd Crowe (1984) described briefly the findings in 5 Thoroughbreds, 2 males and 2 females and one of unknown sex, aged 3 months to 7 years. In each case the Meckel's diverticulum present contributed to the animal's demise. The relevant literature was reviewed including a paper describing the condition in pit ponies and an Icelandic gelding (**Rider 1932**). The Author believed the condition to be uncommon. It had only been encountered in the 5 cases presented out of 15,000 horses necropsied.

Weaver (1987) described the gross findings in a 5 year old Hanoverian mare where the Meckel's diverticulum had become impacted, causing small intestinal obstruction, and ruptured, leading to peritonitis. The diverticulum had been enlarged to 35 x 25 x 25cm and had multiple surface adhesions and splits. The Author believed this to be the only report of fatal impaction of the structure in a horse.

Hooper (1989) described strangulation of the small intestine by a Meckel's diverticulum in a 12 year old Quarter Horse mare. A band of fibrous tissue 2-3cm in diameter, originating from the antimesenteric border of the ileum 40cm proximal to the ileocaecal valve, had looped around and strangulated the ileum and 3.5m of the jejunum. Full examination revealed that the Meckel's diverticulum was in fact 35cm long and 5cm in diameter and communicated with the ileum. The Author believed that this case with the diverticulum knotting around the intestine was unique.

Mesodiverticular bands are caused most commonly by the persistence of a vitelline artery which results in a band extending from the mesentery to the Meckel's diverticulum or in its absence to the antimesenteric border of the small intestines **(Freeman, Koch and Boles 1979)**.

Three cases of small intestinal volvulus and strangulation related to such a structure have been described **(Freeman, et al 1979)**. In the first case, a 7 year old Pinto gelding, volvulus of the jejunum and ileum occurred around a portion of small intestine which had passed through a mesenteric tear and become strangulated. The tear formed one wall of a pocket completed by a large mesodiverticular band. The

findings were similar in the second and third cases, a 9 month old Standardbred filly and an 8 year old Arabian mare. In each case it was proposed that a portion of the small intestines had become incarcerated in a mesenteric herneal sack, formed by the mesentery, the intestine and the mesodiverticular band. One wall had torn allowing strangulation of a portion of the intestines. In addition 2 mesodiverticular bands were found in a 40 day old Standardbred colt euthanised because of an unrelated condition.

FOAL DISORDERS

Diarrhoea is probably the most common clinical abnormality of young foals, up to 80% experience at least one episode in the first 6 months of life (**Urquhart 1981**). There are a number of infectious agents including fungi, protozoa and helminth parasites but bacteria and viruses are probably the most important (**Harbour 1985**).

Urquhart (1981) reviewed the causes of diarrhoea in foals at some length but gave little or no information on the pathological findings. **Harbour (1985)** gave a more concise review but again limited it to the causative agents and relevant literature.

A recent study of the prevalence of enteric pathogens in diarrhoeic Thoroughbred foals in Britain and Ireland confined its investigation to faecal analysis of live foals (**Browning et al 1991**). The prevalence of cryptosporidia, potentially pathogenic *Escherichia coli*, *Yersinia enterocolitica* and *Clostridium perfringens* was not significantly different in diarrhoeic and nondiarrhoeic foals. However, Group A

rotavirises and *Aeromonas hydrophila* were found more frequently in samples from the diseased group. *A. hydrophila* has not been identified previously as a significant pathogen in diarrhoeic foals. **Becht and Semrad (1986)** gave a very useful review of gastrointestinal disease of foals, not limiting themselves to diarrhoea but again pathological details were lacking. Causes of abdominal pain (meconium retention, congenital defects, intestinal accidents, and parasitic infestations) and diarrhoea were described along with their management.

Individual case reports exist on all the common and uncommon disorders. Of the infectious agents *E coli* appear to be of minimal importance in the foal **(Becht and Semrad 1986)**. Salmonellosis in young foals (less than 2 weeks old) often results in sudden death due to septicaemia and bacteraemia without any signs of diarrhoea **(Becht and Semrad 1986)**. **Wenkoff (1973)** reviewed the available literature on salmonellosis and described an outbreak of *Salmonella typhimurium*, the most common equine serotype, in a group of 5 to 6 month old Belgian foals. In the 2 that died there was necrosis and shedding of intestinal mucosa, and infiltration by neutrophils and lymphocytes, haemorrhage and oedema. Necrosis of associated lymph nodes was also noted. A more recent review of salmonellosis in horses states that foals are more susceptible to infection than adults and there may be severe outbreaks on stud farms **(Smith 1981b)**.

A number of different clostridia have been incriminated. **Hibbs, Johnson, Reynolds and Harrington (1977)** described 2 cases of acute haemorrhagic diarrhoea in one week old Quarter Horse foals from the same ranch. One foal had already died having shown similar signs.

Post-mortem examination was performed on one foal and revealed segmented haemorrhage of the ileum. *Clostridium sordelli* was isolated from this animal and Gram positive rods were found in the intestinal content of the other. Haemorrhagic enterotoxaemia (Niilo and Chalmers 1982, Howard-Martin, Morton, Qualls and McAllister 1986) and haemorrhagic enteritis (Pearson, Hedstrom, Sonn and Wedam 1986) caused by *Clostridium perfringens* Type C have both been described in the foal. Haemorrhagic necrotising enteritis associated with a *C perfringens* which was considered to be Type C has also been described (Sims, Tzipori, Hazard and Carroll 1985). The foals were from 1-3 days old, one was found dead (Niilo and Chalmers 1982) and the others died within 15 hours of showing signs of gastrointestinal tract disease. The pathological changes were similar in each case. There was a haemorrhagic or necrotising enteritis involving the jejunum or jejunum and ileum with varying degrees of mucosal sloughing. Histologically the villi were necrotic or sloughed and large numbers of Gram positive rods were seen adhering to the mucosal surface. A fibrinous vasculitis was also present. *C perfringens* Type C or its toxin was identified in tissue or intestinal content samples in each case. *Clostridium difficile* has also been found in association with a haemorrhagic necrotising enterocolitis (Jones et al 1988). The pathological lesions were similar to those caused by *C perfringens* Type C but were more extensive affecting both the small and large intestines. There was a minimal inflammatory response but large numbers of Gram positive rods were colonising the denuded mucosal surface. *C difficile* was isolated from all 4 cases descrobed and its cytotoxin from 2 of these. *C perfringens* was also isolated in large numbers from 2 cases but none of its toxins were identified. All the

foals died within 24 hours of signs of illness and all were 3 days old or less.

Bacteriodes fragilis was cultured from the faeces of 27 out of 40 diarrhoeic foals in one survey in Kentucky **(Myers, Shoop and Byars 1987)**. Ten of these were positive for enterotoxin producing strains and dual infections with rotavirus and salmonella were found in 4 and one of these 10 respectively. Other infections detected included 5 cases with *Salmonella enteriditis* and 6 cases with rotavirus. All foals were less than 2 months old. No pathological details were given.

Corynbecterium equi (now known as *Rhodococcus equi*) is usually associated with respiratory tract infections but is known to cause ulcerative enterocolitis. **Johnson, Prescott and Markham (1983)** experimentally infected 10 foals and reported on the pathology found. Only 2 foals had developed gross lesions at the time of post-mortem examination. These consisted of severe ulcerative typhlocolitis with caecal and colonic lymphadenitis in both, in addition there was enteritis in one. Two further foals had microscopic lesions consistent with infection. The available literature on natural infections was reviewed. **Zink, Yager and Smart (1986)** presented the findings in 131 cases of *C equi* infection on the records at Ontario Veterinary College, all but 3 were foals. Of the 125 foals which were necropsied 46% had both pulmonary and intestinal involvement and 4% had involvement of the intestines only. Of the 60 foals with intestinal lesions 18% had small intestinal involvement, 30% had caecal involvement, 48% had colonic involvement and 72% had involvement of the mesenteric and/or colonic lymph nodes. In 5 foals the main finding was a single large abdominal

primary intestinal infection by *C equi* is possible as well as the more usual involvement secondary to pulmonary infection.

A number of viruses have been implicated either as the sole agent or in conjunction with another process in the production of gastrointestinal disease in foals (**Becht and Semrad 1986**). Rotavirus was isolated from 30% of faecal samples from cases of foal diarrhoea in Kentucky and was believed to be the cause of 4 major disease outbreaks (**Conner and Darlington 1980**). Histopathological examination of the duodenum and ileum revealed mononuclear inflammatory infiltration of shortened, oedematous, club-shaped villi. Ultrastructural examination revealed alteration or absence of microvilli. The Authors believed their results to be an under estimation of the importance of rotavirus. Eleven of the 86 samples examined were positive for *Salmonella sp* and 7 for coronavirus. The latter was not responsible for any major disease outbreaks. Coronavirus has also been isolated from immunodeficient foals with a concurrent cryptosporidium infection (**Mair, Taylor, Harbour and Pearson 1990**).

Adenovirus was found in faecal samples from 3% of diarrhoeic foals, 3 of which had bloody diarrhoea (**McChesney, England and Rich 1973**). Pathological details were minimal but intestinal epithelial cell swelling and viral inclusions were noted.

Ascariasis is common in foals and may produce varying clinical signs eg weight loss (up to 50% depression in weight gain), colic, fever, cough, nasal discharge and nervous disturbances (**Clayton 1978**). By far the most serious problem is obstruction of the small intestines by

Parascaris equorum which can lead to intestinal rupture. This usually occurs after a heavily infected foal is treated with an anthelmintic such as piperazine or an organophosphate which kills the worms quickly, forming a mass not just of dead worms bur also of the entrapped live ones (Becht and Semrad 1986). Di Pietro, Boero and Ely (1983) reported an abdominal abscess associated with *P equorum* infection in a foal. This abscess was centred on the cranial mesenteric artery and contained a single worm. The jejunum, ileum and caecum were adherent to the abscess. Masses of dead worms had obstructed the small and large intestines. The foal had been treated with trichlorfon 3 days earlier.

Cyathostomiasis can affect horses of any age with a previous grazing history (See Part III). Because previous exposure to contaminated pasture is required it is usually young adults that are affected (Love 1992). There is one report of cyathostome colitis and typhlitis in a 6 month old filly (Harmon, Ruoff and Huey 1986) reviewed in Part III, and it has been shown that experimental infection of foals can result in a protein losing enteropathy (Love, Escala, Duncan and McLean 1992). Migration of large strongyles, particulary *Strongylus vulgaris*, is known to cause abdominal pain in the foal (Becht and Semrad 1986). Duncan and Pirie (1975) carried out experimental infection of 9 foals with *S vulgaris* and described the pathology of the 3 stages of its life cycle in great detail. The literature on both experimental and natural infection was reviewed.

There are a number of reports of intestinal protozoal infection. Both *Eimeria leukarti* (Mason and King 1971) and *Cryptosporidia sp* (Gajadhar, Caron and Allen 1985) have been identified as the sole agents in

immunocompetent foals. Cryptosporidia have also been found in immunodeficient Arab foals on their own (Snyder, England and McChesney 1978, Gibson, Hill and Huber 1983) and in conjunction with coronavirus infection (Mair et al 1990b). See Part III.

The congenital disorders, which have been reviewed previously, often result in some sort of displacement eg intussusception (Sprinkle et al 1984). A single report of invagination of the caecal apex in the foal exists (Semrad and Moore 1983) which necessitated amputation of the displaced portion but no cause was established. There is another single report, this time of lymphocytic enteritis in a 7 month old filly (Clark, Deem, Allen and Tyler 1988) reviewed in Part III.

One condition of uncertain aetiology is gastroduodenal ulceration which occurs in both foals and adults (Rebhun, Dill and Power 1982). It is most commonly recorded following a period of stress but clustering of foal cases has implicated an as yet unidentified infectious agent (Acland, Gunson and Gillette 1983). Rebhun and colleagues (1982) described the endoscopic and/or necropsy findings in 3 Thoroughbred and 2 Standardbred foals, 2 of which were from the same farm. Two of the cases also had duodenal ulceration and stricture formation due to fibrous healing of previous ulcers. The pertinent literature was reviewed. Acland, and Colleagues (1983) described the macroscopic and microscopic findings in detail in a group of 9 foals with ulcerative duodenitis. Seven were deemed to have necrotising duodenitis with diffuse peritonitis and also had gastric ulceration. The remaining 2 were cases of granulating duodenitis, which was probably part of the same syndrome in foals that had survived a milder acute incident. Only

one of these foals had gastric ulceration, it also had scarring, fibrosis and stenosis of the duodenum. The Authors failed to identify any cases on their own records prior to 1981 and considered the gastric ulceration found to be less significant than the duodenal lesions. Three of the cases came from the same farm in the same year suggesting an infectious aetiology. A number of bacteria were isolated but were considered insignificant. Four of the foals had experienced a recent stressful incident and 2 of these and a further 4 had received nonsteroidal antiinflammatory therapy which may have been significant.

Becht and Byars (1986) combined the 2 problems and reviewed gastroduodenal ulceration. The Authors discussed the possible aetiopathogenesis based on both the equine and human literature and gave a brief outline of the clinical picture of the various forms and their management.

A recent review of gastroduodenal ulceration in foals (Sweeney 1991) reviewed the available literature and presented the clinical signs, pathology, diagnosis, aetiology, pathogenesis and treatment. Gastric ulcers are most commonly seen in the squamous portion of the stomach and are predominantly asymptomatic. However, those of the glandular portion appear to be of more significance and are rarely asymptomatic. Lesions of the duodenum account for 28% of all ulcers. The only proven cause of gastric ulceration is nonsteriodal antiinflammatory drugs. Investigation has failed to identify an infectious agent and weather stress has only recently come under consideration.

In an editorial in the same publication Baker (1991) discussed gastroduodenal ulceration in adult horses as well as foals. Some Authors consider the syndrome in foals to be a new condition perhaps associated with intensive rearing practices or the spread of some infectious agent but its emergence may just be a consequence of increased awareness and the increasing use of the flexible endoscope. Indeed some Authors initially considered the scarring and stenosis which results from healing of these ulcers to be a congenital lesion so many cases may have been misinterpreted in the past.

Another condition of undetermined aetiology, necrotising enterocolitis, has been described in 2 foals (**Cudd and Pauly 1987**). It closely resembled a condition previously described in babies. The first case, a Thoroughbred filly, had been born prematurely and presented with pulmonary atelectasis and respiratory distress syndrome. The second case, a Thoroughbred colt had been born at full term but foaling had been prolonged. Both developed distension of the abdomen terminally. In both cases a perforation of the ventral colon was identified at post mortem examination. In the second case the large bowel was bubbly and emphysematous. Histopathological examination of tissues from the second case confirmed the emphysema and revealed haemorrhage, oedema, inflammation and scattered microabscesses. In man, the condition is associated with an ischaemic insult to the bowel wall. Both cases had an episode in their history which would account for such an insult. Colonisation by gas forming bacteria is also required eg *Escherichia coli* and *Klebsiella sp.* However, one agent already known to cause necrotising enteritis in foals, namely *Clostridium perfringens* Type C,

is not involved in the pathogenisis of necrotising enterocolitis. The differences in the lesions were described to corroborate this statement.

One known cause of gastrointestinal pathology in the foal is phenylbutazone toxicosis and the foal, like the pony appears to be far more susceptible than the adult horse (**Traub et al 1983**). Experimental dosing of foals resulted in oral ulceration and ulceration of the squamous and glandular zones of the stomach in the majority and colonic ulceration in one. Villus atrophy and colitis were also noted The doses used were in excess of the manufacturer's recommendations.

INFECTIOUS AGENTS

Bacteria

Al-Mashat and Taylor (1986) described the bacteria isolated from multiple gastrointestinal samples, lymph node and liver at post mortem examination of 23 adult horses and 2 foals. Gross gastrointestinal lesions were identified and described in 19 of the animals, the remainder having lesions in other systems. The histopathology was described and illustrated where appropriate. Thirty three species of bacteria were isolated and could be related to the gross or microscopic lesions in some cases. *Clostridium perfringens* Type A, *Actinobacillus equuli*, *Salmonella typhimurium* and *Campylobacter coli* biotype I could all be associated with gastrointenstinal lesions. The Authors believed the case of *C coli* enteritis to be the first such report. *C jejuni* biotype I and *Aeromonas hydrophila* were both recovered and have been

identified as the cause of enteritis in a number of species including horses.

A number of reviews of salmonellosis in the horse exist (**Gibbons 1980, Smith 1981a, Smith 1981b**). According to **Smith (1981b)** there are over 1800 salmonella serotypes but *Salmonella typhimurium* accounts for over 60% of all equine cases. As well as the diseased state both silent and active carriers exist, either of which may break down and become ill following a period of stress. This is the most common time to see clinical disease. Animals which die or are destroyed in the acute phase exhibit a severe fibrinonecrotic typhlitis and colitis. As well as the entire caecum and proximal large colon the distal ileum is usually involved. The gut wall is oedematous with both mucosal and serosal inflammation, possibly with mucosal ulceration and sloughing. The mesenteric lymph nodes are enlarged and engorged. Petechiae are often present on a number of organs. Occasionally there are small abscesses in the large intestinal wall which may lead to adhesion formation and chronic colic at a later date.

Salmonella sp are occasionally identified in conjunction with other infectious agents such as the dimorphic fungus *Histoplasma capsulatum* (**Goetz and Coffman 1984**). **Slocombe and Slauson (1988)** reviewed 19 cases of pulmonary aspergillosis, 16 of which were associated with enterocolitis suggesting an important role for the gastrointestinal tract in this condition along with immunosupression and antibiotic therapy. *Salmonella sp* were incriminated in the intestinal lesions of 14 and *Klebsiella sp* and *Actinobacillus equuli* in one each. *Escherichia*

coli was also isolated from 8 cases but no comment was made on the pathogenic potential of the strains involved.

Gay and Lording (1980) described 3 acute cases and 2 chronic cases of peritonitis due to *A equuli* but only presented minimal pathological information from the one animal necropsied. It had a fibrinous and fibrous peritonitis and pleurisy with extensive verminous lesions in the mesenteric and caecal-colic arteries. The Authors considered the arteries to be the possible point of entry for the bacteria in all cases.

Another bacteriologically induced condition, often with intestinal involvement is tuberculosis. The tubercle bacilli induce chronic granulomatous changes in the intestines and lymph nodes (**Innes 1949, Luke 1958, Baker 1973, Buergelt et al 1987, Cline et al 1991**). The condition is reviewed in Part III.

One condition which appears to be multifactorial but with a bacterial component is colitis X (**Umemara et al 1982**). **Ochoa and Kern (1980)** induced changes similar to those of colitis X by intravenous administration of *Clostridium perfringens* Type A enterotoxin to 3 Shetland ponies. All showed signs of colic within 3 hours 20 minutes. The entire digestive glandular mucosa from the margo plicatus to the small colon was red. There was haemorrhage in the caecum and colon. The intestinal content was fluid with much mucus and there was shedding of villus tips. The mucosa was congested and oedematous except in pony 3 which died after only 22 minutes. Gastritis and enteritis are usually not seen in colitis X. **Umemara and colleagues (1982)** considered that

there was little information available on the pathology of colitis X and attempted to rectify this by recording the findings in 16 cases. There was dark, tarry, unclotted blood in the subcutaneous tissues. Petechiae or larger haemorrhages were present in the large intestinal mucosa which was partially covered by pseudomembranes. The lymphoid follicles were increased in both size and number. The large intestinal content was brown to red, watery and foul smelling. Some mucosal haemorrhages were also present in the stomach and small intestines and all associated lymph nodes were enlarged. Microscopic examination revealed congestion throughout, especially in the large intestines which were also oedematous. The changes varied from mild epithelial loss, through marked haemorrhage, fibrin exudation and inflammation to mucosal necrosis and collapse with multiple thrombi. In the small intestines there was mild exfoliation of epithelial cells in only 2 cases. The Authors considered both the clinical and pathological findings to be consistent with those previously reported. The precipitating events were noted but no infectious agent was proposed.

Another group (**Prescott et al 1988**) reproduced colitis X and isolated a clostridium, which resembled *C cadaveris* as a possible agent. The Authors used lincosamide antibiotics on their own or in combination with intubation with stored colonic content from clinical cases of colitis X. The pathology consisted of a superficial typhlitis and colitis and was similar to that previously described (**Umemara et al 1982**). However, the Authors considered that the lesions could not be distinguished from those of peracute salmonellosis or Potomac horse fever (equine monocytic ehrlichiosis).

Potomac horse fever is an infectious but noncontagious condition of horses in the region of the Potomac River since 1970 (**Cordes, Perry, Rikihisa and Chickering 1986**). **Cordes and colleagues (1986)** reproduced the disease by whole blood transfusion from clinical cases. Lesions were consistently present in the small intestines, caecum and large colon and were also observed in the stomach and small colon. In the small intestines there was hyperaemia, echymoses and ulceration and the content was fluid or mucoid. The large intestinal content was also fluid but had a more foetid odour and the mucosa of the caecum and large colon resembled that of the small intestines. Lesions were only noted in the small colon of 2 animals and consisted of hyperaemia. In the ponies without diarrhoea the content of the rectum was of normal consistency. Microscopic lesions were not marked in the caecum and large colon and consisted of epithelial loss, fibrin exudation and focal necrosis. Some inflammatory cell debris was present within the mucosa and there was a mixed inflammatory cell population in the mucosa and submucosa. Electron microscopy revealed large numbers of ehrlichial organisms in the cytoplasm of deep glandular epithelial cells, mast cells and macrophages, especially in macrophages next to blood vessels. The Authors considered the gastric lesions to be non-specific. The organisms responsible appeared to be closely related to *Ehrlichia sennetsu* but distinct from *E equi*, the agent of equine ehrlichiosis.

Mulville (1991) in a more recent review of equine monocytic ehrlichiosis cited *E risticii* as the causative agent. No vector or reservoir host has as yet been identified. The Author described the clinical signs, clinical pathology, necropsy findings treatment and prevention. The organism is susceptible to a number of antimicrobials but care must be

exercised as tetracycline may itself induce a similar fatal colitis (see section on Toxicoses).

Hattel, Drake, Anderholm and McAllister (1991) described a case of pulmonary aspergillosis associated with acute enteritis which on the basis of serological studies they believed to be caused by *E risticci*. The report deals primarily with the pulmonary lesion and histopathological examination of the diffusely reddened distal small intestines and large colon was not performed.

Fungi

A number of fungi and fungus like organisms have been shown to cause both localised (**Brown and Roberts 1988**) and diffuse (**Dade, Lickfeldt and McAllister 1973**) gastrointestinal lesions. The dimorphic fungus *Histoplasma capsulatum* causes a granulomatous colitis (**Dade et al 1973**) and is reviewed with other chronic inflammatory bowel diseases in Part III.

Pythiosis, caused by a fungus like organism, usually occurs as a cutaneous lesion of horses in warm wet areas but has been identified in intestinal lesions. **Brown and Roberts (1988)** described the findings in a 9 month old Thoroughbred colt with a history of nasal discharge, coughing and ill thrift prior to a single episode of colic due to intestinal obstruction. There was localised peritonitis and omental adhesion to a 20cm diameter nodular mass at the jejunal-ileal junction. On section the mass was seen to be composed of coalescing fibrous nodules and fistulous tracts in communication with the intestinal lumen

which was stenotic but patent. Histological examination revealed necrosis, inflammation and hyphae, the latter usually within mineralised foci. Pythiosis was suspected from the morphological appearance of the hyphae. Immunocytochemical staining of sections confirmed the diagnosis. A second case of enteric pythiosis which also resulted in intestinal obstruction, this time in a 2 year old Quarter Horse, was reported by **Allison and Gillis (1990)**. The horse had shown signs of intermittent colic for over a year. A thick stenotic area was present in the jejunum within which the lumenal content had become impacted. At this point the mesentery was thick and nodular. Histopathologically the mucosa was distorted, there was much inflammatory granulation tissue which contained numerous coalescing granulomata and many eosinophils. Hyphae morphologically consistent with those of *Pythium sp* were identified. Culture of samples of the resected portion proved negative but immunocytochemistry of unstained sections confirmed the presence of pythium-positive hyphae.

Boulton and Williamson (1984) described the acute sequella of a chronic problem. A jejunal intussusception developed in an American Saddlebred mare secondary to a cryptococcal granuloma. The granuloma was a 6 x 4cm, intralumenal, polypoid mass on a narrow stalk and typical cryptococcal organisms were identified in haematoxylin and eosin stained sections.

PARASITISM

Case reports and surveys exist on all forms of equine enteric parasitism; nematodes, cestodes, protozoa and coccidia. Two recent

review papers, one on parasite associated diarrhoea (Love 1992) and one on tapeworms (French and Chapman 1992) include most of the important equine parasites and review the available literature but little information on the resultant pathology is given. A number of editorials also give a broad overview of the general problem, but usually confine their comments to the clinical problems and anthelmintic regimes (Duncan 1985a, Duncan 1985b).

Another type of article is the prevalence study; one article of this type used post-mortem material (Owen, Jagger and Quan-Taylor 1988) and the other faecal flotation (Imrie and Jacobs 1987). The above studies concerned the prevalence of tapeworms in England and Wales. Another study recorded the prevalence and intensity of non-strongyle intestinal parasites of horses in nothern Queensland using post-mortem material (Mfitilodze and Hutchinson 1989). This study revealed the presence of a great many gastric and intestinal parasites but information on associated lesions was confined to a description of the crater-like lesions caused by *Gasterophilus sp.* larvae.

The largest study was carried out by a group in Kentucky who looked at parasites in Kentucky Thoroughbreds at necropsy. In the first paper the emphasis was on stomach worms and tapeworms (Lyons et al 1983). The Authors confined their observations to the stomach, small intestines and caecum. The only lesions described were those of *Drashia megastoma*. A second paper described the prevalence of *Anaplocephala perfoliata* and lesions of *D megastoma* (Lyons et al 1984). Examination was confined to the stomach and caecum. In addition to the lesions associated with *D megastoma* one horse had muscular hypertrophy of the ileum associated

with *A perfoliata*. The third paper looked at the common internal parasites found in the stomach, large intestines and cranial mesenteric artery (**Lyons et al 1987**). Special attention was paid to bots and nematodes in the stomach, tapeworms in the caecum, large strongyles in the cranial mesenteric artery and pinworms. The cranial mesenteric artery was only examined in foals and yearlings. No associated lesions were described.

A number of surveys were undertaken because of a proposed connection between tapeworms and intussusception. The Authors wished to establish the rate of infection in the normal population as opposed to those with an intussusception (**Imrie and Smith 1987, Owen et al 1988**). A number of case reports exist proposing this connection (**Barklay, Phillips and Foerner 1982, Cosgrove, Sheeran and Sainty 1986, Owen, Jagger and Quan-Taylor 1989**).

Other intestinal "accidents" attributed to tapeworms include duodenal rupture due to *Anoplocephala magna* (**Oliver, Jenkins and Walding 1977**). This was described in a 9 month old colt in which an 8cm rupture was present in an extremely inflamed portion of the duodenum. Opening the bowel revealed severe catarrhal inflammation for a length of 45cm containing a mass of tapeworms. There was no other clinical evidence of disease. Caecal perforation in association with *A perfoliata* infection has also been described in 3 horses, one of which also had ileocaecal intussusception (**Beroza et al 1983**).

One group of parasites, the cyathostomes, has only recently emerged as a cause of disease in the horse although their existence has been known

for some considerable time (Love 1992). The resulting colitis is described in Part III (Blackwell 1973, Chiejina and Mason 1977, Jasko and Roth 1984, Giles, Urquhart and Longstaffe 1985, Harmon et al 1986) along with their association with tissue invading cilliates (Gregory, Longstaffe and Giles 1986). Part III also includes the detailed discussion of the literature on cryptosporidia (Gibson et al 1983, Gajadhar et al 1985, Mair et al 1990b) and coccidiosis (Mason and King 1971, Wheeldon and Greig 1977).

Ascariasis (Clayton 1978, Clayton, Duncan and Dargie 1980, Di Pietro et al 1983) and the pathogenesis of *Strongylus vulgaris* infection (Duncan and Pirie 1975) have both been reviewed in the section on foal conditions.

The main effect *S vulgaris* larvae exert on the adult horse is the damage the larvae cause to the cranial mesenteric artery. Rous (1975) believed the main outcome to be the formation of an aneurysm. The Author states that because of the close association of the celiacomesenteric ganglion to the vessels this aneurysm results in alteration in gut motility as well as affecting blood supply to portions of the gut. White (1981) described 18 cases where there was thrombosis of the cranial mesenteric artery due to the presence of larvae and secondary thromboembolic infarction of portions of the gut. The small intestines were involved in 10 horses, the colon in 6 and the caecum in 4. The Author makes no mention of neurological involvement. Greatorex (1975) described diarrhoea associated with ulceration of the caecum and colon resulting from larval migration. This paper is reviewed more fully in Part III, The Enteropathy Series.

TOXICOSES

There are few reports in the horse of environmental poisons which result in damage to the gastrointestinal tract. Acorn poisoning has been recorded although it is a much less common occurrence than it is in the bovine (**Anderson, Mount, Vrins and Ziemer 1983**). The pathology in the single case examined was described in detail and consisted of mesenteric oedema, ulcerative enterocolitis and nephrosis. The results of biochemical and haematological analysis were also given.

Mercury poisoning and the possible sources as previously described in the literature were described by **Schuh, Ross and Meschter (1988)**. The Authors also presented the findings in 2 cases where mercuric blister had been applied in combination with dimethyl sulphoxide (DMSO). The alimentary tract lesions found at necropsy of the more fully examined case consisted or oral ulceration, colonic and caecal segmental infarction, ulceration and fluid distention. Subacute toxic renal tubulonephrosis was also noted. Other possible toxic causes of gastroenteritis and renal failure were listed and the role of the DMSO explained.

There have been a number of reports of adverse drug reactions in the horse. These were reviewed briefly by **Davis (1987)**. The most frequently reported agent is the nonsteroidal antinflammatory drug phenylbutazone (PBZ) (**Snow, Bogan, Douglas and Thompson 1979, Snow et al 1981, MacKay, French, Nguyen and Mayhew 1983, Collins and Tyler 1984, Meschter, Maylin and Krook 1984, Collins and Tyler 1985, Karcher Dill,**

Anderson and King 1990, Meschter at al 1990). These reports are reviewed in detail in Part III.

Adverse reactions to antimicrobial agents in the horse were reviewed by English and Roberts (1983). Baker and Leyland (1973) gave brief descriptions of the post mortem findings in 3 animals which died following the use of oxytetracycline on its own or in combination with chlortetracycline. In one animal only large intestinal oedema and watery intestinal content were noted. In the other 2 there was mild injection of the gastric mucosa, mild catarrhal enteritis and severe typhlitis. The content was watery throughout. In the large intestines there was marked submucosal infiltration by lymphocytes and plasma cells. Mild hepatitis was also noted. Cook (1973) described 3 cases of diarrhoea, this time associated with a combination of oxytetracycline therapy and stress. The one animal which survived had undergone anaesthesia but not surgery but the other 2 had both undergone anaesthesia and prolonged surgery. At necropsy there was congestion of the large and small colons and cloudy swelling in the liver and kidneys. No such problems were encountered with the use of penicillin or a combination of penicillin and streptomycin instead of oxytetracycline.

Baker (1975) reviewed the available literature on diarrhoea in horses associated with tetracyline therapy and concluded like Cook (1973), that even if normal therapeutic doses were given in conjunction with some sort of stress, diarrhoea and death could follow.

Raisbeck, Holt and Osweiler (1981) described the post mortem findings in a Morgan stallion which had died following the consumption of lincomycin

contaminated grain. The entire colon and caecum were black and contained 20-30l of serosanguinous fluid. The submucosa was 5mm thick and haemorrhagic with areas of necrosis from 4-20cm in diameter. Histologically there was sloughing of the mucosa and collapse of the lamina propria. One other horse developed laminitis and diarrhoea but eventually recovered. The similarity to lincomycin induced pseudomembranous colitis as described in man and its possible mechanisms were discussed.

Whitlock (1990) reviewed the feed additives and contaminants which cause equine disease. These include monensin, salinomycin, lasalocid and other ionophorus antibiotics which predominantly cause neurological and cardiovascular signs but will induce colic. Cantharidin or blister beetle poisoning principally induces signs of colic and results in watery diarrhoea. Botulism is also discussed. No information on the pathological lesions caused by any of the agents was given.

Atropine is a parasympatholytic drug used for its spasmolytic properties in cases of equine colic. A paper by **Ducharme and Fubini (1983)** suggests that this action may proceed to gut stasis and gaseous distention. The Authors described the clinical findings in 5 ponies dosed with atropine to determine its effect on gut motility and 3 clinical cases where atropine had been administered. All developed a degree of gut stasis and gaseous distention which lasted up to 12 hours. One of the 3 clinical cases died following torsion and infarction of the large colon. It was likely that the other 2 clinical cases would also have died had decompression of the distended portions of bowel not been performed.

NEOPLASIA

There are a number of well recognised although uncommon neoplasms of the equine gastrointestinal tract. **Head (1976)** reviewed tumours of the lower alimentary tract of domestic animals including those of the horse but the paper is very general describing the histological appearance rather than site and frequency in most cases. Review articles on equine neoplasia include tumours of the stomach and intestines along with those of other systems **(Cotchin 1960, Misdorp 1967, Priester and Mantel 1971, Cotchin 1977, Reid and Howie 1992)**.

Squamous cell carcinoma is the most common tumour of the equine stomach. **Tennant and colleagues (1982)** described the findings in 6 cases. All animals had a history of anorexia and weight loss. In each case the tumour originated from the nonglandular, squamous portion of the stomach, was cauliflower like in appearance, ranged from 8-22cm in diameter and weighed approximately 4kg. There was extension and adhesion to adjacent structures such as the liver, spleen and diaphragm with metastasis to the liver in all 6 cases. Histopathological examination of the mass revealed cords or clusters of epithelial cells separated by connective tissue. In some areas whorls of cells had keratin pearls at the centre. Other areas were gland-like in appearance. Details of 15 cases from the literature were also included. The Authors believed the incidence of gastric squamous cell carcinoma in the horse to be on the increase. The most recent paper on gastric squamous cell carcinoma came from Denmark **(Olsen 1992)**. It dealt predominantly with the clinical findings, weightloss again being a common feature, giving only minimal pathological information. The

tumour weighed from a few grams to 15kg. Metastasis had occurred in all 5 cases and tumour cells were identified in the peritoneal fluid in 3 cases.

The most common form of intestinal neoplasia is alimentary lymphosarcoma. (**Conboy and Powers 1971, Neufield 1973a, Wiseman, Petrie and Murray 1974, Roberts and Pinsent 1975, McConnell, Katada, Fiske and Martens 1982, Humphrey, Watson, Edwards and Wood 1984, Rebhun and Bertone 1984, Reef, Dyson and Beech 1984, Crawely 1985, Wilson, Sutton, Groenendyk and Saewright 1985, Platt 1987**). It is reviewed in detail in Part III.

The remaining tumours are recorded as case reports of one or 2 animals. The majority of these reports concern smooth muscle tumours, leiomyomas and leiomyosarcomas. **Hanes and Robertson (1983)** described the findings in an 11 year old Morgan gelding in which a leiomyoma had obstructed the jejunum and led to gastric rupture. The presence of a leiomyoma may also lead to intussusception of the affected segment. **Collier and Trent (1983)** described such a case where almost the entire jejunum had intussuscepted, including the portion containing a 4cm diameter firm mass which proved to be a leiomyoma. It was composed of a smooth muscle wall and necrotic haemorrhagic intestinal glands which faced the antilumenal surface. There was a well vascularised eosinophilic core with numerous round or oval hyperchromatic nuclei. **Mair, Davies and Lucke (1992)** reported a case where the leiomyoma was present within a small colon intussusception. It consisted of an 8cm diameter intralumenal pedunculated mass with a lobulated dark surface. Histological examination of the mass revealed granulation tissue with

islands of cells similar to those described previously (Collier and Trent 1983).

Ramey and Reinerston (1984) reported the findings in a 15 year old Arabian mare where the primary problem was strangulation of a portion of jejunum. A leiomyoma measuring 15 x 10cm was also identified and removed. It was not involved in causing the jejunal strangulation and appeared to be an incidental finding.

Livesey, Hulland and Yovich (1986) reported the findings in 2 cases, one had a leiomyoma and one a leiomyosarcoma. The leiomyoma was present as an 8cm diameter pedunculated mass on the antimesenteric surface of the proximal jejunum and did not appear to be obstructing passage of ingesta although its removal prevented the recurrence of previously recorded frequent bouts of colic. Histopathological examination of the mass revealed interlacing bundles of smooth muscle fibres at right angles to each other. The nuclei were centrally located. The leiomyosarcoma was associated with a mid jejunal volvulus. The mass measured 7.5 x 6cm and was attached to the antimesenteric border of the bowel. Metastasis had not occurred and resection of the involved segment of bowel proved curative. Histopathological examination of the mass revealed interlacing bundles of smooth muscle fibres at right angles to each other. The nuclei were centrally located.

Clem, DeBowes and Leipold(1987) described a rectal leiomyosarcoma in a 4 year old Quarter Horse. It consisted of a 5cm diameter pedunculated mass with a partially ulcerated surface. The base of the mass arose from the dorsal rectal wall, 6cm proximal to the anal sphincter, but did

not appear to be obstructing the passage of faeces. It was removed, apparently incompletely, under general anaesthesia but regrowth had not occurred 18 months later. **Mair, Taylor and Brown (1990)** described 2 cases in which a leiomyosarcoma had caused obstruction of the duodenum by producing an anular thickening of the wall. In one case the tumour had reduced the diameter of a 3cm length of distal jejunum to 4mm. The intestine proximal to the lesion was dilated. Histopathological examination of the segment revealed that the muscle layers had been replaced by hyaline fibrous tissue and densely packed pleomorphic spindle cells with a high mitotic rate. The cells formed whorling bundles weakly positive for desmin. In the second case there was localised thickening of a 5cm portion of mid duodenum which had led to complete obstruction of the intestinal lumen and ultimately the animal's death. Microscopic examination of the thickened portion revealed sheets and bundles of rounded or elongated oval and spindle shaped cells which stained as muscle with Van Gieson stain.

Adenocarcinomas are an unusual occurrence but they have been reported in the proximal ventral colon (**Wright and Edwards 1984**) and mid jejunum (**Honnas, Snyder, Olander and Wheat 1987**). **Wright and Edwards (1984)** described the findings in a 13 year old Thoroughbred cross mare. A spherical mass 10cm in diameter was present at the base of the caecum and proximal ventral colon. The lumenal surface was haemorrhagic and necrotic. It protruded into the lumen and almost totally occluded the caecocolic orifice. This had led to rupture of the caecum at its base. Although the serosal tear was fresh the mucosal lesion appeared older. The mass was composed of closely packed cords of columnar epithelial cells forming irregular glands which invaded the submucosa and muscular

layers. "Signet-ring" cells, a few mitotic figures and mucin accumulation were seen. Honnas and colleagues (1987) described a 6x6x8cm mid jejunal mass in a 21 year old Arabian cross mare. The small intestines proximal to the lesion were fluid filled and 2-3 times normal size but ingesta could be forced through the mass. The mass, which was both intramural and intralumenal, centred on a large mucosal ulcer which extended through the tunica submucosa to the lamina muscularis mucosa. No metastases were apparent and the animal was still alive 13 months after surgery. Microscopic examination revealed irregularly spaced neoplastic glands which were complex tubular structures lined by low columnar to low cuboidal epithelial cells with little regular polarity and a moderate mitotic rate.

Pascoe (1982) described the findings in a 16 year old Thoroughbred mare with a colonic neurofibroma and reviewed the literature on equine intestinal neoplasms. The tumour was present in the mesenteric side of the small colon, 10cm distal to the transverse colon. It had reduced the lumen to a slit causing gaseous distention of the caecum and large colon. The overlying mucosa was partially ulcerated and when it was incised the tumour mass shelled out easily. Examination of the mass revealed spindle shaped cells in a loose whorling pattern or palisading. No mitoses were seen. The animal was in good condition 12 months post operatively.

Allen, Swayne and Belknap (1989) described a case of small intestinal obstruction due to a ganglioneuroma. The mass was intramural, 4x4x6cm, involved 75% of the circumference of the jejunum and had become strangulated by a loop of adjacent bowel. The mass had reduced the

intestinal lumen and was composed of intertwining fascicles of neurofibrils, many ensheathed by spindle cells, and bundles of poorly differentiated cells. The fascicles were encircled by thin perineural sheaths and separated by collagen fibres. Ganglion cells, which varied in size, shape and nuclei number, were dispersed throughout the tissue.

Orsini and colleagues (1988) described an intestinal carcinoid in an 18 year old Thoroughbred mare. The duodenum and proximal jejunum had passed through a tear in the mesentry of the proximal jejunum resulting in a volvulus of the intestine. The carcinoid was a 6cm diameter, firm mass in the wall of the proximal jejunum and its associated mesentry. Histopathology of the mass revealed nests and acini lined by a single layer of tumour cells with round to vesicular nuclei. The mitotic rate was low, and there was much fibrosis. In man these tumours predispose the involved portion of the gut to volvulus.

Edens and colleagues (1992) reported an intestinal myxosarcoma present as an intralumenal mass, at the base of the caecum, which protruded into the ventral colon of a 12 year old Thoroughbred mare. Post-mortem examination revealed that the mass measured 26x16x10cm and arose from a pedicle 6cm in diameter. It was firm and red-brown with a nodular surface. It partially occluded the caecocolic orifice and proximal right ventral colon. Sectioning revealed a thick fibrous capsule around yellow, gelatinous material interspersed with firm brown tissue. Histopathological examination of the mass revealed widely spaced, fusiform to elongate cells with round to ovoid nuclei. The surrounding matrix consisted of a fibrillary eosinophilic material with scattered round to confluent clear spaces. The stromal component stained

positively for mucopolysaccarides. Cells and stromal components were present within adjacent lymph nodes. The Authors cited the only previous report of equine intestinal myosarcoma which described 2 cases (**Feldman 1932**).

Serosal lipomas are common in horses as an incidental finding and are frequently mentioned in review articles (**Cotchin 1977, Cotchin and Baker-Smith 1975**). They do however, occasionally, cause problems because of their pedunculated nature. The stalk may reach some considerable length and loop round and strangulate a portion of the bowel (**Pascoe and Summers 1981**). **Mason (1978)** described strangulation of the rectum of a 16 year old Thoroughbred mare and **Ramey and Reinerston (1984)** described strangulation of the jejunum in a 16 year old Arabian mare. Ligation of the stalk and removal of the lipoma were curative in the former but in the latter the animal subsequently died as a result of gastric rupture despite removal of the compromised portion of bowel as well as the lipoma. Histologically lipomas resemble adipose tissue but there is a greater variation in cell shape and size. The stalk may twist around resulting in necrosis (**Ramey and Reinerston 1984**).

A number of tumours although not arising from the gastrointestinal tract have been reported to compromise its function. **Shokry and Lotfi (1984)** described a malignant perianal melanoma in a 6 year old grey horse which had grown rapidly and was obstructing the anus. It measured 12 x 12 inches and was fluctuant containing viscid, tarry fluid. Biopsy of a regional lymph node revealed a highly invasive malignant melanoma. **Parks, Wyn-Jones, Cox and Newsholme (1986)** described the unusual finding

of an abdominal testicular teratoma in a 4 day old Shire colt which was causing partial obstruction of the small colon. The right testicle was a 225gm, 7.5cm diameter cystic mass and its spermatic cord had entraped the small colon allowing the passage of some gas and mineral oil but no faeces. Rupture of the small colon was reported by **Wilson and colleagues (1989)**. The small colon was occluded by the taut left uterine horn and suspensory ligament of the left ovary which contained a 25 x 30cm cystic mass. This had resulted in impaction of the cranial portion of the small colon. There was a 7cm perforation in the small colon immediately proximal to the constriction about 60cm cranial to the anal sphincter.

DISPLACEMENTS

Portions of the intestines may be displaced from their normal position and orientation with other organs and either remain within the abdomen (**Rooney 1965**) or protrude beyond the body wall (**Van der Velden and Rutgers 1990**). **Rooney (1965)** recorded 24 cases of volvulus, 15 cases of strangulation incarceration and 5 cases of jejunal intussusception and hypothesised about the possible role played by *Strongylus vulgaris* especially in the cases of volvulus. Individual case reports and case series have been published describing each of these lesions in detail. Two cases of small intestinal intussusception associated with a leiomyoma (**Collier and Trent 1983, Mair et al 1992**) and another associated with a cryptococcal granuloma (**Boulton and Williamson 1984**) have been reviewed in the sections on neoplasia and bacterial agents respectively.

Two cases of chronic intestinal intussusception were described by **Scott and Todhunter (1985)**. The first case was a 7 year old Quarter Horse mare with a history of repeated episodes of colic for more than a year. A jejunal intussusception was identified at laparotomy. The intussuscipiens was firmly adhesed to the intussusceptum, the proximal intestine was greatly distended and the distal portion was reduced in diameter. A patent lumen 3cm in diameter was palpated and the involved segment was resected. The second case, a 2 year old Standardbred filly had a 3 month history of intermittent colic. An ileal intussusception was identified at celiotomy which was only partially reducable and had markedly narrowed the lumen. The affected portion was resected.

In contrast **Van der Velden (1989)** described finding a fully reducable intussusception on routine exploration of the abdomen after correction of a mesenteric hernia. The tissue of the intussusception was considered viable but compromised so the involved portion of distal jejunum was bipassed but not removed.

Greet (1992) described the findings in 16 cases of ileal intussusception, all young Thoroughbreds, 5 being less than 12 months old. The available literature was reviewed and the clinical findings presented. Various procedures were employed, the majority (11 cases) required an ileo-caecal bypass proximal to the lesion after its reduction. Twelve horses eventually recovered completely. Of the remaining 4 the intestinal wall was irreversibly damaged in one and ruptured in another. One foal had a cardiac arrest and the remaining animal was destroyed after 4 months because of adhesion formation. Ten cases of chronic iliocaecal intussusception were described by **Hackett**

and Hackett (1989). The possible causes of chronic colic were reviewed and the signalment, treatment and outcome of each case presented. Only 3 animals were over 12 months of age. Portions of ileum from 2-10cm in length had intussuscepted into the caecum. The jejunum and proximal ileum were dilated and thickened. No cause for the intussusception was found in any of the cases although a number of possibilities were discussed. One animal died during surgery and one died one year post operativly because of adhesion induced jejunal strangulation, the rest were normal or lost to follow up at time of publication.

In a larger study, **Ford and colleagues (1990)** looked at the history, signalment and clinical, surgical and pathological findings in 26 horses with ileocaecal intussusception. Three animals were destroyed but the remaining 23 underwent surgery and the intussusception varied from 6cm to 457cm in length in those with an acute presentation and from 2cm-10cm in those with a history of pain for more than 3 days. Five animals were euthanased because of viscus rupture or abdominal contamination or at the owners request. The Authors considered that this is predominantly a condition of young animals with 81% being less than 4 years old. In most cases no predisposing cause is identified although tapeworms were noted at the ileocaecal orifice of one of the 26 cases.

A number of authors consider tapeworms to be an important predisposing cause of ileocaecal intussusception. **Cosgrove and colleagues (1986)** described one such case, a 2 years old Thoroughbred filly with an exceptionally heavy *Anoplocephala perfoliata* burden. At necropsy there was extensive fibrinous peritonitis and omental adhesion to the small intestines. Large numbers of tapeworms were present around the

ileocaecal valve and in the base of the caecum. The Authors proposed that the intussusception was a sequel to the obstruction caused by inflammatory changes in the mucous membrane around the valve. **Barclay and colleagues (1982)** described a further 5 cases, all 3 years old or younger. The intussusceptions were ileo-caecal in 2 cases the rest being ileal-ileal,caecal-caecal and caecal-colic. The relevant literature was reviewed by **Owen and colleagues (1989)** in an attempt to establish the significance of the *A perfoliata* found in 2 cases of caecal intussusception presented within a one month period. One was a 4 year old Welsh Mountain Pony with a caecal-caecal lesion and the other a 16 year old Hunter mare with a caecal-colic lesion. The Authors concluded that there was little evidence that tapeworms were a major cause of intussusception in the horse.

Two cases of caecal intussusception were described by **Milne, Pogson, Else and Rowland (1989)**. In one case the base had invaginated into the body and in the other almost the entire caecum had invaginated into the right ventral colon. In both cases the invaginated portions were necrotic and microscopic examination revealed that the lesion had been present for some time. One paper describing invagination of the caecal apex in a foal has been reviewed in the section on foal disorders **(Semrad and Moore 1983)**.

Robertson and Tate (1982) described a case of large colon intussusception in a 9 years old Thoroughbred gelding but dealt predominantly with the surgical method used for its resection. A large portion of the pelvic flecture had invaginated into the left dorsal colon. Intussusception of the left ventral colon proximal to the pelvic

flecture in a 1 year old Standardbred filly was described briefly by **Dyson and Orsini (1983)**. Another brief description, this time of intussusception of the left dorsal colon in a 1 year old Appaloosa filly was given by **Wilson, Wilson and Reinertson (1983)**.

Bailey and Hutchins (1987) described a case of type 4 rectal prolapse (intussusception of the peritoneal rectum or small colon through the rectum) managed with a diverting colostomy. The prolapse had developed postpartum and resection and anastomosis were performed. These failed, as did the subsequent colostomy. At necropsy a loop of small intestine was adherent to the anastomosis site resulting in obstruction and gastric dilitation. There was also prolapse of the mucosa of the small colon at the colostomy site. A second case of small colon intussusception, this time in a prepartum mare, was described by **Ross, Stephens and Reimer (1988)**. In this case the leading edge of the intussuscepiens had been visible at the anus 8 days previously. At surgery the intussusception was found to be 75cm in length, packed with dry food material and covered with fibrin. Resection and anastomosis were performed and the animal was well 1 year later.

Turner and Fessler (1980) discussed the 4 types of rectal prolapse and corrective surgical techniques and presented the findings from 11 cases. Treatment was successful in 10 of the cases and a possible cause was identified in 7 of the cases.

Another form of intestinal displacement where the intestine remains within the abdomen, is incarceration through a normal or acquired aperture. **Becht and McIlwraith (1980)** described displacement of the

jejunum through the mesometrium in a pregnant mare. Abdominal exploration revealed incarceration of 5m of apparently viable intestine which were returned to their normal position. Recovery was uneventful.

The small intestines can also become incarcerated through the epiploic foramen. **Turner, Adams and White (1984)** described 15 such cases and believed it to be an uncommon cause of colic. It occurs in 2 forms; 1) incarceration of the intestine from the right peritoneal cavity to the left entering the omental bursa, and 2) left to right where the omentum is ruptured or pushed through the epiploic foramen. In all 15 cases the mid or distal jejunum or ileum was involved and had passed right to left in 12 cases and left to right in 3. **Vasey (1988)** described the findings in a further 15 cases. The paper dealt predominantly with the clinical findings but it was noted that the incarceration occurred from right to left and involved from 26cm to 13m of intestine. It appears to be predominantly a condition of older animals possibly because of a degree of age related liver atrophy.

Incarceration of the small intestine through rents in the gastrosplenic ligament has also been described **(Yovich, Stashak and Bertone 1985)**. The Authors believed it to be an uncommon cause of abdominal crisis in the horse and presented the findings from 5 cases. In each case from 1-6m of distal jejunum and/or ileum had passed cranially through a rent measuring from 3-12cm in diameter.

One possible route of intestinal herniation is through a defect in the diaphragm into the thorax. **Wimberly, Andrews and Haschek (1977)** reviewed the available literature and described 6 additional cases.

Both congenital and acquired hernias occur in the horse although there is not always a history of trauma to account for the latter. The majority of lesions occur in the tendinous portion where the *centrum tendineum* blends into the *pars costalis*, a logical point of weakness. **Proudman and Edwards (1992)** described the diagnosis and surgical repair of a diaphragmatic diverticulum, not in communication with the pleural cavity, which had caused non-strangulating obstruction of the large colon in a 7 year old Thoroughbred gelding. At laparotomy the pelvic flecture was located in the anterior abdomen and a diaphragmatic defect approximately 25cm in diameter was present in the ventral mid line. The intrathoracic sac lay adjacent to, but did not communicate with the pericardial sac and had not collapsed. The defect was almost completely closed and the animal made an uneventful recovery.

The large intestines can also be displaced within the abdomen which results in their function being compromised. Left dorsal displacement of the colon was described in 4 Quarter Horses and 2 Arabians by **Milne and colleagues (1977)**. The Authors believed it to be a previously undescribed condition. At surgery the caecum was distended with gas as was the stomach which was displaced caudally and ventromedially accompanied by the ventral apex of the spleen. The stomach was displaced because of the abnormal position of the sternal and diaphragmatic flexures which were displaced cranial to the stomach between it and the left lobe of the liver. The left ventral and dorsal colons were displaced dorsally between the suspensory ligaments of the spleen and the dorsal body wall and were distended caudally and cranially to the compressed portion. This compression caused partial obstruction of colonic blood flow resulting in blanching of the left

ventral and dorsal colons in one horse. All 6 horses made an uneventful recovery following surgical correction of the displacements. A further 2 cases, both young Thoroughbreds, were described by **Spiers, Hilbert and Blood (1979)**. The Authors reviewed the available literature showing the condition had in fact been described by Forssell in 1908 but was a rare cause of colic. In neither case was the transverse colon displaced anterior to the stomach as was described by **Milne and colleagues (1977)** but a greater length of colon was involved in case 2 which included part of the transverse colon. There was also displacement of the caecum. The reasons for the displacement were only speculative such as tympany or lack of ingesta followed by rolling or strenuous exercise. The left ventral and dorsal colons have no stabilising attachments.

Right dorsal displacement of the large colon was described by **Huskamp and Kopf (1983)**. The Authors could only find one previous report. The findings in 48 cases were described. In each case the pelvic flecture was located cranially accompanied by various degrees of torsion of the left ventral and dorsal colons. One animal was destroyed because of financial constraints but the remaining 47 underwent surgery and made an uneventful recovery.

Hackett (1983) described 32 cases of non-strangulating displacement of the colon and consigned the cases to one of 6 groups at laparotomy. Volvulus of the large colons had occured in 4 cases, volvulus of the large colons and caecum in 8 cases. The degree of rotation was from 180-270° in both groups, and volvulus of the sternal and diaphragmatic flexures, with a 180° twist, occured in one heavily pregnant mare. Cranial displacement of the pelvic flexure was identified in 8 animals,

3 of which also had colonic impaction but it was not clear whether this was cause or effect. Displacement of the left colon to the right, 4 cases, and dorsally, 7 cases, was also encountered. Twenty five animals were discharged from the hospital following surgery although 2 subsequently died of colic 8 and 13 months later.

Volvulus is a form of displacement where the affected portion of intestine remains within the abdomen. It can occur secondary to or in association with another lesion such as a congenital defect (**Grant and Tennant 1973, Freeman et al 1979, Yorich and Horney 1983, Harrison 1989**). These papers have been reviewed in the section on congenital defects. Volvulus of the large intestines without any predisposing lesion has also been recorded. **Barclay, Foerner and Philips (1980)** described 25 cases with involvement of the large colon. At surgery the dorsal and ventral colons were found to be distended with gas, fluid and ingesta and the serosal surface varied from blanched white to light blue. There was mesenteric oedema and congestion or thrombosis of its vessels. Nine horses survived surgery the remaining 16 died or were destroyed during or shortly after surgery. In those horses that were destroyed or died the colonic mucosa was black and sloughing, the mesenteric vessels were thrombosed and the serosa was blue/black or had ecchymotic haemorrhages. The colonic wall was thick and oedematous. Possible causes were given as excessive rolling and verminous arteritis but neither were substantiated.

A much larger report concerning 124 cases of large intestinal volvulus in 122 horses was published by **Harrison (1988)**. It was stated that the

large size and relatively small area of attachment to the body wall predispose the equine large intestines to both strangulating and nonstrangulating displacements. The most common site of volvulus was the region of attachment of the caeco-colic fold, 47 cases. The left ascending colon or sternal/diaphragmatic flexures were involved in 33 cases, the entire caecal base and transverse colon were involved in 26 cases and the right ascending colon cranial to the caecal fold in 3 cases. The remaining 15 cases were not accounted for. Of the 32 cases in which the direction of the volvulus was recorded it was dorso-medial in 30 and dorso-lateral in 2. The Author found that the normal weight of the empty washed out large intestines was about 10kg whereas large intestines subjected to a 360° volvulus at the caecocolic fold weighed about 32kg. This study substantiated a previous claim that ascending colon volvulus was primarily a condition of brood mares as a number had foaled less than 10 days before admission and 20 were pregnant at admission.

Snyder and colleagues (1989a) confined their series of 57 cases to those with strangulating volvulus of the ascending colon. Forty two were mares, 21 of which were postparturient, 8 were stallions and 7 were geldings. It appeared to occur most commonly in the hotter months with only 5 cases in the winter but this may have been related to breeding patterns. Microscopic examination always revealed haemorrhage and oedema in the submucosa and mucosa. Where only minor damage had occurred to the surface and crypt epithelium haemorrhage was confined to the lamina propria next to the muscularis mucosa. Where damage was more extensive the haemorrhage was present throughout the lamina propria. Mucosal damage was characterised by variable epithelial sloughing and

degeneration. This varied from small groups of cells to the entire epithelium. Preservation of crypt epithelium correlated with survival. Degree of rotation also affected survival, 71% of those with a 270° volvulus survived but only 36% with a 360° volvulus survived. The serosa was light blue in the former becoming pink after correction and blue black in the latter. By grading the histopathological changes they could be used as a prognostic guide. Significant differences were noted between survivors and non survivors in the superficial mucosal and crypt epithelium alterations and in the interstitium to crypt ratio. The histopathological findings in 7 of the cases suggested that arterial obstruction preceded venous obstruction. Interestingly a mucosal inflammatory cell infiltrate appeared to be protective increasing the survival rate.

The most recent report of torsion of the large colon concerned a single case of 180° torsion around the mesenteric root in a 20 year old Thoroughbred Gelding (**Mair and Lucke 1992**). The paper dealt predominantly with the unusual associated finding of chyloperitoneum and the possible causes as described in man. Although the precise cause could not be determined in this case it was possibly due to compression by the distended large colon but this has not been recorded previously.

The intestines can also be displaced outwith the abdominal cavity to become externally visible via a number of routes. **Steckel and Nugent (1983)** described the findings in a Quarter Horse yearling filly which had suffered a sudden increase in size of its umbilical hernia and colic. The sac measured 6x8cm and was oedematous, warm and painful. Surgical exploration of the abdomen revealed a segment of small intestine trapped at the orifice. This proved to be a portion of the

proximal ileum. The sac also contained 30ml of serosanguinous fluid. On further examination a 3cm portion of the antimesenteric wall was found to be devitalised and near rupture. The affected length of intestine was removed. This type of hernia is known as a "Rickter's" hernia and had not been previously reported in the horse.

Stolfus (1980) related the consequence of breeding trauma in a miniature Appaloosa mare in a rather jolly, anecdotal style. At presentation an 18 inch length of small intestine was protruding from the vagina and hung halfway to the hocks. The mare had been served by a full size stallion that morning and his penis had torn a rent in the anterior, right lateral vagina. The intestines appeared completely undamaged so were thoroughly cleaned and returned to the abdomen. The rent was sutured blindly. Recovery was uneventful.

Vaginal evisceration of the small intestine in 3 mares was described in slightly more "scientific" terms by **Tulleners, Richardson and Reid (1985)**. Return of the prolapsed viscera via the vaginal lesion was performed in the first case, a 5 year old Thoroughbred mare, but this had to be followed 28 hours later by a laparotomy and removal of 2m of necrotic distal jejunum and ileum. A large mesenteric tear and a bleeding vessel were also identified and repaired. In the second case, a 7 year old Thoroughbred mare, simple replacement and closure of the rent sufficed. In the third case a portion of devitalised small intestine was found to be protruding from the external urethral orifice, presumably from a ruptured urethra or urinary bladder. Standing repair was attempted but gastric rupture ensued and the mare was destroyed.

Schneider, Milne and Kohn (1982) reviewed the findings in 27 cases of acquired inguinal hernia in the horse, 26 of which were stallions and one a gelding. Surgery was performed on 24 of the animals. In a proportion of these the herneated intestine, often the distal ileum, was found to be nonviable and was resected. In 21 of the surgical cases the small intestines were displaced directly through the internal inguinal ring and were in the common vaginal tunic. In the gelding the intestine was displaced through the inguinal canal and was in the subcutaneous fascia of the inguinal region. In the other 2 surgical cases the small intestine was outside the common vaginal tunic. The internal inguinal ring was not enlarged in every case, sometimes measuring only 1 finger in diameter, but many had a history of recent strenuous exercise or breeding activity. The problem of inguinal hernia can be even more serious if it occurs following castration and results in visceral prolapse. Van der Velden and Rutgers (1990) described the findings in 18 such cases concentrating on comparison of the different castration techniques and their safety. The Authors stated that following castration, especially by the open method, omentum or intestine may prolapse through the inguinal ring and vaginal cavity of the inguinal canal into the scrotum. The prolapse could be considered an acquired inguinal hernia. In the 18 cases studied the time between castration and prolapse varied from one hour to 4 days. In 6 cases the tissue involved was omentum, in 9 it was jejunum and in the remaining 3 it was jejunum and ileum. Fifteen animals recovered but one was then destroyed 6 months later because of colic and no post mortem examination was performed.

OBSTRUCTION

Obstruction of the intestinal tract at any level can be the result of one or more of a great many lesions. All of the previous sections in this literature review contain examples of such lesions. Indeed every form of displacement reviewed in the previous displacement section can lead to obstruction of the affected portion of bowel and it is the pain caused by this obstruction which often prompts investigation.

Of the congenital lesions some cause a physical obstruction eg Meckel's diverticulum (Hooper 1989) or atresia coli (Cho and Taylor 1986) and some cause a functional obstruction eg aganglionosis in white progeny of overo spotted horses (McCabe et al 1990). In foals, the small intestines may be blocked by an impacted mass of dead *Parascaris equorum* following anthelmintic treatment (Becht and Semrad 1986).

The presence of a neoplasm either arising from the intestines or from another abdominal organ may lead to obstruction. In the former group the following have been implicated: Intestinal myxosarcoma (Edens et al 1992), leiomyosarcoma (Mair et al 1990a), leiomyoma (Hanes and Robertson 1983), adenocarcinoma (Honnes et al 1987), ganglioneuroma (Allen et al 1989), neurofibroma (Pascoe 1982) and intestinal carcinoid (Orsini et al 1988). In the latter group a malignant perianal melanoma caused direct pressure on the rectum (Shokry and Lotfi 1984) and a strangulating lipoma (Ramey and Reinerston 1984) and a granulosa cell tumour (Wilson et al 1989) entrapped a portion of intestine.

A great many of the miscellaneous conditions in the final section of this review were either the result of intestinal obstruction eg gastric rupture (**Todhunter, Erb and Roth 1986**) or yet another possible cause of it. Of the latter group duodenitis/proximal jejunitis is extremely difficult to differentiate from small intestinal obstruction on clinical grounds (**Johnston and Morris 1987**). Idiopathic muscular hypertrophy, most frequently of the ileum greatly narrows and obstructs the lumen (**Chaffin et al 1992**). Caecal impaction or overload appears to be the result of a physiological rather than a physical obstruction frequently occurring post operatively (**Hilbert, Little, Bolton and McGill 1987**). Grass sickness (**Obel 1955**), mal seco (**Uzal, Robles and Olaechea 1992**) and ileus (**Adams 1988**) are further examples of physiological obstructions.

Of the papers not reviewed elsewhere one by **Edwards (1981)** concerning obstruction of the ileum in 27 horses included conditions from all of the afore-mentioned categories. The most common cause was incarceration through the epiploic foramen which accounted for 7 cases. There were 3 cases each of muscular hypertrophy, strangulation by a pedunculated lipoma and incarceration by a mesenteric tear, 2 cases each of strangulation by a fibrous band, strangulated scrotal hernia, impaction and intussusception and one each of occlusion by a mesenteric anomaly, incarceration by an umbilical hernia and a strangulated diaphragmatic hernia. Thirteen of the animals recovered.

One case of gastric retention associated with acquired pyloric stenosis in a 3 year old Thoroughbred cross gelding was described by **Church, Baker and May (1986)** and the available literature was reviewed. The

diagnosis of pyloric obstruction was made preoperatively and confirmed at laparotomy. The pyloric wall and the majority of the glandular stomach were massively thickened and rigid. The rest of the stomach was distended. The lumen of the affected portion was reduced to less than 1cm in diameter and the wall was greater than 7cm thick. The extent of the lesion and the belief that it was neoplastic prompted euthanasia. At necropsy the pyloric mass was 15x20x15cm and involved the entire circumference of the stomach wall. The mucosal surface was ulcerated with 2 deep clefts and the cut surface was firm and white. The bile duct and biliary tree were enormously dilated because of a 4x2x2cm mass occluding the orifice of the common bile duct. Microscopic examination of both masses revealed only granulation tissue which it was speculated had developed in response to previous ulceration.

Dobson and Lopez (1981) recorded a case of intestinal obstruction and gastric rupture involving a penetrating foreign body in a 6 year old Quarter Horse. The animal died soon after admission and a post mortem examination was performed. This revealed 81 of fluid and fresh ingesta within the abdominal cavity and multiple peritoneal haemorrhages. The stomach had split along its greater curvature. A thick band of fibrous tissue extended from the apex of the caecum to the mid jejunum. Nodules of mature granulation tissue, each containing a piece of wire and measuring 12x4cm and 9x9cm, were present at each end. The nodule in the jejunal wall had caused a fibrous stricture reducing the intestinal lumen and producing distention proximally. Two previous cases of penetration of the bowel wall by a foreign body were described.

A complete review of enteroliths in horses was given by **Lloyd, Hintz, Wheat and Schryver (1987)**. It included details on history, composition, shape, numbers and sizes, rate of growth and factors involved in formation. An apparent higher incidence in Arabians was pointed out but the cause of this is unknown.

A case of duodenal impaction in a 10 year old Thoroughbred mare was reported by **Bohanon (1988)**. At laparotomy the impaction was located 15cm distal to the pylorus and measured 8cm in diameter. It was hard, composed of tightly compressed cracked corn and had resulted in marked distention of the stomach and proximal small intestine. In addition displacement of the caecum and pelvic flexure were detected and easily corrected. Several of the mare's stable mates developed colic at the same time. Two died with a ruptured stomach and firm dry undigested corn obstructing the stomach/and or duodenum was found. In a third horse exploratory laparotomy revealed a nephrosplenic entrapment of the left colons as well as firm ingesta in the proximal duodenum.

Green and Tong (1988) described 2 cases of small intestinal obstruction this time associated with wood chewing. In the first case, an 11 year old pony gelding, the obstruction was located in the descending duodenum adjacent to the right kidney. It had resulted in marked dilatation of the stomach and proximal duodenum. The bowel proximal to the obstruction was dark but apparently not devitalised. On removal the mass was hard and black, measured 8x6cm and was composed of wood splinters and split twigs. It required a saw to divide it. The second case, a 9 year old Halfbred gelding had a hard, egg shaped mass in the jejunum. The stomach and proximal small intestines were dull red and

distended. The mass was composed of felted wood fibres and splinters resembling "chip board" and could not be broken by hand. The Authors considered obstruction of the small intestines by foreign material a rare occurrence and other causes of intestinal obstruction were discussed.

Twelve cases of ileal impaction were reported by **Embertson and colleagues (1985)**. All horses had a mass of ingesta impacted from the ileocaecal orifice cranially for 30-90cm. The intestines proximal to the impaction were distended. One horse had further impacted areas, both in the distal small intestines and one horse had a 2mm perforation proximal to the impaction. Despite surgical exploration of the abdomen in 11 animals and full post-mortem examination of 4 of these and of the animal which died, no mechanical obstructive lesions were found to explain the impactions.

A much larger series of 75 cases of ileal impaction was reported by **Parks and colleagues (1989)**. Analysis of the results revealed that Arabians and mares were significantly over represented. Six horses were destroyed prior to surgery and 4 did not recover from the anaesthesia. Additional lesions encountered at laparotomy included large colon displacements (6), small intestinal torsion (1), jejunal puncture wound (1) and ileal smooth muscle hypertrophy (4). The Authors concentrated on the clinical findings but did note that mucosal damage had occurred both proximal and distal to the site of obstruction.

Boles and Kohn (1977) described 10 cases of fibrous foreign body impaction colic. All were young horses, and in each case the impaction

was caused by rubberised fencing material trapped in the proximal small colon. Other incidences of impaction caused by similar material were reviewed.

Three cases of obstruction of the small colon by a foreign body were reported by **Van Wuijckhuise-Sjouke (1984)**. In the first case, a 3 1/2 year old Dutch half bred gelding, peritonitis developed as a result of rupture of the small colon secondary to its obstruction by a concretion consisting of a guy-rope and hair encrusted with food material and minerals. The second case, a 7 year old Fjord gelding had shown signs of colic for 14 days before it died. At necropsy an enterolith consisting of a piece of bailing twine encrusted with ingesta, sand and minerals was found in the small colon. The colon was damaged proximal to the obstruction. In the final case, a 6 1/2 year old pony mare, the owner elected for euthanasia and at necropsy an enterolith consisting of cloth and fishing line encrusted with ingesta and minerals was present in the small colon.

Although sand accumulated within the equine large intestine does not form a completely solid mass it none the less leads to impaction. The surgical treatment of 48 cases of sand colic was described by **Specht and Colahan (1988)**. In each case various portions of the large intestines were hard, granular, and difficult to indent. A large amount of sand was present in the impacted content. Single impactions were found at the pelvic flecture (14), right dorsal colon (3), left dorsal colon (2) and transverse colon (1). The right dorsal colon (15 cases) and transverse colon (11 cases) were involved most frequently when 2 or more impactions were present but the additional sites could be anywhere from

the ileocaecal junction to the small colon. Eight cases had impactions at 3 sites and 3 cases had impactions at 4 sites. Twenty six cases had concurrent large colon displacement or torsion. Fourteen cases survived surgery and were discharged although one developed colic due to extensive adhesion formation 11 months later and was destroyed. Another study of the surgical treatment of sand colic, this time in 40 horses was made by **Ragle, Meacher, Lacroix and Honnas (1989)**. Multiple sites of accumulation or obstruction were identified in 20 horses. Concurrent large colon displacement or volvulus was recorded in 10 horses.

An intramural haematoma has been cited as the cause of both small and large intestinal obstruction. **Kobluk and Smith (1988)** described one such haematoma in the jejunum of a 15 year old Thoroughbred mare. At laparotomy there was a firm, dilated, 40cm segment of jejunum approximately 150cm proximal to the ileocaecal junction. The small intestines proximal to this were filled with gas and fluid. The serosal surface of the affected segment was dark red, except adjacent to the mesenteric attachment, suggesting an intralumenal haematoma. The associated mesentery and its vessels appeared grossly normal. The animal made an uneventful recovery following resection of the segment. The haematoma was deep to the serosa and completely obstructed the lumen. The cause was not established but histological examination revealed an intense eosinophil infiltrate so the possibility of damage caused by migrating *Strongylus vulgaris* larvae was considered. The mare developed colic, due to volvulus around adhesions at the surgical site, and was destroyed 4 years later.

Three cases of obstruction of the small colon by an intramural haematoma were described by Spiers and colleagues (1981). In case one the haematoma was located 1.5m proximal to the anus and did not communicate with the lumen. The affected portion was resected and examination revealed marked submucosal oedema, haemorrhage, thrombosis and increased numbers of polymorphs. The site of and reason for the haemorrhage were not found. The animal died during the recovery period. In the second case, a 10 year old mare, intestinal content was found free in the abdomen at laparotomy and the animal was destroyed. A mass measuring 27x7cm was identified at necropsy. It appeared to have resulted from focal ulceration of the mucosa with infection of the submucosa to produce severe dissecting cellulitis and blood vessel damage. In the final case, a 13 year old Thoroughbred stallion, fresh blood was noticed on the arm of the referring vet following rectal examination for colic of 3 days duration. Iatrogenic trauma was suspected. An exploratory laparotomy revealed obstruction of the small colon 30cm anterior to the pelvic brim where there was extensive bruising and distention and a 1cm tear in the serosa which oozed bloody fluid. The affected portion was resected and the haematoma was found to communicate with the gut lumen. A further 4 cases with involvement of the small colon, this time described as submucosal haematomas, were reported by Pearson and Waterman (1986). Three cases had a history of colic for 24 hours and the fourth case a history of colic for 5 days with sudden deterioration overnight. In case one a 50cm length of small colon was completely occluded by a massive blood clot between the mucosa and muscularis mucosa which contained several litres of blood. Histological examination revealed thrombosis of many blood vessels and foci of eosinophils, haemorrhage and fat necrosis in the mesentery. In case 2

there was occlusion of 25cm of colon by a submucosal haematoma. No source for the haemorrhage was found on microscopic examination. Gross haemoperitoneum was evident in case 3, along with distention of the large intestines. A 15cm length of small colon was obstructed. There were several large clots and much blood below the mucosa. Examination revealed infarction of small mucosal blood vessels and early granulation tissue at the margins of the lesion. No vascular abnormality was found to account for the haemorrhage. In case 4 there was distention of the small colon proximal to a 20cm portion of occluded gut. Again no source of haemorrhage was identified.

Finally, extralumenal obstruction of the rectum may be caused by perirectal abscesses. Six such cases were described by **Sanders-Shamis (1985)**. All presented with signs of colic. One had a history of dystocia 30 days previously and 2 had received injections one to 6 weeks before presentation. The location of the abscesses varied but all were palpable per rectum and had led to faecal impaction. In relation to the rectum the abscess was lateral in 3 cases, dorsal in 2 and ventral in one. In 2 horses the abscesses involved abdominal organs and peritonitis ensued, one of these animals died. Culture of the aspirated material from 5 cases yielded *Staphlococcus zooepidemicus* and/or *Escherichia coli*.

MISCELLANEOUS CONDITIONS

A final group of conditions exist which in some cases are associated with one of the preceding groups but are still sufficiently different to merit a section on their own. Gastrointestinal ulceration, as reviewed by **Baker (1991)**, is a condition which affects both foals and adults but is most commonly encountered as a clinical entity in the former. The only proven cause is nonsteriodal antinflammatory drug therapy. However, stress has been incriminated with one study showing an increase in prevalence and severity of gastric lesions at post-mortem examination in race horses killed straight from training versus those which had been retired for at least one month.

Four cases of primary gastric impaction were described by **Barclay, Foerner, Phillips and McHarg (1982)**. The diagnosis was made at surgery and no physical lesion was identified that might have obstructed gastric outflow. In each case the stomach contained dry, compacted ingesta which required hydration before it could be massaged through the pylorus. This was despite the fact that the 4 animals had already received large quantities of mineral oil via stomach tube. However, fluid normally bypasses gastric solids in the horse unless the stomach is almost empty. The Authors offered no explanation for the development of these impactions.

Three cases of gastric impaction associated with ragwort poisoning were described by **Milne, Pogson and Doxey (1990)**. The stomach was distended in one case by dry hay and in another case by 24 litres of soft grass-like ingesta and had ruptured premortem in both cases. In the

stomach was impacted by a large amount of dry grass but had not ruptured. No other lesions of the gastrointestinal tract, or its associated sympathetic ganglia, were detected. The liver was grossly abnormal in each case and megalocytosis was detected histopathologically. The relevant literature and the difference between gastric dilation and impaction were discussed and the causes of primary and secondary gastric impaction were listed.

Fifty four cases of gastric rupture, due to a variety of causes, were described by **Todhunter and colleagues (1986)**. The prevalence varied from 1% in a general necropsy survey to 8% in animals with acute abdominal disease. Rupture was preceded in each case by dilatation. The causes of primary dilatation as previously described in the literature were listed. The most likely cause of the dilatation in the 54 cases presented fell into one of 6 categories, primary, idiopathic, obstructive, peritoneal, enteric or traumatic with only one case in the last group and the remaining 53 cases spread roughly equally among the other 5 categories. Approximately 66% of the cases were secondary. No specific risk factor for gastric dilatation was identified and a great many possible aetiologies emerged. In general gastric rupture occurs along the greater curvature and the serosal lesion is larger than the mucosal lesion.

An apparently rare cause of gastric dilatation, acute necrotising pancreatitis, was described by **Lilley and Beeman (1981)**. The animal in question, a 5 year old Arabian gelding, was presented because of acute abdominal pain and nasogastric reflux. A hopeless prognosis was given and the animal was destroyed. At necropsy the abdomen contained a large

amount of serosanguinous fluid. The majority of the intestinal tract was grossly normal but the proximal 30cm of duodenum was dark scarlet and dilated to 1 1/2 times normal size. The serosal surface was darker than the mucosa, was flabby and lacked structural integrity. Several small, 1-7mm diameter, chalky white plaques were dispersed throughout the visceral peritoneum in the area of the pancreas and throughout the interstitium of the most distal aspect of the pancreas. The main body of the pancreas was oedematous. The gastric serosa was split to a length of 15cm along the greater curvature and covered in petechial and echymotic haemorrhages. Microscopic examination of the duodenum revealed a mild catarrhal enteritis with haemorrhage in the muscle and serosal layers. No cause for the pancreatitis was established but possible causes and the likely mechanisms were discussed.

One of the many conditions which can lead to gastric dilatation is duodenitis-proximal jejunitis also known as anterior enteritis or gastroduodenojejunitis. The findings in 20 cases, as encountered during surgery and/or post-mortem examination, were described by White, Tyler, Blackwell and Allen (1987). In all cases the diagnosis was made on gross inspection of the intestines. The duodenum was always involved and the jejunum inconsistantly so, leaving the ileum and large intestines unaffected. The involved intestine and stomach were distended and the contents varied from red to brown. The serosal surface was smooth with numerous petechial and echymotic haemorrhages and multifocal pale pink areas with white and yellow streaks. The mucosa of the proximal small intestines was deep red and contained petechial haemorrhages with the addition of focal necrosis and ulceration in 3 cases. Microscopic lesions were most consistent and

marked in the duodenum and jejunum but were present throughout the alimentary tract from the stomach to the large colon. The lesions varied from mucosal and submucosal hyperaemia and oedema in mild cases to sloughing of villus epithelium, haemorrhage and neutrophil infiltration in moderate cases with extensive sloughing, villus atrophy, haemorrhage, oedema and infiltration in severe cases. Submucosal lymphoid patches were markedly depleted. Bacteriological culture of affected intestines yielded *Clostridium sp* in 3 out of 9 horses and viral isolation was negative in all cases. No aetiology was proposed.

Another paper on duodenitis-proximal jejunitis compared the condition to small intestinal obstruction (**Johnston and Morris 1987**). Thirty four cases of each condition were studied and the importance of a clinical distinction emphasised. Surgical intervention is necessary in cases of obstruction but should be avoided in duodenitis-proximal jejunitis. The small intestinal obstructions were due to a number of lesions such as volvulus at the root of the mesentery (11 cases), strangulation through a rent in the mesentery (5 cases), mesodiverticular band strangulation (5 cases), strangulating lipoma (4 cases), strangulation around abdominal adhesions (2 cases) and single examples of a mixture of conditions. A number of significant differences in the clinical findings emerged and the survival rate in the duodenitis-proximal jejunitis group was much higher, 94%, compared to the obstructed group 29.4%.

No specific conditions of the jejunum have been described. There has been one report of ischaemic necrosis of the jejunum caused by a penetrating foreign body (**Davies 1983**). At surgery 2.4m of mid jejunum

was found to be purple, ischaemic and requiring resection. The lumen of the affected bowel was not obstructed. The associated mesentery was distorted and incorporated a fibrous mass, 10cm in diameter, which was adherent to the jejunum distal to the compromised portion. A fibrous cord containing a 4.5cm length of rusty wire was found within the adhesions. The wire had tracked from the lumen of the small intestine but had been encapsulated by fibrous tissue. Other possible causes of bowel ischaemia were also given.

One condition which predominantly affects the ileum is idiopathic muscular hypertrophy. **Schneider, Kennedy and Leipold (1979)** were the first to record it in the United States but stated that it had been well recognised elsewhere and had been reported in English pit ponies during the early twentieth century. The paper described the findings in a single case with a 3 months history of weight loss. The small intestines had gradually increased in diameter and thickness of wall over the caudal two thirds of their length. Maximum thickness, 2.5-3cm, was attained for a length of 22cm, terminating 10cm cranial to the ileocaecal junction. The most affected area was the circular layer of the muscularis which was whitish and firm with vertical striations. The associated serosa was diffusely thickened and white. At the point of maximum thickness the lumen was closed but cranial to the obstruction it was dilated. The ileocaecal valve and large intestines were within normal limits. Microscopic examination revealed that the inner circular layer of smooth muscle was affected to a greater extent than the outer layer. The vertical clefts noted grossly were due to separation of fibres by oedema. There was an increase in cell size rather than cell number. The submucosa was congested and oedematous with an increase in

mixed inflammatory cells. Some irregularly shaped villi and focal mucosal haemorrhage were noted.

A case of ileal smooth muscle hypertrophy and rupture was reported by **Lindsay, Confer and Ochoa (1981)**. The animal had an 8 week history of recurrent colic. At necropsy a pocket containing ingesta was present in the terminal 12cm of ileum which was joined to the caecal base by fibrous connective tissue. The adjacent serosa was thick and red-tan. The circular smooth muscle layers, especially the inner layer, of the distal 30cm of ileum became progressively thicker reaching 1.5cm. This ended abruptly so that the last 2-3cm and ileocaecal valve appeared normal. The associated mucosa was hyperaemic and contained two stellate scars 1-1.5cm across which extended transmurally. Adjacent to the scars a 2cm diameter tract opened into the pocket. Microscopic examination revealed excess goblet cells in the mucosa, and fibrosis, neovascularisation and mild mononuclear inflammatory cell infiltration of the submucosa. The Authors proposed a number of possible causes for the lesion such as parasitism or a resolved intussusception although no such cause was evident at time of examination.

The most recent report of idiopathic small intestinal muscular hypertrophy concerns 11 cases (**Chaffin et al 1992**). All had a history of colic of from 3-870 days duration. In 5 cases the hypertrophy was confined to the ileum and 3 of these had ileal diverticulae, in 2 cases the hypertrophy was confined to the jejunum and one had a jejunal diverticulum, in 2 cases both the jejunum and ileum were involved with an ileal diverticulum in one and in the remaining 2 cases the duodenum, jejunum and ileum were involved with an ileal diverticulum in one. In

one of the cases where the hypertrophy was confined to the jejunum there was a 1.5m long split in the muscle along the antimesenteric border which allowed the mucosa to bulge through stretching the intact serosa. In 3 cases there was impaction and rupture of the ileum. In all cases no lesion was detected which could have induced secondary hypertrophy of the ileum but the more proximal lesions could have developed secondary to the ileal lesions. Microscopic examination revealed hypertrophy of both the circular and longitudinal muscle layers in each case which was due to enlargement of individual cells. *Haemomelasma ilei* was present in 8 of the cases which seemed more than coincidence to the Authors but the cause and effect of this lesion were not determined.

Kiper, MacAllister and Qualls (1988) reported a case of haematochezia attributable to a cranial mesenteric arterial aneurysm with connecting tracts to the ileum and caecum in an 18 month old stallion. Intermittent bloody diarrhoea had been noted over a 4 week period. At surgery blood clots and fibrin strands were found in the peritoneal cavity. There was a 25-30cm firm lobulated soft tissue mass medial to the caecal base which incorporated the cranial mesenteric artery and caecal wall. Post-mortem examination revealed that the mass was firmly attached to the ileum, caecum and large colon near the ileocaecal junction. Two full thickness defects (3-5mm in diameter) were seen in the mucosa of the terminal ileum and one in the caecum which connected via a cavity in the mass to the cranial mesenteric artery. The abdominal lymph nodes and caecal and colonic walls were oedematous. The lesion was described as a cranial mesenteric arterial aneurysm with connecting tracts to the ileum and caecum and may have been attributable to *Strongylus vulgaris* activity.

Twenty one cases of caecal impaction were described by **Campbell and colleagues (1984)**. They were divided into 3 groups. Six horses presented with caecal rupture, 4 developed caecal impaction and rupture post anaesthesia and surgery and 11 were admitted with colic and underwent exploratory laparotomy. All the horses in the first and second groups were euthanised as were 4 of the third group 2 of which again suffered caecal rupture. In all cases the impacted material was primarily coarse, dry roughage. A previous full thickness tear had apparently occurred in 2 animals and a partial tear in one. The size, direction and site of the rupture varied.

Ross, Martin and Donawick (1985) confined their review to 23 cases with unexpected rupture of the caecum out of a group of 66 horses with primary caecal disease. Thirty four of the remaining animals had detectable caecal impaction before rupture and 9 had other primary caecal diseases or conditions. Of the 23 animals, 10 presented with a ruptured caecum and 13 were already hospitalised. Again the perforations occurred in a number of different sites and in 22 of the 23 animals there was a large firm ingesta-filled caecum and empty colon but no physical obstruction. This was considered to be due to abnormal caecal out-flow and ingesta accumulation secondary to lack of propulsive motility or a functional obstruction.

Hilbert and colleagues(1987) reviewed the relevant literature on caecal overload and rupture and pointed out the increased risk in animals post surgery and in periparturient mares and presented the findings in 12 cases from their files. Six cases had no history of gastrointestinal

tract disease and had undergone minor elective surgery for an orthopaedic problem, and 6 cases had been treated by the referring vet for signs of colic. At necropsy the caecum was filled with ingesta, ie. caecal overload, but not impacted. The rupture occurred at various sites. The Authors appeared to implicate perioperative feeding management as a possible cause.

The particular problem of caecal rupture in parturient mares was studied by **Platt (1983)**. Full post-mortem examinations had been performed on 4 animals. One mare died in labour and the others became ill soon afterwards. In the former, death appeared to have been sudden and peritonitis had not developed. In all cases the rupture occurred at the base of the caecum. In at least 2 cases there were separate mucosal tears elsewhere in the base of the caecum that did not extend through the wall. A review of the literature revealed that the right ventral colon may also rupture at parturition. The Author surmised that as the foal changed position in first and second stage labour it might kick the adjacent caecum. If other predisposing factors, such as caecal tympany were present the organ might rupture. It was noted that the condition had thus far only been described in Thoroughbreds.

Another problem reported in postparturient mares is segmental ischaemic necrosis. The findings in 5 cases were described by **Livesey and Keller (1986)**. In each case the affected portion of bowel was the small colon and tears were present in the mesocolon of the affected area. One animal recovered following resection and anastomosis of the affected portion, 2 sustained fractures during recovery from anaesthesia and one developed peritonitis and one became impacted proximal to the

anastomosis site. Previously reported cases of ischaemic necrosis of the small colon following mesocolonic rupture were associated with type III or IV rectal prolapse. In these cases intussusception of the small colon through the anus resulted in stretching of the mesocolon and possibly rupture and disruption of the vascular arcade leading to ischaemia of the affected portion.

Another well recognised and all too common problem is the rectal rear. The pertinent anatomy and findings in 42 cases were reviewed by **Arnold, Meagher and Lohse (1978)**. An important point to note is the short distance between the peritoneum and anus, only 15-20cm, so what is thought be a caudal pelvic retroperitoneal injury may in fact be a perforation into the peritoneal cavity. Most tears were longitudinal, at the pelvic inset, 20-30cm proximal to the anus and between 10 and 12 o'clock in the circumference of the rectum. By far the most common cause was rectal examination. The Authors speculate that entry of the major blood vessels at the mesocolonic band may result in an area of weakness predisposing the rectum to rupture at this point, ie dorsally.

Three noniatrogenic tears have also been reported (**Slone, Humburg, Jager and Powers 1982**). In one case an initial examination did not detect a tear and no blood was found. The tear was identified during a subsequent examination. It had rounded edges and did not appear to be recent. This was confirmed by histopathological examination. In the second case the tear was identified by palpation and was found to contain dry, impacted faeces and clotted blood. Faecal material was present in the mesocolon and underrunning the serosa of the small colon. In the third case the tear appeared to have been caused by the rectal

examination made by the owner but multiple areas resembling infarcts were identified in the stomach and small intestine at necropsy so a similar lesion may have predisposed the rectum to tearing. Infarction was also the most likely cause of the lesion in the first case.

Abscessation can occur within any abdominal organ but of the 25 cases described by **Rumbaugh, Smith and Carlson (1978)** 17 involved the mesentery. The organisms most commonly cultured from the 8 cases which died were *Streptoccus equi*, *Streptococcus zooepidemicus* and *Corynebacterium pseudotuberculosis*. It is not clear from the case descriptions where these abscesses were located. The paper deals predominantly with clinical signs and diagnosis. **Dyson (1983)** reviewed the findings in 30 cases of peritonitis. A variety of causes were identified in the 9 horses which were destroyed and underwent post mortem examination. In each case the gastrointestinal tract was the location of the primary lesion.

Grass sickness is a curious condition the aetiology of which is still unknown. By the time Obel first described the neuronal lesions found in the sympathetic ganglia the condition and its gross pathology were well recognised (**Obel 1955**). The Author reviewed the literature to that date listing dilation of the stomach and small intestines by fluid, inspissation of the large intestinal content and the presence there of large quantities of mucus as the usual findings. Oesophageal ulceration and splenic enlargement had also been noted. Microscopic examination of the central nervous system and a number of other organs had not revealed any specific lesions. The gross findings in the 9 acute and 5 chronic cases studied were consistent with the previous descriptions. Microscopic examination of the sympathetic ganglia (cranial cervical,

stellate, solar and caudal mesenteric ganglia and the thoracic and abdominal sympathetic trunk) revealed advanced changes. The ganglion cells were swollen with central chromatolysis advancing to total chromatolysis. The nuclei lay eccentrically and the nucleoli and the nuclear membranes were strongly basophilic. In the most advanced cases small vacuoles occurred in the cytoplasma of the chromatolytic cells. These often lay centrally and peripherally with a layer of condensed eosinophilic cytoplasm between. There was often karyopyknosis and karyolysis of the nucleus when these vacuoles occurred. The most marked changes were found in the cranial cervical ganglion, the stellate ganglion and the sympathetic trunk with the solar and caudal mesenteric ganglia less involved. Examination of the intramural ganglia in the gastric and intestinal wall revealed similar changes. The similarity to the histopathological lesions of poliomyelitis in man was noted and the many proposed causes described.

Further studies of the neuropathology of grass sickness were made by Barlow (1969). The Author described the gross and microscopic lesions of 6 "typical" cases. The lesions in 5 of the 6 animals were consistent with those previously described (Obel 1955) with similar changes also noted in the central nervous system of 5 of the cases (all 3 chronic cases and 2 of the acute cases). In the sixth case only small intestinal distention was noted and no neurological lesions were found on histopathological examination. The severity of the central nervous system lesions was directly proportional to the duration of the signs so may possibly be secondary.

Uzal and colleagues (1992) described the findings in 4 horses with mal seco, a grass sickness-like syndrome in Argentina. The stomach and small intestines were either empty or filled with fluid, the large colon and frequently the caecum were distended and filled with dry, hard ingesta and there was congestion of the abdominal organs. Histopathological examination of the celiacomesenteric ganglia revealed swollen, homogenous, eosinophilic neurones. There was loss of Nissl substance and a narrow peripheral rim of basophilia or vacuolation of the cytoplasm, mainly centrally. In addition round or ovoid, smoothly outlined, homogenous, eosinophilic structures were present in the perineuronal tissue and in the perikaryon of normal and degenerating neurones in 3 out of 4 horses. In the degenerating neurones a small dark nucleus was eccentrically located. The Authors believed that the clinical signs and gross microscopic lesions were so similar to those of grass sickness (**Obel 1955**) that mal seco and grass sickness were in fact one and the same condition.

A single case of myenteric ganglionitis resulting in chronic intestinal pseudo-obstruction was described by **Burns, Karcher and Cummings (1990)**. An exploratory laparotomy demonstrated impaction of the entire small colon. Examination of a biopsy sample revealed marked fibrosis and a significant mononuclear cell infiltrate within the ganglia of the myenteric plexus. The ganglionic neuronal population appeared diminished. There was reactive enteroglyal hyperplasia and vascular endothelial cell proliferation within the ganglia. Much collagenous material permeated the ganglia, fasicles and smooth muscle. There was also hyperplasia and oedema of the tunica muscularis. A diagnosis of myenteric ganglionitis and secondary hypoganglionosis was made. At

necropsy the small colon was irregularly distended by ingesta but the remainder of the intestinal tract appeared normal. Histopathological examination of multiple samples of small colon revealed that the acute ganglionitis had significantly subsided but focal areas of nonsuppurative inflammation were still present. The tunica muscularis had reduced slightly in thickness. A mononuclear cell infiltrate in close proximity to the ganglionic nerve cell bodies was discovered in the cranial mesenteric ganglion and several small perivascular cuffs were also observed in the adjacent stroma. Other recognised causes of chronic intestinal pseudo-obstruction syndrome were discussed. The possible aetiology in this particular case was also discussed but not determined.

Another form of intestinal pseudo-obstruction is ileus. The recognition and management were discussed by **Adams (1988)**. Ileus also known as adynamic or paralytic ileus results when the bowel does not function properly due to inhibitory hormonal or neural input. This suppresses propulsive motility or causes incoordination of motility and results in failure of the passage of ingesta. There are a number of causes, most often it occurs following intestinal surgery and may be fatal. The possible mechanisms and sequelae were discussed.

PART I - NECROPSY SERIES

INTRODUCTION

Few studies have been made into the importance of gastrointestinal tract disease either as a cause of death or of subclinical lesions in the horse. It was decided to base such a study on the records of Glasgow University Veterinary School Department of Veterinary Pathology. The post mortem findings of all equines examined in the 5 years 1987-1991 were used to produce the information presented in Part I, the Necropsy Series.

CASE MATERIAL

A search of the Glasgow University Veterinary School Department of Veterinary Pathology records revealed that 213 horses, ponies and donkeys had been fully examined post mortem during the years 1987-91. These animals formed the Necropsy Series under study.

The majority of animals euthanased were shot with a free bullet, but those suspected of having a central nervous system lesion, those killed on the operating table and those killed during the night by the attending clinician received a lethal dose of barbiturates.

METHOD OF STUDY

Archive Material

The necropsy findings of the 213 horses, ponies and donkeys fully examined post-mortem by the Glasgow University Veterinary School Department of Pathology from 1987-1991 inclusive were reviewed. In each case the age, sex, breed, time of year examined post mortem and principal and incidental findings were noted. A full post-mortem examination had been performed by one of the Departmental Pathologists.

Tissue samples, where appropriate, had been taken for histopathological examination. Samples had also been collected for bacteriological and mycological examination and sent to the Bacteriology and Mycology laboratories within the Department of Veterinary Pathology. Samples for parasitological examination were sent to the Department of Veterinary Parasitology. Most of the necropsies, 92 cases, in the period October 1989 - December 1991 inclusive were carried out by the Author.

Necropsy Technique

Horses necropsied by the Author during the period October 1989 - December 1991 inclusive were examined as follows:

The animal was either shot with a free bullet or given a lethal dose of barbiturate. Most were bled out and examined immediately, but those that died or were destroyed during the night were subject to delay.

The external surface, including the eyes, ears, mouth and external genitalia was examined. The animal was suspended by both left legs, and an incision made along ventral mid line from the chin to the anus. The sternum and pelvic symphysis were split. The organs were examined *in situ* and their orientation and anatomical associations noted before being removed as one unit, by cutting through the hyoid apparatus, diaphragm and mesenteric root and around the anus, and laid out on the floor.

The lungs and heart were detached and the thyroid glands removed and sectioned. The trachea and bronchi were opened and this cut was continued down to the tips of the lung lobes. A number of cuts, perpendicular to these, were made into the parenchyma. The pericardial sac was opened and the heart examined by opening each chamber in the pattern of blood flow (right atrium, right ventricle, left atrium, left ventricle).

The liver and spleen were removed and examined and a number of cuts were made into the parenchyma.

The small intestines were cut away from the mesentery at their attachment and the connecting bands which link the caecum colon and small intestines were also cut. The small colon was cut away from its mesentery.

The stomach was opened along its greater curvature then the content and mucoid surface were examined. The entire intestines were opened to

reveal the mucosal surface, again noting quantity, colour, consistency and odour of the content.

The mesenteric lymph nodes were examined and sectioned.

The kidneys, adrenal glands and celiacomesenteric ganglion were dissected from the abdominal fat. The adrenal glands were sectioned. The kidneys were split into two and their capsules removed. The cranial mesenteric artery was opened and its lumenal surface examined. The uterus was opened and the ovaries or testes sectioned where appropriate.

The head was detached. The brain and pituitary gland were removed. The mandible was detached, then the hard and soft palates and tongue were examined.

In those cases where a spinal lesion was suspected the carcass was split through the vertebral bodies and the spinal cord removed. Samples of peripheral nerve, usually the sciatic nerve, were taken.

In those cases where a locomotor problem was suspected the affected limb was removed and subjected to detailed dissection including splitting of the hoof.

Tissue samples were taken from any lesions encountered and from any organ suspected of being involved in the disease process and processed as follows:

Histopathology

Tissue samples collected for histopathological examination were placed in 10% neutral buffered formalin for 24 - 72 hours, post fixed in corrosive sublimate for 24 - 48 hours and processed to paraffin wax in a Miles Scientific V.I.P. (Vacuum Impregnated Processor) 1000 . Sections 3-5 microns thick were cut on a Leitz 1512 Rotary Microtome and stained with haematoxylin and eosin (see Appendix 1). Selected blocks were recut and sections stained by a variety of special techniques (see Appendix 1).

Gastrointestinal Tract Normal Reference Study

Twenty two horses with problems unrelated to the gastrointestinal tract were chosen to provide a reference library of normal tissue for comparison with tissues taken from cases of gastrointestinal tract disease. Each animal was examined as described previously in the Necropsy Technique and the following tissues were sampled and processed identically to diagnostic samples.

Duodenum - 1m distal to the pylorus

Jejunum - 4m distal to the pylorus

Ileum - 0.75m proximal to the caecum

Caecum

Ventral colon

Dorsal colon

Small colon

Liver

Kidney

Pancreas

Samples of stomach were collected from 4 animals and samples of rectum from 7 animals. Each of these animals has been identified by the letter N after the case number in Appendix 2.

RESULTS - NECROPSY SERIES

During the 5 year period of the study, 1987-1991 inclusive, 213 horses and ponies and 2 donkeys were examined post-mortem at the Glasgow University Veterinary School Department of Veterinary Pathology. Henceforth the term horses will be used to denote all 3 types of equidae. The cases are listed in full in Appendix II with details of signalment and final diagnoses.

The Majority of these horses, 172, (81%) had been presented to the Departments of Veterinary Medicine or Surgery as clinical cases and had either died or been destroyed as a consequence of their clinical problems. The remaining horses, 41, (19%) had been donated to the School alive to provide clinical teaching material, had been involved in a research project or were presented dead for a diagnostic post-mortem examination.

Half of the necropsies (107) were performed during the years 1987-89 with the remainder split equally between 1990 and 1991 (Table 1)

Year	Number of Horses
1987	38
1988	33
1989	36
1990	53
1991	53
TOTAL	213

TABLE 1 The number of necropsies performed each year

Sex

The 213 horses consisted of 105 neutered males, 13 entire males, 91 females and 4 where the sex was not recorded (Table 2)

Sex	Number of Horses
Neutered Male	105
Male	13
Female	91
Not recorded	4
TOTAL	**213**

Table 2 The sex of horses in the Necropsy Series

Age

The Recorded ages ranged from a 5 month foetus to 30 years. For 18 horses the age was not recorded. A total of 99 horses were 0-10 years of age, 78 were 11-20 years of age and 18 were 21-30 years of age. The ages are given in detail in Table 3.

Age (Years)	Number of Horses
> - 1	22
1 - 2	6
3 - 4	22
5 - 6	18
7 - 8	20
9 - 10	11
11 - 12	14
13 - 14	13
15 - 16	29
17 - 18	10
19 - 20	12
21 - 22	18
not recorded	18
TOTAL	**213**

Table 3 The age at Death of animals in the Necropsy Series

Breed

The horses were mostly pleasure animals of a number of different breeds (Table 4). This reflects the horse population in the Glasgow Area. Eighty eight were Thoroughbreds or Thoroughbred-type and 85 were ponies. In addition there were 10 heavy horses, 9 European warm bloods, 6 Arabs or Arab crosses, 12 miscellaneous breeds, both horses and ponies, 2 donkeys and only one animal where the breed was not recorded.

Breed	Number of Horses	Number in Group
Thoroughbred	35	
Thoroughbred Cross	24	88
Riding Horse	29	
Pony (Unspecified)	25	
Riding Pony	21	85
Native Pony	39	
Heavy Horse	10	
European Warm Blood	9	
Arab, Arab Cross	6	
Donkey	2	
Miscellaneous	12	
Not Recorded	1	
TOTAL	213	

TABLE 4 The breeds of the horses in the Necropsy Series

System Involvement

The number and percentage of cases with principal and incidental lesions in each organ system is detailed in Table 5. A principal lesion was determined as one which either caused, or played a major role, in the animal's death or destruction. An incidental lesion was determined as

one which either did not contribute to the animals demise or was one component of a disease syndrome.

System	Number of Cases		% of the 213 Cases	
	Principal	(Incidental)	Principal	(Incidental)
Gastrointestinal	90	(79)	42	(37)
Musculoskeletal	53	(11)	25	(5)
Respiratory	24	(10)	11	(4)
Nervous	15	(4)	7	(2)
Hepatic	14	(23)	7	(11)
Cardiovascular	10	(20)	5	(9)
Haemopoietic	9	(2)	4	(1)
Upper alimentary	8	(21)	4	(9)
Integumentary	6	(12)	3	(6)
Reproductive	5	(3)	2	(1)
Endocrine	3	(11)	2	(5)
Urinary	2	(13)	1	(6)
Miscellaneous	17	(3)	8	(1)
No Diagnosis	4		2	
TOTAL	**260**	**(212)**	**123**	**(97)**

TABLE 5 System involvement by the number and percentage of the 213 horses in the Necropsy Series with a principal and (incidental) lesion of each system.

The total number of principal and incidental lesions (318 and 244 respectively) and the cases recorded with a principal lesion (260) is in excess of the total number of horses examined. This is because many had principal and/or incidental lesions in a number of systems or multiple lesions in a single system. The individual cases in which each principal and incidental lesion was encountered are detailed in Appendix III grouped according to the system involved.

The gastrointestinal (GI) system was by far and away the most frequently involved system, both in terms of the number of individual lesions and the number of cases with such lesions. The musculoskeletal system was

the second most frequently involved and the respiratory system the third.

The remaining systems were all represented to a lesser extent. Three had mainly principal lesions ie. the nervous, haemopoietic and reproductive systems and 6 had mainly incidental lesions ie. the hepatic, cardiovascular, upper alimentary, integumentary, endocrine and urinary systems. There was also a group of miscellaneous conditions and 4 cases in the principal findings group where no diagnosis was reached.

Gastrointestinal System

The principal and incidental causes of GI tract disease and the number of cases involved are listed in Table 6. A number of cases were considered to have more than one principal and/or incidental lesion, hence the total number of principal and incidental lesions is greater than the number of horses in these groups.

Condition	Number of Cases	
	Principal	(Incidental)
Peritonitis	25	(5)
Displacement/obstruction	22	
Grass sickness	22	
Enteropathy	20	(1)
Acute regional inflammation	11	(5)
Perforation/rupture	15	
Regional ulceration	13	(29)
Parasitism	7	(35)
Other	3	(5)
Total Occurances	**138**	**(80)**
Total horses	**90**	**(79)**

TABLE 6 The gastrointestinal system: The lesions found in 213 horses and the number of cases in which each lesion was a principal or incidental finding.

Eight conditions and a small group of miscellaneous lesions were identified. Six of these conditions, peritonitis, displacement/ obstruction, grass sickness, enteropathy, acute inflammation and perforation/rupture, were broadly of principal importance. The remaining 2 conditions, ulceration and parasitism, being responsible for the majority of the incidental lesions and only a small proportion of the principal ones. The group of miscellaneous conditions accounted for the remaining principal and incidental lesions.

These conditions are all examined in detail in Results, Chapter II Tables 19 - 35.

Musculoskeletal System

The principal and incidental conditions causing musculoskeletal system disease and the number of cases of each are listed in Table 7. Two cases had 2 principal lesions, so were counted twice. Most of the lesions in this system were considered to be principal.

Condition	Number of Cases	
	Principal	(Incidental)
Joint subluxation/ degenerative joint disease	12	(1)
Laminitis	9	(5)
Fractured limb	9	
Tendon rupture/inflammation	4	
Spinal subluxation	3	
Discospondylitis	3	
Osteomylitis	3	
Fractured spine	2	
Vertebral malformation	2	

Botulism	2	
Osteochondritis dissecans	1	
Angular limb deformity	1	
Discospondylosis	1	
Acute myositis	1	
Postoperative myopathy	1	
Navicular disease		(2)
Osteoporosis		(2)
Sarcocystis		(1)
Muscle abscess		(1)
Total occurrences	**54**	**(12)**
Total horses	**53**	**(11)**

TABLE 7 The musculoskeletal system: The lesions found in 213 horses and the number of cases in which each lesion was a principal or (incidental) finding.

The most frequently recorded principal condition was a combination of joint subluxation and degenerative joint disease, where there were 12 instances. It was also noted as an incidental finding once. Laminitis and fracture of a limb were both recorded 9 times as principal lesions. Laminitis was also present as an incidental finding in 5 cases where the animals were destroyed for another reason.

A further 12 separate conditions were responsible for the remaining 24 principal lesions and another 4 conditions for the remaining 5 incidental lesions.

Of the 64 cases with a lesion involving the musculoskeletal system, 19 also had incidental GI tract lesions, and 2 also had principal GI tract lesions. Eleven were selected for the GI tract normal reference study.

Respiratory System

The principal and incidental respiratory system lesions and the number of cases involved are listed in Table 8. Two cases each had 2 separate lesions so each was counted twice. Most of the lesions in this system, were considered to be principal rather than incidental.

Condition	Number of Cases	
	Principal	(Incidental)
Chronic obstructive pulmonary disease	9	(2)
Strangles	4	
Acute exudative pneumonia	2	
Laryngeal paralysis	2	
Pneumonia	1	(5)
Inhalational pneumonia	1	
Parasitic pneumonia	1	
Asphyxiation	1	
Pleurisy	1	
Guttural pouch empyema	1	
Guttural pouch mycosis	1	
Nasal polyp	1	
Hydatidosis		(2)
Sinusitis		(1)
Bronchiolitis		(1)
Total occurrences	**25**	**(12)**
Total horses	**24**	**(10)**

TABLE 8 The respiratory System:The lesion found in 213 horses and the number of cases in which each was a principal or (incidental) finding.

The most frequently recorded condition was chronic obstructive pulmonary disease. This occurred as a principal finding 9 times and an incidental finding twice. Strangles was noted 4 times and acute exudative pneumonia and bilateral laryngeal paralysis twice each. The latter 2 cases are also included with the hepatic system because although liver failure was the primary ie principal lesion it was the laryngeal

paralysis which led to the demise of both animals so was also a principal lesion. Pneumonia was a principal finding in one case and an incidental finding in 5. A further 10 conditions accounted for the remaining 7 principally and 4 incidentally involved cases.

Of the 34 cases in this group 5 also had an incidental GI tract lesion, 3 had principal and incidental GI tract lesions, and one had a principal lesion of the GI tract. Eight were selected for the GI tract normal reference study.

Nervous System

The principal and incidental nervous system conditions and the number of cases involved, excluding the 22 cases of grass sickness which are included with the GI system, are listed in Table 9. Each animal is only represented once. Most of the conditions were principal rather than incidental.

Condition	Number of Cases	
	Principal	(Incidental)
Spinal cord compression	8	
Cerebral oedema	1	(2)
Ocular lesion	1	(1)
Cauda equine neuritis	1	
Peripheral neuropathy	1	
Chronic adhesive meningitis	1	
Multifocal encephalitis	1	
Vagosympathetic transection	1	
Brain abscess		(1)
Total occurrences	15	(4)
Total horses	15	(4)

TABLE 9 The nervous system: The lesions found in 213 horses and the number of cases in which each lesion was a principal or (incidental) finding.

The most frequently recorded condition was spinal cord compression, noted 8 times as a principal lesion. All but one of these cases is also listed with the musculoskeletal system because of a vertebral lesion. The remaining case (91) is also listed with the integumentary system because it was caused by metastasis from a malignant melanoma.

There were one principal and 2 incidental occurrences of cerebral oedema, one principal and one incidental ocular lesion and 5 single principal and one single incidental lesions.

Six of the 19 cases in this group also had a single incidental GI tract lesion, 5 were selected for the normal GI tract reference study.

Hepatic System

The principal and incidental hepatic conditions and the number of cases involved are listed in Table 10. In this system there were more incidental than principal lesions, and one case had 3 incidental lesions, cirrhosis, haemosiderosis, and hepatic hydatidosis and one case had 2 incidental lesions namely cholangitis and haemosiderosis.

Condition	Number of Cases	
	Principal	(Incidental)
Fatty degeneration	8	(4)
Cirrhosis	2	(5)
Haemochromatosis/ haemosiderosis	1	(9)
Ragwort poisoning	1	(1)
Cholangitis/cholangio-hepatitis	1	(1)
Liver failure	1	
Parasitic hepatitis/cysts		(5)
Hepatic calcification		(1)
Total occurrences	**14**	**(26)**
Total horses	**14**	**(23)**

TABLE 10 The hepatic system: The lesions found in 213 horses and the number of cases in which each lesion was a principal or (incidental) finding.

The most frequently recorded condition was fatty degeneration. This was noted as a principal finding 8 times and as an incidental finding 4 times. Five of the 8 cases were clinically hyperlipaemic and also listed in the "other" category. Cirrhosis was found as a principal lesion twice and incidentally 5 times. Haemochromatosis or haemosiderosis was also predominantly an incidental finding being recorded 9 times as such but only once as a principal finding. Ragwart poisoning (*Senecio Jacobea*) and inflammation were each represented once in both categories and liver failure of undetermined aetiology once on the principal category. Parasitic lesions and calcification, probably also of parasitic origin, accounted for the remaining 6 incidental lesions.

Of the 37 cases in this group 28 also had lesions in the GI tract. There were 14 cases with only principal lesions, 8 with only incidental lesions and 6 with both. Only 3 cases from the GI tract normal reference study were selected for the hepatic system group.

Cardiovascular System

The principal and incidental cardiovascular system conditions and the number of cases involved are listed in Table 11. These were predominantly incidental lesions but one case had 2 principal lesions thrombosis and thromboembolism.

Condition	Number of Cases	
	Principal	(Incidental)
Acute heart failure	4	
Arteritis	2	
Cranial mesenteric arterial thrombosis/aneurysm	1	(12)
Thromboembolism	1	
Ruptured chordae tendinae	1	
Patent ductus arteriosus	1	
Aortic insufficiency	1	
Myocardial infarction	1	(6)
Aortic mineralisation		(1)
Thrombophlebitis		(1)
Total occurrences	**11**	**(20)**
Total horses	**10**	**(20)**

TABLE 11 The Cardiovascular system: The lesions found in 213 horses and the number of cases in which each lesion was a principal or (incidental) finding.

Acute heart failure accounted for 4 of the principal lesions and arteritis for 2. One of the cases of arteritis was due to guttural pouch mycosis and is also listed with the respiratory system and the other (205) had led to ischaemia of the colon and peritonitis so is also listed with the GI system. Damage to the cranial mesenteric artery in the form of either thrombosis or aneurysm was noted as an incidental finding 12 times and a principal finding once. These lesions were attributed to large strongyle activity (*Strongylus vulgaris*) and are also listed with the GI system under parasitism. Ruptured chordae tendinae, a patent ductus arteriosus and aortic insufficiency accounted for the 3 remaining principal lesions and myocardial infarction, aortic mineralisation and thrombophlebitis for the 7 remaining incidental lesions.

Of the 20 horses in this group 6 had incidental lesions, 2 had principal lesions and 10 had both principal and incidental lesions of

the GI tract. There were 4 animals which were selected for the GI tract normal reference study.

Haemopoietic System

The principal and incidental haemopoetic system conditions and the number of cases involved are listed in Table 12. These conditions were largely principal in nature with each case having only one principal or incidental lesion.

Condition	Number of Cases	
	Principal	(Incidental)
Lymphosarcoma	6	
Splenic abscess	1	(1)
Anaemia	1	(1)
Multiple myeloma	1	
Total occurrences	**9**	**(2)**
Total horses	**9**	**(2)**

TABLE 12 The haemopoietic system: The lesions found in 213 horses and the number of cases where each lesion was a principal or (incidental) finding.

Lymphosarcoma was by far the most important condition, noted 6 times. There were 3 cases of alimentary lymphosarcoma, 2 of splenic and one involving a submandibular lymph node in a donkey . There were single principal and incidental recordings of both splenic abscesses and anaemia and a single principal case of multiple myeloma.

Four of the 11 cases in this group also had principal lesions, and one had a principal and an incidental lesion involving the GI tract. Two of

the animals in this group were selected for the GI tract normal reference study.

Upper Alimentary System

The principal and incidental upper alimentary system conditions and the number of cases involved are listed in Table 13. These were predominantly incidental lesions with each case having only one principal or incidental lesion.

Condition	Number of Cases	
	Principal	(Incidental)
Oral squamous cell carcinoma	2	
Abnormal dentition	2	
Ameloblastoma	1	
Cleft palate	1	
Oesophagitis	1	
Stomatitis	1	
Oesophageal ulceration		(18)
Oral ulceration		(2)
Oesophageal papilloma		(1)
Total occurrences	**8**	**(21)**
Total horses	**8**	**(21)**

TABLE 13 The upper alimentary system: The lesions found in 213 horses and the number of cases in which each lesion was a principal or (incidental) finding.

However, 18 of the 21 incidental lesions were oesophageal ulceration, with all but 2 being cases of grass sickness. The other 3 incidental lesions were cases with oral ulceration or an oesophageal papilloma. The principal lesions consisted of 2 oral squamous cell carcinomata, 2 cases with abnormal dentition and single examples of ameloblastoma, cleft palate, oesophagitis and stomatitis.

In this group, in addition to the 16 cases of grass sickness and their associated principal and incidental GI tract lesions there were also 2 cases with a principal, and one case with an incidental and a principal GI tract lesion. Only one of the cases from the GI tract normal reference study was included in this group.

Integumentary System

The principal and incidental integumentary system conditions and the number of cases involved are listed in Table 14.

Condition	Number of Cases	
	Principal	(Incidental)
Melanomata	5	(3)
Dermatitis	1	(1)
Sweet itch		(2)
Photosensitisation		(2)
Papillomata		(1)
Sarcoid		(1)
Dermatophilosis		(1)
Sebaceous cyst		(1)
Total occurrences	6	(12)
Total horses	6	(12)

TABLE 14 The Integumentary system: The lesions found in 213 horses, the number of cases in which each lesion was a principal or (incidental) finding and the number of cases involved.

There were 12 incidental and 6 principal conditions, and each case was only included once. Melanomata accounted for 3 incidental and 5 principal lesions. The 5 principal cases were all malignant with evidence of metastasis.

Dermatitis was noted twice, once principally and once incidentally. Six further conditions accounted for the remaining 8 incidental lesions.

Of the 18 cases in this group 3 had incidental lesions, 3 had both principal and incidental lesions and one had a principal lesion of the GI tract. Only 2 cases from the GI tract normal reference study were included in this group.

Reproductive System

The principal and incidental reproductive system conditions are listed in Table 15.

Condition	Number of Cases	
	Principal	(Incidental)
Inguinal hernia	2	
Vaginal tear	1	
Dystocia	1	
Abortion	1	
Inflammatory hydrocoel		(1)
Scrotal abscess		(1)
Endometritis		(1)
Total occurrences	**5**	**(3)**
Total horses	**5**	**(3)**

TABLE 15 The reproductive system: The lesions found in 213 horses and the number of cases in which each lesion was a principal or (incidental) finding and the number of cases involved.

There were 5 principal and 3 incidental lesions and each case had only one principal or incidental lesion. Inguinal hernia accounted for 2 of the principal cases which were both also included with the GI tract, as was the case with a vaginal tear. There were also single principal cases of dystocia and abortion. The 3 incidental lesions consisted of an inflammatory hydrocoel, a scrotal abscess and endometritis.

Of the 7 cases in this group 3 had principal lesions and 2 had both principal and incidental lesions of the GI tract. No cases in the GI tract normal reference study had a reproductive tract lesion.

Endocrine System

The principal and incidental endocrine system conditions and the number of cases involved are listed in Table 16. One case had incidental pituitary and thyroid nodular hyperplasia. There were only 3 principal conditions, all of which were pituitary adenomas which had resulted in Cushing's Syndrome so these cases are also listed in the "other" category. Pituitary adenomas or nodular hyperplasia were also recorded 5 times as an incidental finding. The remaining 6 incidental lesions consisted of 3 cases of thyroid nodular hyperplasia and one each of thyroid adenoma, thyroid cyst and phaeochromocytoma.

Condition	**Number of Cases**	
	Principal	**(Incidental)**
Pituitary adenoma/hyperplasia	4	(5)
Thyroid nodular hyperplasia		(3)
Thyroid adenoma		(1)
Thyroid cyst		(1)
Phaeochromocytoma		(1)
Total occurrences	**3**	**(12)**
Total horses	**3**	**(11)**

TABLE 16 The endocrine system: The lesions encountered in 213 horses and the number of cases in which each lesion was a principal or (incidental) finding.

Of the 14 cases in this group 5 also had a single GI tract incidental lesion and one had both a principal and an incidental GI tract lesion. This group also included 2 horses which were selected for the GI tract normal reference study.

Urinary System

The principal and incidental urinary tract conditions and the number of cases involved are listed in Table 17.

Condition	Number of Cases	
	Principal	(Incidental)
Ruptured Bladder	1	
Renal necrosis	1	
Renal infarction		(7)
Cystitis		(3)
Renal abscessation		(2)
Klossiellosis		(1)
Interstitial nephritis		(1)
Hypercalcaemic nephropathy		(1)
Total occurrences	**2**	**(15)**
Total horses	**2**	**(13)**

TABLE 17 The urinary system: The lesions found in 213 horses and the number of cases in which each lesion was a principal or (incidental) finding.

There were only 2 principal conditions, one case with a ruptured bladder and one with renal necrosis due to salmonellosis. There were 13 cases with 15 incidental lesions because one case had 3 separate lesions. There were 7 cases of renal infarction, 3 of cystitis, 2 of renal abscessation and one each of klossiellosis, interstitial nephritis and hypercalcaemic nephropathy.

This group of 15 cases included 5 cases with principal GI tract lesions, 3 with incidental GI tract lesions and 2 with both principal and incidental GI tract lesions. It also includes one case selected for the GI tract normal reference study.

Other Conditions

The other principal and incidental conditions and the number of cases involved are listed in Table 18. All but 2 were principal findings.

Condition	Number of Cases	
	Principal	(Incidental)
Hyperlipaemia	5	
Cushing's Syndrome	4	
Salmonellosis	3	
Diaphragmatic rupture	2	
Septicaemia	2	(1)
Poorly differentiated tumour	1	
Haemorrhagic pancreatitis		(1)
Sialoadenitis		
Total occurrences	**17**	**(3)**
Total horses	**17**	**(3)**

TABLE 18 The other lesions encountered and the number of cases in which each was present as a principal or (incidental) lesion.

The 3 incidental conditions were single cases with septicaemia, haemorrhagic pancreatits and sialoadenitis. The principal conditions consisted of 5 cases of hyperlipaemia and 4 cases of Cushing's Syndrome already recorded with the hepatic and endocrine systems respectively. There were also 3 cases of salmonellosis, 2 cases of diaphragmatic rupture and one case with a poorly differentiated tumour which have all been included in the Gi tract list. The final 2 principal cases were of septicaemia.

Of this group of 20 cases, 7 also had a single incidental GI tract lesion, 5 had principal GI tract lesions and 3 had both principal and incidental GI tract lesions. No horses from the GI tract normal reference study group were present in this category.

No Diagnosis

There was a total of 4 cases in which no principal diagnosis was reached although 2 had intestinal ulcertion as an incidental finding.

DISCUSSION - NECROPSY SERIES

Few large studies of the post-mortem findings in horses have been made and these that have tended to concentrate on one particular type of lesion eg. neoplastic (**Cotchin and Baker-Smith 1975**), parasitic (**Lyons et al 1984**).

There is only one truly comparable study in the published literature. In that study **Baker and Ellis** presented their findings in two parts, cause of death (**Baker and Ellis 1981a**) and incidental findings (**Baker and Ellis 1981b**). This study was much larger than the present Necropsy Series comprising 480 horses examined over 22 years. However like the Necropsy Series, it was not possible to determine the relevance of the results in terms of the general population. There were some basic differences between the **Baker and Ellis** series and this Necropsy Series in the groupings chosen and the group into which some lesions were placed. **Baker and Ellis** had one large alimentary tract group. In the Necropsy Series it was decided to separate this group into the upper alimentary system, gastrointestinal system and hepatic system. The Necropsy Series concerned gastrointestinal pathology in particular and the subdivision allowed lesions of that system to be studied separately. Strictly speaking the upper alimentary tract extends to the junction of the squamous and glandular regions of the equine stomach (**Head 1976**) but for the purpose of this study it was considered to terminate at the cardiac sphincter. **Baker and Ellis** placed the cases of grass sickness in the nervous system group (**Baker and Ellis 1981a**). In the Necropsy Series grass sickness was considered a gastrointestinal lesion and was placed in that group.

In the Necropsy Series therefore there were 12 system groups plus a miscellaneous group for both the principal and incidental sections with the addition of "no diagnosis" in the principal and incidental section. **Baker and Ellis** used 8 groups plus "others" and no diagnosis for the "cause of death" series (**Baker and Ellis 1981a**) and 9 groups plus "others" and "no lesions other than cause of death" for the "incidental lesion series" (**Baker and Ellis 1981b**).

The accuracy of any retrospective study is a function of the quantity and quality of available information. This Necropsy Series was no exception so only animals on which full post-mortem examinations had been carried out were included. Approximately half the necropsies were performed by the author but the remainder were shared between 6 different pathologists all of slightly differing attitudes and experience, therefore, some inconsistency was inevitable. Also, each pathologist, while performing a full post-mortem examination, takes special interest in certain systems or types of lesion describing the tiniest changes in one system in detail but perhaps not considering them worthy of mention in other systems. Alternatively when an unusual or novel lesion was encountered, the relevant tissue of all subsequent animals was examined in depth whereas prior to this the tissue might not have been studied if there was no clinical indication of a lesion there. The prime example of this was the routine examination of pituitary glands after a case of Cushing's disease had been encountered. This revealed a number of adenomata and hyperplasias of the pars intermedia which were clinically silent.

Very few of the animals in the necropsy series died, most were destroyed on humane grounds. This meant that the lesions encountered were assumed to be significant because they would account for the clinical signs which ultimately led to the animal's demise although they were not actually directly fatal. Again the designation incidental was subjective being based on the appearance of the lesion and the lack of reported clinical signs which might have been associated with such a lesion.

The results of the series are further slanted by the fact that particular conditions were actively sought by members of staff, both clinicians and pathologists, for their own interest. These conditions were therefore over represented in the Necropsy Series. The 2 main examples were chronic obstructive pulmonary disease (COPD) and causes of chronic diarrhoea and weight loss. Some orthopaedic conditions were under represented because prior to October 1989 in some cases the affected limb was removed post mortem and returned to the clinician on request. A full post-mortem examination was not performed so these cases were excluded from the Necropsy Series. This contributed in a small way to the marked increase in the total number of animals examined in the years 1990 and 1991 which was not necessarily a reflection of an increase in the local horse population but was due to a number of "internal" factors within the Veterinary School. New clinicians attracted more clinical cases so there was a corresponding increase in the number of animals presented for post-mortem examination. As already mentioned all animals presented for post-mortem examination after 1st October 1989 were examined fully and included in the Necropsy Series without exception.

No attempt has been made to compare accurately the clinical details of the horses in the necropsy series to that of the hospital or general population.

The sexes were roughly equally represented, 118 male: 91 female. Only 13 of the males were entire. Almost half the animals, where the age was recorded, were over 10 years old, the actual figure being 96 out of 195. This was a reflection of the local horse population, which was comprised almost exclusively of pleasure animals kept into old age, but in stark contrast to the ages of the animals studied by **Baker and Ellis (Baker and Ellis 1981a, 1981b)**. Almost all their cases were less than 10 years old and 25% were less than one year old.

The pleasure animal nature of the population was again highlighted by the diverse variety of breeds represented and the high proportion of ponies. Because the animals were all cases at a referral centre economic constraints also played a part in distorting the results. Less valuable animals were less likely to be referred for expensive treatment without a guarantee of success. Ponies were therefore probably under represented.

When the frequency of system involvement was considered as a whole the findings were broadly similar to those of **Baker and Ellis (Baker and Ellis 1981a, 1981b)** with the gastrointestinal system being by far the most frequently involved system in terms of both principal and incidental lesions. In many cases multiple lesions were present. These will be discussed in full in Part II, the Gastrointestinal series.

The importance of gastrointestinal tract disease may have beeen over emphasised in this survey because of one clinician's special interest in chronic diarrhoea and cyathostomiasis. This meant that animals were admitted as clinical cases to Glasgow University Veterinary School when they might otherwise have been referred elsewhere or been destroyed on economic grounds by the referring veterinary surgeon.

The musculoskeletal system lagged some way behind in terms of both principal and incidental lesions falling to sixth equal place for the latter. This was probably an underestimate of the importance of lesions of this system because no clinician had a special interest and as explained earlier, some cases in this group presented prior to October 1989 were excluded from the Necropsy Series because a full post mortem examination was not performed. In addition the local horse population contained a low proportion of racing and competitive animals, prone to such problems.

Although the respiratory system lagged behind still further in third place the total number was probably higher than it should have been because cases of chronic obstructive pulmonary disease (COPD), not usually a fatal disease, were actively sought for post-mortem examination by one of the pathologists. This view appears to be borne out by the fact that **Baker and Ellis** placed the respiratory system in fourth place behind the nervous system (**Baker and Ellis 1981a**). The nervous system occupied fourth place in the Necropsy Series. However, in **Baker and Ellis's** series the cases of grass sickness were included with the nervous system rather than the alimentary system. If the 22 cases of grass sickness in the Necropsy Series had been transferred to

the nervous system, that system would have moved to third place but the gastrointestinal system would still have remained on top. Very few incidental lesions were found in the respiratory and nervous systems whereas **Baker and Ellis** placed the respiratory system in third place in terms of incidental lesions (**Baker and Ellis 1981b**)

Interestingly, the hepatic system came an extremely close fifth in terms of principal lesions in the Necropsy Series and second in terms of incidental lesions. **Baker and Ellis** placed lesions of the liver with those of the alimentary system (**Baker and Ellis 1981a, 1981b**). Had they considered the hepatic system separately it would have been in eighth and fourth place in their principal and incidental tables respectively. The latter placing would largely have been due to hydatidosis.

The cardiovascular system was the next most frequently affected by principal lesions lying in sixth place but attaining fourth place in terms of incidental lesions. **Baker and Ellis** found the position to be reversed placing the cardiovascular system fourth in the principal lesion table and sixth in the incidental lesion table (**Baker and Ellis 1981a, 1981b**). The haemopoetic system was seventh in the Necropsy Series and sixth in the **Baker and Ellis** series for principal lesions (**Baker and Ellis 1981a**) and thirteenth and seventh (**Baker and Ellis 1981b**) for incidental lesions.

The upper alimentary system came eighth for principal lesions and third for incidental lesions in the Necropsy Series. **Baker and Ellis** included it with the rest of the alimentary system (recording only a small number of lesions) (**Baker and Ellis 1981a, 1981b**). The integumentary system

system occupied ninth and sixth equal place in the Necropsy Series principal and incidental tables. Baker and Ellis placed it in eighth (Baker and Ellis 1981a) and fifth place (Baker and Ellis 1981b) but their series had fewer system groups. The reproductive system came tenth in the principal and eleventh in the incidental table in the Necropsy Series. No lesions of the reproductive system were considered significant by Baker and Ellis (Baker and Ellis 1981a) but it came ninth in their incidental table (Baker and Ellis 1981b).

The endocrine system came eleventh for principal and sixth equal for incidental lesions in the Necropsy Series. Baker and Ellis did not detect any principal lesions in this system (Baker and Ellis 1981a) but the incidental lesions found placed it in eighth place (Baker and Ellis 1981b). Finally the urinary system came twelfth and fifth for principal and incidental lesions in the Necropsy Series. Compared to Baker and Ellis' placing of seventh (Baker and Ellis 1981a) and sixth (Baker and Ellis 1981b). No diagnosis was achieved in 4 cases (2%) in the Necropsy Series compared to 15 cases (3.1%) in the other series (Baker and Ellis 1981a).

It appears that the findings in the Necropsy Series broadly agreed with those in the only comparable series (Baker and Ellis 1981a, 1981b). The different categories chosen made direct comparison difficult in some areas. In general terms the frequency of principal lesions was not proportional to the frequency of incidental lesions in any one system. Only the gastrointestinal system was at the same position for both types of lesion being by far and away the most frequently involved system. At the other end of the tables the reproductive system was only different

by one placing. In contrast there was a 7 place discrepancy in the position of the urinary system and a 6 place discrepancy in the position of the respiratory, nervous and haemopoetic systems. Some systems were more important in terms of principal lesions and others in terms of incidental lesions.

When the lesions of each system were studied in more detail the same general statements could be made with some conditions causing predominantly principal lesions and others incidental lesions. Many animals had more than one lesion present at time of death. This was most marked in animals with involvement of the gastrointestinal system, some having up to 4 different principal or incidental lesions.

Peritonitis was the most frequently encountered principal lesion. Presumably this was because it occurred as a result of a great many other conditions. However in 4 cases the cause was unknown. In a further 3 cases the peritonitis, usually in the form of fibrous adhesions, was considered to be an incidental finding because of the lack of associated clinical signs and in 2 cases the peritonitis was one of the lesions of granulomatous enteritis.

Displacement or obstruction was encountered almost as frequently as peritonitis and in all 24 cases it was considered to be significant but this was a blanket term which included a number of different lesions of both the small and large intestines which are discussed in more detail in Part II, the Gastrointestinal Series.

Grass Sickness was the most frequently recorded single entity even though this was an underestimate of the true incidence as most cases in Scotland are referred to the Royal "Dick" School of Veterinary Studies.

There were almost as many cases which were considered to have an enteropathy but this was used as a very broad term covering a great many different conditions which were examined in great detail in Part III, the Enteropathy Series. In this group the animals all had a history of chronic weight loss and malabsorption. The broad grouping was chosen to allow extensive comparison between the cases which were then subdivided according to the final diagnosis reached. In addition to the 20 horses in which the enteropathy identified was considered significant there was one case where there was no history of weight loss so the lesions identified were classified as incidental. The animal had presented with acute colic and no history of weight loss. Surgical exploration of the abdomen revealed a small intestinal displacement and much devitalised small intestine which was removed. Unfortunately the animal had to be destroyed as a result of uncontrolled post operative haemorrhage. Microscopic examination of the small intestines revealed distortion of the mucosa and inflammatory cell infiltration with a similar appearance to that of the other 20 cases.

Acute regional inflammation and perforation or rupture each accounted for an equal number of incidents of principal disease. However there were fewer cases in the former group as the inflammation often involved more than one region of the gastrointestinal system. There were also an additional 6 instances where the inflammation was considered to be an

incidental finding. Perforation or rupture of a viscus was always significant.

At the bottom of the table the remaining 2 categories of lesion, regional ulceration and parasitism, were both predominantly incidental. This was partly because the regional ulceration group included the gastric ulceration noted in the majority of grass sickness cases. Parasitism, however accounted for the greatest number of incidental lesions but was only considered to have resulted in a significant lesion in 7 cases. A variety of different parasites were incriminated in both groups.

All the cases with a principal or incidental lesion of the gastrointestinal system were studied in greater detail as described in Part II, the Gastrointestinal Series.

In the Musculoskeletal System joint subluxation or degenerative joint disease was the most important cause of lesions. In themselves they were the end result of a number of different problems eg. septic arthritis or trauma. Degenerative joint disease was detected in one animal with no history of lameness so was considered to be an incidental finding. Laminitis was significant in the majority of cases because although it did not kill the animal it did lead to its humane destruction. These were all chronic cases which had not responded to treatment. Laminitis was considered insignificant in the remaining cases where it was either one component of Cushing's disease or not the most significant lesion at the time of death. Fracture of a limb was recorded with surprising frequency considering that the group studied

was a referral population. Some were cracks or chip fractures of small bones which could be repaired under certain circumstances but many were compound fractures of long bones which probably should not have been transported.

The remaining musculoskeletal lesions occurred much less frequently. The cases with tendon lesions were either examples of tendon rupture which was unrepairable or chronic lesions unresponsive to treatment. A total of 5 different spinal lesions were identified. Only one lesion, a fracture, was considered acute and in one case it had resulted in the sudden death of a horse in a fall and in the other sudden death in a foal which had run into a wall. The remaining lesions were more chronic and had resulted in neurological deficits by spinal cord compression. Of the remaining bony lesions osteoporosis was probably the most interesting. It was recorded twice as an incidental lesion in cases of Cushing's disease. Osteoporosis is well recognised in hormonal abnormalities, such as hyperadrenocoricism, in other species (Jones and Hunt 1983) but has not been recorded previously in equine cases. Of the soft tissue lesions the cases of botulism were both from the same outbreak caused by contaminated big bale silage.

Conditions of the Respiratory System were dominated by chronic obstructive pulmonary disease which would not usually be considered fatal. However examples of this condition were actively sought for post-mortem examination by one of the departmental pathologists. Pneumonia which was recorded 5 times as a principal finding was subdivided into 4 groups, acute exudative, inhalation, parasitic and not specified. There were also cases where the pneumonia was not

considered to be significant. Five upper Respiratory System lesions were encountered. The 2 cases of laryngeal paralysis were extremely unusual, both being bilateral and secondary to liver failure. The sinusitis, nasal polyp and guttural pouch lesions were all amenable to treatment. In reality only the single case of asphyxiation was actually fatal in itself.

Lesions of the Nervous System were dominated by cases of spinal cord compression. All but one of these were caused by a spinal lesion so have been mentioned previously. The remaining case was due to the presence of metastases from a melanoma. Of the brain lesions half were significant and half incidental, the latter group includes one of the cases of Cushing's disease which had a fungal abscess. As discussed earlier the cases of grass sickness were not considered with the Nervous System because significant gross and histopathological lesions were found in the Gastrointestinal System so they were included there.

As already stated the Hepatic System was considered separately in the Necropsy Series, not as part of the Alimentary System. Fatty degeneration was the most important lesion and 5 out of the 8 principal cases were clinically hyperlipaemic. The single case of unqualified liver failure was a young animal which had demonstrated clinical evidence of hepatic dysfunction for the majority of its life. No physical hepatic lesion was detected at post mortem examination.

The remaining 6 lesions were predominantly incidental, and included 5 of the 7 cases of cirrhosis. Ragwort poisoning is a commonly recognised cause of chronic liver disease in the horse in Britain (Milne et al

1990) but only 2 cases, one of which was not considered significant at time of death, were identified in the necropsy series. There are a number of possible explanations for this apparent contradiction but as histopathological examination of the liver is required to make a definitive diagnosis it may be that only a presumptive clinical diagnosis is being made causing other types of lesion to be overlooked. Ragwort poisoning was not recorded by **Baker and Ellis (1981a, 1981b)**.

Lesions of the cardiovascular system were either principal or incidental, with only cranialmesenteric arterial thrombosis/aneurysm identified in both categories. The single case in which it was considered significant was the only case in which associated thromboembolism was identified. This had led to infarction and perforation of the large intestine at the pelvic flecture.

Lesions of the haemopoetic system were dominated by neoplasia, predominantly lymphosarcoma which parallels the results of **Baker and Ellis (1981a)**. This was observed despite the low incidence of this condition in the equine (**Priester and Mantel 1971**) and the lack of known causative retroviruses in this species (**Reid and Howie 1992**). Three of the 6 cases were examples of alimentary lymphosarcoma which have been discussed in full in Part III, the Enteropathy Series.

As already stated the upper alimentary system was considered separately from the gastrointestinal system to allow more detailed study of lesions of the latter. Seven different principal lesions were identified in the upper alimentary system and each was of low incidence and significance. Interestingly, all but one of the principal lesions were oral whereas

all but one of the incidental lesions were oesophageal. The most common of these was oesophageal ulceration which was almost exclusively associated with grass sickness. The upper alimentary tract lesions recorded by **Baker and Ellis** were predominantly incidental and more evenly spread between the two sites (**Baker and Ellis 1981a, 1981b**).

Lesions of the integumentary system were dominated by melanomata. There is some debate as to whether these are true neoplasms or just the result of pigment dilution which occurs as part of the ageing process in grey animals (**Coleman and Sutton 1993**). However, 5 cases were considered malignant on the basis of their cellular morphology, the presence of metastases and the fact that all had been the reason for the affected animals humane destruction. A further two neoplasms, papilloma and sarcoid, were recorded only once as incidental findings. This was despite the relatively high incidence of neoplasms of the integumentary system in the horse (**Priester and Mantel 1971**).

The reproductive system lesions were equally divided between the sexes. The principal lesions in the females were all related to parturition whereas in the male they were both examples of inguinal hernia so involved the gastrointestinal system as well.

Lesions of the endocrine system were dominated by pituitary adenoma/hyperplasia which was considered to be a principal finding when a clinical diagnosis of Cushing's disease had been made. All the incidental lesions in the pituitary were identified after the second case of Cushing's disease was found when a survey of equine pituitary

glands was undertaken. These lesions would not have been detected prior to this date.

In the urinary system 2 of the 3 cases of cystitis were identified in animals with an enteropathy, one had granulomatous enteritis and the other eosinophilic enteritis. These were examples of chronic inflammatory bowel diseases and both have been associated previously with inflammatory lesions in other tissues as described here (Lindberg et al 1985).

The remaining group was a conglomeration of a number of specific conditions which did not fit neatly into any other category. Hyperlipaemia is principally a biochemical problem but it induces lesions in the liver so the cases were also listed with the hepatic system. Cushing's disease has been mentioned repeatedly because of the many different lesions which were present. Only the pituitary adenoma was common to all cases but conversely not all pituitary adenomata were associated with Cushing's disease. Salmonellosis, although principally a condition of the alimentary tract, was systemic in 3 cases, hence its inclusion here. Diaphragmatic rupture was associated with intestinal displacement in only one of the 2 cases and no history of trauma was obtained for either. The tissue of origin for the poorly differentiated tumour could not be identified. The tumour had infiltrated a number of organs and had caused intestinal oedema which in turn had led to rectal perforation during routine examination. The animal was immediately destroyed before peritonitis could develop. Acute pancreatitis was identified in a horse which died following surgery to correct a large intestinal displacement and at necropsy an inguinal hernia involving a

portion of small intestine was identified. Although the pancreatitis was considered acute its possible role in the induction of either of the intestinal lesion could not be proved.

Finally, if a broader view is taken and the lesions, rather than their location, are considered all classes of lesion were identified. Infectious disease was poorly represented which was probably a reflection of the fact that the Necropsy Series was based on a referral, not a first opinion, population and involved few young animals. Chronic disease was probably over represented because many of the animals would have been undergoing investigation or treatment for some time before referral. Traumatic lesions appeared only to be of importance in the musculoskeletal system. Neoplasia was identified in 5 systems. It is probably over represented because the incidence of neoplasia in general increases with age in the horse as it does in other species (Reid and Howie 1992). As stated previously almost half the animals in this series were over 10 years old. Conversely however this would have reduced the incidence of benign neoplasia of the integumentary system which did appear to be under represented when compared with other studies (Priester and Mantel 1972) because their incidence decreases with age (Priester 1973)

So, in summary, this Necropsy Series identified the presence of both fatal and incidental lesions in all equine organ systems and identified the gastrointestinal system as the most frequent site of these lesions. In general the findings are in agreement with those of the only other extensive equine necropsy series in the literature (Baker and Ellis 1981a, 1981b).

PART II - GASTROINTESTINAL SERIES

INTRODUCTION

As already stated the purpose of the Necropsy Series was to establish the importance of diseases of the gastrointestinal systam as a cause of death and of subclinical lesions. The gastrointestinal system was found to be the most frequent site of both principal and incidental lesions. These lesions are described more fully in this section, Part II, the Gastrointestinal System.

CASE MATERIAL AND METHOD OF STUDY

The Case Material and Method of study have been described at the beginning of Part I, the Necropsy Series.

RESULTS - GASTROINTESTINAL SERIES

The various different conditions responsible for the 90 cases of principal and 79 cases of incidental gastrointestinal (GI) disease as summarised in Table 6 are examined in greater detail in Tables 19-35. The individual cases in which each principal and incidental GI lesion was found are detailed in Appendix IV grouped by the lesion present.

Peritonitis

The most frequently recorded principal finding was peritonitis (Table 6). This was found in 25 (28%) of the 90 cases with principal GI disease.

Cause	Number of cases Principal	(Incidental)
Gastric rupture	5	
Intestinal leakage	5	
Large Intestinal perforation	3	
Rectal perforation	2	
Small intestinal perforation	1	
Inguinal hernia	1	
Large intestinal necrosis	1	
Sand colic	1	
Ruptured bladder	1	
Splenic abscesses	1	
Granulomatous enteritis		(2)
Unknown	4	(3)
Total occurrences	**25**	**(5)**
Total Horses	**25**	**(5)**

TABLE 19 The most probable cause of the 30 cases of peritonitis and the number of cases in which each cause produced a principal or (incidental) lesion.

The cause (Table 19) was grossly apparent in all but 4 of the cases. The majority of cases also had at least one other principal lesion of the GI system and are also listed under the condition which caused the peritonitis eg displacement/obstruction or perforation/rupture and partly account for the total number of cases of principal GI disease appearing to be 138 not 90.

The most common cause of peritonitis was GI tract rupture, of which there were 10 cases, with post operative intestinal leakage accounting for another 5 cases (Table 19).

There were 3 cases in which peritonitis, usually in the form of old fibrous adhesions, was considered to be an incidental finding and in a further 2 cases it was one of the components of granulomatous enteritis.

Displacement/obstruction

Displacement or obstruction of a portion of the intestinal tract was found in 22 (24%) of the cases with principal GI lesions (Figures 1-4). There was a total of 24 instances because one case had a small intestinal intussusception as well as large intestinal volvulus and one case had volvulus of both the small and large intestines.

Lesion	Number of Cases
Small intestine	
Volvulus/torsion	5
Obstruction	4
Hernia	3
Intussusception	2
Entrapment	1
Large intestine	
Volvulus	5
Entrapment	3
Obstruction	1
Total occurrences	**24**
Total Horses	**22**

Table 20 The types of displacement/obstruction found in 22 horses and the number of cases in which each occurred.

Involvement of the small intestine occurred in 15 cases and involvement of the large intestine in 9 cases (Table 20). Eleven of the 22 cases also had a principal finding of peritonitis and in one case it was an incidental finding.

Grass Sickness

A diagnosis of grass sickness was confirmed in 22 (24%) of the principal GI tract lesions group (Figure 7). In each case histopathological examination of the celiacomesenteric ganglion revealed changes consistent with those of grass sickness.

The number of cases varied greatly from year to year (Table 21) with 8 cases recorded in 1987 and only one in 1989.

Year	Number of Cases
1987	8
1988	6
1989	1
1990	2
1991	5
Total	**22**

Table 21 The number of the 22 cases of grass sickness examined each year.

The majority of cases were examined in May and June (Table 22), with a further 6 in August and September and the remaining 2 in February, both in 1988.

Month	Number of Cases
February	2
March	0
April	0
May	8
June	6
July	0
August	3
September	3

TABLE 22 The number of the 22 cases of grass sickness examined each month.

Examination of the 22 cases of grass sickness in more detail reveals that one principal and 41 incidental GI lesions were also present (Table 23).

Lesion	Number of Cases	
	Principal	(Incidental)
Gastric rupture	1	
Oesophageal ulceration		(16)
Gastric ulceration		(10)
Splenic enlargement		(5)
Cyathostomes		(4)
Typhlitis		(1)
Intestinal ulceration		(1)
Haemomelasma ilei		(1)
Cranial mesenteric arterial thrombosis		(1)
Lipoma		(1)
Total occurrences	1	(40)
No other lesions noted	3	

TABLE 23 The additional principal and (incidental) GI system lesions present in the 22 cases of grass sickness.

The one principal lesion was gastric rupture. The most commonly recorded incidental lesions were oesophageal (16 cases) and gastric (10 cases) ulceration (Figure 6). Parasitism was encountered in 6 cases and splenic enlargement in 5 cases. In 3 cases no other lesions were recorded.

Enteropathy

An enteropathy was found to be a principal lesion in 20 (22%) of the cases with principal GI tract lesions and an incidental finding in one case (10%).

Diagnosis	Number of Cases Principal	(Incidental)
Alimentary lymphosarcoma	3	
Enterotyphlocolitis	3	
Granulomatous enteritis	2	
Eosinophilic enteritis	2	
Cyathostomiasis	2	
Protein-losing enteropathy	1	
Coccidiosis	1	
Granulomatous typhlocolitis	1	
Granulomatous colitis	1	
Typhlocolitis	1	
Enteritis	1	
Lymphoid hyperplasia	1	
Dental exhaustion	1	
Chronic enteropathy		(1)
Total occurrences	**20**	**(1)**
Total Horses	**20**	**(1)**

TABLE 24 The diagnoses in the 21 cases of enteropathy and the number of cases in which each was a principal or (incidental) finding.

In each of the principle cases there was a clinical history of weight loss, and evidence of malabsorption. A number of conditions were identified (Table 24). In 11 cases a definitive diagnosis was achieved; alimentary lymphosarcoma, granulomatous enteritis, eosinophilic enteritis, cyathostomiasis, protein-losing enteropathy secondary to Phenylbutazone toxicity and intestinal coccidiosis. In 9 cases only a morphological diagnosis was made; enterotyphlocolitis, typhlocolitis, granulomatous typhlocolitis, granulomatous colitis, enteritis, lymphoid hyperplasia and dental exhaustion. These 20 cases are studied in depth in results Chapter III.

Acute regional inflammation

Acute regional inflammation was identified as a finding in 11 (12%) of the 90 cases with principal GI tract lesions and 5 (7%) of the 79 with incidental lesions. There were however 15 principal and 6 incidental occurrences of acute inflammation because 2 cases both had enteritis, typhlitis and colitis as a principal finding and one case had typhlitis and colitis as an incidental finding (Table 25).

Diagnosis	Number of Cases	
	Principal	(Incidental)
Colitis	9	(1)
Typhlitis	3	(1)
Enteritis	2	
Gastritis	1	
Diarrhoea		(4)
Total occurrences	**15**	**(6)**
Total Horses	**11**	**(5)**

Table 25 The diagnosis in the 16 cases of acute regional inflammation and the number of cases in which each condition was found as a principal or (incidental) lesion.

By far the most frequently involved region was the colon with 10 cases of colitis recorded (Figure 8). The 4 cases in which diarrhoea was an incidental finding were also included in this group. Only one of these cases had another gross lesion involving the GI tract, namely ulceration. The principal finding in this case was a fractured limb and in the other case was hyperlipaemia. No samples were taken for histopathological or bacteriological examination to determine the cause in these 4 cases.

Perforation/rupture

Perforation or rupture was identified as a principle finding in 15 (17%) of the cases of principal GI disease. The locations of the lesions are detailed in Table 26.

Location	Number of Cases Principal
Stomach	6
Rectum	4
Caecum	2
Colon	2
Small intestine	1
Total occurrences	**15**
Total Horses	**15**

TABLE 26 The location of the lesions in the 15 cases of intestinal perforation/rupture and the number of cases of each location.

Perforation of the rectum accounted for 4 of the 15 cases. One of the 2 cases of caecal rupture was an animal with a caecal canula. Necrosis of the adjacent caecal wall had resulted in rupture. The other case had occurred at foaling along with a vaginal tear. One of the 2 cases of colonic perforation was secondary to colonic volvulus and the other to arterial thromboembolism and infarction (Figure 10). No cause for the case of small intestinal perforation was found where a single small 1cm diameter hole was present in the duodenum. This had resulted in acute fibrinous peritonitis and adhesion of adjacent loops of bowel.

Cause	Number of Cases Principal
Duodenal obstruction	3
Small intestinal volvulus	1
Grass sickness	1
Hyperlipaemia	1
Total Occurances	6
Total Horses	6

TABLE 27 The most probable cause of the 6 cases of gastric rupture and the number of cases in which each caused a principal lesion.

The 6 cases of gastric rupture are detailed in Table 27. Three were due to duodenal obstruction, 2 of these had a foreign body and one an intussusception (Figure 1). The remaining 3 cases were due to small intestinal volvulus, grass sickness and hyperlipaemia.

Regional Ulceration

Regional ulceration was largely an incidental finding recorded in only 13 (14%) of the 90 cases with a principle GI lesion, but in 32 (40%) of the 79 cases with an incidental lesion (Table 6). It was recorded 37 times as an incidental lesion because 3 cases all had ulceration of two separate regions (Figure 9) (Table 28).

Location	Number of Cases Principal	(Incidental)
Large intestine	9	(10)
Small intestine	4	(6)
Stomach		(21)
Total Occurrences	13	(37)
Total Horses	13	(32)

TABLE 28 The location of the 42 cases of regional ulceration and the number of cases where the lesion was a principal or (incidental) finding at each location.

Ulceration was recorded 9 times as a principal and 10 times as an incidental finding in the large intestine and the associated principal lesions are detailed in Table 29. It was recorded 4 times as a principal and 6 times as an incidental finding in the small intestine and the associated principal lesions are detailed in Table 30.

There were 21 cases in which there was incidental ulceration of the stomach. These are detailed in Table 31.

Diagnosis	Number of Cases of Ulceration	
	Principal	(Incidental)
Ulcerative colitis	1	
Fungal enteritis	1	
Granulomatous typhlocolitis	1	
Acute haemorrhagic typhlocolitis	1	
Cyathostomiasis	1	
Caecal perforation	1	
Protein-losing enteropathy	1	
Necrotising colitis	1	
Enteritis	1	(1)
Colonic volvulus		(3)
Alimentary lymphosarcoma		(2)
Poorly differentiated tumour		(1)
Cirrhosis		(1)
Granulomatous enteritis		(1)
Fractured leg		(1)
Total Occurances	**9**	**(10)**
Total Horses	**9**	**(10)**

TABLE 29 The additional principal lesions associated with large intestinal ulceration and the number of principal or (incidental) cases of ulceration with each additional lesion.

Diagnosis	Number of Cases of Ulceration	
	Principal	(Incidental)
Granulomatous enteritis	2	
Duodenal obstruction	1	
Salmonellosis	1	
Enterotyphlocolitis		(1)
Poorly differentiated tumour		(1)
Osteomyelitis		(1)
Laminitis		(1)
Fractured leg		(1)
Arthritis		(1)

Total Occurrences	**4**	**(6)**
Total Horses	**4**	**(6)**

TABLE 30 The additional lesions associated with small intestinal ulceration and the number of principal or (incidental) cases of ulceration with each additional lesion.

Diagnosis	**Number of Cases of (Incidental) Ulceration**
Grass sickness	(9)
Lymphoid hyperplasia	(1)
Enterotyphlocolitis	(1)
Necrotising colitis	(1)
Eosinophilic enteritis	(1)
Colonic volvulus	(1)
Salmonellosis	(1)
Chronic obstructive pulmonary disease	(1)
Polyarthritis	(1)
Laminitis	(1)
Cushing's Syndrome	(1)
Inconclusive	(1)
Total Occurrences	**(21)**
Total Horses	**(21)**

TABLE 31 The additional principal lesions associated with gastric ulceration and the number of (incidental) cases of ulceration with each additional lesion.

The most frequent cause of gastric ulceration was grass sickness. This accounted for 9 of the 21 cases. Gastric ulceration was recorded as an incidental finding associated with a further 11 different conditions and in one case in which a definitive diagnosis was not reached.

Parasitism

Parasitism was largely an incidental lesion only being recorded 7 times as a principle lesion in 8% of the 90 cases with a principle GI tract lesion, but it was recorded 44 times as an incidental lesion representing 48% of the total 91 occurrences, in 35 (50%) of the 79 cases of incidental GI tract involvement (Figures 6,10-15). Eight cases

in this group had 2 separate lesions and one case 3 lesions indicative of parasitological activity (Table 32).

Parasite/Parasitic lesion	Number of Cases Principal	(Incidental)
Cyathostomes	5	(14)
Cranial mesenteric arterial thrombosis/aneurysm	1	(13)
Eimeria leukarti	1	
Haemomelasma ilei		(12)
Large strongyles		(3)
Ascarids		(2)
Tape worms		(2)
Gasterophilus intestinalis		(2)
Total Occurrences	**7**	**(48)**
Total Horses	**7**	**(39)**

Table 32 The causes of parasitism and the number of cases in which each caused a principal or (incidental) lesion.

The most commonly recorded cause of lesions in this group were the cyathostomes (small strongyles). These were a principal finding in 5 cases and an incidental finding in 14 cases. The time of year the animals were examined is detailed in Table 33.

Month	Number of Cases Principal	(Incidental)
January		
February		(1)
March		(1)
April		
May	1	(5)
June		(2)
July		
August		(1)
September	1	
October		(2)
November	3	(1)
December		(1)
Total Horses	**5**	**(14)**

TABLE 33 The month of the year cyathostomes were identified postmortem in 19 horses and the number of cases in which they were a principal or (incidental) finding.

The spread is quite even throughout the year with no cases recorded in January, April or July but one principal and 5 incidental cases in May, 3 principle and one incidental case in November, one principal case in September, and 2 incidental cases in June, and October and a single incidental case in February, March, July and December. It must be noted that the principal case in September and one of the principal cases in November were both experimentally induced. Two of the principal cases are discussed in greater detail in Part III, the Enteropathy Series.

Large strongyle activity (*Strongylus vulgaris*) as evidenced by cranial mesenteric arterial thrombosis or aneurysm or nodules in the large intestinal wall was noted in 14 cases (Figures 10-12). It was a principal finding in only one of these where ischaema and perforation of the large colon had occurred following thromboembolism from a cranial mesenteric arterial verminous thrombus (Figure 10).

Haemomelasma ilei, an incidental finding in 12 cases, is also believed to result from large strongyle activity (Figure 13a). This lesion did appear to have a seasonal incidence (Table 34) being recorded from October to June with no cases recorded July to September.

Month	**Number of Cases (Incidental)**
October	(2)
November	(1)
December	(1)
January	(5)
February	(0)
March	(1)
April	(1)
May	(0)
June	(1)
Total Horses	**(12)**

TABLE 34 The month of the year *Haemomelasma ilei* was identified postmortem in 12 horses and the number of cases in which it was an (incidental) finding.

Eight of the 12 cases occurred between November 1990 and March 1991 inclusive. The remaining 4 cases were noted in October 1987, June 1988 and April 1990

The other parasites noted in the gastrointestinal system were coccidia, tapeworms, bots and ascarids (Figures 38,15,6 and 14 respectively).

Other

The final group consists of a miscellaneous collection of lesions. These were responsible for 3 (3%) of the 90 cases with principal lesions and 5 (6%) of the 79 cases with incidental lesions. There were single incidents of 3 principal lesions, arteritis, thromboembolism and mesenteric lipoma (Table 35). The latter had caused strangulation of the small colon.

Condition	Number of Cases	
	Principal	(Incidental)
Arteritis	1	
Thromboembolism	1	
Lipoma	1	(4)
Gastric impaction		(1)
Total Occurrences	**3**	**(5)**
Total Horses	**3**	**(5)**

Table 35 The other lesions encountered in 8 horses and the number of cases each was a principal or (incidental) finding.

One case is also recorded with the group of parasitological lesions because the embolus originated from a verminous thrombus in the cranial

mesenteric artery. Of the incidental lesions there were 4 mesenteric lipomata and one case of gastric impaction. The latter was found incidentally at post mortem examination. The animal had been showing signs of hepatic encephalopathy with no indication of GI disease.

DISCUSSION - GASTROINTESTINAL SERIES

A review of the literature failed to reveal any detailed post-mortem studies of equine gastrointestinal system lesions. Limited studies confined to parasitic lesions have been made (Lyons et al 1984) and the gastrointestinal system was examined by Baker and Ellis in their survey of the post mortem findings in 480 horses but the results were not presented in detail (Baker and Ellis 1981a, 1981b).

This study was part of the Necropsy Series involving only the 90 animals with gastrointestinal system lesions so the same limitations prevail ie. it was based on a referral population and certain conditions were over represented. However, the results have been presented in more detail allowing in depth assessment and comparison of factors such as the time of year the post mortem examination was performed and any possible association between lesions.

When the cases of peritonitis were broken down 11 causes were identified, five of these causes, which accounted for 14 of the 24 principal cases, were a form of gastrointestinal perforation and four of the remaining nine causes also involved damage to the intestinal wall (Figures 1,10,11 and 25). Therefore, of the 23 cases in which the cause

of the peritonitis was identified only two were due to nonintestinal lesions namely a ruptured bladder and a splenic abscess. These findings are in agreement with those of a previous study of 30 cases of peritonitis where post mortem examination of the 9 horses which did not survive revealed that severe ulceration and, or, rupture of the intestinal tract was the cause of the peritonitis (Dyson 1983).

The high incidence of displacement/obstruction of the intestines was partly explained by De Boom in his review of the anatomy of the equine alimentary tract where he highlighted the many "design faults" predisposing the horse to such lesions (De Boom 1975) (Figures 1,2,3 and 4). In this series more cases of displacement/obstruction were identified in the small intestines compared with the large intestines which agrees with previous findings (Baker and Ellis 1981a). Interestingly one of the 5 cases of large intestinal volvulus also had a small intestinal volvulus and another had a small intestinal intussusception. Baker and Ellis did not identify any cases of entrapment but this Necropsy Series contained 4, and only one of intussusception which they considered an incidental finding (Baker and Ellis 1981a, 1981b). One form of obstruction common in other countries, such as the United States, but absent from the Necropsy Series was the enterolith. This may be due to factors such as the type of roughage fed and the wet climate in the West of Scotland (Lloyd et al 1987).

Grass sickness was the most frequently identified primary condition despite the fact that it was considered to be more of an east of Scotland problem and most cases were referred to the Royal (Dick) School of Veterinary Studies in Edinburgh for specialist care. This would

suggest that grass sickness was actually under represented in the Necropsy Series. When the 22 cases were examined more closely much variation in the number presented for post mortem examination each year was noted. Eight cases were examined in 1987 compared to only one in 1989. A seasonal pattern emerged with no cases observed November to January and all but 2 of the cases occurred in the five months period May - September. The cases tended to occur in clusters of 2 or 3 in close succession from different premises.

A number of different gross lesions were detected in the majority of cases of grass sickness (Figure 7). The most common of these was oesophageal and/or gastric ulceration (Figure 6) which along with splenic enlargement and gastric rupture have been recorded previously (**Barlow 1969, Platt 1982**). Of the remaining lesions typhlitis and intestinal ulceration may have been directly related to the grass sickness but the parasitic lesions and the lipoma were not.

All but one of the cases of enteropathy has been considered more fully in Part III. The remaining case was considered to be an incidental finding because there was no associated history of weight loss or diarrhoea. Detailed study of the cases in the Enteropathy Series which include extensive histopathological examination, yielded 11 separate diagnoses and one case where no morphological intestinal abnormality was detected. Some of the conditions were uncommon but well recognised in countries other than the U.K. eg. granulomatous enteritis and eosinophilic enteritis (**Lindberg et al 1985**). Other conditions have been reported in the United Kingdom eg. coccidiosis (**Wheeldon and Greig 1977**) and lymphosarcoma (**Wiseman et al 1974**).

Acute inflammation was detected in all portions of the gastrointestinal system but was most common in the colon (Figure 8); it has been suggested that this may simply be a function of its size (**Wingate 1986**). However studies have shown that the colonic mucosa, especially that of the right dorsal colon, is an extremely active tissue both excreting and absorbing various substances, (**Karcher et al 1990**). This suggests some intrinsic differences in the mucosa and, or, osmolarity in this portion of the gastrointestinal tract which might predispose it to toxic insult or injury.

Perforation or rupture was recorded at all levels of the gastrointestinal system. It was most common in the large intestine predominantly because of rectal tears which were likely to have been iatrogenic although there have been reports of noniatrogenic tears (**Slone et al 1982**). The cases of gastric rupture were generally secondary to small intestinal obstruction (Figure 1) as has been recorded previously (**Todhunter et al 1986**). The 2 exceptions were one animal with grass sickness, a disease in which gastric rupture is a recognised complication (**Platt 1982**) and one animal with hyperlipaemia. The latter was interesting when one considers the fact that gastric impaction is known to occur in association with Ragwort poisoning which like hyperlipaemia can result in liver failure (**Milne et al 1990**). The mechanism for obstruction of outflow in these cases is not known.

Again, ulceration was recorded at all levels of the gastrointestinal system but was significant most often in the large intestine and the same predisposing factors of size and activity apply (**Karcher et al**

1990). No common conditions were identified among the 9 cases of significant large intestinal ulceration and 7 separate associated principal lesions were identified in the 10 cases of incidental large intestinal ulceration(Figure 9). The 5 cases with ulceration of two regions all had large intestinal involvement. The small intestinal ulceration was again associated with a number of different principal lesions 4 of which involved the musculoskeletal system rather than the gastrointestinal system. This was also noted in the cases of gastric ulceration (Figure 5) but here almost half were secondary to grass sickness.

Parasitism was the most frequently encountered cause of incidental lesions (Figures 10-15) but it only accounted for a small number of principal lesions, a finding consistent with previous studies (**Baker and Ellis 1981a, 1981b**). More than one sort of parasitism was encountered frequently in the same animal. Two of the three causes of principal lesions, cyathostomes and *Strongylus vulgaris* were also the most common causes of intestinal lesions. In a previous study parasitic infections were usually mixed and nearly all cases had a heavy burden of cyathostomes although the term *Trichonema sp* was used (**Baker and Ellis 1981a**). No seasonal incidence was observed for cyathostomiasis, ie. the co-ordinated mass emergence of small strongyles, even though the condition has been likened to Type II ostertagiasis in cattle in the spring (**Love 1992**).

A seasonal incidence was observed for *Haemomelasma ilei* (Figure 13a) with 5 cases noted in January alone and none July to September inclusive. The lesion is believed to be caused by the migration of

large strongyles, *Strongylus edentatus* in particular (**Baker and Van Dreumel 1985**). This suggests that the seasonal incidence may be due to seasonal exposure to infective larvae. Lesions identical to those of the *H. ilei* were observed from two weeks after experimental infection of foals with *S. vulgaris* (**Duncan and Pirie 1975**). However, the more common and more significant lesion observed with *S. vulgaris* infection is thrombosis of the cranial mesenteric artery (Figures 10,11 and 12) (**Rous 1975, Lyons et al 1987**) which can lead to thrombotic infarction of the intestines (**White 1981**). This was believed to have occurred in one case in this study which was also included in the final group as the case of thromboembolism (Figure 10).

This small group of remaining lesions was dominated by pedunculated lipomata which in one of the 5 affected animals had resulted in strangulation of the small colon. Such lipomata have been considered common incidental findings (**Cotchin and Baker-Smith 1975, Cotchin 1977**) but have been observed previously to strangulate the small colon (**Pascoe and Sellars 1981**) as well as other portions of the intestines (**Mason 1978, Ramey and Reinerston 1984**). **Baker and Ellis** did not record any in their series (**Baker and Ellis 1981a, 1981b**). The single case of gastric impaction, which was considered to be an incidental finding, was in a 6 months old foal with liver failure of unknown aetiology. As discussed already gastric impaction has been recorded in association with liver failure due to ragwort poisoning (**Milne et al 1990**).

In conclusion very many types of lesion were observed in the equine gastrointestinal system. In general a different group of conditions resulted in significant lesions compared to that which caused the

incidental lesions but there was some overlap. Despite the widespread availability of effective anthelmintics, parasitism appeared to be still common and significant disease and death from parasitism was not infrequent. Some conditions, both parasitic and of unknown aetiology, appeared to have a seasonal incidence, while others did not. It was apparent however that disease affecting the equine gastrointestinal system occurred throughout the year in animals of all ages and breeds.

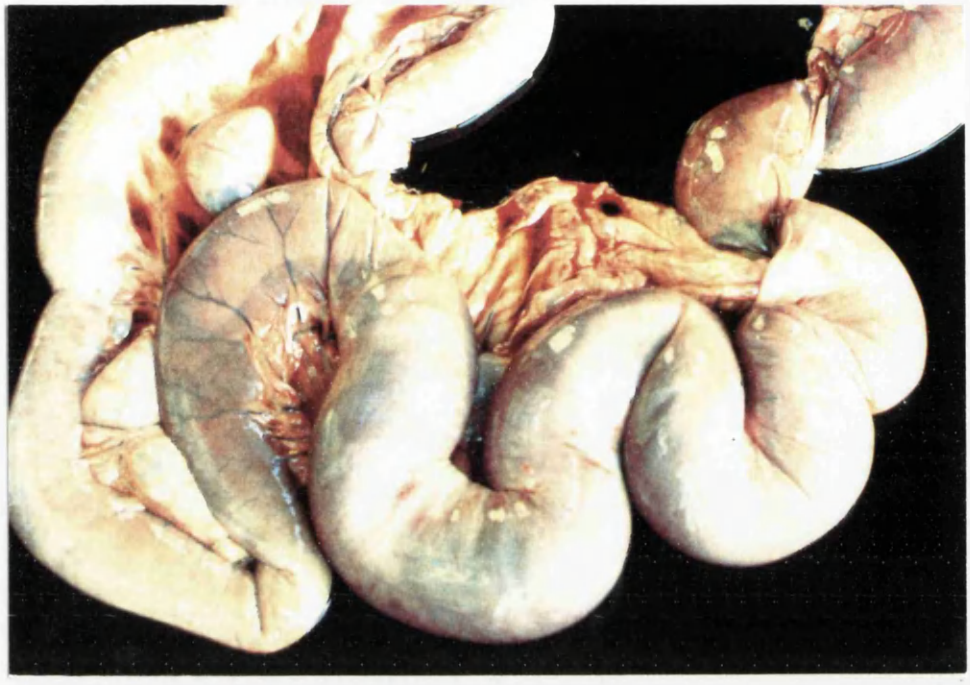
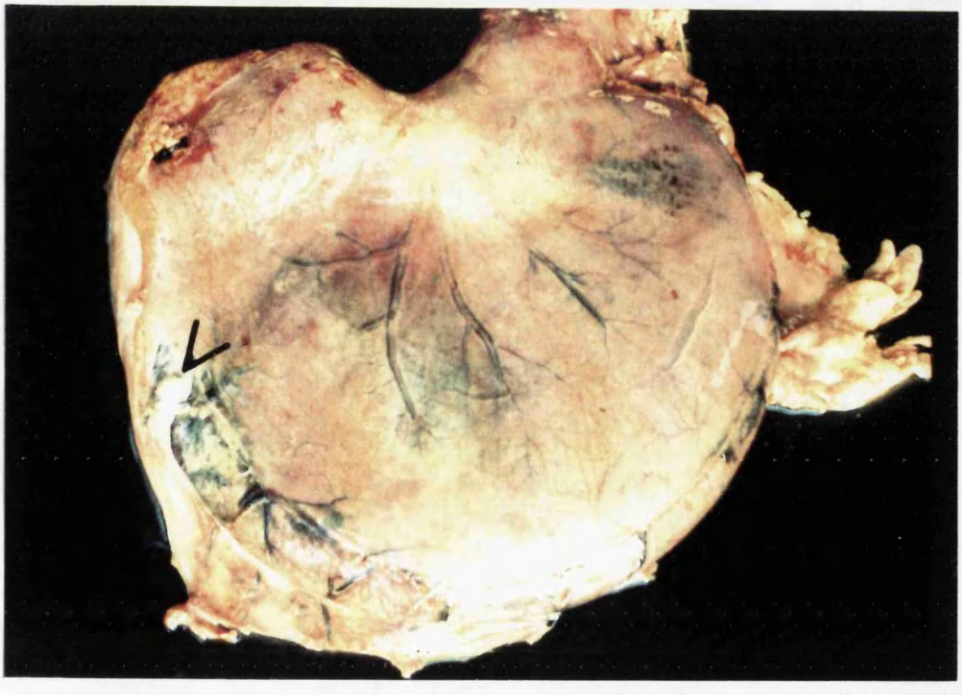


Fig.1 Case No. 166. A. Jejunal intussusception has led to obstruction. There is dilatation of the proximal intestine, on the right, and gastric rupture (B).



B. Gastric rupture. The muscle lesion along the greater curvature is extensive but the mucosal perforation is small (Arrow).

Fig. 2 Case No. 109. Obstructed fluid filled loops of small intestine secondary to a torsion at the mesenteric root (Arrow).

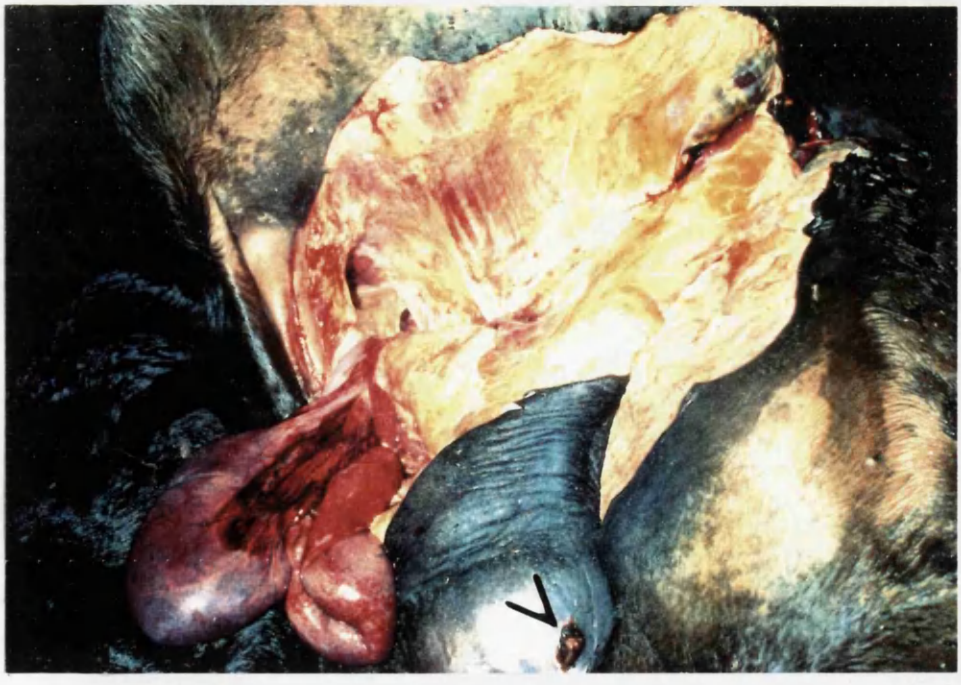


Fig. 3 Case No. 207. Inguinal hernia. The left hind leg is raised and the left inguinal canal opened to reveal two loops of incarcerated small intestine. The crusty lesion in the scrotal skin is overlying an abscess (Arrow).

Fig. 4 Case No. 191. The small intestinal mesenteric tear (*) has been partially repaired. The dilated loops of intestine are due to post operative ileus.

Fig. 5 Gastric ulceration. There is deep ulceration of the squamous and glandular portions and scarring at the pyloric sphincter.

Fig. 6 Grass sickness. Superficial ulceration of the squamous portion of the stomach and *Gasterophilus intestinalis* larvae.

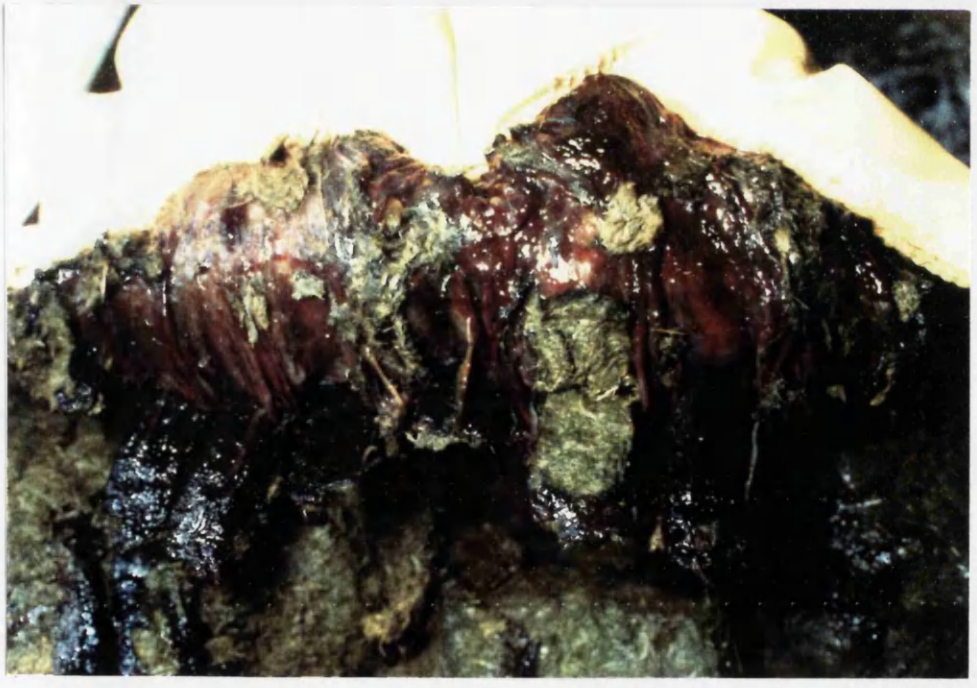


Fig. 7 Grass sickness. The colon has been opened to reveal the red-brown sticky mucus attaching the mucosa to the extremely dehydrated content.

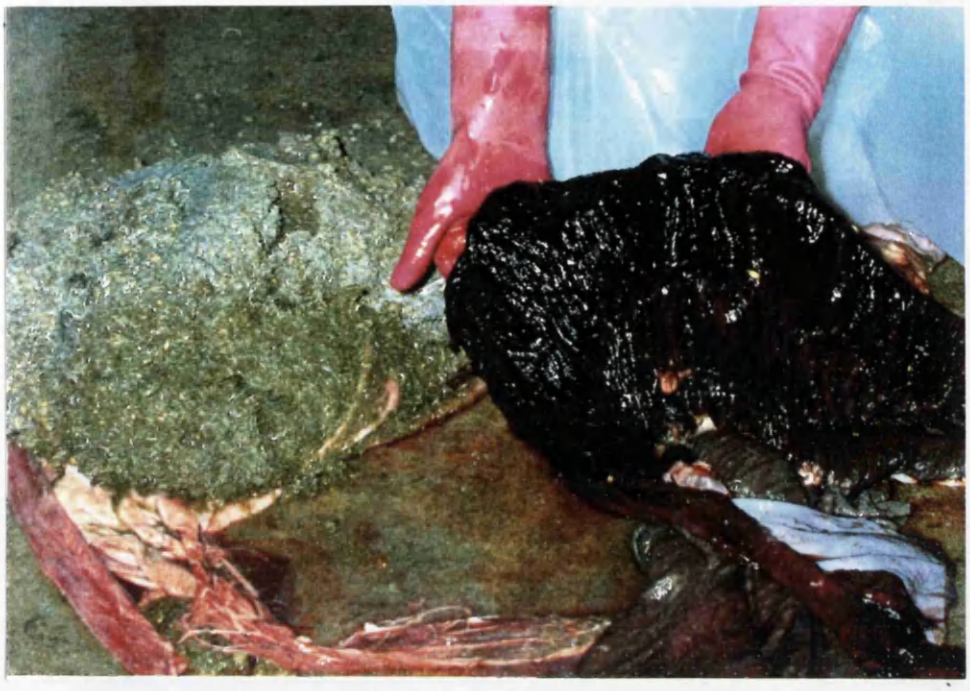
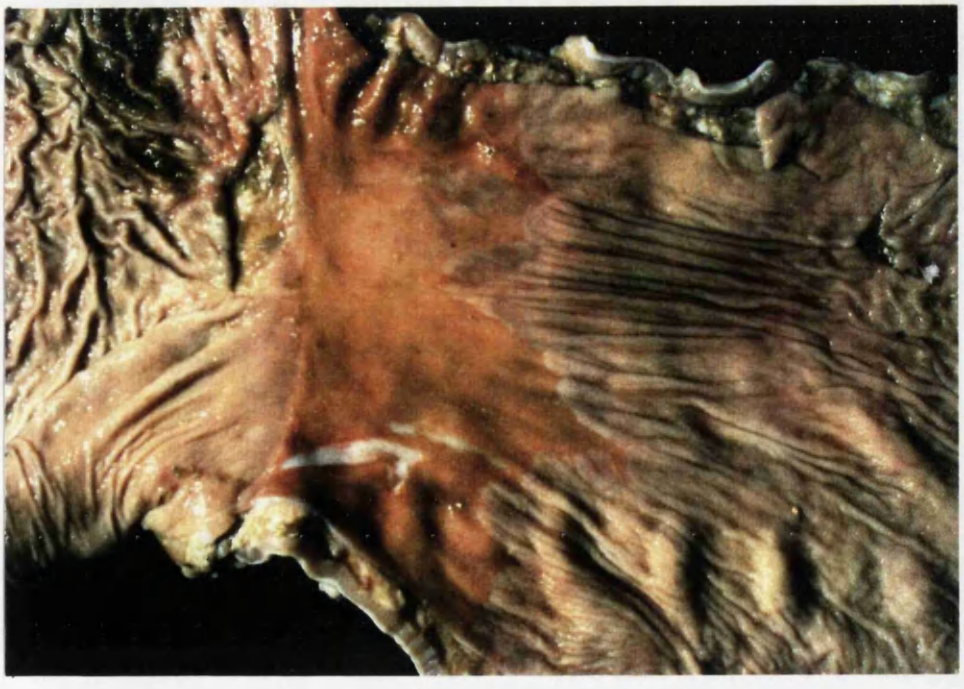


Fig. 8 Case No. 162. Acute colitis due to acidosis. There is a large amount of barley in the opened stomach on the left and the colonic mucusa is black and haemorrhagic.

Fig. 9 Case No. 113. Incidental intestinal ulceration in a horse with chronic osteomyelitis on long term Phenylbutazone therapy. A. Small intestine.

B Colon



C Colon

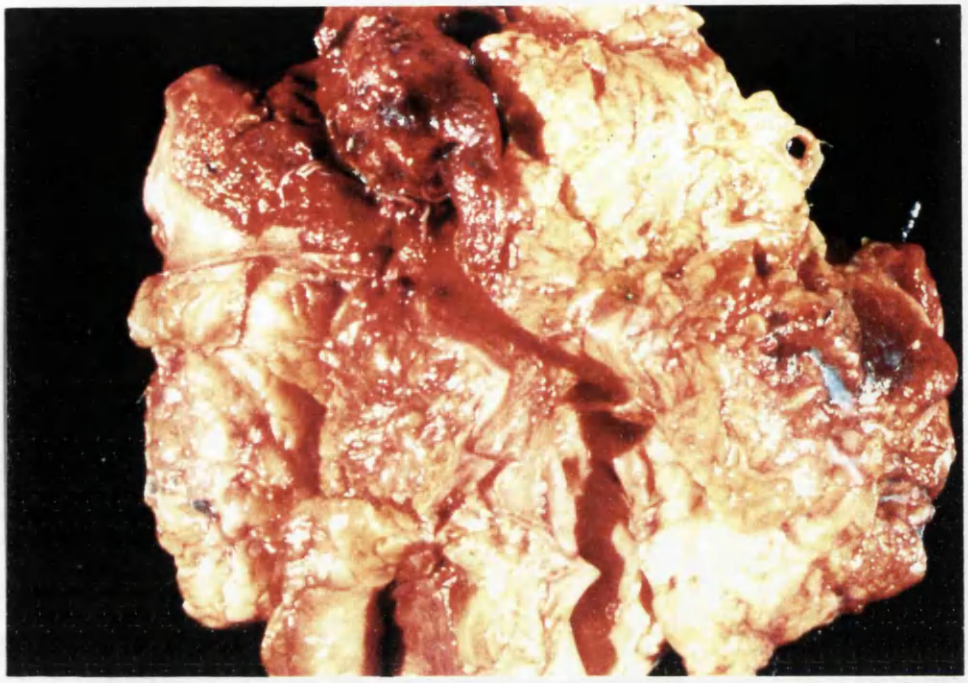


Fig. 10 Case No. 203. *Strongylus vulgaris*.
A. There is a large fresh thrombus in the cranial mesenteric artery which contains many *S vulgaris* L4 larvae.

B Thromboembolism. There is a wedge shaped area of infarction at the pelvic flexure which has perforated releasing intestinal content and causing peritonitis.

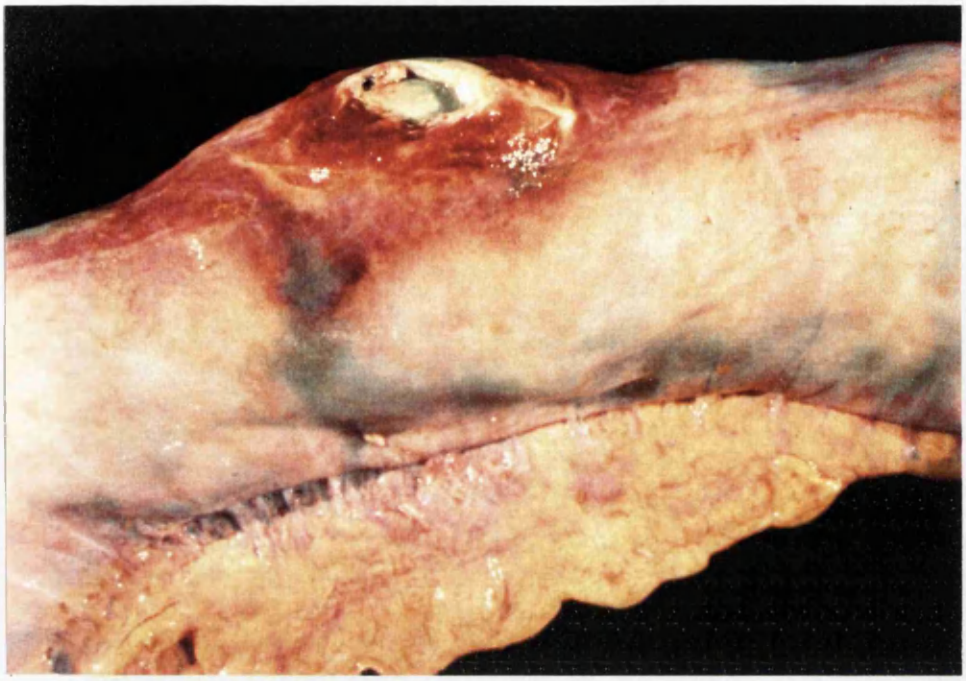
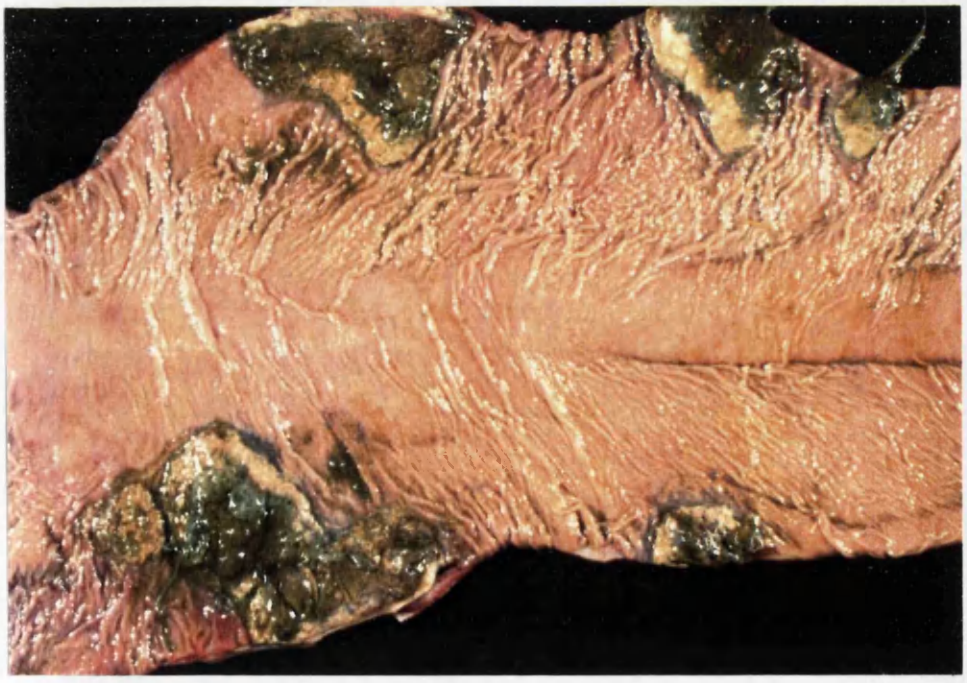


Fig. 11 Case No. 205. Verminous thromboembolism. A. Area of infarction in the dorsal colon.



B The colon has been opened along the antimesenteric border to reveal wedge shaped areas of infarction each supplied by a single blood vessel.

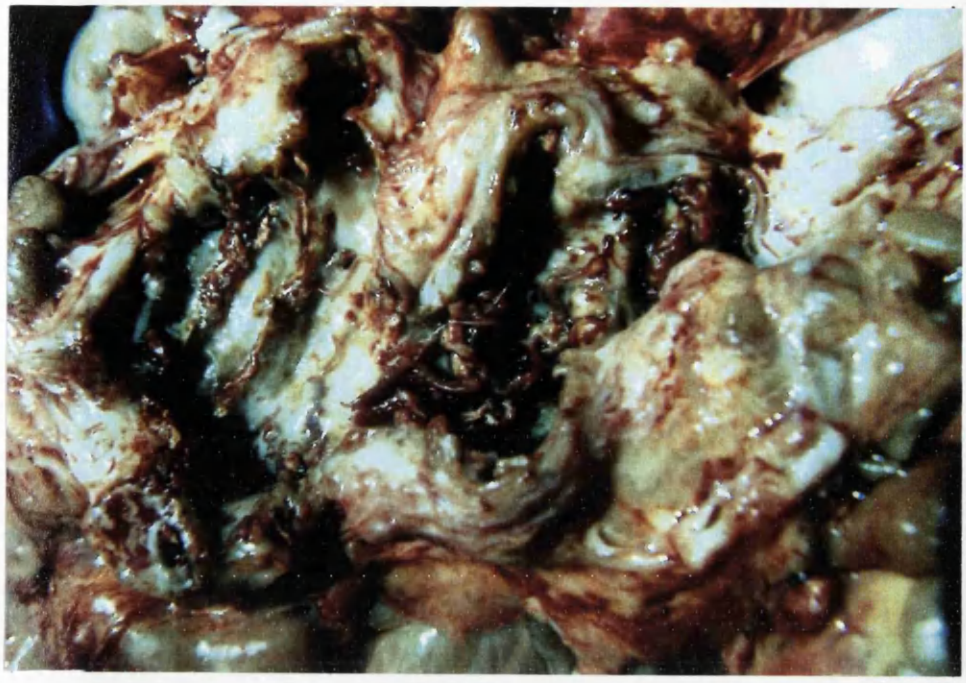


Fig. 12 Case No. 136. A *S vulgaris* L4 larvae in a cranial mesenteric arterial aneurysm. No thromboembolic lesions were noted at necropsy.

B Large pustular nodules in the colonic mucosa occupied, or recently vacated, by adult *S vulgaris*.

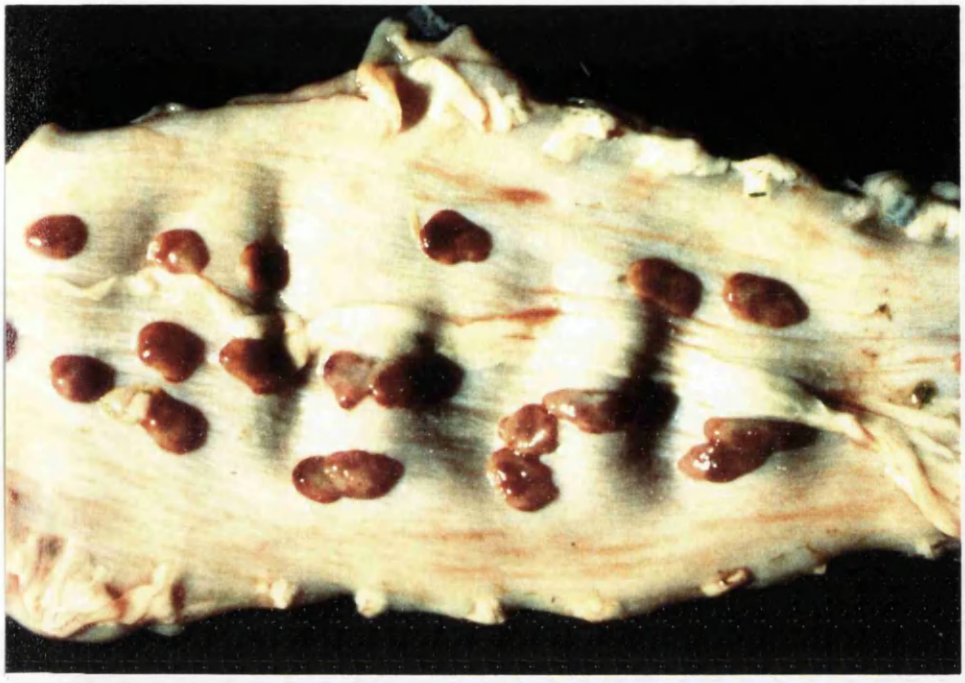
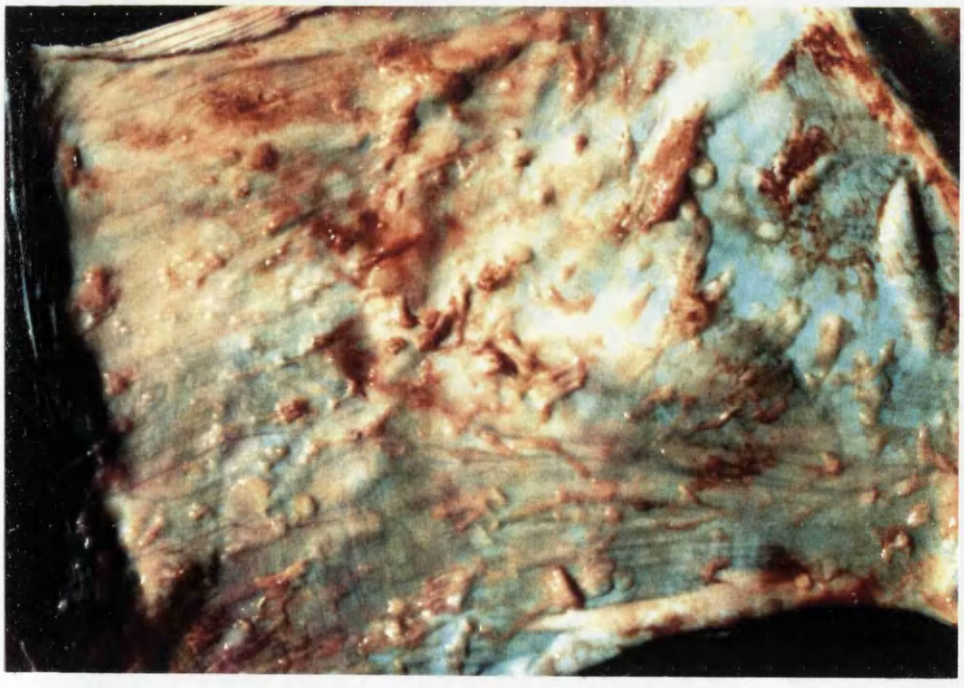


Fig. 13 Case No. 150. *Strongylus edentatus* A *Haemomelasma ilei*



B Peritoneal fibrous tags

Fig. 14 Case No. 206. *Parascaris equorum* spilling out of the incised small intestine.

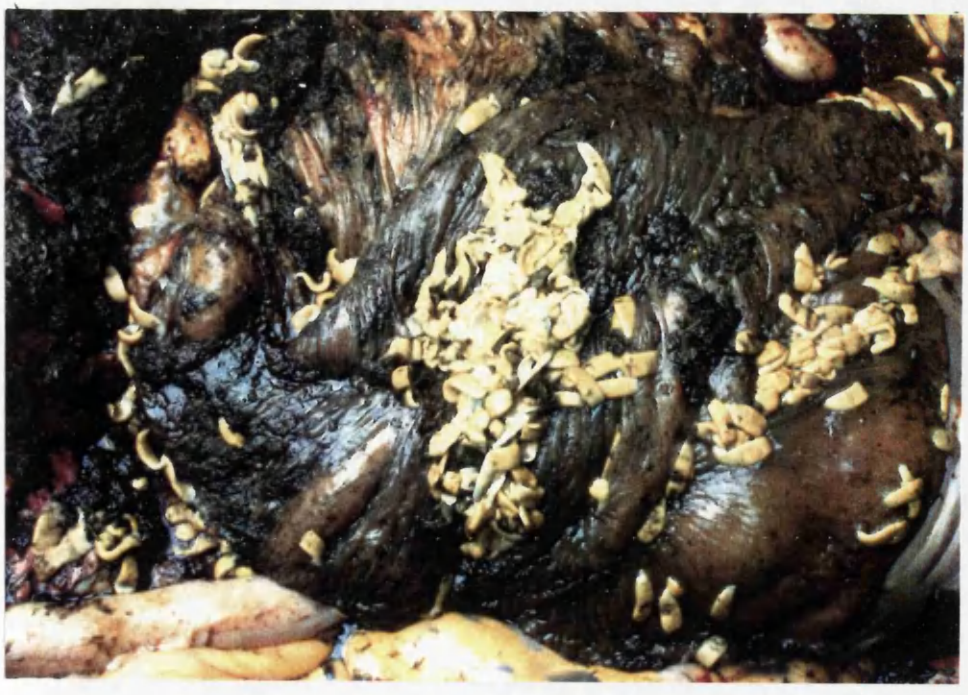


Fig. 15 Case No. 132. Caecum at the ileocaecocolic valve. Large number of *Anoplocephala perfoliata* and mucosal oedema.

PART III - ENTEROPATHY SERIES

INTRODUCTION

With the availability of greatly improved techniques for the assessment of intestinal function, chronic enteropathies with malabsorption were being recognised increasingly by the equine clinician (**Roberts 1985**). Tests such as the oral glucose tolerance test (**Mair, Hillyer, Taylor and Pearson 1991**) and the xylose absorption test (**Bolton et al 1976**) can pin-point the lesion to the small intestines or alternatively rule out significant pathology in that organ.

The cause of one subset of the chronic enteropathies, the inflammatory bowel diseases, remains largely unknown (**Roberts 1985**). So the challenge to the pathologist is not only to categorise these chronic enteropathies and provide the clinician with a definitive diagnosis, but also to investigate and further the understanding of these conditions in an effort to determine their aetiology.

To this end, the clinical records and postmortem findings of 20 animals from the necropsy series found to have a chronic enteropathy or evidence of malabsorption were reviewed in detail.

LITERATURE REVIEW

A review of the literature revealed a relatively small number of publications on the subject of chronic enteropathies in the horse. Those that concerned the inflammatory bowel diseases came predominantly from North America, Australia and Sweden. Some of these conditions have not been described in detail in this country although they apparently do occur (**Platt 1986, Mair et al 1991, Love, Mair and Hillyer 1992**). In general the case series from the United Kingdom grouped together a number of different chronic enteropathies (**Platt 1986**) often because of a common presenting sign (**Love et al 1992, Mair et al 1991**). In contrast case series from abroad tended only to consider a single condition based on the final pathological diagnosis and described the details in depth (**Pass and Bolton 1982, Cimprich 1974**).

The only case series describing chronic enteropathies in the horse was published by **Platt (1986 and 1987)** and he confined his observations to the small intestines. In this series of 20 animals which were almost all young Thoroughbreds, there were 9 cases of alimentary lymphosarcoma, which were described separately (**Platt 1987**), 5 of chronic inflammation with eosinophils, 3 of equine granulomatous enteritis, 2 of intestinal lesions accompanying mycobacterial infections and one of chronic post-infarctive inflammation.

A review of 30 cases of peritonitis (**Dyson 1983**) included one case of granulomatous enteritis out of the 9 horses necropsied but no pathological details were given. **Love and colleagues (1992)** described the clinical findings in 51 cases of chronic diarrhoea in adult horses.

No pathological details were given but the diagnoses included 14 cases of cyathostomiasis, 5 of alimentary lymphosarcoma, 9 of chronic colitis, one of villous atrophy and one of generalised eosinophilic disease.

When Bolton and colleagues (1976) from the U.S.A. assessed the D-xylose absorption test, they listed villous atrophy, necrosis of villous lamina propria and oedema of the lamina propria as findings in their group of abnormal horses but made no attempt to make more than a morphological diagnosis. Mair and colleagues (1991) in their paper assessing the usefulness of the oral glucose tolerance test, included a number of cases of inflammatory bowel disease in the test group but did not describe their pathology.

Roberts has published two general review articles concerning inflammatory bowel disease in the horse (Roberts 1983, Roberts 1985). In his paper on protein-losing enteropathy in the horse (Roberts 1983) the recognised causes were listed and also possible nonenteric routes of protein loss given. The available diagnostic tests were discussed and the pathological findings in three cases of protein-losing enteropathy were described. In his second paper on malabsorption syndromes in the horse Roberts appeared to have extrapolated directly from the human literature (Roberts 1985). However some of the syndromes to which he referred have not been described in the horse. He briefly reviewed the equine literature on the major inflammatory bowel diseases and again described the available diagnostic tests and included possible therapies.

Reviewing these case series revealed that a small number of conditions were consistently identified by the different authors, namely alimentary lymphosarcoma, granulomatous enteritis, eosinophilic enteritis and cyathostomiasis. These conditions and a number of less common ones, have all been described in more detail in other papers. Lymphosarcoma is the most commonly recorded neoplasm of the equine haemopoietic system according to **Van den Hoven and Franken (1983)**. **Neufield (1973a)** reviewed all published cases of equine lymphosarcoma from the case described by Leisering (1853), only 8 years after it was first described in man. His descriptions were confined largely to the gross pathology but age and sex comparisons were made. In a second paper **Neufield (1973b)** reviewed the cases of equine lymphosarcoma on the files of the Ontario Veterinary College and found involvement of the gastrointestinal tract in only one of the 8 listed.

Wiseman et al (1974) described 2 cases with a common presentation of diarrhoea which they believed to be unusual. There was small intestinal involvement in one and small and large intestinal involvement in the other. Of the 3 cases described by **Roberts and Pinsent (1975)** 2 had a history of diarrhoea. There was small intestinal involvement in each case and comparisons were made with alpha-chain disease in man because of the high proportion of plasma cells. **McConnell and colleagues (1982)** described a single case with multifocal involvement of the duodenum and jejunum which had been diagnosed clinically as equine infectious anaemia. **Van den Hoven and Franken (1983)** reviewed the clinical aspects of 16 cases of lymphosarcoma; 3 of these cases were of the alimentary form but pathological details were minimal. A similar case to that of **McConnell and colleagues (1982)** with involvement of the entire small

intestine was described by Humphrey and colleagues (1984). Reef and colleagues (1984) described 3 cases of lymphosarcoma, 2 of which involved the gastrointestinal tract. Their paper dealt principally with the associated immune mediated haemolytic anaemia and thrombocytopaemia but did include the pathological findings. A single case with a history of diarrhoea and involvement of the entire intestinal tract, which included diffuse ulceration was described by Crawley (1985). Wilson and colleagues (1985) described a single case of diffuse intestinal involvement, most marked in the duodenum and jejunum, with cutaneous manifestations.

Reports of a chronic granulomatous enteritis first appeared in the literature from the U.S.A. in 1974 (Cimprich 1974). The condition was likened to Crohn's disease of man and affected, almost exclusively, Standardbreds. Apparently, however, it was not a new disease since cases had occurred sporadically in Sweden for decades (Lindberg 1984).

Cimprich (1974) described the pathological findings in a group of 10 young adult horses (9 Standardbreds and one Thoroughbred) with a history of weight loss. The lesions varied slightly between cases but were predominantly small intestinal, the ileum being most severely affected and in all cases there was villous atrophy and a transmural granulomatous inflammatory infiltrate consisting of lymphocytes and macrophages or epithelioid cells with the addition of plasma cells and occasional multinucleate giant cells. Lesions were also present in a number of other organs notably the mesenteric lymph nodes and liver. Cimprich was also involved in a paper presenting the clinical details as well as the pathological findings in 9 cases of granulomatous enteritis

(7 Standardbreds and one Thoroughbred) (Merritt, Cimprich and Beech 1976). This group apparently included some of the horses first reported by Cimprich (1974) with the addition of some older animals. Cimprich published a continuing education article (Cimprich 1981) reviewing both the clinical and pathological findings. He discussed the similarity between equine granulomatous enteritis and Crohn's disease and the two opposing theories concerning their aetiology ie, an as yet unidentified infectious agent or a defect in the individual's immune response. He finally suggested it might be a combination of the two.

Lindberg (1984) gave detailed descriptions of the pathological findings in a group of 13 young adult Standardbreds. These findings were consistent with those described previously (Cimprich 1974) but lesions were also present in a number of other organs including the stomach. He reviewed the available case reports and discussed the various aetiological possibilities. Lindberg and Karlsson (1985) looked at the mucosal changes in detail in 4 cases using small intestinal biopsy samples which were examined by light and electron microscopy.

After the first case series (Cimprich 1974) a number of case reports were published. Meuten, Butler, Thomson and Lumsden (1978) described the clinical and pathological findings in 2 young Thoroughbreds but approached the report more from the malabsorption, protein-losing enteropathy angle. Therapy was attempted with some clinical improvement in the second case. Bester and Coetzer (1978) described the clinical and pathological findings in a young Thoroughbred from South Africa. In this case although the pathology was similar to that described by other workers there was no transmural involvement. Roberts and Kelly (1980)

published a similar report of a young Standardbred from Australia which again lacked transmural involvement.

The similarity between granulomatous enteritis and Crohn's disease has not gone unnoticed by those working on human gastroenterology. **Mayberry, Rhodes and Heatly (1980)** looked at infections which cause ileocolic disease in animals including equine granulomatous enteritis, and discussed their relevance to Crohn's disease.

The most recent case report **(Sweeney, Sweeney, Sack and Lichensteiger 1986)** described the clinical and pathological findings in 3 sibling Standardbreds. Two were diagnosed as granulomatous enteritis but the third, had greatly elevated numbers of eosinophils and was diagnosed as eosinophilic granulomatous enteritis. The authors proposed that eosinophilic enteritis and granulomatous enteritis may in fact just be either end of the spectrum of a single condition.

Lindberg and colleagues (1985) had however published a paper comparing and contrasting the two conditions. The clinical and pathological findings in 36 horses, predominantly young adults, were presented in 2 groups, 19 cases of eosinophilic gastroenteritis (18 Standardbreds, one Thoroughbred X Orlov trotter) and 17 cases of granulomatous enteritis (all Standardbreds). Consistent marked differences emerged, the most obvious being constant small intestinal involvement in granulomatous enteritis with only discrete large intestinal lesions and constant, diffuse large intestinal involvement in eosinophilic gastroenteritis with limited small intestinal involvement.

Pass and Bolton (1982) in Australia published the first paper in English on eosinophilic gastroenteritis although it had been described previously in Sweden (Lindberg and Persson 1979). They described the clinical and pathological findings in 3 Thoroughbreds and one Standardbred, all young adults. Lesions were present in the small and large intestines and mesenteric lymph nodes in all cases. There was also inconsistent involvement of the pancreas, liver and skin. Microscopically the lesions consisted of masses of eosinophils surrounded by macrophages, epithelioid cells and occasional multinucleate giant cells or diffuse infiltration of the small intestinal lamina propria, submucosa, muscularis mucosa and serosa by the above cells with the addition of lymphocytes and plasma cells. No evidence of parasitological activity was found and the authors concluded that the condition was a chronic but ongoing immediate hypersensitivity reaction of unknown aetiology.

Breider, Kiely and Edwards (1985) described a single case in an aged Quarter Horse with involvement of the skin (all 4 coronary bands), pancreas, liver and large intestine. The authors proposed an immunological basis suggesting because of the distribution, that it was an exaggerated immune response to parasitic antigens.

Wilkie and colleagues (1985) described the findings in five horses from Canada with a chronic eosinophilic dermatitis. All 5 had pancreatic lesions consistent with those previously described for eosinophilic gastroenteritis (Pass and Bolton 1982, Breider et al 1985). Three had lesions in the biliary system, 3 had involvement of the abdominal lymph nodes and one had ulcerative colitis. The authors suggested that their

cases represented one end of the spectrum of the eosinophilic dermatitis/enterocolitis complex.

This conclusion was supported by the findings in one Standardbred and one Thoroughbred from Australia (**Gibson and Alders 1987**). Both had chronic diarrhoea, weight loss, intestinal lesions and eosinophilic dermatitis. Both responded to therapy, one returned to stud but the other was destroyed because of acute colitis.

The two most recent papers described the condition as multisystemic eosinophilic epitheliotropic disease. (**Sanford 1989, Hillyer and Mair 1992**). The first (**Sanford 1989**) concerns a 5 year old Standardbred stallion with weight loss, diarrhoea and patchy alopecia. At necropsy there was hyperkeratosis of the oesophagus and the squamous portion of the stomach, fibrous thickening of the duodenum, the ileum, and the common and intrahepatic bile ducts and firm nodules throughout the pancreas. There was marked enlargement of the mesenteric and caeocolic lymph nodes. Thickening of the ileum had caused stenosis of the lumen and the intestine had become dilated proximally. The histopathological findings were similar to those previously described (**Cimprich 1974**). The second paper is as yet the only detailed report from the United Kingdom (**Hillyer and Mair 1992**). The most dramatic lesions were those of the skin (crusting dermatitis) and the pancreas (two large fibrous masses). There was a mononuclear and eosinophil cell mucosal infiltrate throughout the intestines which had resulted in a flat absorption curve, following the oral glucose tolerance test and diarrhoea. No eosinophilic granulomata were present in the intestines, but they were found in the lungs and pancreas.

A number of inflammatory bowel disorders have been described in the horse predominantly as single case reports, in which the authors considered the findings to be inconsistent with those previously recorded and again the aetiology was not established.

Pass, Bolton and Mills (1984) described a case of what they termed basophilic enterocolitis. There was widespread submucosal oedema, areas of hyperaemia and diphtheraesis or mucosal ulceration with infiltration by lymphocytes, plasma cells, macrophages, eosinophils, basophils and occasional mast cells. There were elevated numbers of basophils in the bone marrow. The possibility that the condition was a variant of eosinophilic enteritis was suggested.

Clark and colleagues (1988) described a single case of lymphocytic enteritis in a filly. This is somewhat confusing, because within the text comparisons were made, and the general similarities pointed out, with lymphocytic-plasmacytic enteritis in other species. They did however explain the different pressure gradients required for the production of protein poor and protein rich fluid.

MacAllister, Mosier, Qualls and Cowell (1990) described a further 2 cases of lymphocytic-plasmacytic enteritis. The infiltrate was confined to the small intestine in one and predominantly to the small intestine in the other and was composed entirely of lymphocytes and plasma cells. The second case had a subclinical disseminated intravascular coagulopathy. The high incidence of a hypercoagulative state in humans with Crohn's disease was pointed out and the only report of a comparable

equine case highlighted. In that report Morris, Vaala and Sartin (1982) described a single case of protein-losing enteropathy in a filly with subclinical disseminated intravascular coagulation and autoimmune haemolytic disease. Their proposed diagnosis was idiopathic ulcerative colitis, a rare condition in man not previously reported in the horse, but the paper dealt more with the haematological findings.

In addition to the inflammatory bowel disorders already described, where the aetiology is at best speculative, a number of conditions of known aetiology exist in the horse and have been described in detail. The agents involved include nematodes (Blackwell 1973), protozoa (Wheldon and Greig 1977), fungi (Dade et al 1973) and bacteria (Innes 1949).

Love (1992) reviewed the major parasitological causes of diarrhoea in the horse, including the clinical and pathological details and gave a brief review of the relevant literature. Parasitism in the form of large strongyles has long been associated with intestinal disease, usually acute but occasionally chronic (Greatorex 1975) and granulomatous in nature (Platt 1986).

The paper by Greatorex (1975) dealt primarily with the clinical findings and treatment of 91 cases of diarrhoea due to *Strongylus vulgaris* activity in adult horses but also gave a reasonable account of the pathology of 35 cases necropsied. The common findings were vascular thrombosis and mucosal ulceration, both acute and chronic. The mucosal infiltrates were composed of mononuclear cells and eosinophils.

Barklay, McCracken, Phillips and Foerner (1987) also briefly described the clinical and pathological findings in a group of 7 horses, the most likely cause of which was *S. vulgaris*. The pathological descriptions were based on examination of resected portions of bowel in all 7 cases and post- mortem examination in 2 cases. The lesions were small intestinal in 3 animals and large intestinal in the remaining 4. The histopathology was described as severe chronic inflammation involving the full thickness of the wall in each case but inconsistent with that of granulomatous enteritis. The common aetiology proposed was nonoclusive mesenteric ischaemia or temporary ischaemic strangulation obstruction of the intestine most likely as a result of *S. vulgaris* activity.

Cyathostome (small strongyle, trichoneme) larvae may also cause a chronic colitis and diarrhoea as a result of their mass emergence in the spring (Duncan 1985b). This has been described by a number of authors. Blackwell (1973) briefly presented the salient clinical and pathological findings describing a granulomatous colitis with extensive infiltration of the lamina propria and submucosa by chronic inflammatory cells. Chiejina and Mason (1977) illustrated their general observations on the syndrome with the findings in a single case but did not examine its intestine microscopically; oedema and congestion were the most striking gross lesions. They also pointed out that even the best anthelmintic programmme did not affect the inhibited larvae. The case series published by Giles and colleagues (1985) contains only minimal pathological detail. Jasko and Roth (1984) described a single case, the first from North America, and diagnosed a granulomatous colitis. The second case from North America (Harmon et al 1986) dealt predominantly

with the management of the animal's electrolyte and acid base balance but did describe the curled cyathostome larvae within the mucosa. They also noted that the most intense reaction was around the nematodes in the submucosa rather than those within the mucosa.

A number of protozoa have been implicated as the cause of intestinal disease in the horse but their precise aetiological significance remains obscure. **Gregory and colleagues (1986)** described a case where there was invasion of the large intestinal mucosa by *Polymorphella ampulla* in association with a marked cyathostome burden. Other papers describe the clinical findings in horses believed to be suffering from giardiasis **(Kirkpatric and Shand 1985)** or trichomoniasis **(Bennett and Franco 1969)**. The diagnosis was based on the response to specific therapy in the former and faecal examination in the latter. **Laufenstein-Duffy (1969)** stated that no pathology was associated with trichomoniasis and that it must be a functional not physical disturbance.

Wheeldon and Greig (1977) described a case of *Eimeria leukarti* (a coccidial protozoan) infection of the small intestine of a pony which had become emaciated as a result of chronic diarrhoea. No other agent was implicated. **Mason and King (1971)** described finding developing *E leukarti* oocysts in the small intestinal lamina propria of a foal which had died of intractable diarrhoea. They seemed unsure of the aetiological significance also finding large areas of mucosal loss in the small intestine and thrombosis of blood vessels in the large intestine. Indeed, oocysts of *E leukarti* have been isolated from the faeces of asymptomatic foals by other authors **(Lyons,Drudge and Tolliver**

1988, McQueary, Whorley and Catlin 1977) so clearly the parasite does not always cause disease.

Another coccidian parasite isolated from asymptomatic carriers is cryptosporidia (Tzipori and Campbell 1981). There has been only one report of disease caused by this organism in fully immunologically competent foals (Gajadhar et al 1985) in which numerous organisms were present attached to the surface of the villous epithelium, no inflammatory or physical changes were noted in the intestines. The remaining reports have all been in Arabian foals with severe, combined immunodeficiency syndrome. Snyder and colleagues (1978) described findings similar to those of Gajadhar and colleagues (1985) but Gibson and colleagues (1983) and Mair and colleagues (1990b) described villous fusion and atrophy as well as surface cryptosporidia.

Another cause of granulomatous colitis is infection by the dimorphic fungus *Histoplasma capsulatum*. This has been described as the sole aetiological agent (Dade et al 1973) or in conjunction with *Salmonella sp.* (Goetz and Coffman 1984). No cases have been recorded in the United Kingdom.

Reports of tuberculosis in the horse have increased in frequency (Buergett et al 1988). Tuberculosis has been reviewed by a number of Authors. Innes (1949) quotes Griffiths (1936) who stated that all but 2 of the 55 cases examined in England were of the bovine type. Luke (1958) commented on the low incidence of tuberculosis in the horse, quoting a figure of less than one in 15,000 (Calmette 1923). He also stated that the lesions in horses differ from those of other domestic

animals because they may be confined to the digestive tract and resemble Johnes disease in cattle. **Larsen, Moon and Merkal (1972)** reviewed the equine cases of *M Paratuberculosis* and investigated the horse's susceptibility to that organism. They were able to infect a group of animals which in turn infected a control group. **Baker (1973)** described a single case of *M paratuberculosis* of the avian type which included involvement of the large intestine. Two papers describing the clinical **(Merrit et al 1976)** and pathological **(Cimprich 1974)** findings in a group of horses with granulomatous enteritis refer to one animal in the group from which avian *M tuberculosis* was cultured from the faeces.

Three equine cases of avian mycobacteriosis were described and the recent literature on the subject reviewed by **Buergelt and colleages (1988)**. The first case was a 2 year old, female Paso Fino horse with a 6 month history of anorexia, weight loss and diarrhoea which lived in close contact with chickens and pigeons. Examination of small intestinal, colonic and mesenteric lymph node biopsies revealed a granulomatous enteritis and lymphadenitis with acid-fast organisms. The horse was destroyed. At necropsy the Peyer's patches of the distal small intestine were prominent and the mesenteric lymph nodes were enlarged. Multiple 0.1-0.4cm diameter white nodules were present thoughout the liver, and lungs. The intestinal infiltrate consisted of epithelioid macrophages, lymphocytes, plasma cells and eosinophils and the normal Peyer's patch population had been replaced by macrophages. Small granulomata were present adjacent to submucosal lymphatics. The mesenteric modes contained macrophages and multinucleate giant cells which in turn contained numerous acid-fast organisms. The pulmonary and hepatic nodules were composed predominantly of eosinophils. The

organisms grown from faecal and tissue samples were identified as *Mycobacterium avium-intracellulare* complex serotypes 1, 8/1 and 5. Case 2 was a 7 year old Argentine gelding which had developed skin lesions and lost a lot of weight. At necropsy in addition to multiple skin nodules several white nodules were also present in the serosa of the ventral and small colons and the liver contained white foci. The mesenteric lymph nodes were of expected size but bulged on section. Microscopic examination of the serosal lesions revealed granulation tissue and the hepatic lesions were granulomata which contained acid-fast organisms. The organisms were identified as *Mycobacterium avium* complex serotype 8. The third case was a 6 year old Quarter Horse stallion with a history of inappetence, weightloss, oedema and intermittent colic. Biopsy samples of large colon and mesenteric lymph node were interpreted as equine granulomatous colitis and lymphadenitis and the animal was treated with anabolic steroids and sent home. The animal deteriorated over the next 2 months and was destroyed. At necropsy the right dorsal colon wall was thickened by multiple closely packed white nodules. Mesenteric and colonic lymph nodes were enlarged and bulged on section. Microscopic examination of the dorsal colon revealed dense sheets of lymphocytes, plasma cells, macrophages and a few neutrophils and multinucleate giant cells. A small number of acid-fast bacterial rods were present. Colonic lymph nodes contained macrophages and multinucleate giant cells. Again *Mycobacterium avium-intracellulate* complex, serotype 8 was isolated. The Authors noted that horses susceptible to *M.avium* infection usually develop the intestinal form of the disease suggesting oral uptake of bacilli and the lesions more closely resemble those of *Mycobacterium paratuberculosis* than mammalian tuberculosis.

Cline and colleages (1991) described a single case of abortion and granulomatous colitis due to *Mycobacterium avium* complex infection in a horse. A 17 year old Standardbred mare had a 6 month history of diarrhoea prior to aborting a 160 day foetus. Examination of the foetus and foetal membranes revealed numerous noncaseating granulomata containing acid-fast bacilli. The mare gave a positive reaction to the avian intradermal tuberculin test and was destroyed. At necropsy there was a large lingual ulcer, thickening and ulceration of the right dorsal colon and focal ulceration of the small colon. A number of lymph nodes were moderately enlarged. Microscopic examination of the colonic lamina propria revealed many macrophages and multinucleate giant cells and multiple granulomata were present in the lymph nodes. Occasional single or clustered acid-fast bacilli were seen in both the colons and the nodes. The organisms cultured were identified as *M.avium* complex but could not be serotyped.

Not all known causes of gastrointestinal disease in the horse are infectious. Toxic doses of phenylbutazone (PBZ), a nonsteroidal anti-inflammatory drug (NSAID), induce marked lesions in the gastrointestinal tract of the equine, ponies being extremely intolerant of the drug (Snow et al 1981). The lesions are acute in nature and are not classified as a chronic enteropathy but do result in malabsorption as well as a protein- losing enteropathy (Snow et al 1981). Snow and colleagues (1979) were the first to report the toxic effects of PBZ in ponies which included oral, gastric and large intestinal ulceration as well as peritonitis. A second paper on the biochemical and pathophysiological changes was published in 1981 and included more detailed pathology (Snow

et al 1981). Massive intestinal ulceration, mainly large intestinal, but also oral, gastric and duodenal was reported. In the large intestines the ulcers coalesced to form extremely large areas of damage with associated submucosal oedema. There was peritonitis in one animal. Microscopic changes consisted of surface necrosis with vasculitis and vascular thrombosis below the larger ulcers. Premortem the animals were found to be hypoproteinaemic. Radio labelled Cr-plasma protein studies pinpointed the site of protein loss as the gastrointestinal tract.

MacKay and colleagues (1983) used much higher doses of PBZ and reported a number of lesions in addition to those previously described (Snow et al 1981). Renal lesions were observed in all the animals, pulmonary vascular lesions in 4, liver lesions in 2, an acute diaphragmatic hernia in one and colonic thrombosis and infarction in one. The authors proposed that the mechanism of intestinal damage may be related to interference with the protective effect of prostaglandins or decreased epithelial repair and replacement.

Meschter and colleagues (1984) studied the vascular pathology in Thoroughbreds given intravenous PBZ. The intestinal ulceration was most marked in the duodenum and again renal lesions were noted. The basic lesion was degeneration of the wall of small veins and acute ischaemic necrosis of the intestinal mucosa leading to ulceration.

The clinical findings in 7 clinical cases of PBZ toxicity were described by Collins and Tyler (1984) and brief descriptions of the pathology of the 3 animals that were necropsied were included. Gastrointestinal ulceration, renal lesions and oedema were recorded. In a second paper

Collins and Tyler (1985) reported that they had induced changes consistent with those of PBZ toxicity and greatly reduced them with synthetic prostaglandin E_2. They concluded that prostaglandins are important in maintaining the integrity of the alimentary mucosa and in the cellular protective responses when the mucosal barrier is broken. Meschner and colleagues (1990) reviewed the literature on the subject of PBZ toxicity and made a detailed study of the biochemical changes and duodenal and colonic pathology in a group of experimental animals. They also measured the mucosal prostaglandin concentrations but the results were not significant. After much discussion they concluded that the lesions induced by excessive doses of PBZ were mediated by non-prostaglandin pathways.

Twenty one cases of right dorsal colitis were described by Karcher and colleagues (1990). In all but 3 of the cases there was a history of PBZ or flunixin meglumine (another NSAID) therapy often in combination with a degree of dehydration. The Authors proposed that right dorsal colitis, characterised by multiple to coalescing ulcerative foci with discrete islands of mucosal regeneration, was an as yet unreported manifestation of NSAID toxicosis. Lesions were not found elsewhere within the gastrointestinal tract. The animals either died in the acute phase or progressed to a chronic phase and eventualy died or were destroyed on humane grounds. Scarring and stenosis of the right dorsal colon was present in some of the chronic cases. The Authors reproduced the lesions, which were again confined to the right dorsal colon, in 2 experimental horses by a combination of PBZ and partial water deprivation. It was noted that the right dorsal colon is the only segment of the large intestine with a net water flux in a secretory

direction suggesting some intrinsic mucosal membrane differences which might explain the localised nature of the colitis.

CASE MATERIAL

The clinical records and necropsy reports of all the animals in the Necropsy Series were reviewed. All cases with a history of weightloss and significant pathology confined to the gastrointestinal tract or with weightloss and clinical evidence of malabsorption but no significant pathology were identified. The resultant group of 20 animals, 8 of which had been examined post-mortem by the Author formed the Enteropathy Series. Each animal was assigned an Enteropathy Series Case Number, 1 - 20, as detailed in Table 36. These are the numbers which will be used exclusively in this section.

METHOD OF STUDY

The recorded macroscopic findings and tissue sections from the 20 animals, were re-examined. In all but one case multiple intestinal sections and a variety of other tissues were available. Where appropriate, fresh sections were cut and stained with one or more special stains (see Appendix 1). All the horses had been necropsied as previously described in Part I, Necropsy Technique.

RESULTS - ENTEROPATHY SERIES

Once all the information was collated it was decided to divide the 20 horses in the Enteropathy Series into 2 groups, (Table 36). Group A consisted of Cases 1 - 11 in which a definitive diagnosis was made. Group B consisted of the remaining 9 cases, Cases 12 - 20 where only a morphological diagnosis was achieved.

ENTEROPATHY SERIES CASE NUMBER	NECROPSY SERIES CASE NUMBER	GROUP	DIAGNOSIS
1	24	A	Alimentary Lymphosarcoma
2	26	A	Alimentary Lymphosarcoma
3	3	A	Alimentary Lymphosarcoma
4	155	A	Granulomatous Enteritis
5	161	A	Granulomatous Enteritis
6	172	A	Eosinophilic Enteritis
7	21	A	Eosinophilic Enteritis
8	11	A	Cyathostomiasis
9	150	A	Cyathostomiasis
10	126	A	Coccidiosis
11	28	A	Protein-losing Enteropathy
12	70	B	Enterotyphlocolitis
13	1	B	Enterotyphlocolitis
14	159	B	Enterotyphlocolitis
15	92	B	Granulomatous Typhlocolitis
16	62	B	Granulomatous colitis
17	2	B	Typhlocolitis
18	80	B	Enteritis
19	31	B	Lymphoid Hyperplasia
20	112	B	Dental Exhaustion

Table 36 The Enteropathy and Necropsy Series case numbers, group and diagnosis for the 20 cases in the Enteropathy Series.

The Enteropathy series comprised 11 females and 9 neutered males. Both horses and ponies, of a number of different breeds were represented [7

ponies, 2 Welsh mountain ponies, 5 Thoroughbreds, 2 Thoroughbred crosses, 2 riding horses (1 Arab cross and 1 Welsh cob)] which had been presented at times throughout the year. The average age was 15 with only 3 animals less than 10 years of age. The clinical histories varied but all animals had lost weight, 13 had suffered from diarrhoea and 9 from colic. The duration of signs prior to presentation ranged from 2 weeks to 18 months. Tables 37 and 38 summarise the signalment and clinical presentation of the animals in Groups A and B respectively.

CASE NUMBER	BREED	AGE	SEX	DURATION	DIARRHOEA	WEIGHT LOSS	COLIC
1	Pony	13	Mn	4M	+	+	-
2	Pony	15	F	3 1/2M	+	+	-
3	Pony	15	F	9W	+	+	-
4	Arab X	13	F	8M	-	+	+
5	Pony	15	Mn	6W	+	+++	-
6	TB	20	F	10W	+	+++	-
7	RH	20	Mn	NR	+	+++	-
8	TBX	15	Mn	6W	+	+++	-
9	Pony	13	F	2W	+	+++	+
10	WC	14	Mn	3W	+	+	+
11	RH	15	Mn	5M	-	+	-

Table 37 The signalment and clinical presentation of the horses in Group A. Mn=neutered male, F=female, M=months, W=weeks, TB=Thoroughbred, RH = riding horse, WC=Welsh Cob, NR=not recorded

CASE NUMBER	BREED	AGE	SEX	DURATION	DIARRHOEA	WEIGHT LOSS	COLIC
12	Pony	8	F	5W	+	+	-
13	TB	15	F	3M	-	+	+
14	WMP	20	Mn	6M	-	++	+
15	Pony	12	F	5-6M	+	+	+
16	TBX	19	F	8W	+	+++	-
17	TB	15	F	3M	-	+	+
18	TB	6	F	4-6W	-	+	+
19	TB	9	Mn	6W	-	+	-
20	WMP	30	Mn	18M	+	+	+

Table 38 The signalment and clinical presentation of the horses in Group B. Abbreviations as Table 37. WMP=Welsh Mountain Pony.

The major biochemical abnormality was hypoalbuminaemia, a finding in 15 cases. The most consistent haematological abnormality was neutrophilia as found in 11 cases although anaemia was noted in 2 (Cases 15 and 20). Rectal biopsies had been performed on 10 animals, one showed evidence of an enteropathy (Case 1) and the rest were inconclusive or lacked any detectable abnormalities. Other findings were noted in a number of cases eg reduced appetite, oedema or peritonitis. Tables 39 and 40 summarise the clinical test results and other findings in Groups A and B respectively.

CASE NUMBER	HYPOALBUMINAEMIA	ABNORMAL OGGT	RAISED WCC	RECTAL BIOPSY	OTHER
1	+	+	+	Enteropathy	Prepucial Oedema Peritonitis
2	+	-	+	NAD	Ascites Cholangiohepatitis
3	+	Partial	+	NAD	
4	+	+	+	NP	Peritonitis
5	+	Delayed Peak	-	NAD	Anorexic Systolic murmur
6	+	+	+	NAD	Reduced appetite Ventral oedema Polydipsic
7	+	NP	-	NP	Grade I murmur
8	+	Partial	+	NAD	
9	+	NP	+	NP	Ventral oedema
10	+	NP	+	In-conclusive	Ventral oedema
11	-	+	+	NAD	Good appetite

TABLE 39 The clinical test results and other findings from the horses in Group A.

OGGT - oral glucose tolerance test, WCC - white cell count, NR - not recorded, NP - not performed, NAD - no abnormalities detected

CASE NUMBER	HYPOALBUM-INAEMIA	ABNORMAL OGGT	RAISED WCC	RECTAL BIOPSY	OTHER
12	+	NP	-	NP	Polydipsic
13	NR	NP	NR	NP	Thick rectal wall
14	+	NP	+	NP	Good appetite
15	+	NP	+	NP	Anaemic Thick rectal wall, Hepatic - Haemosiderosis
16	NR	NP	NR	NP	
17	-	partial	-	Inconclusive	

18	+	NP	NR	NP	Anorexic
19	-	+	-	Inconclusive	
20	+	+	NR	NP	Anaemic

Table 40 The clinical test results and other findings from the horses in Group B Abbreviations as Table 39.

The oral glucose tolerance test had been performed on 11 animals (see Table 41). In one case the result was normal (Case 2), one showed a delayed peak (Case 5), 6 cases showed complete malabsorption (Cases 1,4,6,11,19,20) and 3 showed partial malabsorption (cases 3,8 and 17).

The pathological findings for each Case in Groups A and B (Figures 16-50) are described in full in Appendix 5 and summarised in Tables 42 - 47. They varied greatly both macroscopically and microscopically, from oedema, which was present in all cases, to diffuse involvement of either small or large intestines. Other changes specifically noted were villous atrophy, increased numbers of intraepitheleal lymphocytes, a cellular infiltrate and ulceration.

RESULT	CASE NUMBER	SMALL INTESTINES OEDEMA	VA	IEL	INF	ULCERATION	LARGE INTESTINES OEDEMA	IEL	INF	ULCERATION	DIAGNOSIS
Normal	2	+		+			+		+	+	Alimentary Lymphosarcoma
Delayed Peak	5	+	+	+	+	+	+	+	+	+	Granulomatous Enteritis
Complete Malabsorption	1	+	+	+	+	+	+		+	+	Alimentary Lymphosarcoma
	4	+	+	+	+	+	+	+			Granulomatous Enteritis
	6	+	+	+	+		+	+	+	+	Eosinophilic Enteritis
	19	+	+	+	+		+		+		Lymphoid Hyperplasia
	11	+			+		+	+			Coccidiosis
	20	+					+	+			Dental Exhaustion
Partial Malabsorption	3	+					+	+	+	+	Alimentary Lymphosarcoma
	8	+					+		+		Cyathostomasis
	17	+			+		+		+		Typhlocolitis

Table 41 The oral glucose tolerance test results for the horses in the Enteropathy Series, the small and large intestinal lesions present at necropsy and the final diagnosis made.

VA - villous atrophy, IEL - intraepithelial lymphocyte numbers increased, INF - infiltration by inflammatory cells

CASE NUMBER	STOMACH	SMALL INTESTINES					
		Oedema	VA	IEL	INF	Ulceration	Other
1	Oedema	+	+	+	+	+	
2	Ulceration	+		+			Galt Reactive
3							
4	Hyperkerat-osis	+	+	+	+	+	Fibrosis
5	Hyperkerat-osis	+	+	+	+	+	Fibrosis Petechiae
6	Ulceration	+	+	+	+		Diptheritic
7		+	+		+		
8		+					
9							
10		+			+		Galt Reactive Peritonitis
11		+					Coccidia

Table 42 The gastric and small intestinal pathology present at necropsy in the horses in Group A. Abbreviations as Table 41. GALT=Gut associated lymphoid tissue.

CASE NUMBER	LARGE INTESTINES					OTHER ORGANS INVOLVED		
	OEDEMA	IEL	INF	ULCERATION	OTHER	PANCREAS	LIVER	NODES
1	+		+	+				+
2	+	+	+	+				+
3	+	+	+	+	Petechiae			+
4	+	+						+
5	+	+	+	+				+
6	+	+	+	+		+	+	+
7	+	+	+					+
8	+		+		Larvae			
9	+				Larvae			
10	+				Rectal tear Peritonitis		+	
11	+				Parasitism			

Table 43 The large intestinal, pancreatic, hepatic and lymphoid pathology present at necropsy in the horses in Group A. Abbreviations as Table 41

CASE NUMBER	STOMACH	SMALL INTESTINES					
		OEDEMA	VA	IEL	INF	ULCERATION	OTHER
12	Erosions	+		+	+		
13	Scarring	+	+	+	+		*H.ilei*
14		+			+	+	Mucosa necrotic
15		+	+	+	+		

16		+	+	+	+		
17		+			+	+	Ileal mucosa thickened
18		+	+	+	+		Peritonitis GALT Reactive
19	Ulceration	+	+	+	+		*H.ilei* GALT hyperplastic
20		+					

Table 44 The gastric and small intestinal pathology present at necropsy in the horses in Group B. Abbreviations as Tables 41 and 42. H.ilei=*Haemomelasma ilei*.

	LARGE INTESTINES					OTHER ORGANS INVOLVED		
CASE NUMBER	OEDEMA	IEL	INF	ULCERATION	OTHER	PANCREAS	LIVER	NODES
12		+	+	+	Bacterial invasion			+
13	+	+	+		GALT reactive			
14	+		+	+	Rectal tear Peritonitis GALT reactive			
15	+	+	+	+	GALT reactive			+
16	+	+	+					
17	+		+		GALT reactive			
18	+		+	+	Peritonitis			+
19	+		+		GALT reactive			
20	+	+						

Table 45 The large intestinal, pancreatic, hepatic and lymphoid pathology present at necropsy in the horses in Group B. Abbreviations as Tables 41 and 42.

The cellular infiltrate, where present, varied both in its components and in the proportions of these components (Tables 46 and 47). There was marked variation between cases but also between different areas in a single case. The mucosa was generally the most severely affected with spread to involve the submucosa, or even the entire thickness of the wall (Cases 4 and 5) (Figure 23).

DIAGNOSIS	CASE NUMBERS	SMALL INTESTINES	LARGE INTESTINES
Alimentary Lymphosarcoma	1	Neoplastic Lymphoblasts	Neoplastic Lymphoblasts
	2, 3		Neoplastic Lymphocytes
Granulomatous Enteritis	4, 5	Macrophages Lymphocytes Plasma Cells Eosinophils	Eosinophils
	5	Mast Cells	Lymphocytes Plasma Cells Mast Cells Eosinophils
Eosinophilic Enteritis	6, 7	Eosinophils Lymphocytes Plasma Cells	Eosinophils Lymphocytes Plasma Cells
Cyathostomasis	8	-	Lymphocytes Plasma Cells Eosinophils
	9	Not Examined	Not Examined
Protein-losing Enteropathy (Phenylbutazone Toxicity)	10	Lymphocytes Plasma Cells	
Coccidiosis	11	-	-

Table 46 The Group A cases listed by diagnosis, and the nature of the small and large intestinal infiltrate in each case.

DIAGNOSIS	CASE NUMBERS	SMALL INTESTINES	LARGE INTESTINES
Enterotyphlocolitis	12	Lymphocytes Plasma Cells Eosinophils Neutrophils (mild)	Lymphocytes Plasma Cells Eosinophils Neutrophils (mild)
	13, 14	Lymphocytes Plasma Cells Eosinophils Globule Leukocytes	Lymphocytes Plasma Cells Eosinophils Globule Leukocytes
Granulomatous Typhlocolitis/ Colitis Enteritis	15/16	Lymphocytes Plasma Cells Eosinophils	Lymphocytes Plasma Cells Macrophages Eosinophils

Typhlocolitis	17	Eosinophils (mild)	Macrophages Eosinophils (mild)
Enteritis	18	Lymphocytes Plasma Cells	Eosinophils
Lymphoid Hyperplasia	19	Eosinophils	Eosinophils (mild)
Dental Exhaustion	20	-	-

Table 47, The Group B cases, listed by diagnosis, and the nature of the small and large intestinal infiltrate in each case.

As stated earlier once all the information was collated, the animals could be placed in one of 2 broad groups. The first group, Group A, consisted of 11 animals in which the gross and histopathological features were consistent with one of the previously described conditions. A definitive diagnosis was made in each case. There were 3 cases of alimentary lymphosarcoma, 2 of granulomatous enteritis, 2 of eosinophlic enteritis, 2 of cyathostomasis and one each of protein-losing enteropathy and coccidiosis. This information is summarised in Table 48.

DIAGNOSIS	NUMBER OF CASES	CASE NUMBERS
Alimentary Lymphosarcoma	3	1, 2, 3
Granulomatous Enteritis	2	4, 5
Eosinophilic Enteritis	2	6, 7
Cyathostomiasis	2	8, 9
Protein-losing Enteropathy (Phenylbutazone Toxicity)	1	10
Coccidiosis	1	11

Table 48, The definitive diagnosis for each of the cases in Group A and the number of cases with each condition.

In the second group, Group B, of 9 animals the inflammatory infiltrate lacked a precise pattern or was inconsistent with the pathological

findings of previously described conditions. In each case only a morphological diagnosis was achieved. There were 3 cases of enterotyphlocolitis, 2 of granulomatous typhlocolitis and enteritis, and one each of typhlocolitis, enteritis. lymphoid hyperplasia and dental exhaustion. This information is summarised in Table 49.

DIAGNOSIS	NUMBER OF CASES	CASE NUMBERS
Enterotyphlocolitis	3	12, 13, 14
Granulomatous Typhlocolitis/ Colitis Enteritis	2	15, 16
Typhlocolitis	1	17
Enteritis	1	18
Lymphoid Hyperplasia	1	19
Dental Exhaustion	1	20

Table 49, The morphological diagnosis for each of the cases in Group B and the number of cases with each condition.

In Group A the 3 cases of alimentary lymphosarcoma (Figures 16-21) were characterised by a dense mucosal infiltrate consisting of neoplastic lymphocytes which spilled over into the submucosa (Figures 19B, 20A and 21A). This involved the large intestines in all three cases and also the small intestines in Case one. There was mucosal ulceration and involvement of the lymph nodes(Figures 16,17B,18B,19A and 20B). See Tables 42, 43 and 46.

The major lesions in the 2 cases of granulomatous enteritis were small intestinal. Case 4 had multiple fibrous thickenings and mucosal ulcers (Figure 22) and Case 5 a single thickening (Figure 25). There was serosal involvement and omental adhesion in both cases. The infiltrate consisted

of lymphocytes, plasma cells, macrophages and eosinophils and extended through the full thickness of the wall (Figures 23,27B). See Tables 42, 43 and 46.

The 2 cases of eosinophilic enteritis differed in that Case 6 had the classical diffuse large intestinal and limited small intestinal involvement consisting of eosinophilic granulomata (Figures 28 and 29). In Case 7 granulomata were only present in the ileum (Figure 31) and there was a dense mucosal and submucosal eosinophil infiltrate throughout the large intestine. See Table 46.

Histopathological examination was not performed on the second of the two cases of cyathostomasis but both had a similar macroscopic picture with marked large intestinal oedema and many emerged and unemerged larvae (Figures 35 and 37). See Table 43.

Unfortunately the case of protein losing enteropathy believed to be due to phenylbutazone toxicity was quite autolytic at post mortem examination but the recognisable pathology was consistent with such a diagnosis. See Tables 42 and 43.

A diagnosis of coccidiosis was made in Case 11 based on the presence of giant microgametocytes and macrogametocytes of the species *Eimeria leukarti*, however there was apparently no cellular immune response and no mucosal distortion (Figure 38). See Tables 42, 43 and 46.

In Group B the 3 cases of enterotyphlocolitis each had a similar infiltrate at all levels of the intestines but it was marked in the large

intestines. This consisted of lymphocytes, plasma cells and eosinophils (Figures 39-42). In Case 12 there were also polymorphs and in Cases 13 and 14 the micropathology was dominated by globule leukocytes. See Table 47.

In the 2 cases of granulomatous typhlocolitis with enteritis lymphocytes, plasma cells and eosinophils were present at all levels, again the infiltrate was most marked in the large intestines where it was joined by macrophages but was confined to the mucosa and submucosa and did not involve the full thickness of the wall (Figures 45B and 46). See Table 47. This distinguished them from the cases of granulomatous enteritis in group one.

The single case of typhlocolitis had only occasional eosinophils in the small intestines considered to be within normal limits. In the large intestines however, there was a layer of macrophages, with finely granular cytoplasm just below the surface epithelium. See Table 47. These granules proved to be PAS (Periodic Acid Shiff) positive and ZN (Zeihl-Neelsen) negative (Figure 47). (See Appendix 1)

The lesions in Case 18 were dominated by peritonitis. However, microscopic examination of the small intestines revealed oedema, a marked mononuclear inflammatory cell infiltrate, villus stunting and fusion and an increase in the intraepithelial lymphocyte numbers (Figure 48).

In Case 19 the changes were confined to the small intestines where there was marked hyperplasia of the gut associated lymphoid tissue in both the jejunum and ileum. The overlying mucosa was flattened at these points and

there was a marked increase in the intraepithelial lymphocyte numbers (Figure 49). See Tables 44, 45 and 47.

In the last case, Case 20, no significant intestinal pathology was identified. There was only oedema of the small intestines and changes associated with mild parasitism in the large intestines. See Tables 44 and 45. However, the cheek teeth were extremely worn and uneven, some were missing. The remaining teeth varied in height from 0-3cm and had trapped a lot of food material and damaged the buccal epithelium (Figure 50). Their impaired function was evidenced by the long strands of unchewed hay in the stomach.

DISCUSSION - ENTEROPATHY SERIES

This series is the first to present comprehensive clinical and pathological findings in a group of horses with different chronic enteropathies and/or malabsorption.

The only previous series dealing with a number of different conditions involved almost exclusively young Thoroughbreds, confined its observations to the small intestines and did not include clinical findings or details of the large intestinal lesions (Platt 1986, Platt 1987). The pathological descriptions that were included were somewhat brief and confusing. It was not clear whether the 5 cases of chronic inflammation with eosinophils were examples of eosinophilic enteritis or the result of parasitological activity. The author appeared to favour the latter explanation. Again it was not clear whether there was transmural involvement of the intestinal wall in the 3 cases of granulomatous enteritis. Most authors consider this to be an important finding, if not a prerequisite for such a diagnosis (Cimprich 1974).

The detailed information gleaned from the present series allows interesting comparisons and connections to be made not just between the clinical and pathological findings in single cases but also between a number of examples of a single condition and between a number of conditions. Comparisons can also be made with cases recorded in the literature. A number of different breeds were represented, they were predominantly pleasure animals which reflects the local horse population in the Glasgow area but contrasts markedly with case series in the literature which deal almost exclusively with Standardbreds and

Thoroughbreds (Cimprich 1974, Platt 1986, Platt 1987). One particularly interesting finding in relation to breed concerned the 2 cases of granulomatous enteritis. One was an Arab cross and the other a pony. The condition had previously only been recorded in Standardbreds and Thoroughbreds (Cimprich 1974).

The animals in this series were all aged, only 3 being less than 10 years old (Cases 12, 18 and 19) and none being less than 6 years old. Again this contrasts with the previously published findings which predominantly concerned young adults.

The clinical findings varied but 13 of the animals from both groups of the case series had a history of diarrhoea. In each case there was extensive large intestinal involvement; small intestinal involvement was inconsistent. In 6 of the 7 cases with no history of diarrhoea there was no significant large intestinal pathology. The exception was Case 14, which had a history of weight loss without diarrhoea but a diagnosis of enterotyphlocolitis. Presumably the lesions had resulted in a protein losing enteropathy or small intestinal malabsorption but no large intestinal malabsorption and so there was no diarrhoea. Unfortunately the oral glucose tolerance test was not performed on Case 14 so this theory cannot be confirmed. Usually protein losing enteropathies do result in diarrhoea because of the resultant hypoproteinaenemia and oedema of the gut wall but this is not always the case (Roberts 1983).

The correlation found here between diarrhoea and large intestinal disease fits with the generally accepted view that diarrhoea is the result of an overload of water and electrolytes within the intestinal lumen (Roberts

1985). This can be explained by the fact that the caecum and colon must resorb the equivalent of the entire extracellular fluid volume every 24 hours (Argenzio 1975). So, although there is a reserve capacity to reabsorb 3 to 4 times that volume, if the large intestine is compromised to such an extent that its absorptive function is seriously impaired the fluid content of the faeces will dramatically increase resulting in diarrhoea and often dehydration (Roberts 1985).

More usually the term malabsorption is used in connection with small intestinal function. This is the major site of digestion and absorption of nutrients in the horse (Roberts 1985). A number of tests have been proposed eg d(+) xylose absorption test (Bolton et al 1976) starch tolerance test (Loeb, McKenzie and Hoffsis 1972), the oral lactose tolerance test (Roberts 1985) and the oral glucose tolerance test (Mair et al 1991). The oral glucose tolerance test is the most simple to perform and assay and was performed on 11 of the 20 animals. The results were abnormal in 10 of the 11. In the remaining case (Case 2) with a normal absorption curve no significant small intestinal pathology was evident although there was small intestinal oedema. Of the abnormal group one had a delayed peak and significant small intestinal pathology (Case 5). Six had a flat absorption curve. There was significant small intestinal pathology in 4 of these (Cases 1,4,6,19) and 2 had only oedema of the small intestine (Cases 11 and 20). Three cases had partial malabsorption. In 2 of these 3 significant pathology was confined to the large intestines but again with oedema of the small intestines (Cases 3 and 8) and in one case there was oedema and a mild eosinophil infiltrate in the small intestines and oedema and a more significant histiocytic infiltrate in the large intestines (Case 17).

This supports the finding that the oral glucose tolerance test is a useful, if not exact indicator of small intestinal pathology (**Mair et al 1991**). The results also suggest that oedema of the mucosa and submucosa can affect its function and should be considered significant (**Cases 3,8,11,17 and 20**) although the results in case 2 would contradict that statement.

This conclusion was also reached by **Bolton and colleagues (1976)** following their assessment of the d(+) -xylose+ absorption test. They divided their horses with abnormal absorption curves into 3 groups and the pathology of the third group, all of which had a low peak absorption value, was limited to oedema of the lamina propria. Another interesting point to note from this paper was the fact that the mucosal epithelial cells of one of the horses in the second group although normal on light microscopy, were one third normal height when examined by electron microscopy.

With reference to **Case 20** the poor state of the animal's dentition was also considered significant (Figure 50). The theory proposed was that its reduced ability to masticate had resulted in long fibrous lengths of food material which were harder to digest. These were still present in the animal's stomach after the overnight fast and may have trapped the glucose delaying gastric emptying. This cannot be proved and in the light of the findings in **Cases 3, 8 and 11** may be irrelevant, and the small intestinal oedema was the significant lesion in **case 20**.

Hypoalbuminaemia was a finding in 15 of the animals. No significant renal lesion was evident so it can be assumed that it was due to intestinal

malabsorption, excessive intestinal loss or more likely a combination of the two (Roberts 1983). Unfortunately radio labelled Cr-albumin studies (Roberts 1983) were not performed so the presence of a protein losing enteropathy cannot be confirmed in any of the cases. Only one animal (Case 2) had hepatic lesions but these were mild and unlikely to have affected protein production.

There are a number of mechanisms responsible for enteric protein loss eg ulceration or leakage through cell junctions because of mucosal infiltration and distortion (Roberts 1983). Both of these were common findings in the hypoalbuminaemic animals.

Rectal biopsies were performed on 10 animals, in only one were the findings reported as abnormal (Case 1) even though 6 of the remaining cases had large intestinal lesions at necropsy. These results would suggest that the procedure is not relevant. However, it is relatively safe and simple to perform and does not require major invasion or general anaesthesia.

Despite the number of clinical tests available a definitive diagnosis was achieved pre mortem in only 3 of the animals (Cases 8 and 9, cyathostomiasis, and Case 11, with protein-losing enteropathy secondary to phenylbutazone toxicity). A final definitive diagnosis was achieved post mortem in 11 of the animals, those in Group A, and a morphological diagnosis was made in the remaining 9, Group B, which indicated the site and nature of the lesions for comparison with other and future cases.

In the first group of cases (Group A) although each case was generally consistent with a previously described condition (**Wheeldon and Greig 1977, Roberts 1983, Jasko and Roth 1984, Lindberg et al 1985, Platt 1987**) there were a number of notable differences. The 3 cases of alimentary lymphosarcoma. (**Cases 1-3**) were all predominantly large intestinal with a history of diarrhoea. There was small intestinal involvement only in **Case 1** (Figures 17 and 19). Cases in the literature are predominantly small intestinal (**Roberts and Pinsent 1975, McConnell et al 1982, Humphrey et al 1984, Platt 1987**) although involvement of the large intestines or the entire tract is not unique (**Wiseman et al 1974, Crawley 1985, Wilson et al 1985**).

A marked plasma cell presence was noted by **Roberts and Pinsent (1975)** in 2 of their 3 cases with small intestinal involvement. This was not a feature of **Cases 1-3**. Ulceration was marked in **Cases 1 and 2** and has been previously described (**Wiseman et al 1974, Roberts and Pinsent 1975, Reef et al 1984, Crawley 1985, Wilson et al 1985**). The infiltrate varied slightly between each case but was basically composed of neoplastic lymphoid cells. Those of **Cases 2 and 3** apparently were mature while those of **Case 1** were immature blast cells (Figures 19-21).

In **Case 2**, although there was diffuse large intestinal involvement it was apparently centred on the dorsal colon where changes were most marked. Lymphoid follicles were present thoughout the infiltrated areas but were the predominant finding in the caecum where the infiltrate was much more mixed (Figure 20A). The question arises, were these follicles the first stage in involvement of that organ, or were these merely collections of reactive, but non-neoplastic lymphoid cell perhaps stimulated by the high

numbers of activated lymphocytes elsewhere? Unfortunately this question cannot be answered without the use of specific cell markers which would be required to subtype the cells and compare them to those of the dorsal colon. The technique could also be applied to the Peyer's patches of the ileum for the same reason.

In **Case 3**, although there was no gross large intestinal ulceration there was loss of surface epithelium. At these points there was invasion of the mucosa by protozoa from the normal commensal population (Figure 21B). Individual protozoa are not an uncommon finding on the mucosal surface. Presumably invasion occurred here because an altered lumenal environment had allowed the protozoa to multiply to a pathogenic level or damaged epithelium no longer formed a barrier; or more likely a combination of the two was responsible. There have been reports of protozoa being the primary cause of intestinal disease in the horse or being a contributing factor (**Gregory et al 1986**). Their significance here is unclear but they probably were an incidental finding in view of the severity of the primary lesion. There were elevated numbers of intraepithelial lymphocytes in each case, a finding not previously described in cases of alimentary lymphosarcoma but which would doubtless further compromise mucosal function, compounding the diarrhoea problem.

The 2 cases of granulomatous enteritis (**Cases 4 and 5**) fulfilled the main diagnostic criteria previously proposed (Figures 22-27) (**Cimprich 1974, Lindberg 1984, Lindberg et al 1985**). There was granulomatous infiltration and fibrosis of the small intestines, especially of the ileum (Figures 22B and 25A). This infiltrate was full thickness in many areas but most intense in the mucosa and submucosa (Figures 23). This had resulted in

flattening of the mucosa and the presence of a great many intraepithelial lymphocytes (Figures 26 and 27). Case 4 was apparently more severely affected having multiple gross lesions (Figure 22) and more advanced mucosal lesions (Figure 26). In both cases the large intestines were not directly involved and there was no granulomatous infiltrate just oedema and ulceration with elevated numbers of intraepithelial lymphocytes in Case 4.

The major contrasting features between these 2 cases and those in the literature were clinical rather than pathological. Case 4 was a 13 year old Arab cross and Case 5 was a 15 year old pony. The condition has been reported previously to affect exclusively Standardbreds and Thoroughbreds and predominantly young adults (Cimprich 1974, Bester and Coetzer 1978, Meuton et al 1978, Roberts and Kelly 1980, Hodgson and Allen 1982, Lindberg 1984, Sweeney et al 1986). Cases have been recorded in older animals, but again only in Standardbreds and Thoroughbreds (Merrit et al 1976).

The aetiology remains obscure and has been extrapolated from the theories on Crohn's disease in man, a condition which granulomatous enteritis closely resembles (Cimprich 1974). There are 2 main schools of thought. In one an as yet unidentified infectous agent is incriminated, in the other a basic defect in the animal's immune response is proposed (Cimprich 1981, Roberts 1985). Evidence exists among the equine cases for both theories. *Mycobacterium avium* has been isolated from horses with idential lesions but more usually no acid-fast organisms can be demonstrated (Cimprich 1974, Lindberg 1984). In support of the second theory reduced phagocytic activity of peritoneal macrophages has been

demonstrated in clinical cases (**Merrit et al 1976**). In Crohn's disease there is a strong familial and racial prevalence (**Kirsner 1976**) which is also apparent in the horse with the marked over representation of Standardbreds (**Cimprich 1974 Lindberg 1984**) and the report of granulomatous enteritis in 3 sibling Standardbreeds with no other unrelated animals on the stud affected (**Sweeney et al 1986**). The most likely explanation is a combination of the 2 theories (**Roberts 1985**). Individual animals, possibly because of some inherited predisposition, mount an exaggerated, abnormal immune response to an as yet unidentified agent. This agent may be as simple as a parasitic antigen or a commonly encountered bacterium and cause no harmful effects in normal, unpredisposed animals.

Eosinophilic enteritis is another condition of undetermined aetiology (**Hillyer and Mair 1992**) and is characterised by the accumulation of eosinophils in a number of tissues most notably the intestinal tract, pancreas and skin (**Lindberg et al 1985**).

The function of the eosinophil has not been fully elucidated, hampering the investigation of the condition. It is believed to be involved in protection against metazoan parasites and in the modification of hypersensitivity reactions. Unfortunately one side effect of eosinophil activation is damage to the animal's own tissues (**Hillyer and Mair 1992**). This is the most likely cause of the condition, which has been recently termed multisystemic eosinophilic epitheliotropic disease (**Hillyer and Mair 1992**). The initial agent causing the eosinophilic stimulation has still to be defined but the distribution would suggest an enteric parasite, possibly *Strongylus equinus* (**Breider at al 1985**).

The first cases described in the horse were predominantly intestinal (Pass and Bolton 1982) hence the term eosinophilic gastroenteritis. One of these cases also had skin lesions containing a similar infiltrate. Subsequent case reports dealt with both enteric and cutaneous lesions (Breider et al 1985, Lindberg et al 1985, Gibson and Alders 1987) or predominantly with cutaneous lesions (Wilkie et al 1985, Hillyer and Mair 1992). These cases were all linked by the presence of large numbers of eosinophils, often forming granulomata surrounded by macrophages and multinucleate giant cells. Pancreatic lesions consisting of fibrosis and eosinophilic granulomata were described in all but one of the reports (Gibson and Alders 1987). It appears that these cases represented the two ends of the spectrum of a single condition (Wilkie et al 1985) which is also recognised in man and termed multisystemic eosinophilic epitheliotropic disease (Hillyer and Mair 1992).

One paper considered the condition to be closely linked to granulomatous enteritis (Sweeney et al 1986) but there are too many consistent differences in both clinical presentation and pathology for this to be likely (Lindberg et al 1985).

The 2 cases identified in the Enteropathy Series (Cases 6 and 7) varied quite markedly in their pathology and neither had any cutaneous involvement. Case 6 had all the classical lesions (Figures 28-30) with diffuse involvement of the large intestines, limited involvement of the small intestines and pancreatic fibrosis (Lindberg et al 1985). Discrete eosinophilic granulomata were present in the large intestinal mucosa, proximal small intestinal mucosa, intestinal lymph nodes and pancreas (Figures 29 and 30). There was dilatation and fibrosis of both the bile

and pancreatic ducts and eosinophil infiltration of the major and minor duodenal papillae. There were changes throughout the small intestinal mucosa consisting of mononuclear cell infiltration, villus stunting and oedema which although apparently mild was enough to cause malabsorption.

In contrast in the second case, **Case 7**, although there was again diffuse large intestinal involvement it consisted of a dense mucosal and submucosal eosinophil infiltrate. Eosinophilic granulomata were confined to the ileum (Figure 31). There was eosinophil infiltration of the mucosa and submucosa thoughout the small intestine along with a mononuclear cell infiltrate and villus stunting (Figures 32 and 33). The intestinal lymph nodes contained many eosinophils in their capsules and connective tissue trabeculae (Figure 34).

The mucosal granulomata were particularly interesting in both cases. They sat on the muscularis mucosa within depressions lined on either side, in most instances, by intact epithelium (Figures 29 and 31). These lesions appeared remarkably discrete, although there was also more superficial infiltration of the large intestinal mucosa in **Case 6** which had apparently caused surface epithelial loss and ulceration.

It has been proposed that the pancreatic lesions as found in **Case 6** (Figure 30B) are just the horse's nonspecific response to pancreatic injury (**Jubb, Kennedy and Palmer 1985**). Pancreatic disease is apparently rare in the horse so comparison is difficult. Neither animal showed any convincing evidence of parasitism and the pathological findings gave no clue as to the aetiology. There has been one report of the successful

treatment of this condition by the use of steroid therapy (**Gibson and Alders 1987**) but the outcome is usually fatal (**Hillyer and Mair 1992**).

One recently recognised cause of intestinal disease in the horse is the small Strongyle or Cyathostome (**Duncan 1985b**). It exerts its effects, usually in the spring, by *en masse* emergence of inhibited larvae from the large intestinal mucosa (**Love 1992**).

Examination of sections of large intestinal mucosa from **Case 8** revealed that there was remarkably little reaction directed specifically against the inhibited larvae (Figure 36B) and examination of other horses has revealed that quite large numbers can be tolerated without signs of disease. Occasionally granulomata form around small fragments of parasite, especially in the submucosa. This was also noted by **Harmon and colleagues (1986)**.

It is the mass emergence of larvae and resultant mucosal damage which produces clinical signs. The function of the large intestinal mucosa is compromised resulting in diarrhoea (**Blackwell 1973**). Also the damaged mucosa is now no longer a barrier to the loss of protein thus resulting in hypoalbuminaemia. This in turn contributes to the oedema of the large intestinal wall (Figures 35 and 36A) further compromising the mucosal function. There is also oedema of the small intestines which probably caused the partial malabsorption demonstrated in **Case 8** (**Love et al 1992**).

Although the condition is most often recorded in young animals (**Blackwell 1973, Jasko and Roth 1984, Giles et al 1985**), any animal with a previous grazing history can be affected. The 2 cases in the present series were

aged 15 and 13. No predisposing cause was identified in **Case 8**, not it seems that one is required, but **Case 9** had recently received an anthelmintic treatment for the first time in years. It was proposed that this treatment killed the adult cyathostomes present within the gut lumen. No treatment apparently affects the inhibited stages so these subsequently emerged *en masse*, having lost the inhibitory effects exerted by the presence of adults, thus causing disease (**Chiejina and Mason 1977**).

The paper published by **Jasko and Roth (1984)** apparently contradicts both the present findings, and those observed by **Giles and colleagues (1985)**. They described a granulomatous colitis due to the presence of inhibited L4 cyathostome larvae, ie there was a marked inflammatory response and no mass emergence. It may be that the life cycle is different in North America because of different climatic conditions (**Love 1992**). Cyathostome infection appears to be common in the United Kingdom, occasionally leading to clinical disease which is rarely fatal (**Love et al 1992**).

The single case of protein-losing enteropathy, although not an example of a chronic enteropathy, was included because it presented with a history of weight loss and there was malabsorption in the form of diarrhoea. The diagnosis of Phenylbutazone (PBZ) toxicity as made in **Case 10** was a clinical one from the finding of marked hypoalbuminaemia and from the animal's history. Unfortunately the carcase had already undergone a degree of post-mortem degeneration at time of examination. The lesions still evident which corroborated this diagnosis were massive intestinal oedema and if not actual mucosal ulceration in the large intestine there were apparently areas of necrosis and petechiae. There was no marked

inflammatory or neoplastic mucosal infiltrate and no convincing evidence of parasitism (Snow et al 1981).

Phenylbutazone is known to be far more toxic to ponies than to horses. (Snow et al 1981). The dose administered in this case was not known and it may be that the animal, a Welsh cob, was dosed appropriately for its height and weight, as a horse, whereas being a native breed it should have been considered to be a large pony and given a lower dose.

Although supportive therapy including intravenous administration of plasma in an attempt to raise the total protein level was employed the outcome was fatal, the final event being the extension of a partial thickness rectal tear to a full thickness tear and resultant peritonitis. The partial thickness tear had been identified by the clinicians at presentation and the post mortem examination confirmed that it had been present for some time before death. The extremely fragile, oedematous gut apparently tore through without the intervention of rectal examination or external trauma. No renal lesions were identified in this case (McKay et al 1983, Collins and Tyler 1984) but subtle changes within the kidneys or indeed within the gastrointestinal tract could have been masked by the post mortem degeneration.

In the final case in Group A, Case 11 an example of coccidiosis, the lesions present failed to explain some of the clinical findings. The animal had lost weight over a 5 month period and was found to have complete malabsorption following the oral glucose tolerance test. Microgametocytes and macrogametes of *Eimeria leukarti* were identified within the small intestinal villus lamina propria, sometimes 2 or 3 to a

single villus (Figure 38). However, there was no associated cellular response and apparently no epithelial damage. The only additional finding was mild oedema of both the small and large intestines. In a previously published case of *Globidium leukarti* infection in a horse the findings were similar but the oedema was much more marked at the villus tips rendering them "club" shaped and the infection seemed heavier. Again there was little or no cellular response (**Wheeldon and Greig 1977**). It would appear that the parasite has a marked effect on intestinal function, disproportionate to the physical changes seen.

As stated earlier **Bolton and Colleagues (1976)** considered oedema of the lamina propria to be sufficient to cause malabsorption. They also found abnormalities when they examined their tissue samples under the electron microscope that had not been seen with the light microscope. Such a technique might well prove beneficial to further our understanding of this condition.

In the cases in Group B the lesions found did not resemble those described for any previously recognised conditions and only a morphological diagnosis was achieved.

In the first 3 cases, **Cases 12 - 14**, a similar infiltrate was present throughout the intestinal tract, although it was most marked in the large intestines, and a diagnosis of enterotyphlocolitis was made. No cause was found to explain the presence of the infiltrate (Figures 39-42).

In **Case 12**, the infiltrate was predominantly composed of lymphocytes, plasma cells and eosinophils but included neutrophils (Figure 39). The

neutrophils were probably present in the large intestines in response to the superficial necrosis and bacterial invasion. However, in the small intestines the epithelium was intact. The oral glucose tolerance test was not performed so no assessment was made of small intestinal function. Diarrhoea was a feature of the clinical history demonstrating that despite the lack of mucosal distortion the large intestinal infiltrate was sufficient to impair the function of that organ.

In Cases 13 and 14 the infiltrate was dominated by globule leukocytes which are believed to be discharged mast cells (Figures 41 and 42). This suggests some sort of allergic reaction, mast cells being central to Type I (anaphylactic type) hypersensitivity. However mast cells are also central to the Ige mediated destruction of parasites (**Cotran, Kumar and Robbins 1989**). Every horse kept under natural field conditions will be exposed to parasites however good the anthelmintic regime yet these two animals, Cases 13 and 14 were the only ones to have a marked globule leukocyte infiltrate and only occasional mast cells were encountered in other animals.

Eosinophils and plasma cells are also required for the Ige-mediated process. Both of these cell types were a feature of the infiltrate. Another point to note was the presence of eosinophils and mast cells within the epithelium in Case 13 this was a unique finding. It may be that these 2 animals were demonstrating an allergic response to a slight intestinal parasite burden when other, unsensitised animals only develop a more normal, protective immune response which would not require large numbers of mast cells. There was evidence of previous parasitic activity in both cases, *Haemomelasma ilei* in Case 13 and cranialmesenteric arterial

thrombosis in **Case 14**. Parasites were not obvious within the intestinal lumen or walls at post mortem examination. The pathology of **Case 14** was complicated by a rectal tear, peritonitis and necrosis and sloughing of part of the small intestinal mucosa. Whether it was these acute, probably terminal events which induced the marked reaction within the Gut Associated Lymphoid Tissue (GALT) and lymphocytolysis or whether this was part of the more long standing disease process is not clear. It is probable that the rectal tear was precipitated by the marked intestinal oedema, but the oedema would also almost certainly have been exacerbated by the resultant peritonitis. So in short, although the enterotyphlocolitis was chronic in nature in all 3 cases there were superimposed, acute lesions in **Case 14** which precipitated the animal's demise.

In the next grouping of cases, **Cases 15 and 16**, the infiltrate differed between the small and large intestines. Lymphocytes, plasma cells and eosinophils were present at all levels but in the large intestines where they were most numerous they were joined by macrophages (Figures 45B and 46). This finding was reflected in the morphological diagnosis chosen, granulomatous typhlocolitis, the more significant lesion, and enteritis. This differentiated these cases from **Cases 12, 13 and 14** and gave some information about the nature of the large intestinal infiltrate. It also distinguished these cases from the 2 cases of granulomatous enteritis, **Cases 4 and 5** even though the infiltrate was similar. In **Cases 15 and 16** the large intestinal infiltrate although granulomatous in nature lacked distinct organisation and no granulomata *per se* were found. Also the infiltrate was confined to the mucosa and submucosa with no muscle or serosal involvement. In contrast granulomatous enteritis is predominantly

small intestinal with an infiltrate that is transmural in nature (Cimprich 1974).

In Case 15 there was marked large intestinal ulceration (Figure 43). The ulcers were not sharply defined and punched out but instead the edges sloped with the mucosa gradually increasing in thickness and covered by attenuated epithelium. This suggested attempted healing. There was only superficial epithelial loss and ulceration in Case 16. Only the ileum and small colon were examined histopathologically in Case 16 so the extent of the lesions cannot be assessed. Both animals were diarrhoeic which correlates well with the large intestinal lesions found, but small intestinal function was not tested. Case 15 was hypoalbuminaemic, the extensive large intestinal ulceration would explain the protein loss through exudation of protein rich serum and lymphatic fluid. Again in neither case was a cause for the mucosal infiltrate or structural changes identified. It appears that a definitive diagnosis of nonspecific granulomatous colitis or typhlocolitis would be appropriate for these cases.

In Case 17 the changes were extremely limited. A band of pale, histiocytic cells was present in the lamina propria of the large intestines, below the surface epithelium (Figures 47A and 47B). These cells proved to be macrophages with pale, finely granular cytoplasm. These granules were quite uniform and did not resemble cell debris. The use of further staining techniques revealed that they were PAS (Periodic Acid Schiff) positive (Figure 47C) and ZN (Ziehl-Neelsen) negative (see Appendix I) but they could not be typed further. The only other finding was oedema of the submucosa thoughout the intestines and apparently an

increase in mucosal thickness in the ileum but this is an extremely subjective observation. No other lesions were observed to account for the malabsorption found clinically so presumably, as discussed previously, this was due to the small intestinal oedema. This animal would be another candidate for further examination, such as electron microscopy, to study the mucosal surface topography and macrophage granules in further detail.

The pathology in **Case 18** was dominated by both an acute fibrinous peritonitis and a chronic peritonitis with abscessation (Figure 48B). The cause was not established. In addition there was small intestinal oedema and large intestinal mucosal ulceration. However, microscopic examination of the small intestinal mucosa revealed a marked mononuclear cell infiltrate which had resulted in stunting and fusion of the villi, many of which had florid frond-like tips. There was also oedema and an increase in intraepthelial lymphocytes (Figure 48A).

There are three possible, obvious explanations for the small intestinal pathology:

1) The infiltrate was a response to altered gut motility because of adhesions which resulted in stasis of the content and absorption of abnormal substances.

2) The changes were merely the mucosa's response to the toxins from the serosal lesions.

3) There was a primary chronic enteropathy.

There is evidence for and against each theory. There was almost certainly altered gut motility as evidenced by episodes of colic but examination of small intestinal samples from cases of grass sickness does not reveal an infiltrate and there definitely is gut stasis in these animals. The cell type in the mucosal infiltrate was similar to that of the chronic peritonitis but why were the submucosa and muscle layers not involved? A primary enteropathy would not explain the peritonitis but there may well be two separate conditions.

No assessment was made of small intestinal function so no comment can be made about the significance of the lesions. Hypoalbuminaemia was recorded, this could be explained both by the peritonitis and large intestinal ulceration. Both would be sites of potential protein loss from the blood stream. However, no explanation for the colonic ulceration can be given as only oedema, which could well have been secondary to the ulceration, and eosinophils were seen. Again this case merits further investigation to subtype the mononuclear cell infiltrate and to compare the mucosal changes with other cases of peritonitis of known aetiology.

In **Case 19** there was no mucosal infiltrate *per se* but hyperplasia of the GALT (Figure 49). Normally lymphoid nodules (Peyer's patches) are only observed singly and in the ileum. In **Case 19** large reactive nodules were present in groups of 4 or 5 in the mucosa, muscularis mucosa and submucosa of both the jejunum and ileum. They coalesced to form large aggregations spilling lymphocytes and plasma cells into the surrounding tissue. At these points the overlying mucosa was flattened with no villi and there was a marked increase in intraepithelial lymphocyte numbers. These areas must represent the focal areas of flattened mucosa seen grossly. Although

the abnormal areas seem localised there were a great many of them. This combined with the mucosal and submucosal oedema present throughout the small intestines would explain the malabsorption detected clinically. Interestingly the GALT in the large intestines was reactive but within normal limits although there was no obvious reason for this. There were increased numbers of protozoa apparent but these were on the mucosal surface or in crypts and had not invaded the lamina propria.

The nature of the small intestinal lesion was not established and no agent was identified that might have induced such a reaction. It did however resemble two conditions identified in man, low grade B-cell mucosal associated lymphoma and florid lymphoid hyperplasia of the terminal ileum (**Rubin and Isaacson 1990**). The former only very rarely metastasises and tends to remain quite localised. It is derived from and closely resembles normal reactive Peyer's patches with a pale central active area, a marginal zone and an increase in intraepithelial lymphocytes in the overlying mucosa. It is the marginal zone and the intraepithelial component that are neoplastic (**Spencer, Diss and Isaacson 1990**). Florid reactive lymphoid hyperplasia of the terminal ileum can only be distinguished from low grade lymphoma by the use of immunocytochemistry to detect light chain restriction in the latter. **Case 19** again merits further study with specific cell markers to establish whether or not there is a monomorphic population and to identify the condition.

Examination of **Case 20** was singularly unrewarding. As discussed earlier there was clinical evidence of malabsorption which could only be explained by the small intestinal oedema, the poorly masticated nature of the stomach content (Figure 50) or a combination of the 2. Although there was

evidence, in the form of a large cranial mesenteric arterial aneurysm, of previous large strongyle activity none was apparent at time of post mortem examination. The cyathostome burden was considered insignificant. It may be that subtle epithelial changes were missed on examination with the light microscope and examination of samples with an electron microscope might prove useful (Bolton et al 1976).

Close examination of the cases in Group B revealed that although they were dissimilar to conditions previously reported in the horse it was still possible to make a diagnosis on morphological grounds and in some cases propose a firm diagnosis by extrapolation from man (Case 19) or by defining them as conditions in their own right (Cases 15 and 16).

Certain findings were common to the majority of cases in both groups but oedema and ulceration has been noted previously in connection with a number of conditions (Roberts 1985). In contrast the presence of increased numbers of intraepithelial lymphocytes has not been commented upon previously. These are considered an important finding in human intestinal pathology (Marsh 1990) and believed to affect epithelial function and distort mucosal architecture. Once again this is a topic worthy of further investigation.

In conclusion, it is clear from the results of this case series that all the major inflammatory bowel diseases do occur in the United Kingdom and are not confined to Thoroughbreds and Standardbreds as the previous reports would suggest (Cimprich 1974, Lindberg et al 1985). What has also emerged is that a number of as yet unclassified conditions exist (Cases 12 -20). Further tools such as electron microscopy and specific cell markers

are required to shed further light, not just on the second group of cases, but also on the first group in an attempt to establish their aetiology and further our understanding of them.

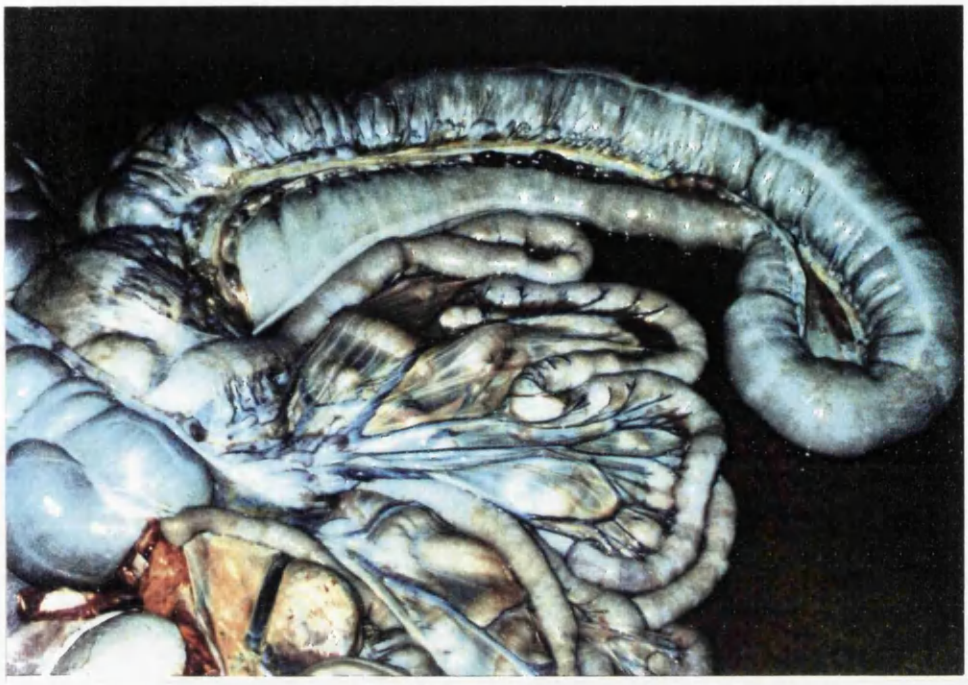
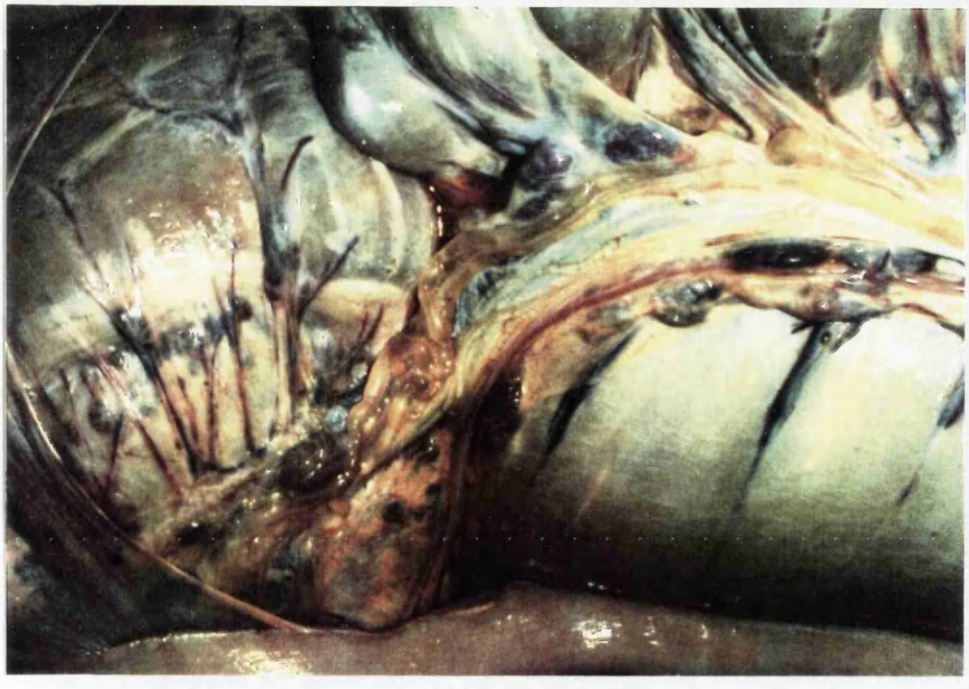


Fig. 16 Case No. 1. Lymphosarcoma A The mesenteric and colonic lymph nodes are markedly enlarged



B Detail of the caecocolic junction. The lymph nodes are enlarged and the lymphatic vessels dilated and tortuous

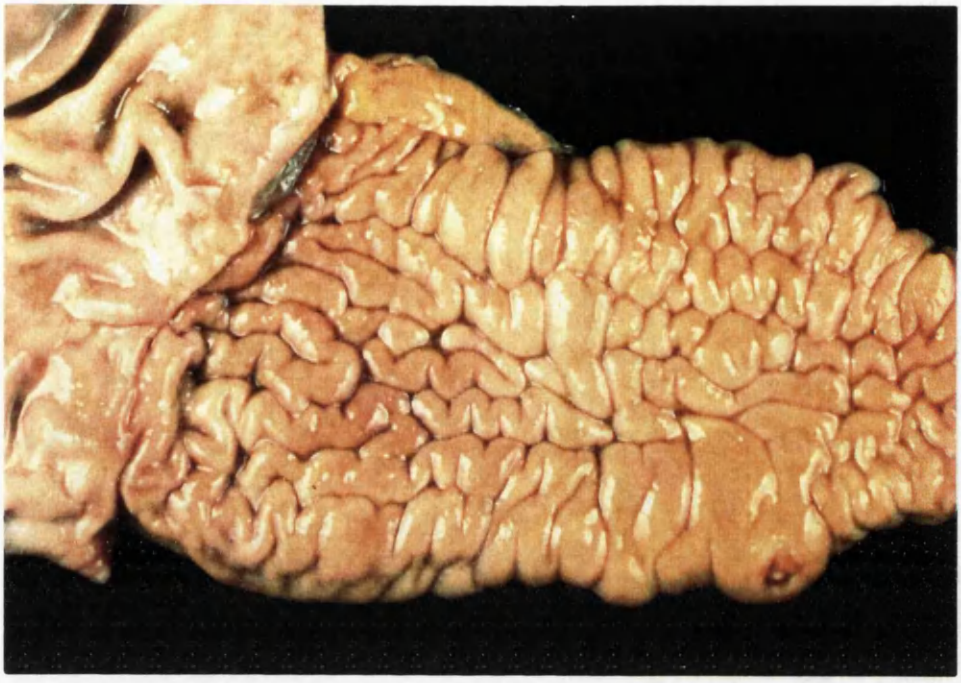
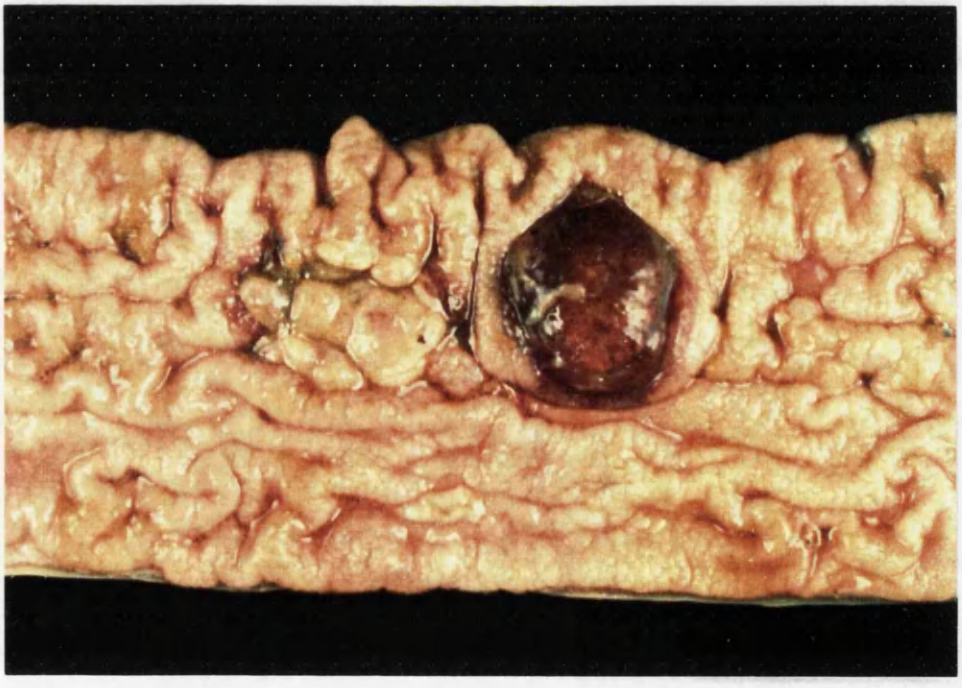


Fig. 17 Case No. 1. Lymphosarcoma A The duodenal mucosa is extremely oedematous



B The small intestinal mucosa is oedematous and the area of diphtheresis overlays a Peyer's Patch

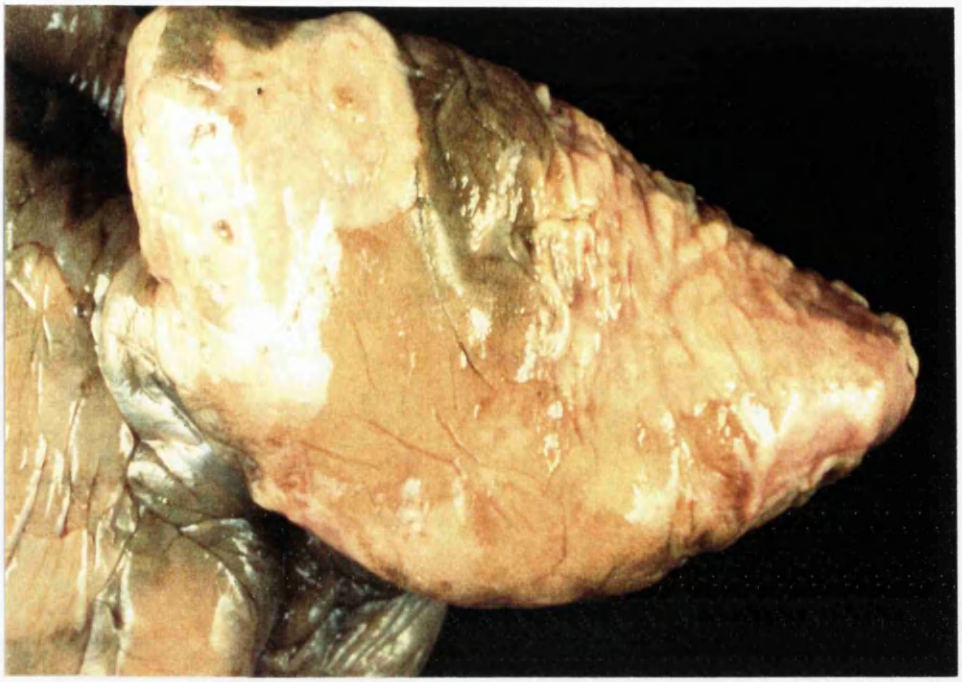
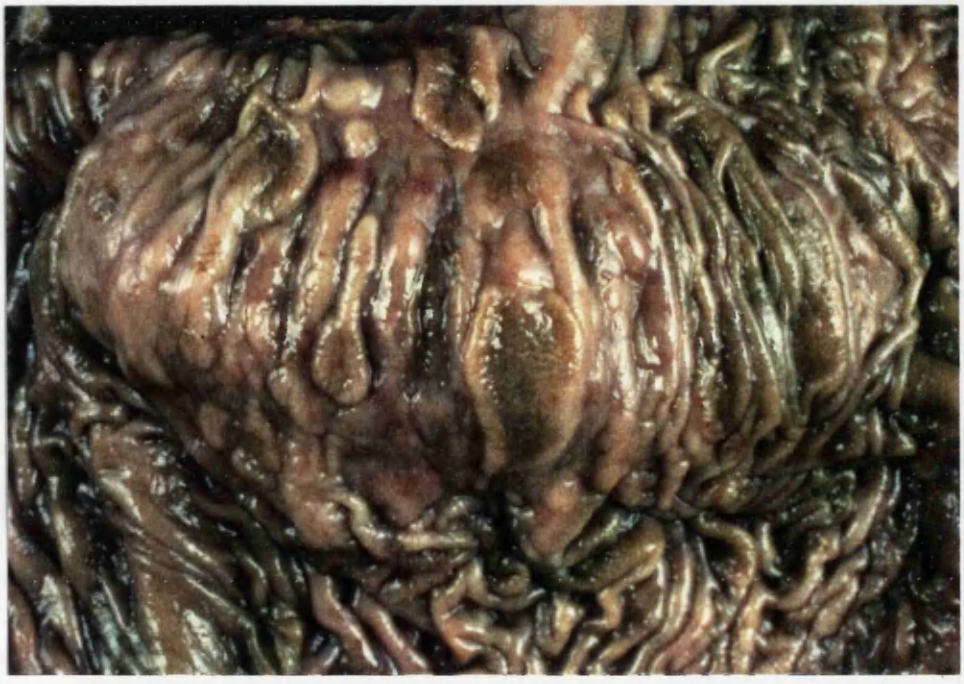
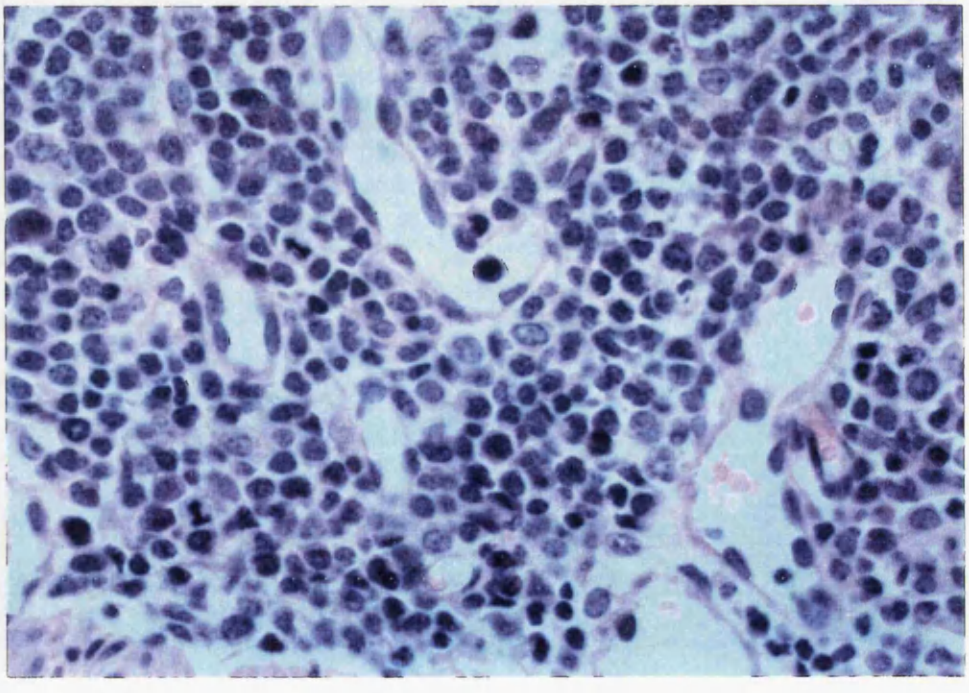


Fig. 18 Case No. 1. Lymphosarcoma A The caecal apex is infarcted and necrotic. It has been inverted to show the mucosal surface



B The colonic Peyer's patches have become thickened plaques and the mucosa is ulcerated

Fig. 19 Case No. 1. Lymphosarcoma A There is ulceration of the small intestinal mucosa. The adjacent villi are short and broad and there is an increase in the crypt to villus ratio (H and E X 4)



B The small intestinal lamina propria contains a dense infiltrate of large lymphoblastic cells will a high mitotic rate (H and E X 40)

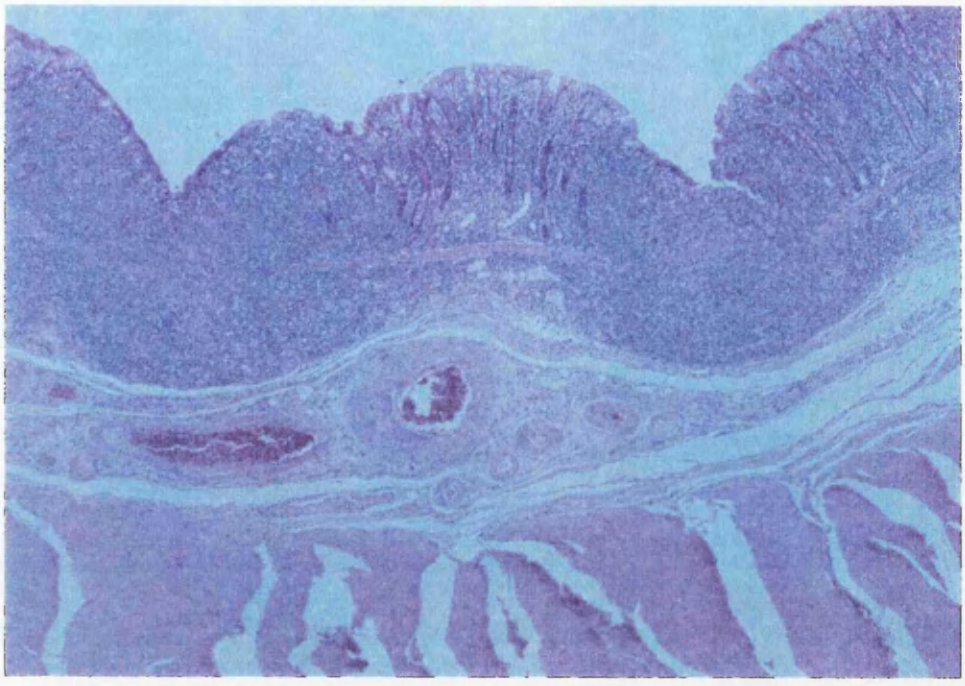
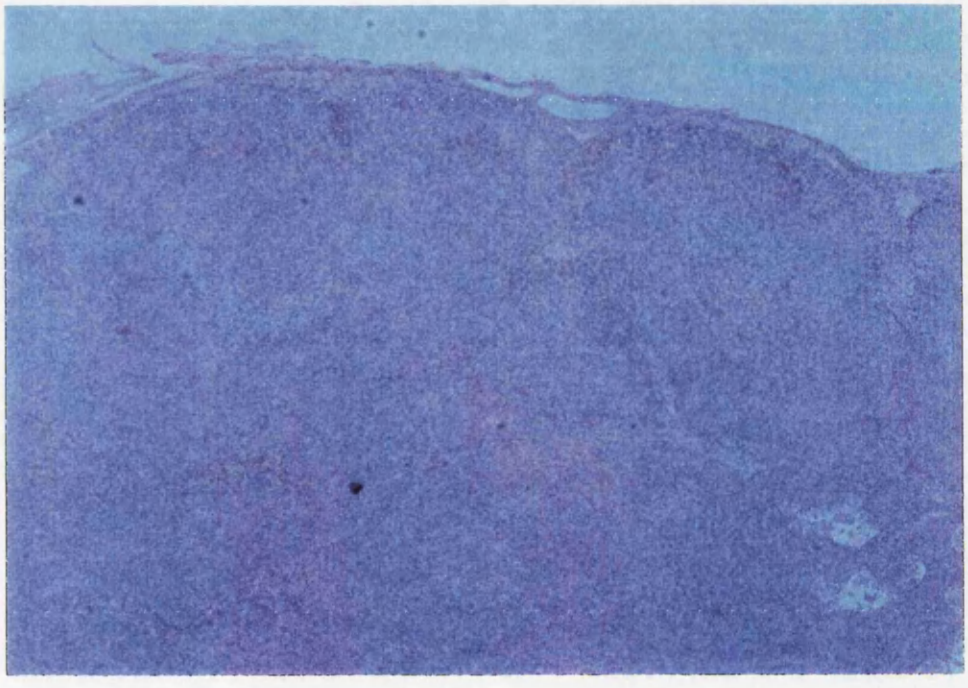


Fig. 20 Case No. 2. Lymphosarcoma A There is diffuse infiltration of the large intestinal mucosa by neoplastic lymphoid cells. There is focal involvement of the submucosa with follicle formation (H and E X 4)



B There is loss of normal architecture in the colonic lymph nodes with solid sheets of neoplastic lymphoid cells in the medulla (H and E X 4)

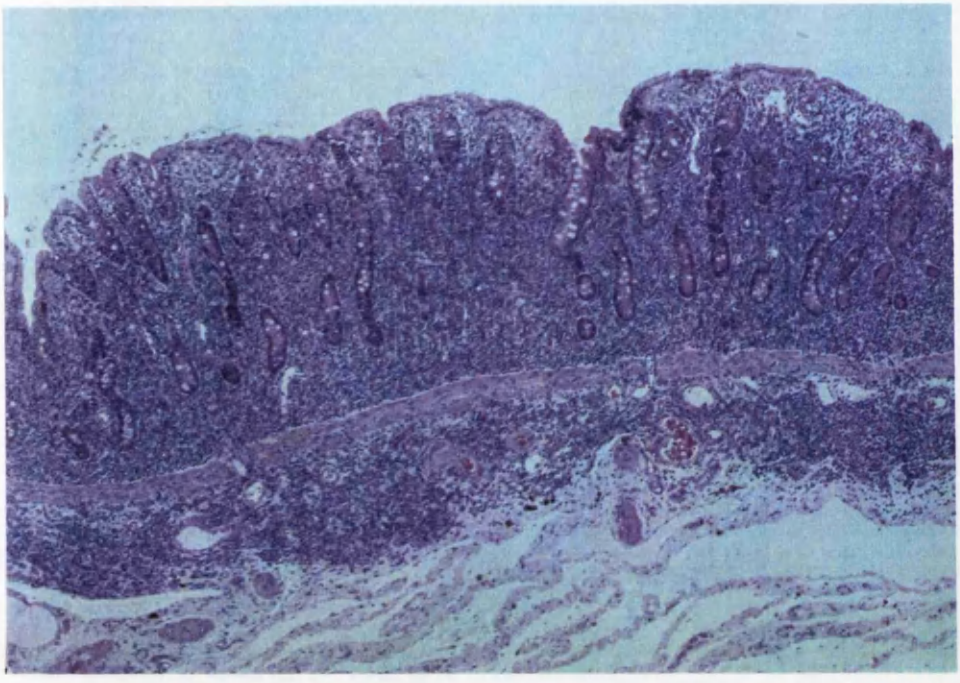
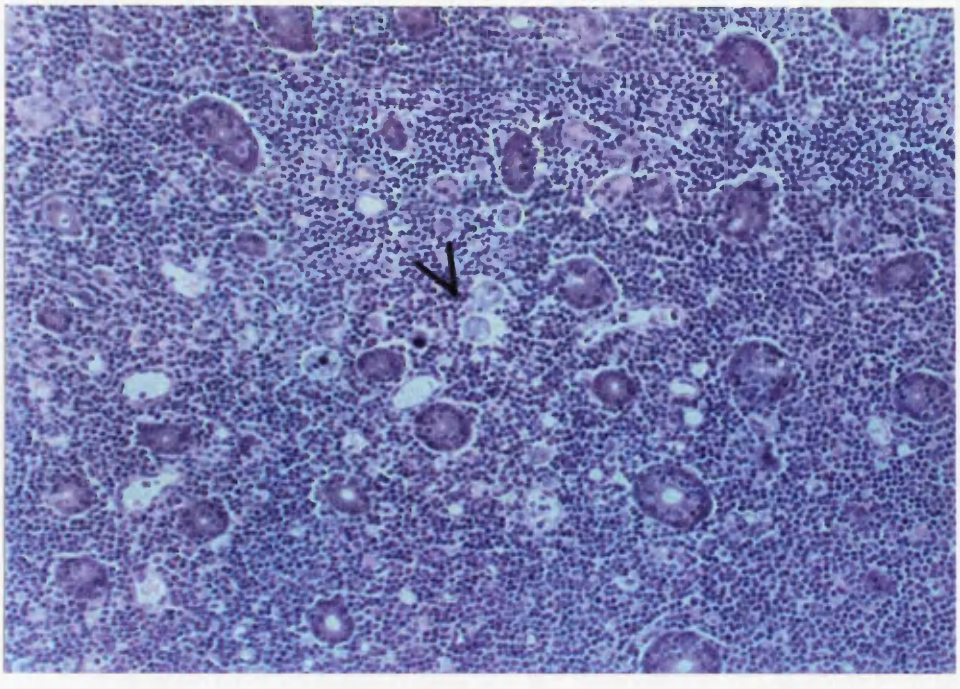


Fig. 21 Case No. 3. Lymphosarcoma A There is a dense large intestinal mucosal infiltrate of neoplastic lymphoid cells. This has "lifted" the crypts off the muscularis mucosa and spilled over into the submucosa (H and E X 4)



B The caecal mucosa has been invaded by commensal protozoa (arrow) (H and E X 20)

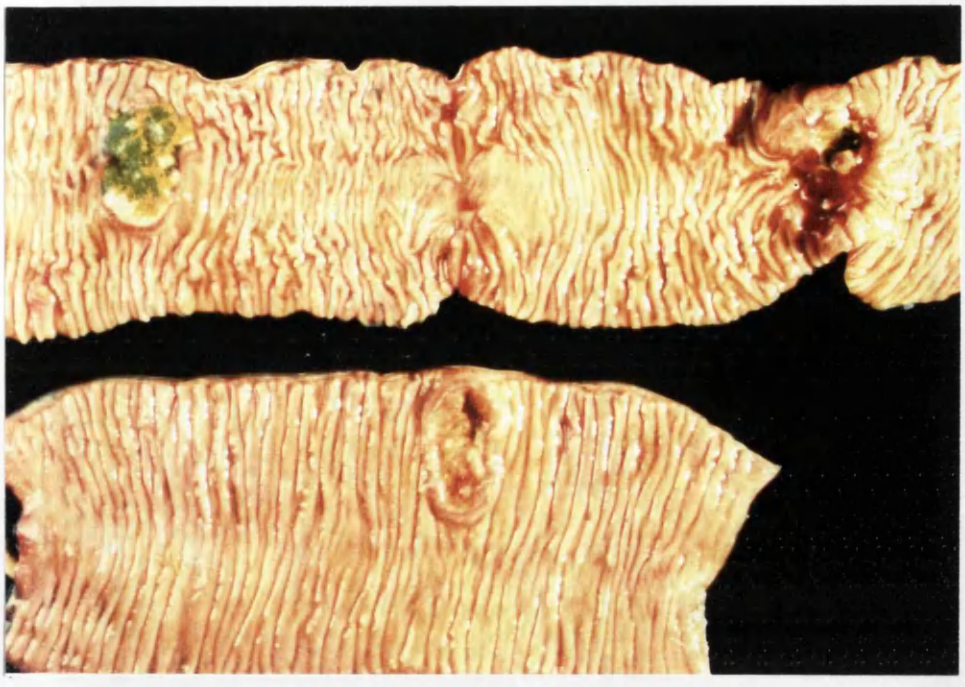
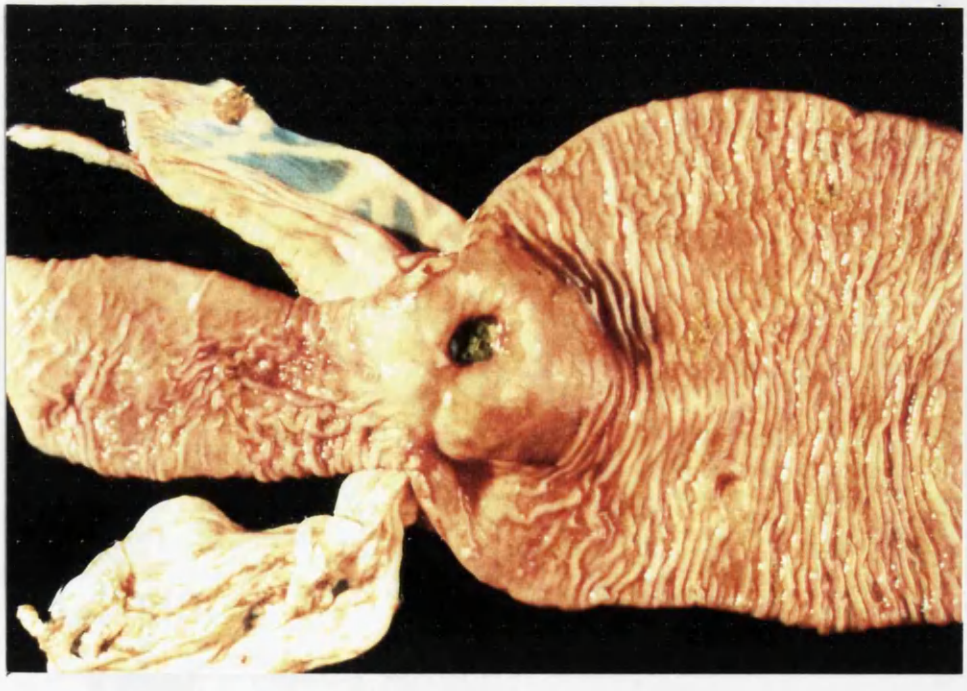
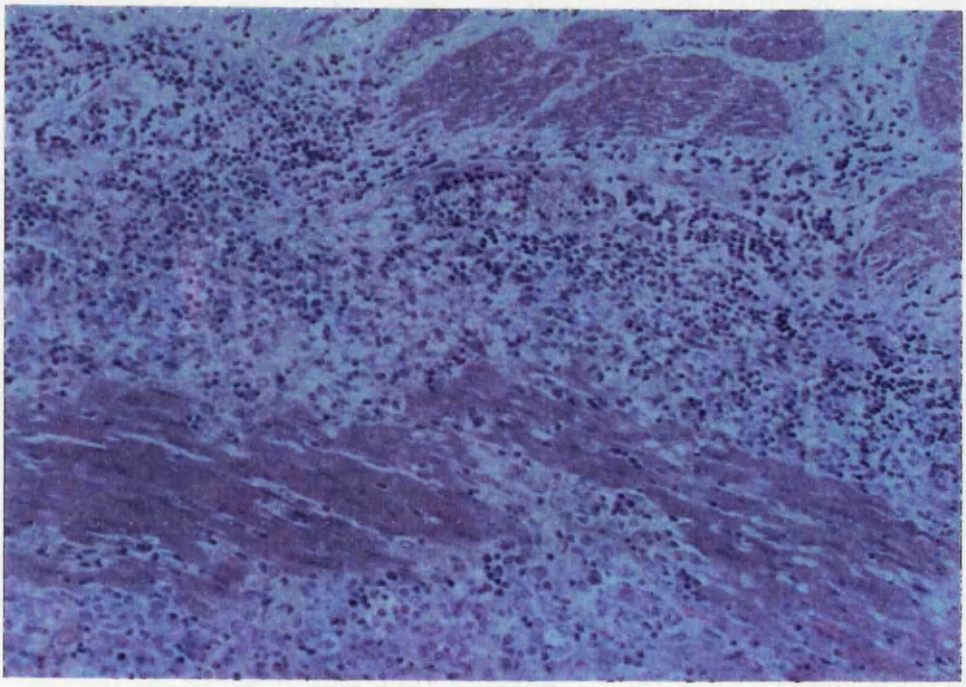


Fig. 22 Case No. 4. Granulomatous Enteritis A The small intestinal mucosa contains deep necrotic ulcers with surface diphtheresis and scarring



B One of the three fibrosed ileal nodules opened to reveal mucosal ulceration. The omental adhesions are due to localised peritonitis and the proximal dilitation, on the right, is due to obstruction

Fig. 23 Case No. 4. Granulomatous enteritis A The small intestinal villi are short, broad and oedematous. the granulomatous infiltrate involves the epithelium, lamina propria, muscularis mucosa and submucosa (H and E X 10)



B At focal points the granulomatous infiltrate is full thickness involving the outer muscle layers as well (H and E X 20)

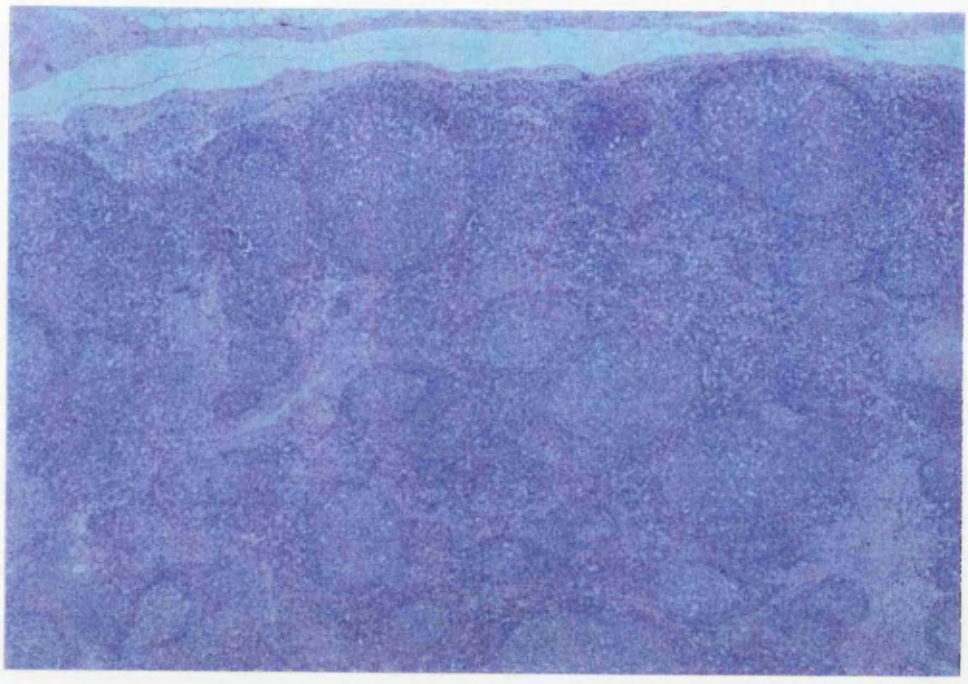


Fig. 24 Case No. 4. Granulomatous enteritis. In the mesenteric lymph node there is B-cell expansion with reactive follicles in both the corex and the medulla. Death of individual cells at the follicle centres has led to a "starry-sky" effect (H and E X 4)

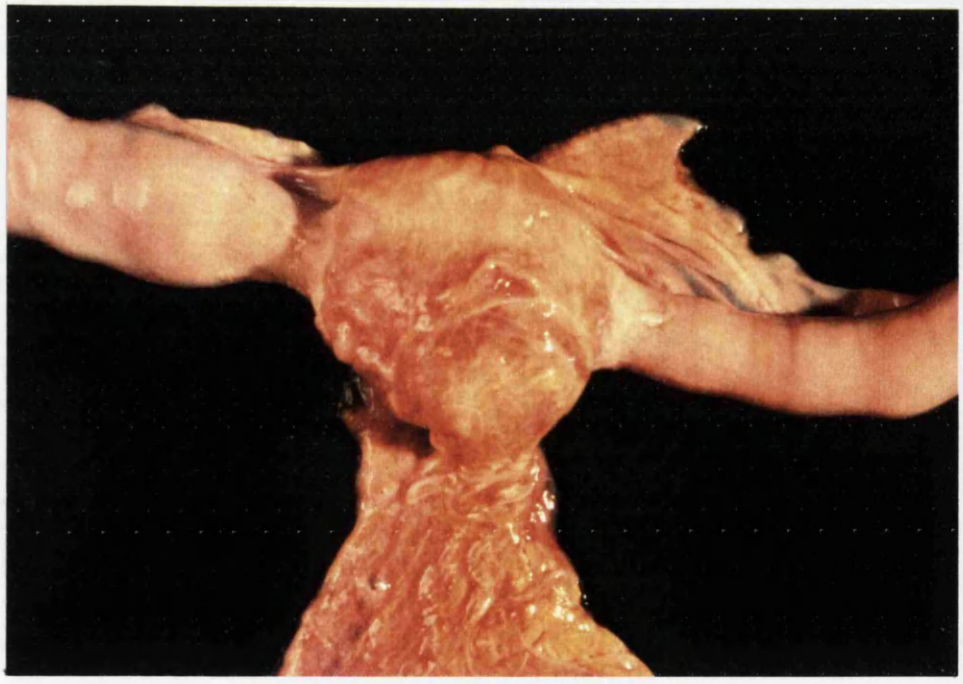
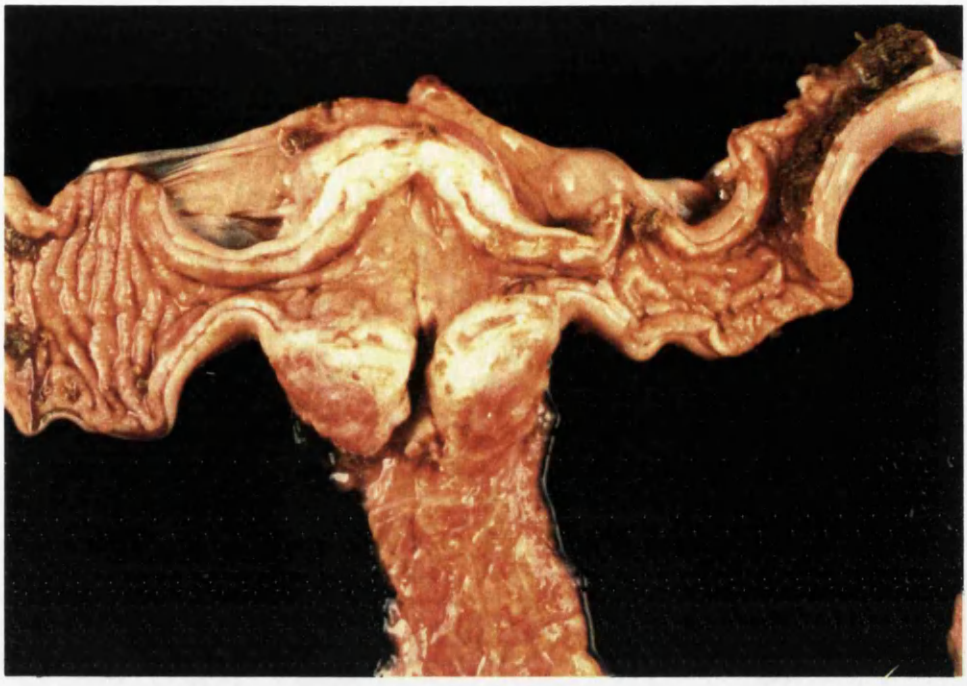


Fig. 25 Case No. 5. Granulomatous enteritis. A A focus of fibrosis in the ileum causing partial obstruction with dilatation of the proximal intestine, on the left. There is localised peritonitis which has led to adhesion of the omentum



B The ileal nodule has been opened to reveal deep ulceration and thickening of the adjacent mucosa

Fig. 26 Case No. 5. Granulomatous enteritis. Adjacent to the ileal nodule there is mucosal flattening, subtotal villous atrophy, dilated, dysplastic crypts and attentuated surface epithelium (H and E X 4)

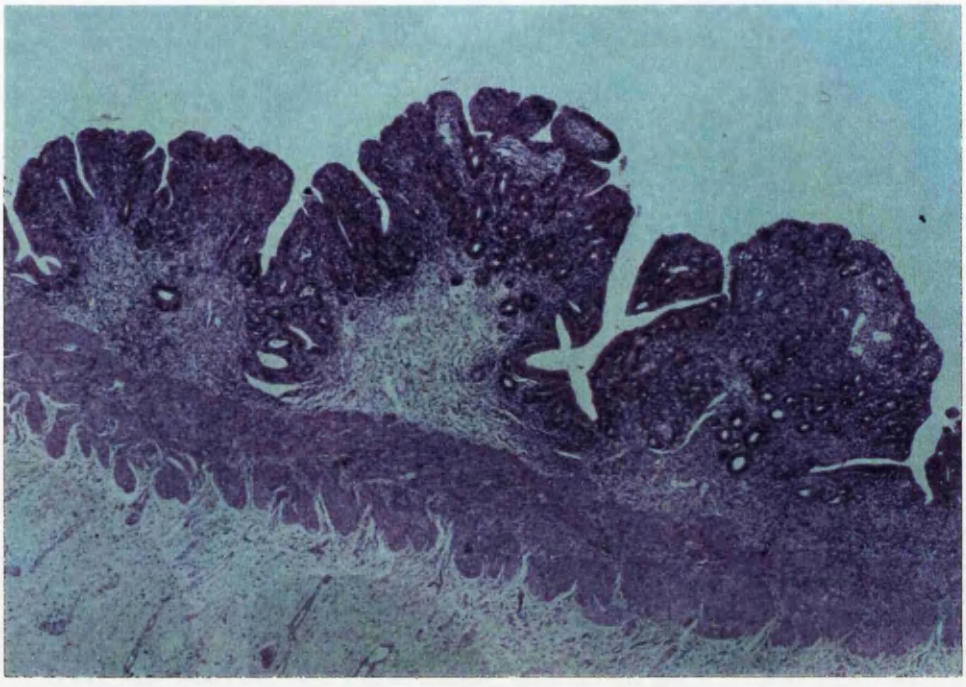
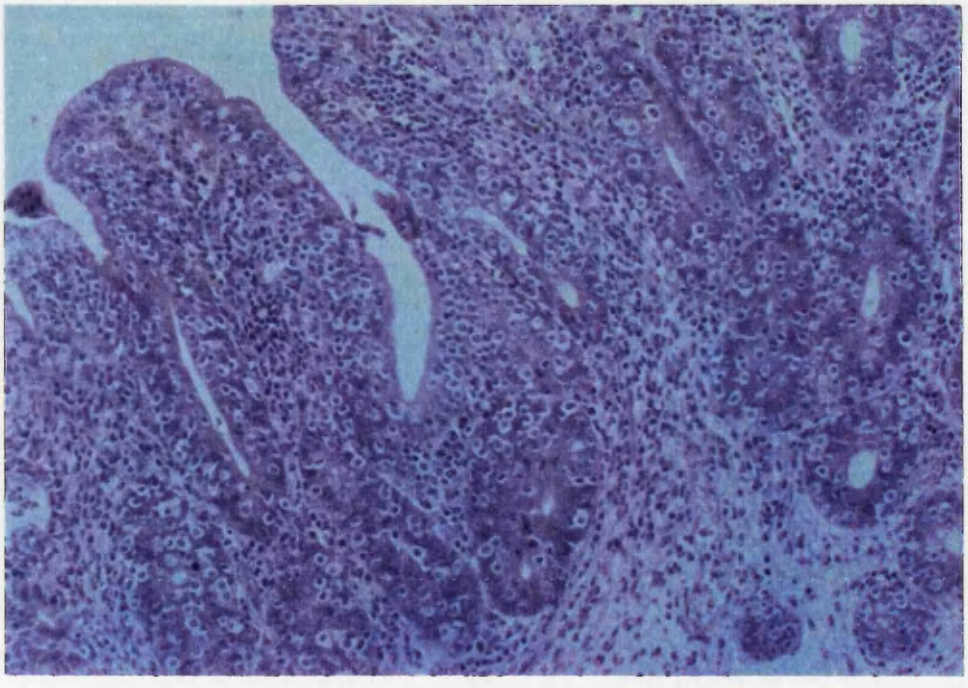


Fig. 27 Case No. 5. Granulomatous enteritis. A The jejunal mucosa is irregular with stunting and fusion of villi and loose, oedematous fibrous tissue in the lamina propria (H and E X 4)



B Higher power view revealing an intense lymphoid epithelial infiltrate with similar cells in the lamina propria (H and E X 20)

Fig. 28 Case No. 6. Eosinophilic enteritis. The large intestinal mucosa is peppered with dark granular, apparently ulcerated areas up to 1cm in diameter

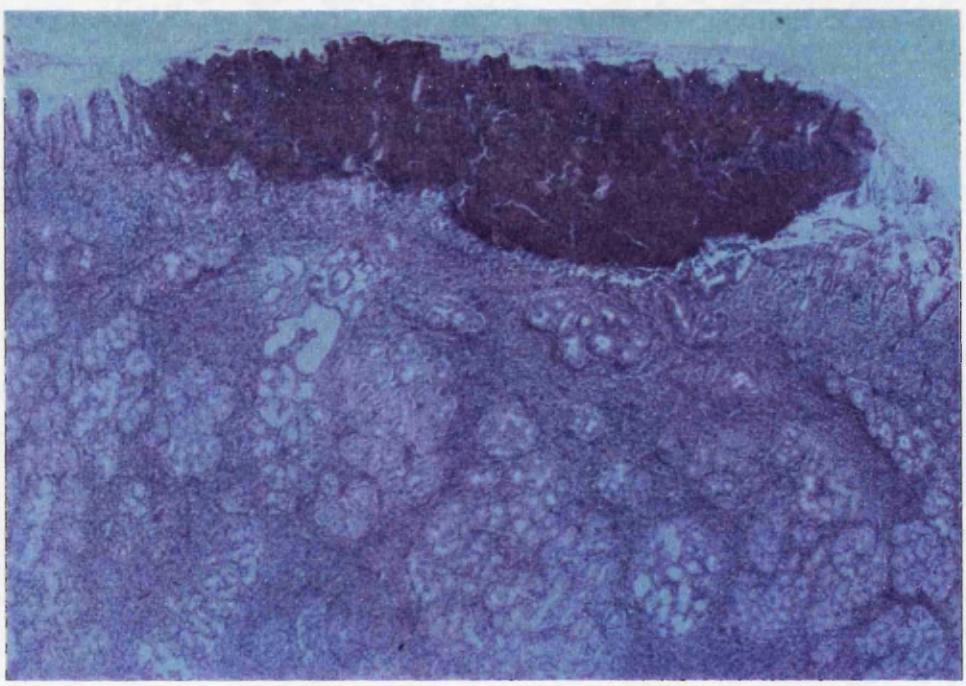


Fig. 29 Case No. 6. Eosinophilic enteritis. There is a mass of eosinophils sitting in a depresion in the mucosa of the major duodenal papilla. Eosinophils have also infiltrated the submucosal tissues (H and E X 4)

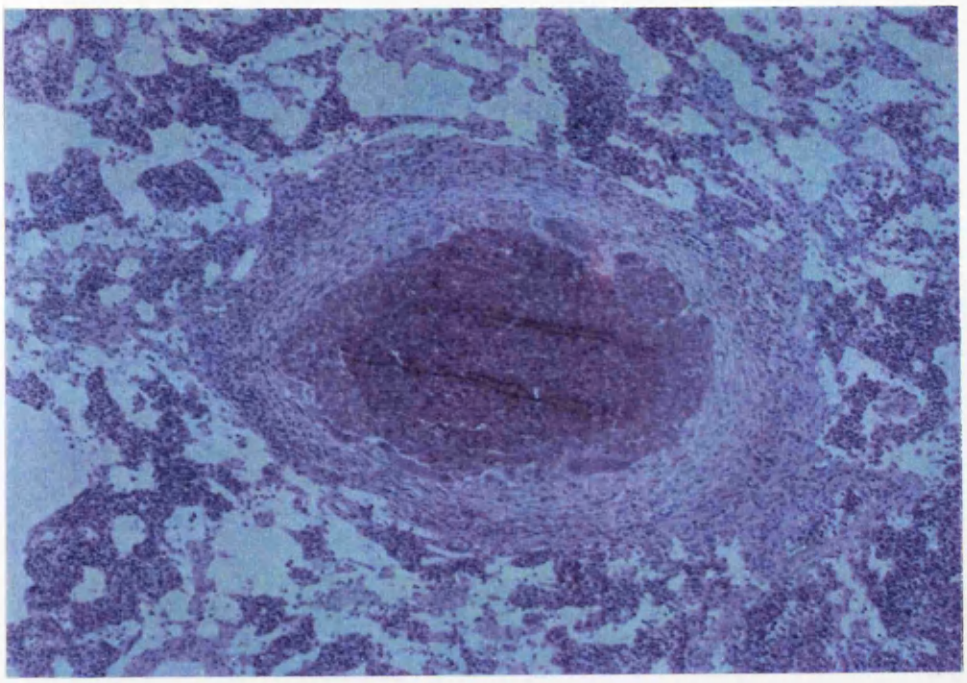
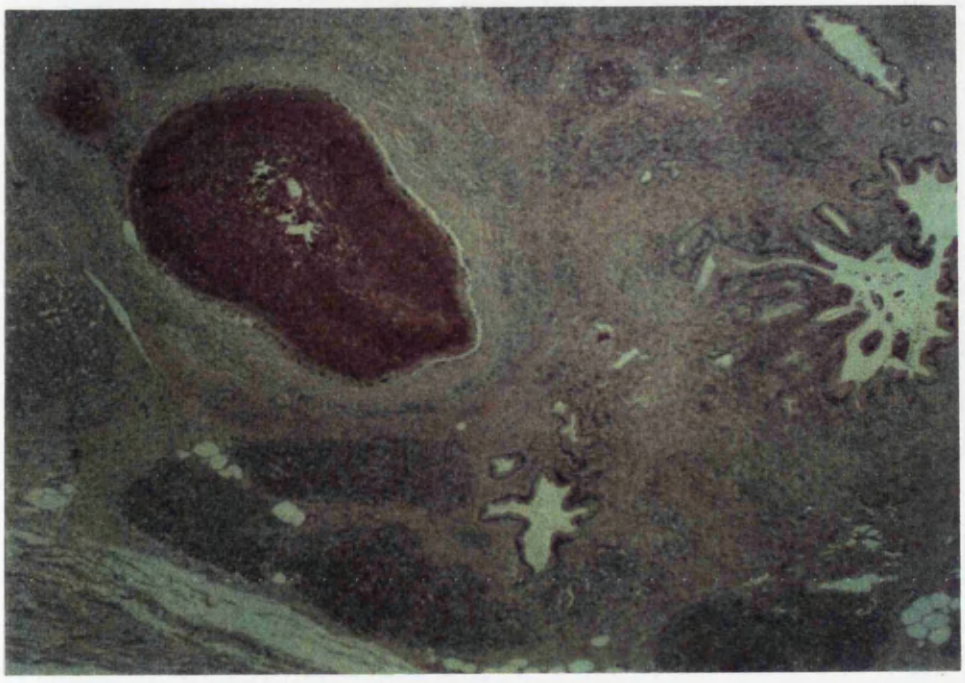


Fig. 30 Case No. 6. Eosinophilic enteritis. A Eosinophilic granulomata composed of a central mass of eosinophils surrounded by macrophages, multinucleate giant cells and a fibrous capsule are present in the colonic lymph nodes (above) and pancreas (B, below)



B Eosinophilic granuloma in the pancreas

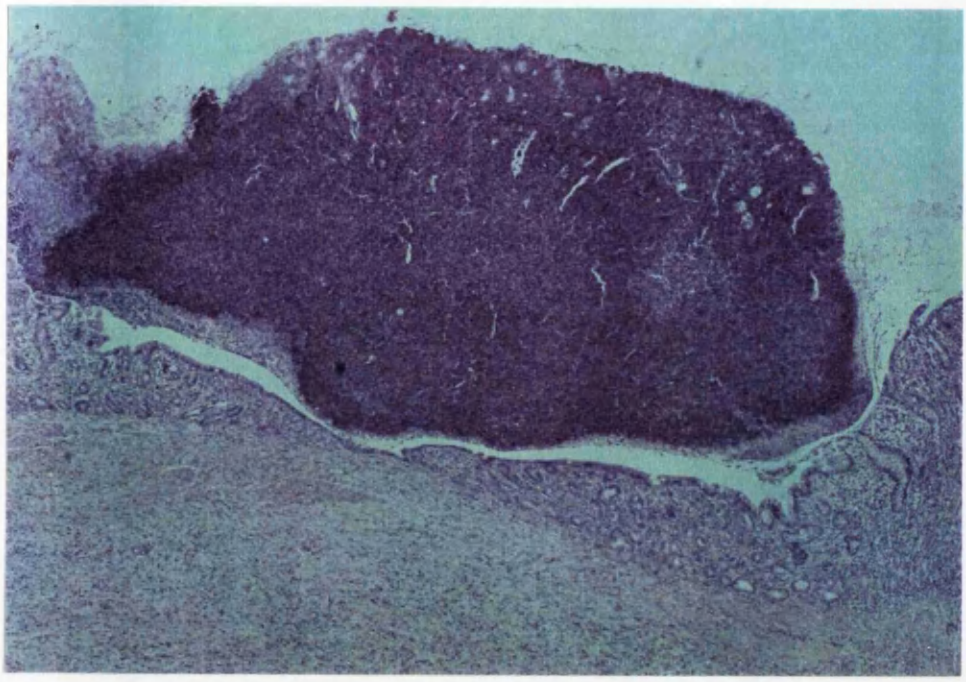


Fig. 31 Case No. 7. Eosinophilic enteritis. An eosinophilic granuloma composed of a central core of eosinophils with a peripheral layer of macrophages sitting in a depresion in the intact ileal mucosa. The adjacent villi are stunted and fused. (H and E X 10)

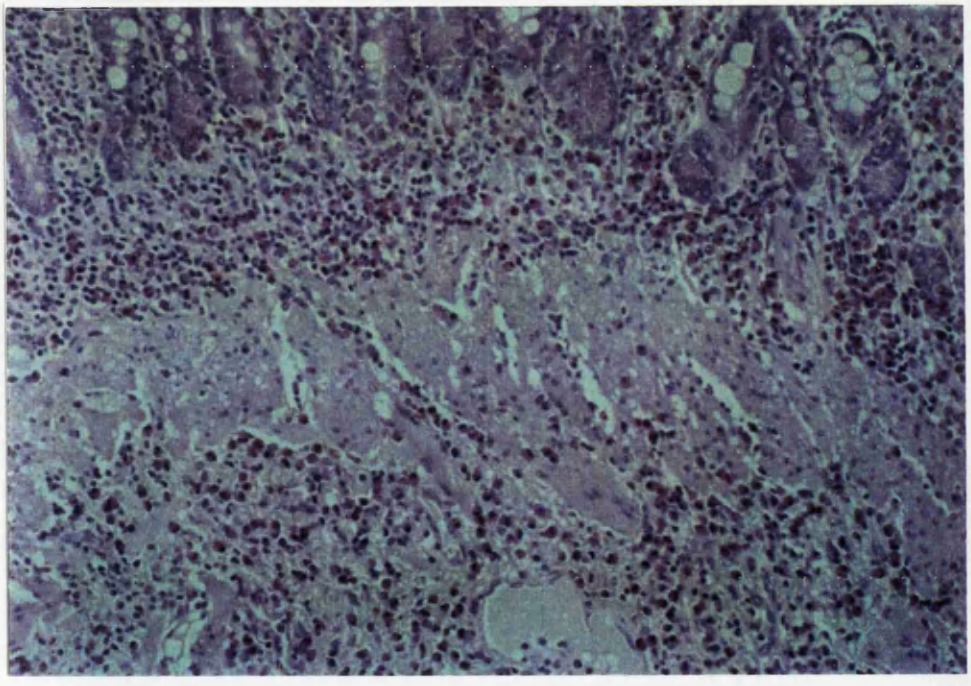


Fig. 32 Case No. 7. Eosinophilic enteritis. The ileal lamina propria and submucosa is densly infiltrated by eosinophils and there is dilatation of submucosal lymphatics (H and E X 20)

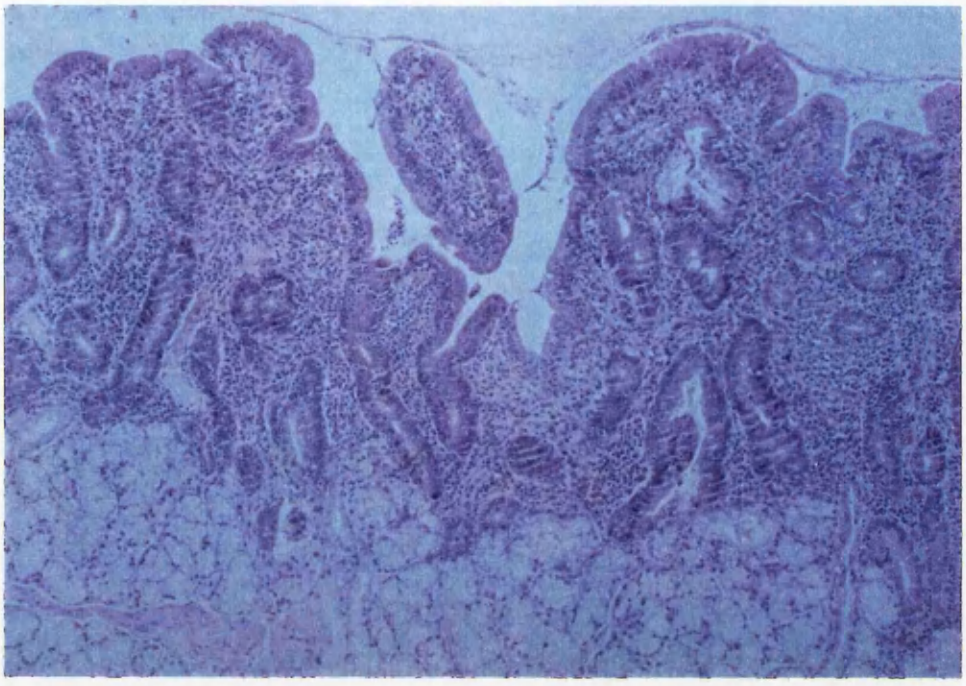


Fig. 33 Case No. 7. Eosinophilic enteritis. The duodenal villi are stunted and fused and there is marked mononuclear inflammatory cell infiltration of the lamina propria (H and E X 10)

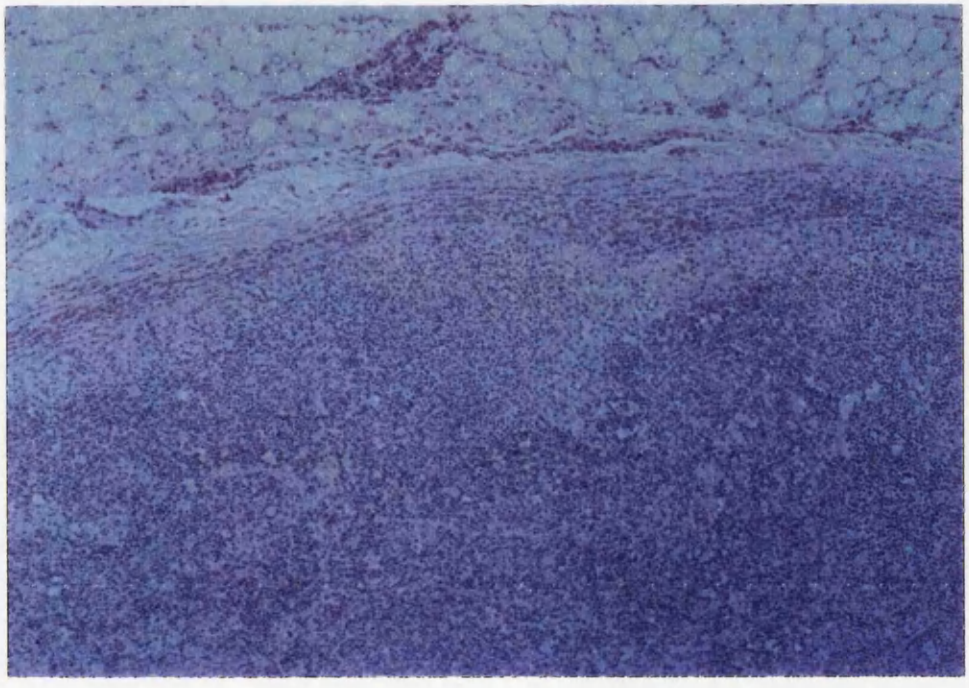
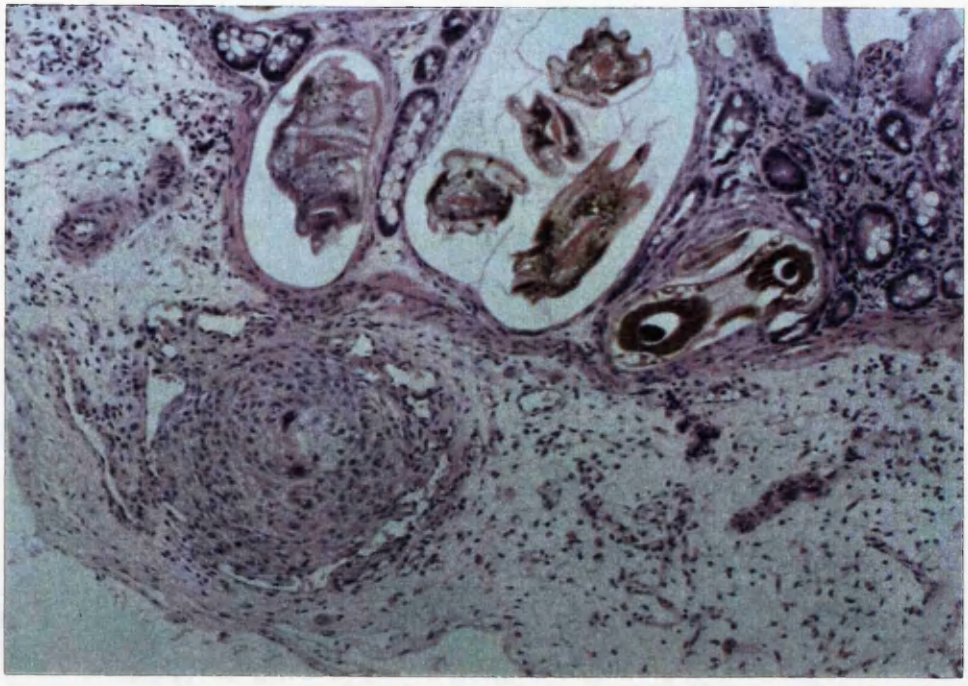


Fig. 34 Case No. 7. Eosinophilic enteritis. The perinodal fat and capsule of the mesenteric lymph node are heavily infiltrated by eosinophils (H and E X10)

Fig. 35 Case No. 8. Cyathostomiasis. The large intestinal mucosa is extremely oedematous and thickened and contains multiple pinpoint haemorrhages and remaining, unmerged *Cyathostome sp* larvae

Fig 36 Case No 8 Cyathostomiasis A In the large intestine there is massive submucosal oedema and unmerged *Cyathostome sp* larvae in the mucosa H and E X 1)



B *Cyathostome sp* larvae in the large intestinal mucosa provoke little or no inflammatory reaction but here is a granulomatous reaction associated with those in the submucosa (H and E X 20)

Fig. 37 Case No. 9. Cyathostomiasis. The mucosa of the ventral colon is peppered with dark spots representing as yet unemerged *Cyathostome sp* larvae

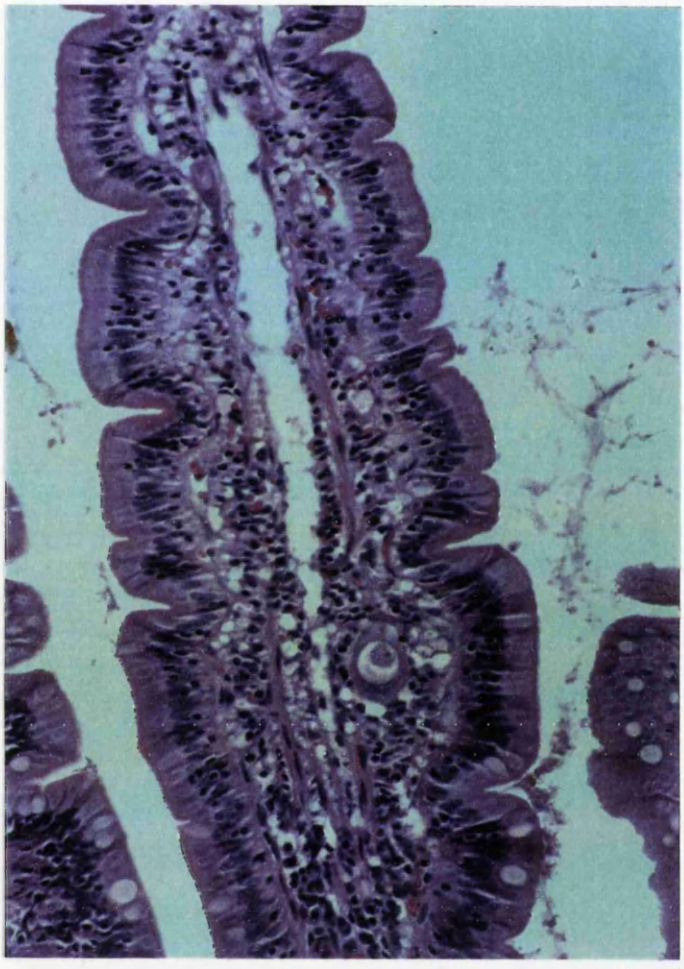


Fig. 38 Case No. 11. Coccidiosis. The small intestinal villi are oedematous and contain macrogametocytes and microgametocytes of *Eimeria leukarti* and E X 20)

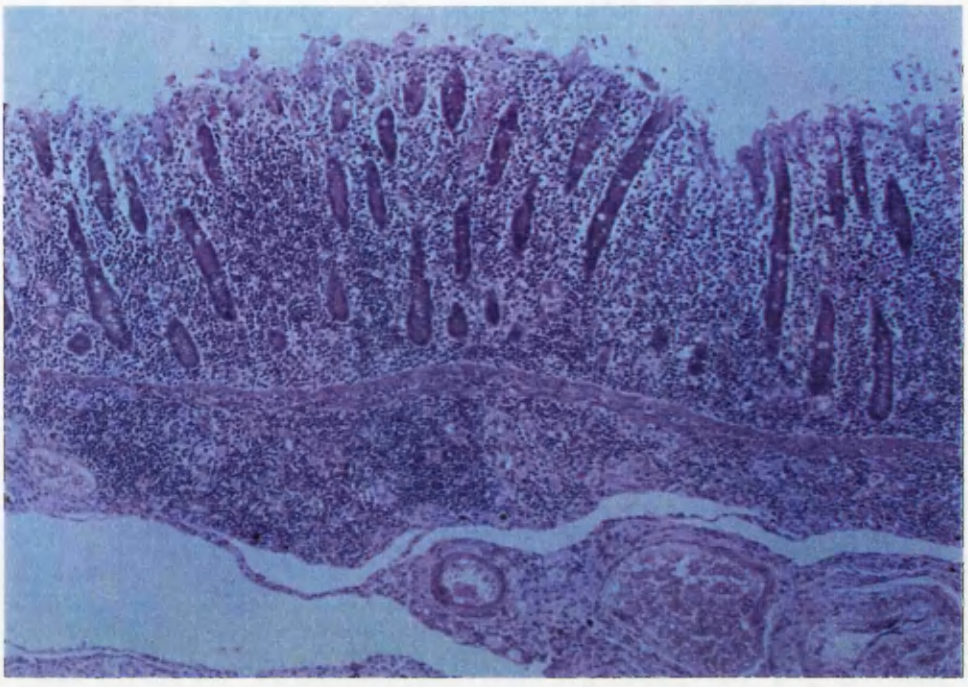
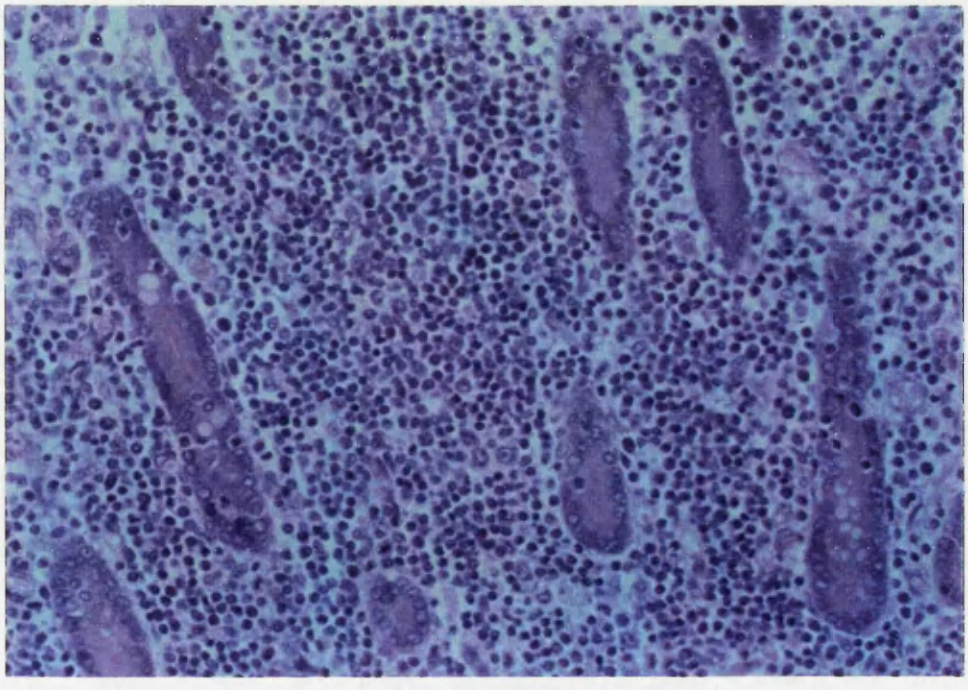


Fig. 39 Case No. 12. Enterotyphlocolitis. A There is a marked mononuclear inflammatory cell infiltrate in the large intestinal mucosa which is spilling over into the oedematous submucosa (H and E X 10)



B This higher power view shows that the infiltrate is composed of lymphocytes and plasma cells (H and E X 24)

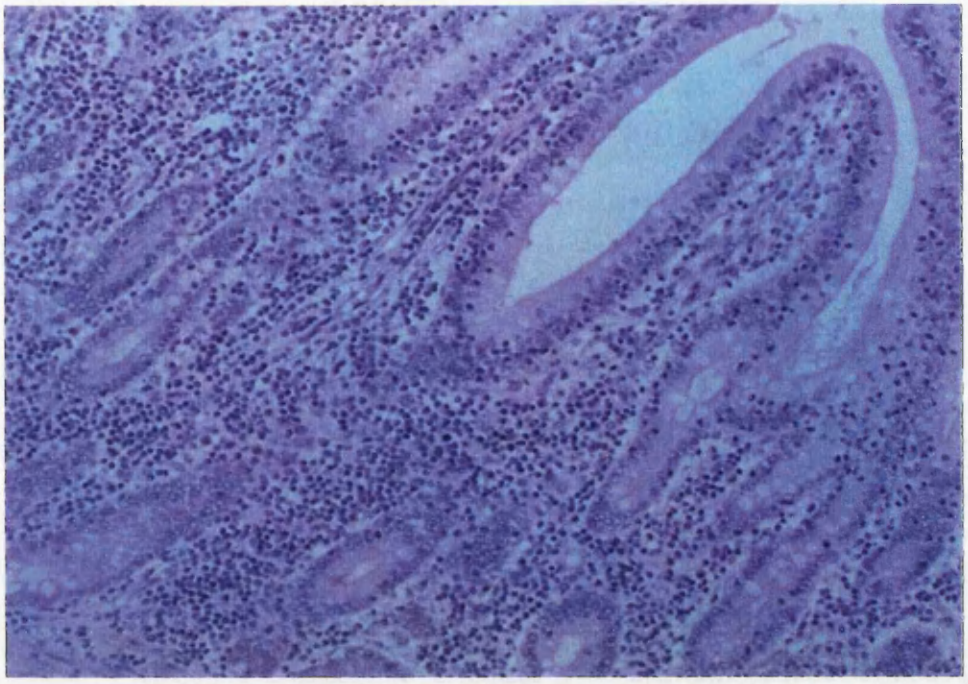


Fig. 40 Case No. 12. Enterotyphlocolitis. The small intestinal lamina propria contains a mononuclear inflammatory cell infiltrate and there is a slight increase in the number of intraepithelial lymphocytes (H and E X 20)

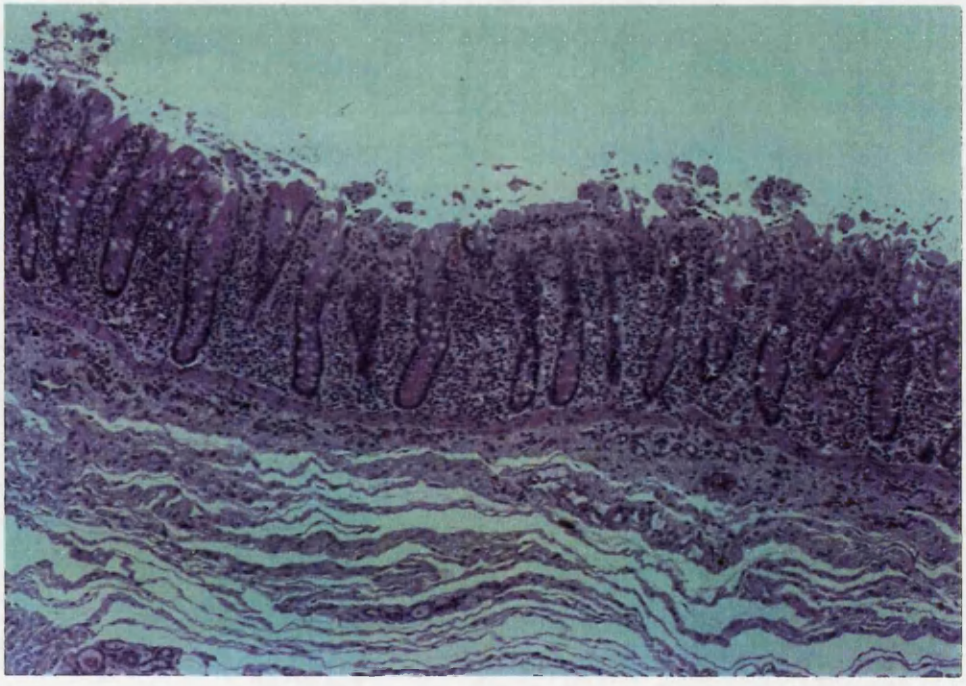


Fig. 41 Case No. 13. Enterotyphlocolitis. There is oedema of the large intestinal mucosa and submucosa and a moderate mucosal infiltrate composed of eosinophils and globule leukocytes as well as mononuclear inflammatory cells (H and E X 10)

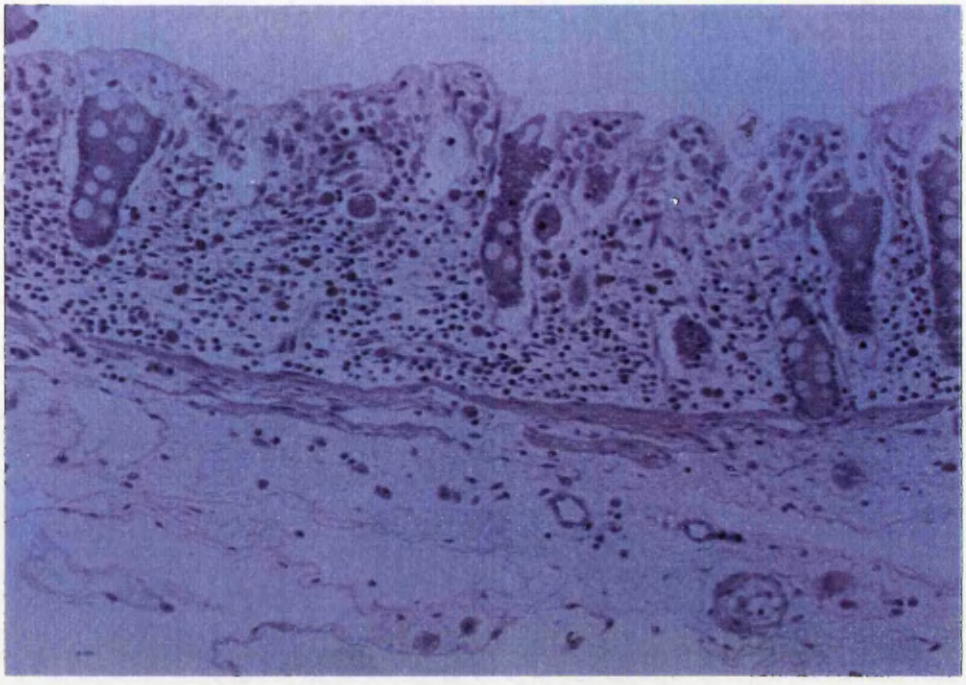
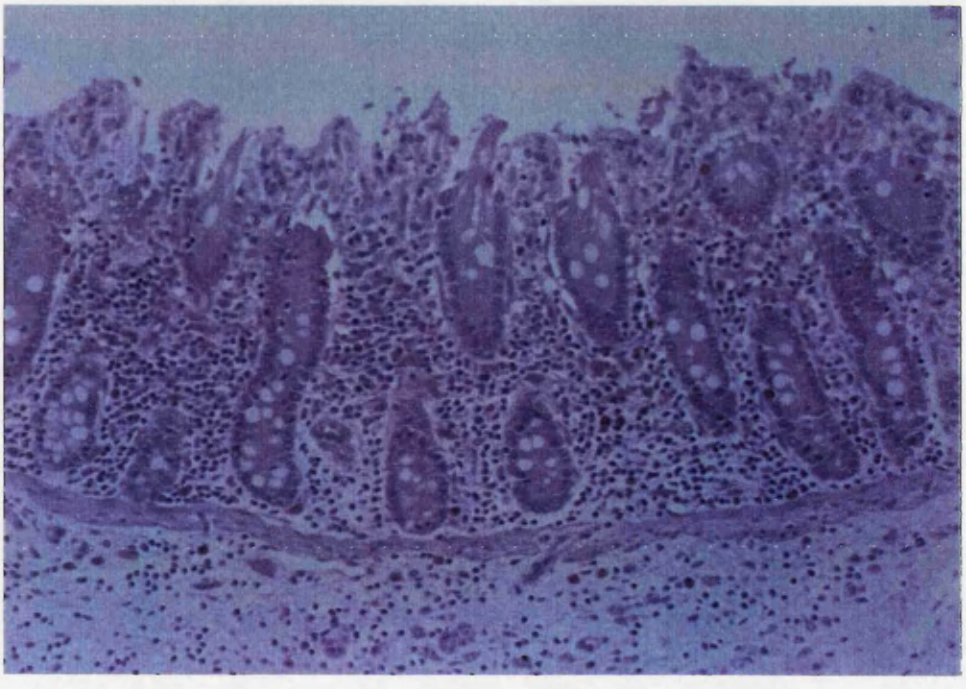


Fig. 42 Case No. 14. Enterotyphlocolitis. A The caecal mucosa and submucosa are oedematous and the mucosa contains eosinophils and globule leukocytes (H and E X 20)



B In this area of the ventral colon the mucosal infiltrate is more intense and the eosinophils and globule leukocytes have spilled over into the submocsa (H and E X 20)

Fig. 43 Case No. 15. Granulomatous typhlocolitis and Enteritis. The colonic mucosa is oedematous and contains multiple ulcerative foci.

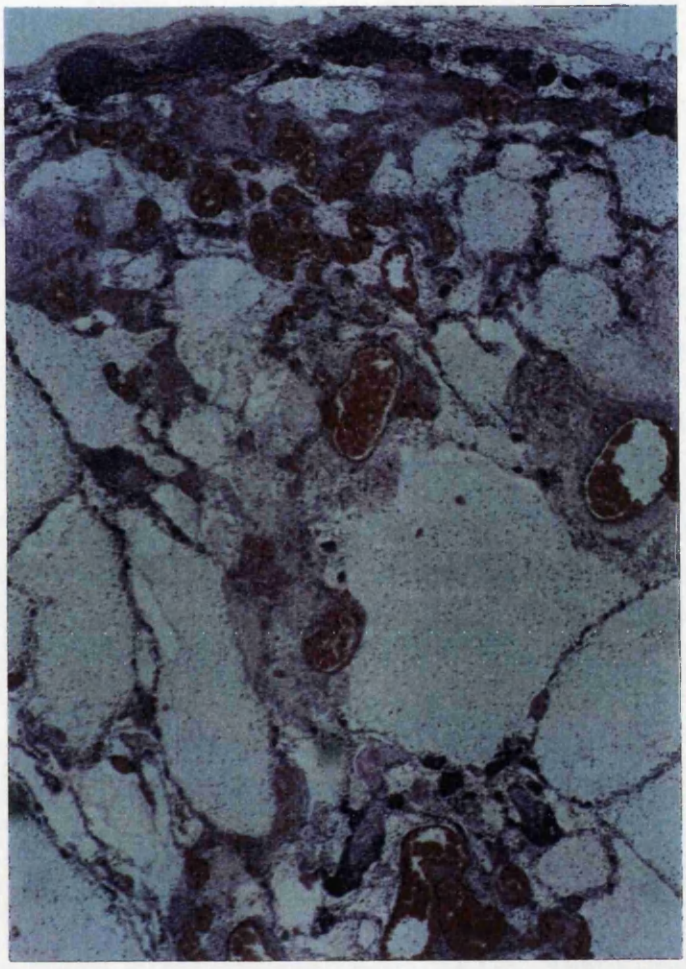


Fig. 44 Case No. 15. Granulomatous typhlocolitis and Enteritis. There is congestion and marked oedema of the colonic lymph nodes (H and E X 4)

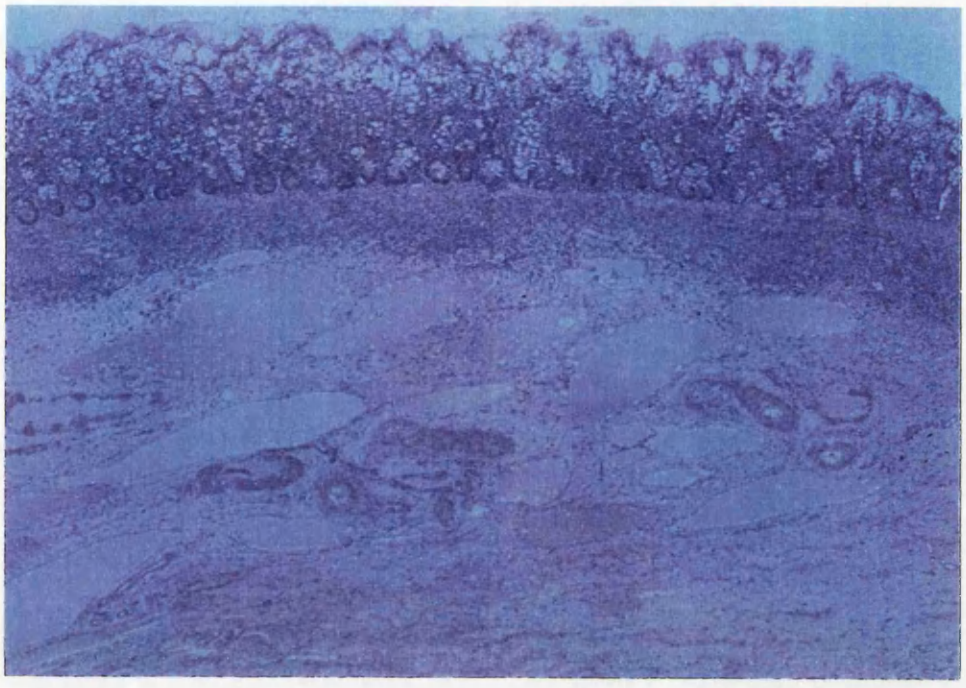
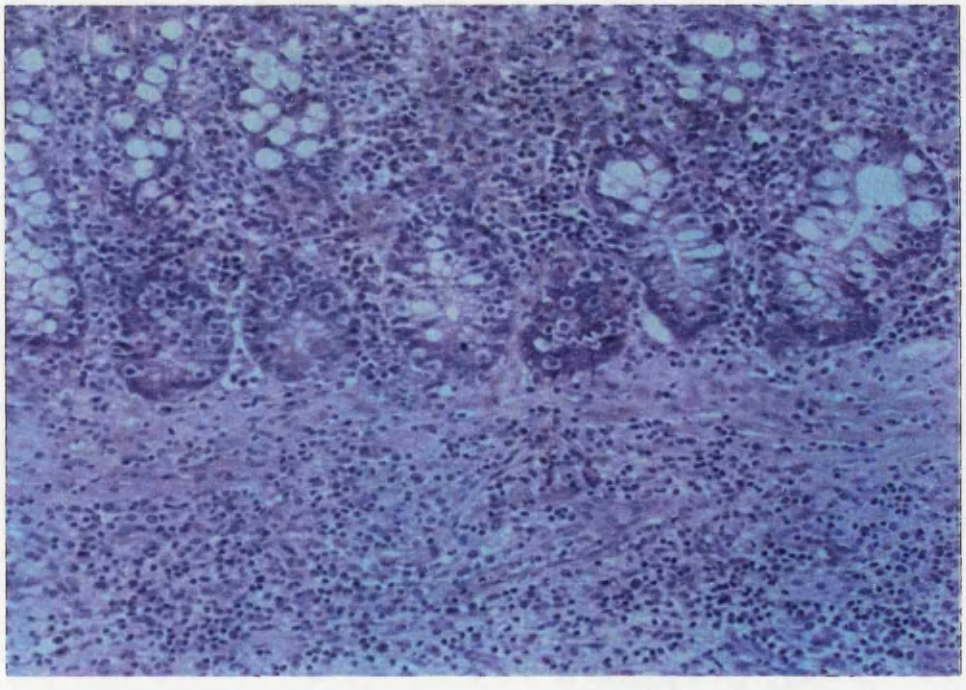


Fig. 45 Case No. 15. Granulomatous typhlocolitis and Enteritis. A There is marked oedema of the submucosa in the large intestine (H and E X 4)



B A higher power view showing the intense granulomatous infiltrate in the mucosa, muscularis mucosa and submucosa and an increase in the number of intraepithelial lymphocytes (H and E X 20)

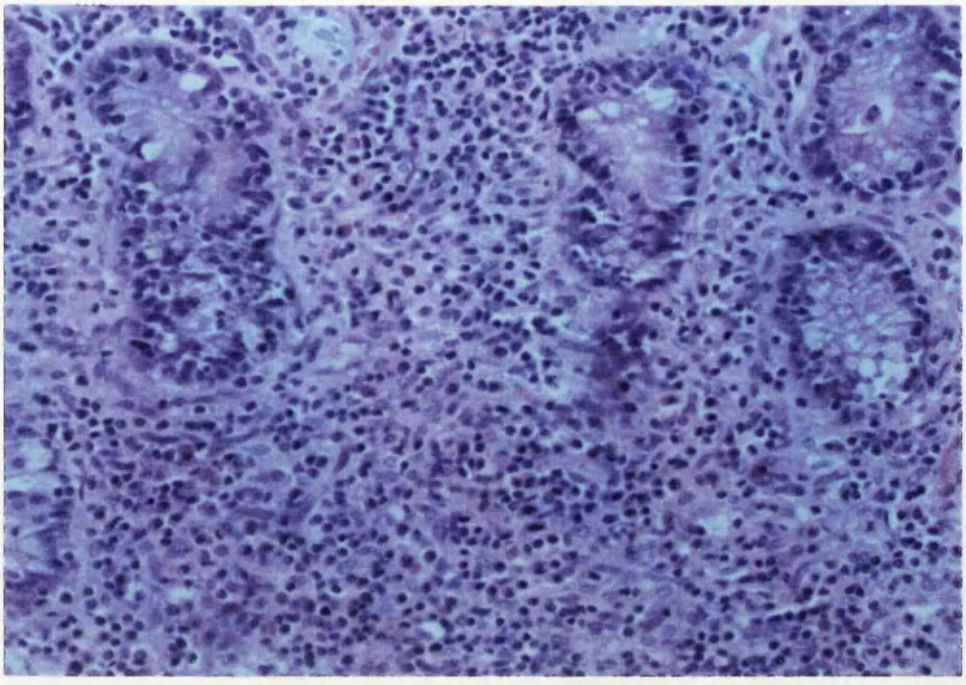


Fig. 46 Case No. 16. Granulomatous colitis and Enteritis. There is an intense granulomatous infiltrate in the mucosa, muscularis mucosa and submucosa and a marked increase in the number of intraepitheleal lymphocytes (H and E X 24)

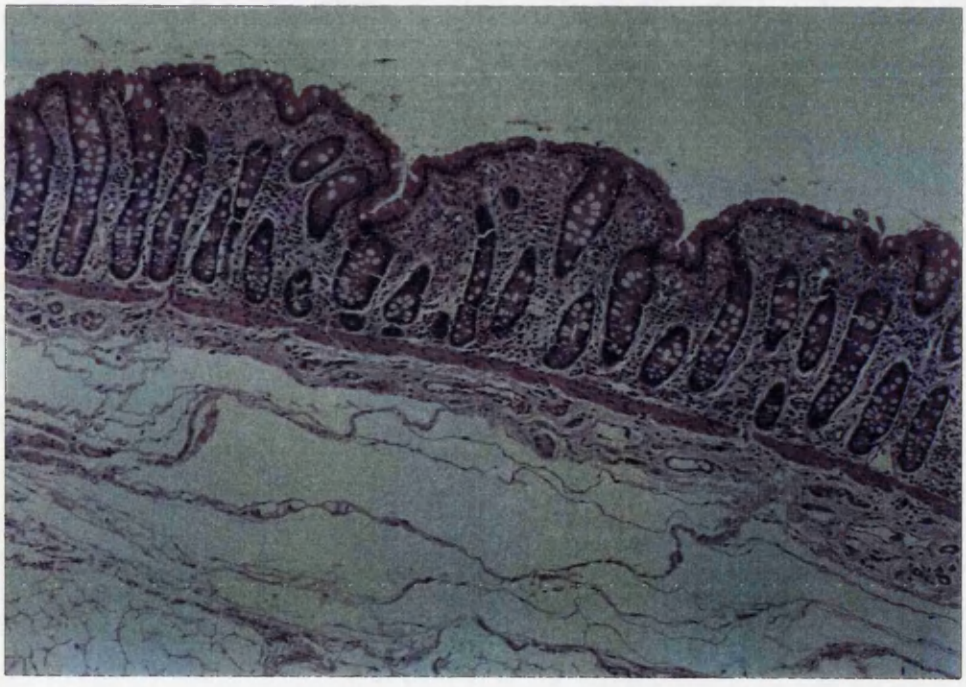
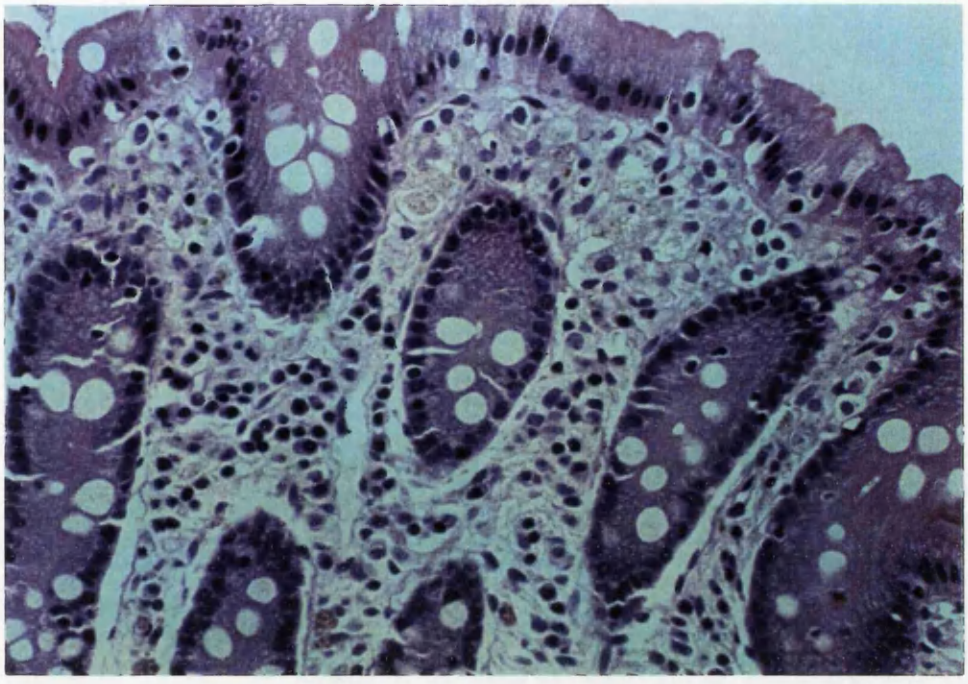
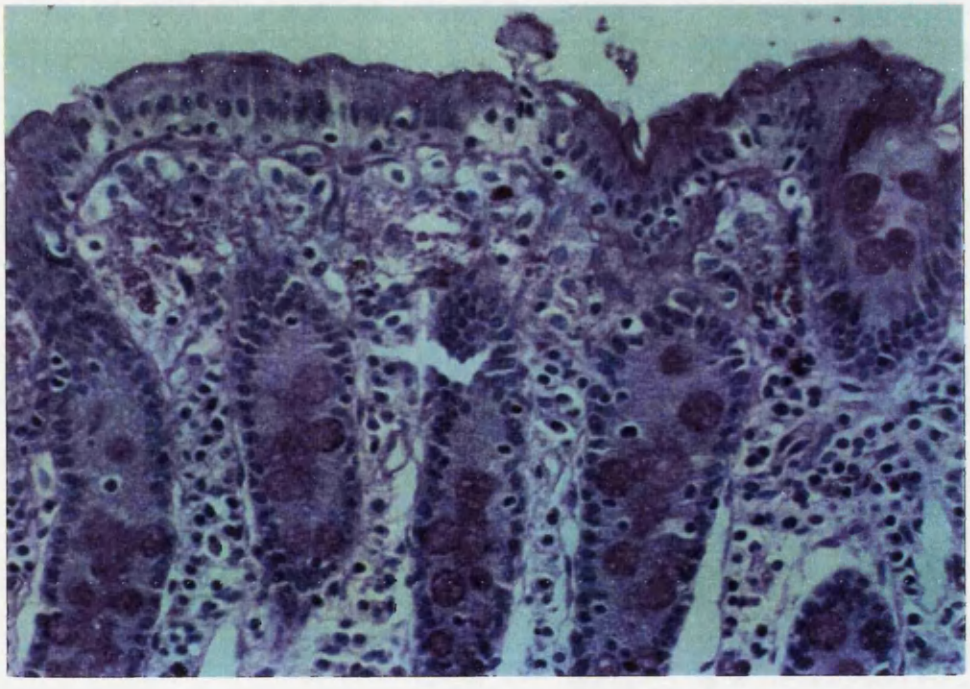


Fig. 47 Case No. 17. Typhlocolitis. A There is a sparsly cellular zone below the surface epithelium and marked submucosal oedema in the colon (H and E X 4)



B The higher power view shows that the zone contains macrophages with granular cytoplasm (H and E X 40)



C The use of the Periodic-Acid Schiff (PAS) stain highlights these granules (PAS X 40)

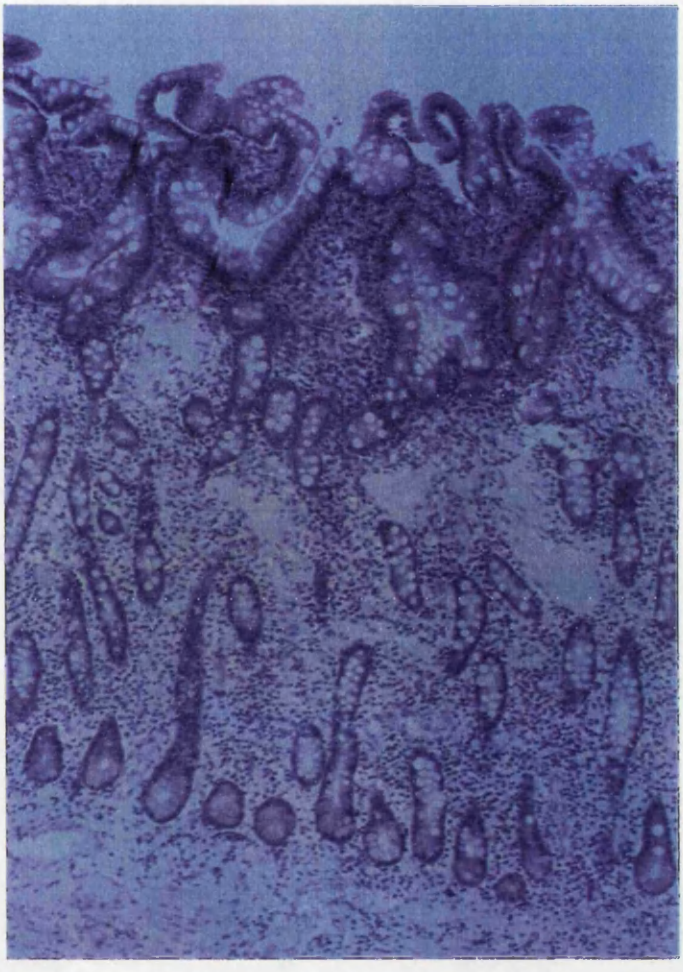
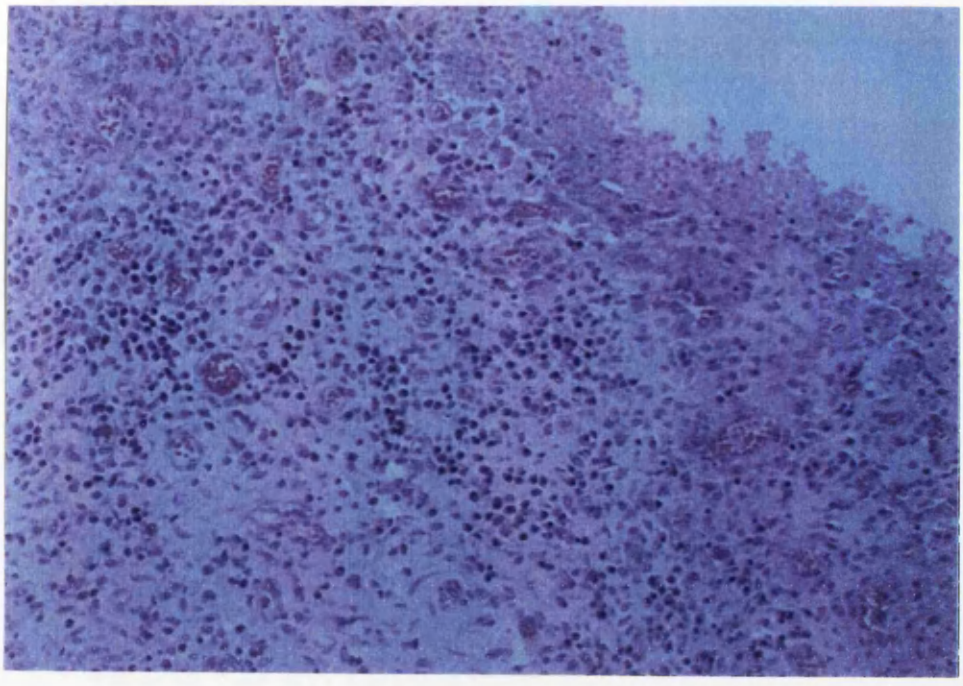


Fig. 48 Case No. 18. Enteritis. A There is mucosal oedema, a mucosal monoculear inflammatory cell infiltrate and stunting and fusion of the villi (H and E X 10)



B The serosa is thick, oedematous and fibrous with a mononuclear inflammatory cell infiltrate and surface layer of granulation tissue (H and E X 20)

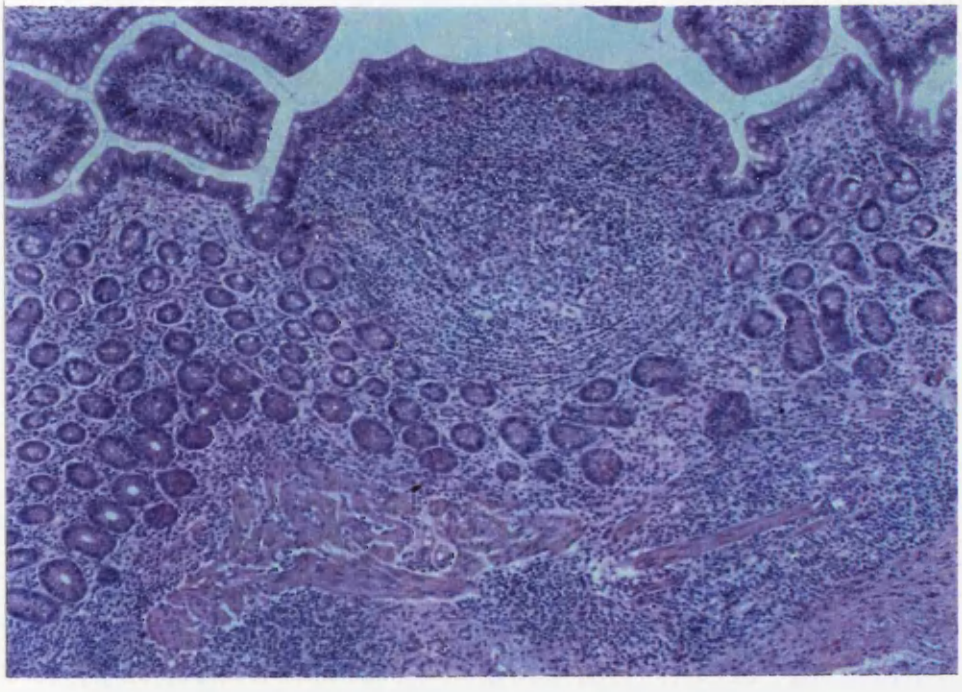


Fig. 49 Case No. 19. Lymphoid hyperplasia. There is a large lymphoid nodule in the jejunal mucosa spilling cells into the surrrounding mucosa, muscularis mucosa and submucosa (H and E X 10)

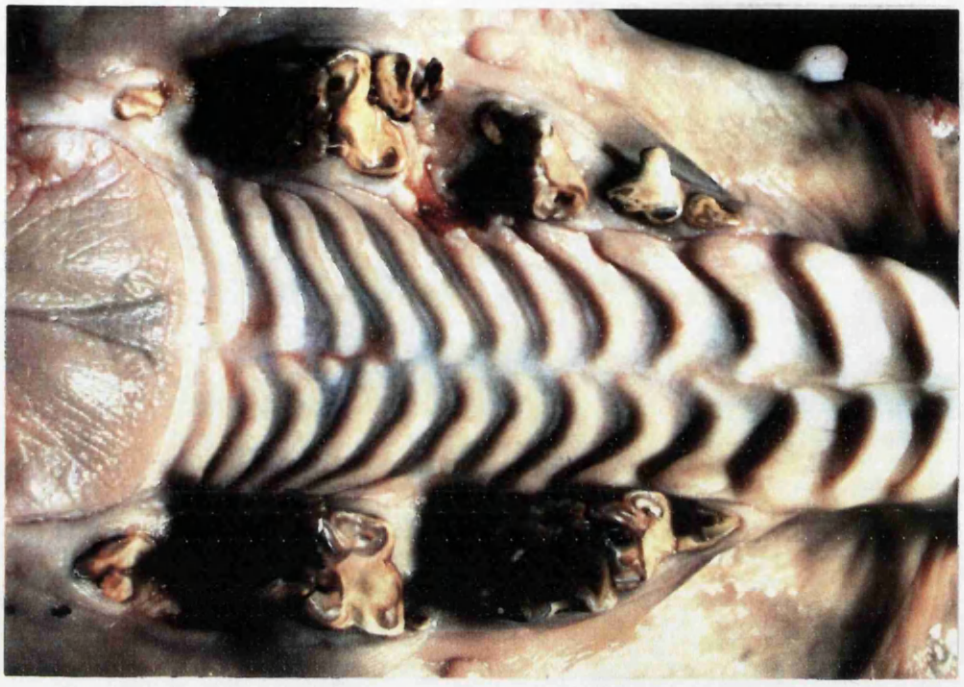


Fig. 50 Case No. 20. Dental exhaustion. The cheek teeth are extremely uneven, some are missing, and the gums are ulcerated.

CONCLUSION

The results of the Necropsy Series indicate that fatal and incidental lesions occur in all organ systems of the horse. However, the gastrointestinal system is by far the most common site of both types of lesion.

In the Gastrointestinal Series grass sickness was the most frequently identified primary condition and it would appear to have a seasonal incidence and to be of varying significance from year to year. It appears that parasitism is still common despite the widespread availability of effective anthelmintics and can be a cause of death not just of incidental lesions.

The Gastrointestinal Series also indicated that chronic enteropathies are a not infrequent cause of serious disease in the horse. The results of the Enteropathy Series prove that all the major chronic inflammatory bowel diseases do occur in the United Kingdom and are not confined to Standardbreds and Thoroughbreds. However, there are still a number of unclassified conditions which require a great deal of further study before they can be fully understood and classified.

APPENDICES

APPENDIX 1: STAINING METHODS

The staining methods used, with the exception of Astra Blue for mast cells, were based on those of **Bancroft and Stevens (1982)** and the stains employed are listed below along with the method for Astra Blue.

Haematoxylin and Eosin

Oil Red-O methods for lipids

Perl's Prussian Blue reaction for ferric iron

Gordon and Sweet's method for reticular fibres

Ziehl-Neelsen stain for tubercle bacilli

Gram stain for paraffin sections

Astra Blue for Mast Cells

Fixation, not critical

Sections, all types

1) Sections to water via iodine and sodium thiosulphate
2) Stain in 0.1% astra blue in 0.7 Normal Hcl for 30 minutes
3) Wash with running tap water 1 minute
4) Rinse in 0.7 normal HCl
5) Wash with running tap water 1 minute
6) Counterstain in 1% safranin (dilute 1/20)
7) Dehydrate through alcohols, clean in xylol, mount in DPX

Results: Mast cell granules - blue

Background - pink - red

APPENDIX 2: NECROPSY SERIES

All the animals studied are listed below. An E following the case number denotes that the animal was in the Enteropathy Series and an N denotes that the animal was part of the normal reference study.

Month	Case Number	Age	Sex	Breed	PRINCIPAL & Incidental Findings
1987					
January	1E	15Y	F	TB	ENTEROTYPHLOCOLITIS *Haemomelasma ilei* Hepatic Haemosiderosis
	2E	7Y	M	TB	TYPHLOCOLITIS Oesophageal papilloma Inflammatory hydrocoele
February	3E	15Y	F	RP	ALIMENTARY LYMPHOSARCOMA Cyathostomes Hepatic Haemosiderosis Thrombophlebitis Sialoadenitis
March	4	5Y	Mn	TB	SPLENIC LYMPHOSARCOMA Cardiovascular mineralisation Hypercalcaemic nephropathy Anaemia
	5	12Y	F	X	HYPERLIPAEMIA diarrhoea
	6	7Y	F	Shetland	HYPERLIPAEMIA diarrhoea
	7	2D	NR	X	INHALATIONAL PNEUMONIA
April	8	6Y	F	Trotter	INCONCLUSIVE Cranial mesenteric arterial thrombosis
May	9	3Y	Mn	Arab	GRASS SICKNESS Cyathostomes Oesophageal ulceration Gastric ulceration
	10	25Y	Mn	RP	CIRRHOSIS
	11E	18Y	Mn	TBX	CYATHOSTOMIASIS
	12	20Y	Mn	RH	GRASS SICKNESS GASTRIC RUPTURE PERITONITIS Oesophageal ulceration

	13	3Y	F	Arab	GRASS SICKNESS Oesophageal ulceration Gastric ulceration Cyathostomes
	14	12Y	F	RH	GRASS SICKNESS
June	15	5Y	F	Pony	STRANGLES
	16	6Y	Mn	RH	GRASS SICKNESS Oesophageal ulceration Gastric ulceration Splenic enlargement
	17	3Y	F	Clydesdale	CERVICAL VERTEBRAL MALFORMATION COMPRESSIVE MYELOPATHY
	18	7Y	F	Pony	NECROTISING LAMINITIS Gastric ulceration Renal infarction Myocardial infarction
	19	6Y	F	RH	GRASS SICKNESS Oesophageal ulceration Gastric ulceration Splenic enlargement
	20	NR	F	WMP	GRASS SICKNESS Oesophageal ulceration
	21E	20Y	Mn	RH	EOSINOPHILIC ENTERITIS Hepatic hydatidosis Pulmonary hydatidosis Renal infarction
	22	15Y	Mn	Pony	MALIGNANT MELANOMATA Laminitis
July	23	1D	M	Pony	ASPHYXIATION
August	24E	13Y	Mn	Pony	ALIMENTARY LYMPHOSARCOMA Intestinal ulceration
	25	11Y	Mn	TB	STRANGLES
	26E	15Y	F	Pony	ALIMENTARY LYMPHOSARCOMA Gastric ulceration Intestinal ulceration Cholangiohepatitis Hepatic haemosiderosis Renal infarction
	27	8Y	Mn	Pony	GRASS SICKNESS Colitis Typhlitis

					Typhlitis
September	28E	15Y	Mn	RH	INTESTINAL COCCIDIOSIS
October	29	Aged	Mn	Pony	ARTHRITIS
	30	Adult	F	Pony	BOTULISM
	31E	9Y	Mn	TB	LYMPHOID HYPERPLASIA Gastric ulceration *Haemomelasma ilei* Splenic Microabscesses
	32	15Y	F	Pony	BOTULISM Pituitary nodular hyperplasia
	33	13Y	Mn	TB	COLITIS Laminitis *Haemomelasma ilei* Cranial mesenteric arterial aneurysm
November	34	27Y	Mn	Pony	SAND COLIC LARGE INTESTINAL OBSTRUCTION PERITONITIS
	35	15Y	F	Shetland X	STRANGLES
December	36	22Y	F	Pony	CERVICAL SCOLIOSIS COMPRESSIVE MYELOPOATHY
	37	8Y	Mn	TB	RUPTURED TENDON
	38	15Y	F	Welsh Cob	FRACTURED PEDAL BONE
1988					
January	39	30Y	Mn	RH	SMALL INTESTINAL OBSTRUCTION Cranial mesenteric arterial thrombosis
	40	12Y	Mn	TB	POST OPERATIVE MYOPATHY
	41	6Y	Mn	Welsh Cob	VAGOSYMPATHETIC TRANSECTION Strongyles
February	42	3Y	Mn	Shire	GRASS SICKNESS
	43	3Y	Mn	Arab X	GRASS SICKNESS Oesophageal ulceration Gastric ulceration Intestinal ulceration

	44	20Y	Mn	X	CHRONIC OBSTRUCTIVE PULMONARY DISEASE
March	45	25Y	F	TB	CHRONIC OBSTRUCTIVE PULMONARY DISEASE
	46	26Y	Mn	RH	PALATINE SQUAMOUS CELL CARCINOMA
May	47	6Y	F	Shetland X	GRASS SICKNESS Oesophageal ulceration
	48	14D	M	Clydesdale	HAEMOLYTIC ANAEMIA
	49	1d	F	Welsh Cob	SEPTICAEMIA
June	50	17Y	Mn	Trotter	GRASS SICKNESS Cranial mesenteric arterial thrombosis *Haemomelasma ilei*
	51	13Y	NR	Pony	ACUTE HEART FAILURE
June	52	25Y	Mn	Pony	ULCERATIVE COLITIS DIAPHRAGMATIC RUPTURE PERITONITIS Cyathostomes Cranial mesenteric arterial thrombosis Multiple melanomata Bronchiolitis
	53	25Y	Mn	Pony	HAEMOCHROMATOSIS
	54	20Y	F	RP	Sweet Itch
	55	19Y	Mn	RH	PITUITARY ADENOMA CUSHING'S SYNDROME Laminitis Brain abscess
August	56	Adult	F	RH	MALIGNANT MELANOMATA
	57	22Y	Mn	TBX	CHRONIC OBSTRUCTIVE PULMONARY DISEASE Thyroid adenoma
September	58	22Y	Mn	RH	FUNGAL ENTERITIS Intestinal ulceration Interstitial nephritis Ragwort poisoning
	59	3Y	Mn	Arab	GRASS SICKNESS Oesophageal ulceration Gastric ulceration

	60	4Y	F	Clydesdale	GRASS SICKNESS Oesophageal ulceration
	61	6Y	Mn	Irish Draft	RUPTURED CHORDAE TENDINEA
	62E	19Y	F	TBX	GRANULOMATOUS COLITIS ENTERITIS *Haemomelasma ilei* Splenic microabsesses
October	63	12Y	F	TB	OSTEOMYELITIS
	64	12Y	Mn	RH	CHRONIC OBSTRUCTIVE PULMONARY DISEASE Peritonitis
	65	4Y	Mn	TB	Dermatitis
	66	1D	F	Hackney	CLEFT PALATE
	67	16Y	F	Irish Draft	MALIGNANT MELANOMATA Chronic obstructive pulmonary disease Osteoarthritis Hepatic hydatidosis
November	68	11Y	Mn	RH	CERVICAL SUBLUXATION COMPRESSIVE MYELOPATHY
	69	4M	F	Pony	SALMONELLOSIS
December	70E	8Y	F	RP	ENTEROTYPHLOCOLITIS Gastric ulceration
	71	Adult	F	Donkey	LYMPHOSARCOMA
1989 January	72	20Y	Mn	RH	PNEUMONIA Pituitary nodular hyperplasia
	73	5Y	Mn	NR	FRACTURED LUMBAR SPINE
	74	16Y	F	RH	COLONIC VOLVULUS Gastric ulceration Large intestinal ulceration
	75	Adult	F	Shetland	CAECAL CANULA LEAKAGE PERITONITIS
	76	2Y	F	TB	SMALL INTESTINAL VOLVULUS SMALL INTESTINAL STRANGULATION GASTRIC RUPTURE PERITONITIS

					Cranial mesenteric arterial thrombosis
March	77	10Y	Mn	RP	FRACTURED PHALANX
April	78	7Y	F	Fell	HYPERLIPAEMIA GASTRIC RUPTURE
	79	18Y	Mn	TBX	LARGE COLON VOLVULUS ILIAL INTUSSUSCEPTION Intestinal ulceration Tapeworms Myocardial infarction
	80E	6Y	F	TB	ENTERITIS PERITONITIS Colonic ulceration
	81	0	M	RP	DYSTOCIA
May	82	5Y	F	WMP	DIAPHRAGMATIC RUPTURE ENTRAPMENT
	83	10Y	F	RH	INCONCLUSIVE Gastric ulceration
	84	9Y	Mn	RP	INTESTINAL LEAKAGE SMALL INTESTINAL VOLVULUS PERITONITIS
	85	7Y	F	Highland	HYPERLIPAEMIA LAMINITIS
June	86	1D	M	Clydesdale	PATENT DUCTUS ARTERIOSUS
	87	13Y	Mn	WMP	GRASS SICKNESS Oesophageal ulceration Splenic enlargement
July	88	25Y	Mn	TBX	AORTIC INSUFFICIENCY
	89	13Y	Mn	Fell	LAMINITIS Cranial mesenteric arterial thromboembolism Hepatic fatty degeneration
	90	63D	Mn	Hanovarian	RUPTURED BLADDER PERITONITIS VENTRAL ABDOMINAL HERNIA
	91	14Y	F	RH	MALIGNANT MELANOMATA COMPRESSIVE MYELOPATHY

August	92E	16Y	F	Pony	GRANULOMATOUS TYPHLOCOLITIS ENTERITIS Large intestinal ulceration Hepatic haemosiderosis
	93	4Y	Mn	Pony	DISCOSPONDYLITIS
October	94	NR	F	RP	OSTEOARTHRITIS
	95	3Y	Mn	Trotter	ILEOSACRAL SUBLUXATION Myocardial infarction
	96	8Y	F	RP	ACUTE HEART FAILURE ARTHRITIS
	97	6Y	Mn	TB	ACUTE HAEMORRHAGIC TYPHLOCOLITIS Large intestinal ulceration
	98	15Y	Mn	TB	POORLY DIFFERENTIATED TUMOUR RECTAL TEAR Small intestinal ulceration Large intestinal ulceration
November	99	8Y	F	RH	LAMINITIS
	100N	9Y	Mn	RH	OSTEOARTHRITIS Myocardial infarction
	101	17Y	F	Pony	PITUITARY ADENOMA CUSHING'S SYNDROME Gastric Ulceration Laminitis Renal infarction Thyroid nodular hyperplasia
	102	10Y	Mn	TB	TENDONITIS Renal infarction
	103	3Y	F	Pony	CYATHOSTOMIASIS INTESTINAL ULCERATION
	104	7Y	Mn	Irish Draft	SMALL INTESTINAL ENTRAPMENT
December	105N	NR	F	Pony	ARTHRITIS
	106N	14Y	Mn	Highland	MALIGNANT MELANOMATA

	107N	15Y	F	RP	CHOLANGITIS OESOPHAGITIS PNEUMONIA
1990					
January	108	4Y	F	Polo Pony	FRACTURED LIMB
	109	18Y	F	TBX	SMALL INTESTINAL MESENTERIC TORSION Oesophageal ulceration
March	110N	20Y	F	Pony	CHRONIC OBSTRUCTIVE PULMONARY DISEASE Cranial mesenteric arterial aneurysm
	111	15Y	F	Pony	CHRONIC OBSTRUCTIVE PULMONARY DISEASE
	112E	30Y	Mn	WMP	DENTAL EXHAUSTION CHRONIC ENTEROPATHY Oral ulceration Cyathostomes Hepatic haemosidersosis Cranial mesenteric arterial thrombosis Renal abscess
	113	5Y	Mn	Dutch WB	OSTEOMYELITIS Small intestinal ulceration
	114	22Y	F	TB	PERITONITIS Post operative intestinal leakage Chronic enteritis Hepatic fatty change
	115	4Y	Mn	TB	INCONCLUSIVE -(AUTOLYTIC)
April	116	17Y	F	TBX	CAECAL PERFORATION PERITONITIS CAECAL ULCERATION VAGINAL TEAR Cyathostomes
	117	4Y	F	Highland	DUODENAL PERFORATION PERITONITIS Strongylosis *Haemomelasma ilei*
May	118	2D	M	Clydesdale	PLEURISY ACUTE EXUDATIVE PNEUMONIA SEPTICAEMIA CONGENITAL OCULAR LESION

	119	5Y	Mn	TB	SMALL INTESTINAL MESENTERIC VOLVULUS Peritonitis *Gasterophilus intestinalis*
	120	11Y	Mn	WMP	FRACTURED LIMB
	121	15Y	Mn	RP	CIRRHOSIS LARYNGEAL PARALYSIS Photosensitisation
	122N	13Y	Mn	Dutch WB	DISCOSPONDYLOSIS CERVICAL MYELOPATHY Cyathostomes
	123N	12Y	Mn	TBX	PERIPHERAL NEUROPATHY Cyathostomes
	124N	15Y	Mn	Connemara	NASAL POLYP Navicular disease
	125	16Y	Mn	TBX	ACUTE HEART FAILURE
June	126E	14Y	Mn	Welsh Cob	PROTEIN-LOSING ENTEROPATHY RECTAL TEAR PERITONITIS INTESTINAL ULCERATION Hepatic lipidosis Myocardial infarction
	127N	18Y	Mn	TBX	CHRONIC OBSTRUCTIVE PULMONARY DISEASE Gastric ulceration
	128N	15Y	Mn	Highland	FRACTURED LIMB
	129	11Y	F	Arab	CAUDA EQUINA NEURITIS Cystitis
	130N	13Y	F	Clydesdale	DISCOSPONDYLITIS CERVICAL MYELOPATHY
	131N	10Y	Mn	Danish WB	POLYARTHRITIS Gastric ulceration
July	132	3Y	F	Clydesdale	LAMINITIS Small intestinal ulceration Tapeworms
	133N	Aged	F	Highland	Ocular collapse
	134N	20Y	Mn	RH	MULTIPLE MYELOMA

	135N	4Y	Mn	TB	GUTTURAL POUCH MYCOSIS ARTERITIS Thyroid cyst Renal infarction
	136	12M	Mn	Clydesdale	ARTHRITIS Strongylosis Cranial mesenteric arterial thrombosis Papillomata
August	137N	15Y	Mn	RH	SPLENIC LYMPHOSARCOMA
	138N	4M	M	TBX	CERVICAL FRACTURE
	139N	21Y	F	RP	GUTTURAL POUCH EMPYEMA Chronic obstructive pulmonary disease Melanomata Cirrhosis Pheochromocytoma
	140	8Y	Mn	Hanovarian	GRASS SICKNESS
September	141	24Y	Mn	New Forest	GRASS SICKNESS Oesophageal ulceration Sweet itch Lipomata
	142	8Y	Mn	WMP	HYPERLIPAEMIA DERMATITIS LAMINITIS
	143	7Y	F	TBX	COLONIC VOLVULUS
	144	18Y	Mn	RP	DUODENAL OBSTRUCTION GASTRIC RUPTURE PERITONITIS INTESTINAL ULCERATION
	145	12M	F	Shetland	CYATHOSTOMIASIS HEPATIC LIPIDOSIS
October	146	Aged	Mn	RH	Dermatophilosis Lipoma
	147N	Adult	Mn	TB	FRACTURED SCAPULA
	148N	9Y	Mn	TB	MULTIFOCAL ENCEPHALITIS ARTHRITIS Cyathostomes
November	149	18M	F	WMP	CYATHOSTOMIASIS HEPATIC LIPIDOSIS

	150E	13Y	F	RP	CYATHOSTOMIASIS Cirrhosis Hepatic Haemosiderosis Hepatic hydatidosis Photosensitisation
	151	5M	F	TBX	ABORTED FOETUS
November	152	23Y	Mn	TBX	INCONCLUSIVE
	153	Adult	F	Pony	CAECAL FISTULA BREAKDOWN PERITONITIS
	154	2 1/2Y	Mn	RH	ANGULAR LIMB DEFORMITY Parasitic hepatitis *Haemomelasma ilei* Cerebral oedema Sarcoid
	155E	13Y	F	ArabX	GRANULOMATOUS ENTERITIS INTESTINAL ULCERATION Peritonitis Hepatic haemosiderosis Cystitis
	156	6M	M	Connemara	HEPATIC ENCEPHALOPATHY LIVER FAILURE Gastric impaction
December	157	12Y	Mn	WMP	SUBLUXATED FETLOCK DEGENERATIVE JOINT DISEASE
	158	4Y	Mn	Arab	CHRONIC ADHESIVE MENINGITIS
	159E	20Y	Mn	WMP	ENTEROTYPHLOCOLITIS RECTAL PERFORATION PERITONITIS Intestinal ulceration Cranial Mesenteric arterial thrombosis
	160	5Y	Mn	TBX	TENDONITIS *Haemomelasma ilei*
January	161E	15Y	Mn	RP	GRANULOMATOUS ENTERITIS INTESTINAL ULCERATION Peritonitis Oesophageal ulceration Hepatic lipidosis Muscle abscess Sarcocystis
	162	15Y	F	RP	NECROTISING COLITIS INTESTINAL ULCERATION *Haemomelasma ilei*

					Haemomelasma ilei Gastric ulceration
	163	23Y	Mn	TB	*Haemomelasma ilei*
January	164	17Y	Mn	TB	PITUITARY ADENOMA CUSHING'S SYNDROME *Haemomelasma ilei* Cranial mesenteric arterial thrombosis and aneurysm Hepatic hydatid cysts Pneumonia Septicaemia Osteoporosis Klossiellosis
	165	10Y	F	WMP	LAMINITIS *Haemomelasma ilei*
February	166	10Y	F	TB	SMALL INTESTINAL INTUSSUSCEPTION GASTRIC RUPTURE PERITONITIS Cranial mesenteric arterial aneurysm
	167	2Y	Mn	Connemara	PERITONITIS
March	168	15Y	F	Highland	STRANGULATED SMALL COLON LIPOMA
	169P	10Y	F	RP	PERITONITIS Intestinal ulceration *Haemomelasma ilei* Cirrhosis
	170	Adult	Mu	TBX	CERVICAL SUBLUXATION
	171	20Y	F	WMP	AMELOBLASTOMA Thyroid nodular hyperplasia
	172E	16Y	F	RH	EOSINOPHILIC ENTERITIS Gastric ulceration Hepatic haemosiderosis Pituitary adenoma Endometritis Cystitis Renal abscess Renal infarction Focal pneumonia Pulmonary hydatidosis Sebaceous cyst

April	173	Adult	NR	TB	DUODENAL OBSTRUCTION GASTRIC RUPTURE PERITONITIS
	174	15y	F	Pony	FRACTURED LIMB
May	175	Old	Mn	RP	CHRONIC OBSTRUCTIVE PULMONARY DISEASE Navicular disease Laminitis
	176	3m	F	Connemara	TENDON RUPTURE
	177	7Y	Mn	TBX	GRASS SICKNESS Oesophageal ulceration
	178	Adult	Mn	RH	GRASS SICKNESS Oesophageal ulceration
	179	8Y	Mn	TB	GRASS SICKNESS Gastric ulceration
	180	Old	F	RP	Pituitary nodular hyperplasia Thyroid nodular hyperplasia Lipoma
May	181	7y	Mn	TBX	ACUTE HEART FAILURE
June	182	3Y	Mn	WMP	GRASS SICKNESS Oesophageal ulceration Gastric ulceration Cyathostomes
	183	6M	M	Dutch WB	DISCOSPONDYLITIS Compressive myelopathy
	184	16Y	Mn	TBX	ORAL SQUAMOUS CELL CARCINOMA
	185	42D	M	TB	INGUINAL HERNIA Small intestinal obstruction Pneumonia
	186	20Y	F	Donkey	HAEMORRHAGIC COLITIS IRREGULAR DENTITION Oral ulceration Gastric ulceration Myocardial infarction
	187	5Y	Mn	TB	Peritonitis Intestinal ulceration

	188	22Y	F	Shetland	STRANGLES
	189	2M	M	Danish WB	FRACTURED LIMB Diarrhoea Intestinal ulceration
July	190	98D	F	TBX	FRACTURED LIMB
	191	13Y	F	Irish Draft	INTESTINAL LEAKAGE (POST OPERATIVE) PERITONITIS
	192	12Y	F	Shetland	HYPERLIPAEMIA Diarrhoea
August	193	4Y	Mn	Welsh Cob	GRASS SICKNESS Oesophageal ulceration Gastric ulceration Cyathostomes
August	194	3M	F	Clydesdale	ACUTE MYOSITIS
	195	7Y	Mn	TB	SMALL INTESTINAL OBSTRUCTION PERITONITIS SPLENIC ABSCESS
	196	5D	F	TBX	SALMONELLOSIS RENAL NECROSIS
	197	3Y	Mn	TB	DEGENERATIVE JOINT DISEASE
September	198	92D	F	TB	SALMONELLOSIS Gastric ulceration Intestinal ulceration
	199	12Y	F	RH	COLONIC VOLVULUS SMALL INTESTINAL VOLVULUS COLONIC RUPTURE PERITONITIS
October	200	7Y	F	TBX	EPIPLOIC ENTRAPMENT GASTRITIS Lipoma
October	201	18Y	Mn	TBX	CHRONIC OBSTRUCTIVE PULMONARY DISEASE Melanomata
	202	7Y	F	TBX	COLONIC VOLVULUS Intestinal ulceration Cyathostomes
	203	6Y	Mn	Highland	COLONIC PERFORATION PERITONITIS CRANIAL MESENTERIC ARTERIAL THOMBOSIS

					THROMBOEMBOLISM
November	204	15Y	Mn	Highland	RAGWORT POISONING LARYNGEAL PARALYSIS Cerebral oedema Pituitary adenoma
	205	16Y	Mn	RP	ARTERITIS LARGE INTESTINAL NECROSIS LARGE INTESTINAL ULCERATION PERITONITIS Cirrhosis
November	206	6M	F	Hanoverian	OSTEOCHONDROSIS DISSECANS ARTHRITIS Intestinal ulceration Ascarids Cyathostomes *Gasterophilus intestinalis*
	207	4Y	Mn	Holstein	INGUINAL HERNIA SMALL INTESTINAL OBSTRUCTION PERITONITIS Haemorrhagic pancreatitis Scrotal abscess
	208	16Y	F	RH	CHRONIC LAMINITIS Sinusitis
December	209	16Y	F	RH	PITUITARY ADENOMA CUSHING'S SYNDROME LAMINITIS Osteoporosis Ascarids Cyathostomes
	210	3Y	Mn	RP	STOMATITIS PARASITIC PNEUMONIA
	211	12Y	Mn	TB	VERTEBRAL MALFORMATION
	212	5y	Mn	TB	ARTHRITIS
	213	Adult	F	Pony	RECTAL TEAR Hepatic calcification.

APPENDIX 3: NECROPSY SERIES SYSTEM INVOLVEMENT

The Cases studied in the Necropsy Series are listed below grouped by the system in which lesions were present. All the lesions of each system and the cases with those lesions were a principal or incidental finding are detailed.

Musculo skeletal System

LESION	CASE NUMBERS	
	PRINCIPAL FINDING	INCIDENTAL FINDING
Joint Subluxation/ degenerative joint disease	29,95,96,100,105, 131,136,148,157, 197,206,212	67
Fractured limb	38,77,108,120,128, 147,174,189,190	
Tendon rupture/inflammation	37,102,160,176	
Spinal Subluxation	36,68,170	
Discospondylitis	93,130,183	
Osteomyelitis	63,94,113	
Fractured spine	73,138	
Vertebral malformation	17,211	
Botulism	30,32	
Osteochondritis dissecans	206	
Angular Limb deformity	154	
Discospondylosis	122	
Acute myositis	194	
Postoperative myopathy	40	
Navicular disease		124,175
Osteoporosis		164,209
Sarcocystis		161
Muscle abscess		161

Respiratory System

CONDITION	CASE NUMBER	
	PRINCIPAL FINDING	INCIDENTAL FINDING
Chronic obstructive pulmonary disease	44,45,57,64,110, 111,127,175,201	67,139
Strangles	15,25,35,188	
Acute exudative pnuemonia	107,118	
Laryngeal paralysis	121,204	
Pneumonia	72	104,172,185,164
Inhalational pneuomia	7	
Paracytic pneumonia	210	
Asphyxiation	23	
Pleurisy	118	
Guttural pouch empyema	139	
Guttural pouch mycosis	135	
Nasal polyp	124	
Hydatidosis	21,172	
Sinusitis	208	
Bronchiolitis	52	

Nervous System

CONDITION	CASE NUMBER	
	PRINCIPAL FINDING	INCIDENTAL FINDING
Spinal cord compression	17,36,68,91,93,122, 130,183	
Cerebral oedema	156	154,204
Ocular lesions	118	133
Cauda equina neuritis	129	
Peripheral neuropathy	123	
Chronic adhesive meningitis	158	
Multifocal encephalitis	148	
Vagosympathetic transection	41	
Brain abscess		55

Hepatic System

CONDITION	CASE NUMBER	
	PRINCIPAL FINDING	INCIDENTAL FINDING
Fatty degeneration	5,6,78,85,142,145, 149,192	89,114,126,161
Cirrhosis	10,121	139,150,169,203, 205
Haemochromatosis/ haemosoderosis	53	1,3,26,92,106, 112,150,155,172
Ragwort poisoning	204	58
Cholagitis/ cholangiohepatitis	107	26
Liver failure	156	
Parasitic hepatitis/cysts		21,67,150,154,164
Hepatic calcification		213

Cardiovascular System

CONDITION	CASE NUMBER	
	PRINCIPAL FINDING	INCIDENTAL FINDING
Acute heart failure	51,96,125,181	
Arteritis	135,205	
Cranial mesenteric arterial thrombosis/aneurysm	203	8,33,39,50,52,76, 89,110,136,159,164
Thromboembolism	203	
Ruptured chorda tendinea	61	
Patent ductus arteriosis	86	
Aortic insufficency	88	
Myocardial infarction		18,79,95,100,186
Aortic mineralisation		4

Haemopoietic System

CONDITION	CASE NUMBER	
	PRINCIPAL FINDING	INCIDENTAL FINDING
Lymphosarcoma	3,4,24,26,71,137	
Splenic abscess	195	31
Anaemia	48	4
Multiple myeloma	134	

Upper Alimentary System

CONDITION	CASE NUMBER	
	PRINCIPAL FINDING	INCIDENTAL FINDING
Oral squamous cell carcinoma	46,184	
Abnormal dentition	112,186	
Ameloblastoma	171	
Cleft palate	66	
Oesophagitis	107	
Stomatitis	210	
Oesophageal ulceration		9,12,13,16,19,20, 43,47,59,60,87, 109,141,161,193, 177,178,197
Oral ulceration		112,186
Oesophageal papilloma		2

Integumentary System

CONDITION	CASE NUMBER	
	PRINCIPAL FINDING	INCIDENTAL FINDING
Melanomata	22,56,67,91,106	52,139,201
Dermatitis	142	65
Sweet itch		54,141
Photosensitisation		121,150
Papillomata		136
Sarcoid		154
Dermatophilosis		146
Sebaceous cyst		172

Reproductive System

CONDITION	CASE NUMBER	
	PRINCIPAL FINDING	INCIDENTAL FINDING
Inguinal hernia	185,207	
Vaginal tear	116	
Dystocia	81	
Abortion	151	
Inflammatory hydrocoel		2
Scrotal abscess		207
endometritis		172

Endocrine System

CONDITION	CASE NUMBER	
	PRINCIPAL FINDING	INCIDENTAL FINDING
Pituitary adenoma/hyperplasia	55,101,164,209	32,72,172,180,204
Thyroid nodular hyperplasia		101,171,180
Thyroid adenoma		57
Thyroid cyst		135
Phaeochromocytoma		139

Urinary System

CONDITION	CASE NUMBER	
	PRINCIPAL FINDING	INCIDENTAL FINDING
Ruptured bladder	90	
Renal necrosis	196	
Renal infarction		18,21,26,101,102, 135,172
Cystitis		129,155,172
Renal abscessation		112,172
Klossielosis		164
Interstitial nephritis		58
Hypercalcaemic nephropathy		4

Miscellaneous Conditions

CONDITION	CASE NUMBER	
	PRINCIPAL FINDING	INCIDENTAL FINDING
Hyperlipaemia	5,6,78,142,192	
Cushing's syndrome	55,101,164,209	
Salmonellosis	69,196,198	
Diaphragmatic rupture	52,82	
Septicaemia	49,118	164
Poorly differentiated tumour	98	
Haemorrhagic pancreatitis		207
Sialoadenitis		3
No diagnosis	8,83,115,152	

APPENDIX 4 NECROPSY SERIES: GASTROINTESTINAL SYSTEM INVOLVEMENT

The cases with lesions in the gastrointestinal system which were examined in this study are listed below grouped by the gastrointestinal condition present at post mortem examination. For each condition the pertinent details and the cases principally or incidentally affected are given.

CAUSE	CASE NUMBER	
	PRINCIPAL FINDING	INCIDENTAL FINDING
Gastric rupture	12,76,144,166,173	
Intestinal leakage	75,84,114,153,191	
Large intestinal perforation	116,199,203	
Rectal perforation	126,159	
Small intestinal perforation	117	
Inguinal hernia	207	
Large intestinal necrosis	205	
Sand colic	34	
Ruptured bladder	90	
Splenic abscesses	195	
Granulomatous enteritis		155,161
Unknown	52,80,167,169	64,119,187

Table 4-1. The 30 cases of peritonitis where it occured as a principal or incidental lesion and the most probable cause in each case.

LESION	CASE NUMBER
Small intestine	
Volvulus/torsion	76,84,109,119,199
Obstruction	39,144,173,195
Hernia	90,185,207
Intussusception	104
Large intestine	
Volvulus	74,79,143,199
Entrapment	82,168,200
Obstruction	34

Table 4-2. The 22 cases of intestinal displacement/obstruction, where it occured as a principal finding, and the actual lesion responsible in each case.

YEAR	CASE NUMBER
1987	9,12,13,14,16,19,20,27
1988	42,43,47,50,59,60
1989	87
1990	140,141
1991	177,178,179,182,193

Table 4-3. The 22 cases of grass sickness and the year in which each case occured.

MONTH	CASE NUMBER
February	42,43
March	
April	
May	9,12,13,14,47,177,178,179
June	16,19,20,50,87,182
July	
August	27,140,103
September	59,60,141

Table 4-4. The 22 cases of grass sickness and the month in which each case occured.

LESION	CASE NUMBER	
	PRINCIPAL FINDING	INCIDENTAL FINDING
Gastric rupture	12	
Oesophageal ulceration		9,12,13,16,19,20, 43,47,59,60,87, 141,177,178,182,193
Gastric ulceration		9,12,13,16,19,43, 59,179,182,193
Splenic enlargement		16,20,87,177,178
Cyathostomes		9,13,182,193
Typhlitis		27
Intestinal ulceration		43
Haemomelasma ilei		50
Cranial mesenteric arterial thrombosis		50
Lipoma		141
Sweet itch		141

Table 4-5. The 22 cases of grass sickness and the additional principal and incidential GI lesions present in each case.

DIAGNOSIS	CASE NUMBER	
	PRINCIPAL FINDING	INCIDENTAL FINDING
Alimentary lymphosarcoma	3,24,26	
Enterotyphlocolitis	1,70,159	
Granulomatous enteritis	155,161	
Eosinophilic enteritis	21,172	
Cyathostomiasis	11,150	
Protein-losing enteropathy	126	
Coccidiosis	28	
Granulomatous typhlocolitis	92,62	
Granulomatous colitis	62	
Typhlocolitis	2	
Enteritis	80	
Lymphoid hyperplasia	31	
Dental exhaustion	112	
Chronic enteropathy		114

Table 4-6. The 21 cases with an enteropathy as a principal or incidental finding and the final diagnosis made in each case.

DIAGNOSIS	CASE NUMBER	
	PRINCIPAL FINDING	INCIDENTAL FINDING
Colitis	33,52,58,69,97, 162,186,196,198	27
Typhlitis	69,97,198	27
Enteritis	69,198	
Gastritis	200	
Diarrhoea		5,6,189,192

Table 4-7. The 16 cases with acute regional inflammation as a principal or incidental finding and the lesion responsible in each case.

LOCATION	CASE NUMBER
Stomach	12,76,78,144,166,173
Rectum	98,126,159,213
Caecum	116,153
Colon	199,203
Small Intestine	117

Table 4-8. The 15 cases of intestinal rupture and the location of the rupture in each case.

CAUSE	CASE NUMBER
Duodenal obstruction	144,166,173
Small intestinal volvulus	76
Grass sickness	12
Hyperlipaemia	78

Table 4-9. The 6 cases of gastric rupture and the most probable cause in each case.

LOCATION	CASE NUMBER	
	PRINCIPAL FINDING	INCIDENTAL FINDING
Large Intestine	52,58,92,97,103, 116,126,162,205	24,26,74,79,80,98, 161,169,189,202
Small Intestine	144,155,161,198	98,113,132,159, 189,206
Stomach		9,12,13,16,18,19, 26,31,43,59,70,74, 83,101,127,131, 162,172,182,193, 198

Table 4-10. The 42 cases of regional ulceration as a principal or incidental finding and the location in each case.

ASSOCIATED PRINCIPAL LESIONS	CASE NUMBERS PRINCIPAL ULCERATION	INCIDENTAL ULCERATION
Ulcerative colitis	52	
Fungal enteritis	58	
Granulomatous typhlocolitis	92	
Acute haemorrhagic typhlocolitis	97	
Cyathostomiasis	103	
Caecal perforation	116	
Protein-losing enteropathy	126	
Necrotising colitis	162	
Enteritis	205	80
Colonic volvulus		74,79,202
Alimentary lymphosarcoma		24,26
Poorly differentiated tumour		98
Cirrhosis		169
Granulomatous enteritis		161
Fractured leg		189

Table 4-11. The additional principal lesions associated with the 16 principal and incidental cases of large intestinal ulceration and the cases with which each lesion was associated.

ASSOCIATED PRINCIPAL LESIONS	CASE NUMBERS PRINCIPAL ULCERATION	INCIDENTAL ULCERATION
Granulomatous enteritis	155,161	
Duodenal obstruction	144	
Salmonellosis	198	
Enterotyphlocolitis		159
Poorly differentiated tumour		98
Osteomyelitis		113
Laminitis		132
Fractured leg		189
Arthritis		206

Table 4-12. The additional principal lesions associated with the 9 cases of principal and incidental small intestinal ulceration and the cases with which each was associated.

ASSOCIATED PRINCIPAL LESIONS	CASE NUMBER INCIDENTAL ULCERATION
Grass sickness	9,12,13,16,19,43,59, 182,193
Alimentary lymphosarcoma	26
Lymphoid hyperplasia	31
Enterotyphlocolitis	70
Necrotising colitis	162
Eosinophilic enteritis	172
Colonic volvulus	74
Salmonellosis	198
Chronic obstructive pulmonary disease	127
Polyarthritis	131

Laminitis	18
Cushing's Syndrome	101
Inconclusive	83

Table 4-13. The additional principal lesions associated with the 20 cases of incidental gastric ulceration.

	CASE NUMBER	
PARASITE/PARASITIC LESION	**PRINCIPAL FINDING**	**INCIDENTAL FINDING**
Cyathostomes	11,103,145,149,150	3,9,13,52,112,116, 122,123,148,182, 193,202,206,209
Cranial mesenteric arterial thrombosis/aneurysm	203	8,33,39,50,52,76, 89,110,112,136, 159,164,166
Eimeria leukarti	28	
Haemomelasma ilei		1,31,33,50,117, 154,160,162,163, 164,165,169
Large strongyles		41,117,136
Ascarids		206,209
Tapeworms		79,132
Gasterophilus intestinalis		119,206

Table 4-14. The 42 cases with parasitism as a principal or incidental lesion and the cause of the lesion in each case.

	CASE NUMBER	
MONTH	**PRINCIPAL**	**INCIDENTAL**
January		
February		3
March		112
April		
May	11	9,13,116,122,123
June		52,182
July		
August		193
September	145	
October		148,202
November	103,149,150	206
December		209

Table 4-15. The 18 cases in which cyathostomes were a principal or incidental finding at necropsy and the month in which each case was examined.

MONTH	**CASE NUMBER**
October	31,33
November	154
December	160
January	1,162,163,164,165
February	

March	169
April	117
May	
June	50

Table 4-16. The 10 cases in which *Haemomelasma ilei* was an incidental lesion at necropsy and the month in which each case was examined.

CONDITION	CASE NUMBER	
	PRINCIPAL FINDING	INCIDENTAL FINDING
Arteritis	205	
Thromboembolism	203	
Lipomata	168	141,146,180,200
Gastric impaction		156

Table 4-17. The 8 cases with a miscellaneous lesion of the GI tract and the cases where each lesion was present as a principal or incidental finding.

APPENDIX 5: ENTEROPATHY SERIES:

THE POST MORTEM EXAMINATION FINDINGS IN DETAIL

The Necropsy Series numbers have been placed within brackets

Case 1 (24E)

13Y Mn Pony

ALIMENTARY LYMPHOSARCOMA

Intestinal ulceration

The carcass was emaciated and oedematous. The intestines were fluid filled and dilated. The serosal lymphatics, especially those of the large intestines, were also dilated. There was prominence of the intestinal lymph nodes, most marked at the ileocaecocolic junction. The intestinal wall was thickened throughout with extensive thickened, oedematous or corrugated plaque-like areas on the mucosal surface. Some of these areas were ulcerated, as were areas of the mucosa, where there was superficial diptheresis. There was patchy oedema of the gastric and rectal walls. A few fibrous tags were present over the peritoneum and there were a small number of nodules on the pleural surface of both lungs, both consistent with previous parasitism. The carcass lymph nodes were not enlarged but the splenic white pulp was prominent.

Microscopic examination of the small intestines revealed diffuse infiltration of the lamina propria by large round lymphoblastic cells with a moderate mitotic rate and occasional abnormal mitotic figures. The

infiltrate had caused marked flattening of the mucosa with stunting and fusion of the villi and lengthening of the crypts. There was a marked increase in intraepithelial lymphocyte numbers. In some areas the infiltrate spilled over into the submucosa. There were distinct, sharply demarcated areas of ulceration extending in some areas to the muscularis mucosa, with fibrin, bacteria and mixed inflammatory cells on the surface. There was oedema of the submucosa.

Examination of the large intestines revealed areas of marked infiltration of the lamina propria, producing a solid highly cellular mucosa. The infiltrate was similar to that of the small intestine. In other areas the infiltrate was less marked and the mucosa was oedematous. There was marked oedema of the submucosa throughout along with occasional haemosiderin laden macrophages and mast cells.

The intestinal lymph nodes were reactive with large follicles in the cortex. The medulla was more solid, populated by sheets of homogenous cells.

Case 2 (26E)

15Y F Pony

ALIMENTARY LYMPHOSARCOMA

Gastric Ulceration

Intestinal ulceration

Cholangiohepatitis

Hepatic Haemosiderosis

Renal Infarction

At necropsy the intestinal mucosa was thickened with irregular ulcers in the dorsal colon and at the caecolic junction. There was also shallow and deep ulceration of the squamous portion of the stomach. The mesenteric lymph nodes were swollen and enlarged with a rounded outline. In addition there was slight mottling of the liver along with more distinct haemorrhagic areas up to 2cm in diameter. There was mild scarring and streaking of the renal cortices and occasional haemorrhages on the splenic capsule.

Microscopic examination of the small intestines revealed oedema of the lamina propria and submucosa throughout. Many reactive Peyer's patches with pale centres were present in the ileum. This involved both the mucosa, where there was a localised increase in intraepithelial lymphocytes, and muscularis mucosa where cells spread out some distance on either side.

Changes were present throughout the large colon and caecum. These were most marked in the dorsal colon where there was extensive infiltration of the mucosa, muscularis mucosa and submucosa. In some areas this resulted in a solid sheet of cells overlying the muscle layers with repeated attempts at follicle formation in the submucosa. The infiltrate was composed predominantly of large round fairly monomorphic lymphoid cells with a moderate mitotic rate and only occasional plasma cells, eosinophils and globule leukocytes. The mucosal architecture was distorted with loss of crypts. Areas of deep ulceration extending to the muscularis mucosa and lined by a layer of fibrin and polymorphs were present. In other areas the surface epithelium had become low cuboidal. In the ventral colon the infiltrate was less intense but similar in distribution and composition with attempts at follicle formation in the submucosa. There was only shallow ulceration or loss of surface epithelium with less distortion of mucosal architecture. As a result more crypts were evident and there was an increase in intraepithelial lymphocytes. Examination of the caecum revealed an intense but mixed mononuclear cell and eosinophil mucosal infiltrate with only slight overspill into the submucosa. The submucosa was oedematous and did however contain many reactive lymphoid follicles at the centre of which were large pale cells with a high mitotic rate. There was a general increase in intraepithelial lymphocytes througout the mucosa. In the colonic lymph nodes there was loss of normal architecture with the medulla replaced by solid sheets of lymphoid cells with a moderate to high mitotic rate and occasional bizzare mitotic figures. Occasional, more normal reactive follicles were apparent in the superficial cortex.

In the liver there was focal haemorrhage and necrosis with an increase in fibrous tissue. In the periportal areas there was a mononuclear inflammatory cell infiltrate and a large amount of haemosiderin within macrophages.

Examination of sections of stomach confirmed the ulceration, which in some areas extended through the subepithelium and revealed that the renal lesions were infarcts.

Case 3 (3E)

15Y F Riding Pony

ALIMENTARY LYMPHOSARCOMA

Cyathosomes

Hepatic Haemosiderosis

Thrombophlebitis

Sialoadenitis

The animal was in poor bodily condition. Petechiae were present in the caecum. There were petechiae and small nodules, resembling those of cyathostome larvae, in the colonic mucosa especially of the dorsal colon. The small intestines, abdominal nodes, liver, pancreas, and kidneys appeared grossly normal. In addition there was abscessation and necrosis of the left parotid salivary gland and thrombo-phlebitis of the right jugular vein.

Microscopic examination of the small intestines revealed only moderate oedema of the submucosa, which extended to involve the lamina propria in the ileum, and a mild eosinophil infiltrate in the lamina propria and submucosa of the duodenum and ileum.

Examination of the caecum and large colon revealed a very dense mucosal infiltrate, composed almost entirely of lymphoid cells, which had greatly increased the depth of the mucosa in the dorsal colon. These cells spilled over into the submucosa, filling it in some areas of the caecum and ventral colon and a great many were also present in the epithelium. In other areas there was loss of surface epithelium with a minimal to absent inflammatory cell response. At these points the mucosa had been invaded by large protozoa resembling those of the normal commensal population. In addition, occasional lymphoid follicles were present in the oedematous submucosa. In the small colon the infiltrate was much less marked with minimal overspill into the submucosa and only a moderate number of intraepithelial cells. Normal, reactive Peyer's patches were also apparent. The hepatic and pancreatic lymph nodes contained cells similar to those of the large intestinal infiltrate and a great many haemosiderin laden macrophages. The latter were also present, periportally in the liver along with a mild mixed mononuclear inflammatory cell infiltrate. Bile pigment was obvious in the sinusoids.

Case 4 (155E)

13Y F Arab X

GRANULOMATOUS ENTERITIS

INTESTINAL ULCERATION

Peritonitis

Hepatic Haemosiderosis

Cystitis

At necropsy the small intestinal wall contained a number of fibrous thickenings some causing stenosis with dilatation proximally. There was adhesion of the omentum and thickening of the mesentry at the majority of these thickenings. At the remainder the serosa was markedly reddened. The duodenal wall was thickened generally and the serosa rough. Further lesions became apparent on opening the small intestines. The proximal 1.5m were thickened. The next 1.5m were also thickened and contained two of the fibrous thickenings, each 5cm in diameter with necrotic, deeply ulcerated centres, two deep ulcers, each 4 x 2.5cm, a shallow ulcer and an area of scarring. The remaining small intestine contained two shallow ulcers with associated scarring, 5.5m and 8.5m distal to the pylorus and a large necrotic nodule 2.5m proximal to the caecum. The stomach, large intestines and abdominal lymph nodes appeared uninvolved. In addition, the bladder mucosa was hyperaemic and thickened.

Microscopic examination of the small intestines revealed a great many changes and much variation between sections examined In some areas the mucosa was relatively normal with the expected crypt to villus ratio,

minimal infiltration by inflammatory cells, and few intraepithelial lymphocytes with only mild oedema of the associated submucosa. In most areas however, the villi were short and broad, possibly with florid, frond-like tips. There was a moderate mixed granulomatous infiltrate in the lamina propria, composed of lymphocytes, plasma cells and macrophages, which along with oedema, pushed the crypts apart.

These changes were occasionally accompanied by multiple large, reactive, lymphoid follicles, spanning the deep mucosa, muscularis mucosa and superficial submucosa, and spilling cells into the surrounding tissue. Where the follicles were confined to the mucosa there was loss of overlying epithelium. Many pale, foamy macrophages were present in the superficial submucosa. The muscle layers and serosa were thickened and oedematous with occasional inflammatory cells.

Adjacent to the small intestinal lesions, seen grossly, the mucosa was extremely abnormal with a great many intraepithelial lymphocytes. The granulomatous infiltrate was much more marked and accompanied by eosinophils. This had resulted in flattening of the mucosa with subtotal villus atrophy and superimposed ulceration. There were areas of crypt hyperplasia altering the crypt to villus ratio or crypt dysplasia and mucosal collapse.

At the fibrous thickenings the granulomatous and eosinophil infiltrate spanned the whole thickness of the wall from the deeply ulcerated lumen, with fibrin and granulation tissue, to the thickened, oedematous serosa.

Many haemosiderin laden macrophages were apparent. At some points the wall was composed entirely of infiltrated fibrous tissue with occasional lymphoid follicles or granulomas.

Examination of the large intestines revealed marked oedema and moderate eosinophil infiltration of the mucosa and submucosa. In the caecum and large colon there was some loss of surface epithelium, a marked increase in the intraepithelial lymphocytes and lymphocytolysis in the Peyer's patches. In addition there was calcification of the intima of submucosal blood vessels.

In the mesenteric lymph nodes there was B-cell expansion. Reactive follicles filled both the cortex and medulla. The cells at the follicle centres were quite large and pale with a high mitotic rate and individual cell death producing a starry sky effect.

The squamous portion of the stomach was thickened and hyperkeratotic. There was mild cuffing of the submucosal vessels in the glandular portion by mononuclear cells.

In the liver there was a mild mononuclear periportal infiltrate and a moderate amount of haemosiderin in periportal macrophages and hepatocytes.

The bladder wall is thickened with a hyperplastic epithelium and lymphoid follicles in the connective tissue.

No abnormalities were detected in the pancreas.

Case 5 (161E)

15Y Mn Riding Pony

GRANULOMATOUS ENTERITIS

INTESTINAL ULCERATION

Peritonitis

Oesophageal Ulceration

Hepatic Lipidosis

Muscle Abscess

Sarcocysts

At necropsy the animal was in extremely poor bodily condition. There was marked wear of the toes of both front hooves and an abscess in the ventral midline abdominal wall. A short length of the ileum, 1m proximal to the caecum, was markedly fibrosed to form an 8cm diameter nodule to which the omentum was adhesed. Opening the intestines revealed that the small intestinal mucosa was thickened and rough throughout. Shallow 1-2cm diameter ulcers were present from 4m distal to the stomach which increased in frequency distally. There were also areas of petechial haemorrhages in the mucosa. The fibrous mass was cavitated and in communication with the lumen. The large intestines were oedematous throughout. The mucosa, including that of the rectum contained multiple shallow 2-15cm diameter ulcers. A deeper necrotic area, possibly at the biopsy site, was also present in the rectal mucosa. Shallow ulcers were present in the

oesophagus, this was most marked distally. In addition the right thyroid was three times the size of the left and there was a moderate amount of yellow fluid in the pericardial sac.

Microscopic examination of the proximal duodenum revealed only marked oedema of the lamina propria and submucosa and an increase in the intraepithelial lymphocytes. More caudally in the duodenum and jejunum marked mucosal changes became apparent. There was stunting and fusion of villi progressing to subtotal villus atrophy with attenuation or loss of the surface epithelial cells. There were a great many intraepithelial lymphocytes and a marked mucosal infiltrate composed predominantly of lymphocytes and plasma cells but including macrophages polymorphs and mast cells, the latter occasionally present in small clumps. There was variable overspill of the infiltration into the extremely oedematous submucosa and occasional lymphoid follicles. In some areas the lamina propria was composed largely of loose oedematous fibrovascular tissue suggestive of scarring. The serosa was generally thickened and oedematous. In the ileum there was stunting and fusion of villi, oedema of the lamina propria and submucosa, mononuclear inflammatory, eosinophil and mast cell mucosal infiltrate and large numbers of intraepithelial lymphocytes. Large lymphoid follicles in the muscularis mucosa were spilling cells into the submucosa. Adjacent to the ileal nodule there was flattening of the mucosa with subtotal villus atrophy, dilated, dysplastic crypts, attentuated surface epithelium and high numbers of intraepithelial lymphocytes. There was a marked granulomatous inflammatory infiltrate in the mucosa spilling over

into the extremely oedematous submucosa which also contained many large lymphoid follicles and granulomata.

Examination of the nodule itself revealed massive fibrosis with granulation tissue at both the mucosal and serosal surfaces. There was a marked granulomatous infiltrate including discrete lymphoid nodules and granulomata spanning the full thickness of the wall.

Examination of the caecum revealed oedema of the submucosa and mucosa. There was a moderate lymphocyte and plasma cell infiltrate, with occasional mast cells and eosinophils in the mucosa spilling over into the submucosa and reactive lymphoid follicles in the submucosa. Many lymphocytes were also present in the epithelium.

The deep area of necrosis in the rectum was composed of granulation and fibrous tissue with bacterial clumps on the lumenal surface. Throughout both the small and large intestines there was moderate mononuclear inflammatory cell infiltration of the submucosal autonomic nerve plexuses and some neuronal degeneration.

There was slight hyperkeratosis of the squamous portion of the stomach.

The mesenteric lymph nodes were reactive and contained quite a high proportion of large, apparently immature lymphoid cells and macrophages in the cortex.

In the liver there was diffuse hydropic change and periportal fatty change. There was a marked mononuclear infiltrate around the major bile ducts, which was much less marked round the minor ducts.

In addition to necrosis and inflammation in the ventral abdominal wall a large number of sarcocysts were present within the muscle.

Case 6 (172E)

16Y F Riding Horse

EOSINOPHILIC ENTERITIS

Gastric Ulceration

Endometritis

Cystitis

Renal Abscess

Renal Infarction

Focal Pneumonia

Pituitary Adenoma

Hepatic Haemosiderosis

Pulmonary Hydatidosis

Sebaceous Cyst

The animal was emaciated. The abdomen contained a small amount of yellow fluid. The large intestinal and mesenteric lymph nodes were enlarged, pale and oedematous. There was a 1cm ulcer at the edge of the glandular zone of the stomach. The major and minor duodenal papillae were enlarged and covered by orange necrotic diptheritic 0.5cm plaques, which were also

present on the adjacent mucosa for approximately 15cm. The rest of the small intestinal mucosa appeared slightly oedematous. The ileocaceal valve was hyperaemic. The large intestines were also oedematous and the contents extremely fluid. The mucosa of the caecum and large colon was completely covered by multiple dark granular apparently ulcerated areas, up to 1cm in diameter. These coalesed in some areas.

The pancreatic and bile ducts were both dilated, thickened and fibrous. The body of the pancreas was firm and fibrous with dilated cystic spaces.

In addition both kidney cortices contained three old infacts and there was a 1.5cm diameter abscess in the right kidney. The bladder mucosa was peppered with petechial haemorrhages.

The uterus was distended by white fluid. The lungs contained four small hydatid cysts and two suppurative areas each 6x3x2cm. There was a sebaceous cyst in the skin over the right pectoral region.

Microscopic examination of the small intestines revealed marked oedema of the mucosa and submucosa at all levels. The villi appeared slightly short and broad and those of the ileum had florid, frond-like tips. There was a slight increase in intraepithelial lymphocytes. The mucosa of the duodenum around the papillae, was oedmatous, flattened and dysplastic with subtotal villus atrophy. Large masses of eosinophils were present on the mucosal surface. These masses had invaded the mucosa and appeared to stream out and coalesce, especially over the surface of the major duodenal papilla,

to form a thick membrane. These masses also extended down into the duct. There was a mononuclear inflammatory cell and eosinophil infiltrate within the mucosa and submucosal glands and throughout the papillae. Large clumps of Gram positive rods and some Gram negative cocci were present on the surface of the mucosa and over the eosinophilic debris.

The changes at the ileocaecal valve were similar with eosinophil masses streaming out over the surface of a dysplastic mucosa. There was some loss of surface epithelium and a moderate mononuclear mucosal infiltrate with only low numbers of eosinophils. This infiltrate spilled over and almost obliterated the oedematous submucosa where it contained a much higher proportion of eosinophils.

Examination of the caecum and large colon revealed that the dark areas seen grossly were masses of eosinophils but these tended to be discrete, rounded and sitting within the mucosa on the muscularis mucosa. The mucosal epithelium on either side was intact forming vertical walls. Occasionally less discrete masses spread over the mucosal surface but only penetrated the mucosal surface for a short distance. There was a moderate mononuclear mucosal infiltrate which spilled over into an extremely oedematous submucosa where it was joined by large numbers of eosinophils.

There was a slight increase in the number of intraepithelial lymphocytes and occasional protozoa were apparent on the mucosal suface and in crypts.

The large intestinal lymph nodes were reactive and oedematous. Large granulomata with a central mass of eosinophils surrounded by macrophages, epitheloid cells, multinucleate giant cells and a variable amount of fibrosis were present in the medulla and capsule along with large numbers of free eosinophils.

The pancreas was extremely fibrous. The ducts were fibrosed and dilated. Their epithelium and connective tissue was densly infiltrated by mononuclear cells and eosinophils. Eosinophilic granulomata, similar to those in the lymph nodes were present in the fibrous tissue along with many eosinophils and mononuclear cells.

In the liver there was a mild mononuclear periportal infiltrate and a moderate amount of haemosiderin within hepatocytes.

In the lung lesions there was cuffing of airways, predominantly by mononuclear cells, and large numbers of polymorphs within the bronchioles and also in the alveoli and alveolar walls. Many eosinophils were present in the fibrous connective tissue.

The bladder wall was thickened and oedematous. There was superficial congestion and haemorrhage and a mixed inflammatory infiltrate below.

Examination of the pituitary gland revealed an adenoma of the par intermedia which had invaded the pars nervosa and displaced the par distalis.

Case 7 (21E)

20Y Mn Riding Horse

EOSINOPHILIC ENTERITIS

Hepatic Hydatidosis

Pulmonary Hydatidosis

Renal Infarction

The animal was extremely emaciated. The mucosa of the small intestines was thickened and that of the ileum contained small nodules. The large intestinal mucosa was oedematous and peppered with multiple small nodules and dark spots. In addition the heart was flabby with a dilated right ventricle but there was no evidence of heart failure. The liver surface was covered by fibrous tags and there were areas of necrosis, especially around the hilus on the diaphragmatic surface. One small hydatid cyst was present in the liver and another in the right lung.

Microscopic examination of the duodenum revealed shortening and thickening of the villi, a mild plasmacytic infiltrate in the lamina propria with occasional eosinophils and oedema of the lamina propria and submucosa. The jejunal villi were similar but the infiltrate was more marked, with more eosinophils and it spilled over into the muscularis mucosa and submucosa. Again, there was oedema of the lamina propria and submucosa. The ileum was generally similar to the jejunum but with still more eosinophils and with the addition of large lymphoid nodules in the mucosa. However, occasional large masses of eosinophils were present sitting in depressions in the

mucosa. Adjacent to these masses the mucosa and submucosa contained an intense eosinophil infiltrate with a milder mononuclear cell infiltrate in the submucosa along with occasional lymphoid nodules.

Examination of the large intestines revealed marked oedema of the submucosa. There was a moderate eosinophil and mononuclear cell mucosal infiltrate which became much more intense in the superficial submucosa. There was a moderate increase in intra epithelial lymphocytes.

The intestinal nodes were reactive and contained a dense eosinophil infiltrate in the capsule and in the fibrous tissue within the node. The infiltrate was more moderate throughout the rest of the nodes and perinodal fat.

Case 8 (11E)

18 Y Mn Thoroughbred

CYATHOSTOMIASIS

The walls of the caecum and large colon were greatly thickened to approximately 2cm, by marked oedema of the mucosa. The mucosal surface was peppered with multiple dark 1-2mm spots and haemorrhages, approximately 20-30 per square cm. Close examination of the dark spots revealed coiled parasites. The small intestinal mucosa appeared thickened.

Microscopic examination of the small intestines revealed marked oedema of the lamina propria and dilatation of submucosal lymphatic vessels. In the

large intestine many worms were present within the mucosa. There was a generalised mixed mononuclear inflammatory mucosal infiltrate with overspill into the submucosa. Only a small number of eosinophils were apparent. In the mucosa the infiltrate was not specifically directed against the worms but in the submucosa it was most marked adjacent to the parasites. Occasional granulomata were present in the mucosa and submucosa centred on what appeared to be parasitic remnants. A small number of crypts contained cellular debris and polymorphs. There was marked oedema of the mucosa and submucosa and many submucosal lymphatics were plugged by lymphoid cells.

Case 9 (150E)

13y F Riding Pony

CYATHOSTOMIASIS

Cirrhosis

Hepatic Haemosiderosis

Hepatic Hydatidosis

Photosensitisation

The pony was in poor bodily condition. There was oedema of the ventral body wall and limbs. Two small (0.5cm diameter) and one large (1.5cm diameter) crusty plaques were present on the unpigmented skin of the muzzle. There were multiple 0.5cm diameter shallow ulcers in the nasal mucosa. The abdomen contained a small amount of straw coloured fluid.

The small intestines appeared empty but the large intestinal content was fluid and contained a large number of cyathostome larvae. The mucosa was extremely oedematous and peppered with multiple 2-3m pale nodules and pinpoint dark spots containing as yet unemerged larvae.

The liver was enlarged and pale with an irregular, slightly nodular surface and rounded edges. It contained two calcified hydatid cysts.

Microscopic examination of the intestines was not performed.

The hepatic pathology consisted of extensive fibrosis of the portal areas and portal bridging. The fibrous tissue contained many inflammatory cells, mostly polymorphs and haemosiderin laden macrophages which were also present below the thickened capsule. Haemosiderin was also seen within hepaocytes. Regenerative nodules were apparent. In the skin there was extensive epidermal necrosis and acute inflammation which extended into the dermis where there was marked vascular congestion and oedema. There was hyperkeratinisation and acantholysis with the clumps of bacteria on the surface and in the necrotic tissue.

Case 10 (126E)

14Y Mn Welsh Cob

PROTEIN-LOSING ENTEROPATHY

RECTAL TEAR

PERITONITIS

INTESTINAL ULCERATION

Myocardial Infarction

Hepatic Lipidosis

At necropsy there was extensive ventral oedema. There was a fibrinous peritonitis, most severe in the pelvic area, arising from an 8x10cm tear in the ventral wall of the rectum about 45cm proximal to the anus. There was no evidence of acute or recent haemorrhage. The edges of the tear were thick and fibrous and there were surrounding fibrous adhesions and pelvic fat necrosis. Acute fibrinous exudates and gut content were present over the caecum and pelvic flecture of the large colon. The large intestinal wall, especially that of the caecum was greatly thickened. This was due to mucosal oedema. No distinct ulceration was found but many pale, apparently necrotic areas, with surface petchiation were present in the terminal dorsal colon. The small intestinal wall was thickened and oedematous and the ileal gut associated lymphoid tissue was prominant, intestinal content was soft throughout and there was a moderate amount of grit in the caecum. The liver was pale mottled and friable. There was no evidence of active parasitism nor associated mesenteric arteritis. The only other finding was a small 0.5cm diameter scar in the wall of the left ventricle.

Microscopic examination of tissue from this horse revealed advancing autolysis which obscured much of the intestinal pathology. Examination of the small intestine confirmed the marked oedema and reactivity of the lymphoid deposits seen grossly with large reactive follicles present in both the jejunum and ileum. Cellular detail in the mucosa was poor but only a mild mononuclear cell infiltrate was apparent with occasional cells also present in the submucosa along with many dilated and congested vessels. The serosa was thick congested and oedematous with a layer of fibrin and inflammatory cells on the surface.

The picture in the large intestines was generally similar but the serosal lesions were much more marked especially in the small colon where there was thrombosis of serosal vessels and clumps of bacteria within the necrotic debris. The mucosal preservation was better and this revealed marked oedema and minimal/no inflammation.

Examination of the liver revealed diffuse fatty change and an acute inflammatory reaction and bacteria on the capsule surface.

No abnormalities were detected in the kidneys.

Case 11 (28E)

15Y Mn Riding Horse

INTESTINAL COCCIDOSIS

The major findings were limited to the alimentary tract. The mucosa of the small intestine was thickened throughout with a rather granular or cobble-stone appearance particularly in the distal ileum which was reminiscent of Johne's disease in cattle. The large intestine appeared macroscopically normal as did the majority of abdominal lymph nodes. A group of three small nodes attached to the ileum were dark red in colour.

In addition there were occasional small fibrous tags on the diaphragmatic surface of the liver and an area of fibrosis of 6cm maximum diameter on the margin of the dorsal lobe.

Microscopic examination of the small intestines revealed a moderate number of distinctive giant microgametocytes and macrogametes of *Eimeria Leukarti*. These were located in the lamina propria of the villi, with highest numbers present in the ileum. There was apparently no cellular response and only oedema of the villi throughout which spread to involve the crypt area in the ileum, and slight broadening of some villi again in the ileum.

Examination of the large intestines merely revealed changes consistent with mild parasitic activity. There was moderate submucosal and mild mucosal oedema, a slight mononuclear cell and eosinophil mucosal infiltrate which spilled over into the submucosa in places and the occasional reactive

lymphoid follicle in the mucosa or submucosa. There was calcification of the intima of submucosal arterioles in both the small and large intestine. The lymph nodes were reactive and oedematous with a moderate number of eosinophils.

Case 12 (70E)

8Y F Riding Pony

ENTEROTYPHLOCOLITIS

Gastric Ulceration

The animal was emaciated with very little carcase or visceral fat. There were few macroscopic abnormalities. The intestines from the ileocaecal junction caudally were fluid filled and the colonic mucosa appeared slightly granular. The mesenteric lymph nodes were enlarged and oedematous but had a normal cortical and medullary pattern. The small intestines were macroscopically normal and the only abnormality in the stomach was the presence of a few erosions at the plica. No other abnormalities were detected and there was no macroscopic evidence of parasitism.

Microscopic examination of the distal duodenum revealed slight submucosal oedema. There was a mild, mixed mononuclear cell infiltrate at the base of the crypts, occasional eosinophils in the lamina propria, a small number of neutrophils in the villi and a slight increase in intraepithelial lymphocyte numbers. In the ileum the oedema was more marked and extended into the lamina propria. There was slight inconsistent stunting and fusion

of the villi, the mononuclear cell infiltrate extended into the muscularis mucosa and there was a moderate increase in intraepithelial lymphocyte numbers.

Examination of the large intestines revealed a dense mononuclear cell infiltrate, composed of plasma cells and lumphocytes, in the mucosa and superficial half of the submucosa. There was minimal mucosal distortion but small superficial areas of necrosis with bacterial invasion were present and occasional crypts contained neutrophils. Intraepthelial lymphocyte numbers were markedly elevated and there were a number of lymphoid nodules in the mucosa, muscularis mucosa and submucosa. Only occasional eosinophils were apparent at any level. The intestinal lymph nodes were reactive, oedematous and congested.

In addition the liver was also congested with diffuse hydropic change.

Case 13 (1E)

15 Y F Thoroughbred

ENTEROTYPHLOCOLITIS

Haemomelasma ilei

Hepatic Haemosiderosis

Examination revealed no abnormalities other than old fibrous tags on the peritoneal surface of the liver and a solitary area of scarring, consistent with a healed ulcer in the stomach and red nodules on the serosal surface of the ileum resembling *Haemomelasma ilei*.

Microscopic examination of the small intestine revealed oedema of the mucosa and submucosa and shortening and broadening of the villi to give them a slightly stumpy appearance. There was slight loss of epithelium at the villus tips in the jejunum exposing dilated capillaries below. There was a mild mononuclear inflammatory cell and moderate eosinophil mucosal infiltrate with slight overspill into the submucosa. In addition occasional globule leukocytes could be seen within this infiltrate. Intraepithelial lymphocyte numbers were moderately elevated. Examination of the ileum confirmed that the serosal lesions were *Haemomelasma ilei* and composed of fibrous tissue containing a large amount of haemosiderin.

Examination of the caecum and large colon revealed a moderate mononuclear cell mucosal infiltrate which was slightly distorting the mucosa by pushing the crypts apart and spilled over into the submucosa. This infiltrate also contained a moderate number of eosinophils and globule leukocytes which were present occasionally in the epithelium along with a great many lymphocytes. Haemosiderin laden macrophages were apparent in the superficial submucosa. There was oedema of the mucosa and submucosa.

In the liver there was a moderate amount of haemosiderin within macrophages both scattered throughout the parenchyma and clumped in the periportal connective tissue.

Case 14 (159E)

20Y Mn Welsh Mountain Pony

ENTEROTYPHLOCOLITIS

RECTAL PERFORATION

PERITONITIS

Intestinal Ulceration

Cranial Mesenteric Arterial Thrombosis

The abdomen contained a large amount of bloody fluid and food material. A full thickness, 12cm tear was present in the ventral rectal wall 30cm from the anus. The entire intestinal wall was markedly thickened and oedematous, especially the caecum where it was greater than 1cm thick, and the ventral colon. The mucosa of the small intestines was necrotic and sloughing. The intestinal content was generally of normal consistency. At the junction of the ventral and dorsal colon it was dry and adherent to the mucosa which was haemorrhagic for a length of approximately 45cm. The small colon contained scanty dry faeces. In addition a thrombus was present in the cranial mesenteric artery overlying roughened endothelium. The liver was enlarged with slightly rounded edges and the kidneys were slightly enlarged and pale. The lungs were macroscopically normal.

Examination of the duodenum revealed loss of epithelium progressing to loss of villi, oedema of the remaining mucosa and a mild mixed mononuclear cell infiltrate in the crypt area. The serosa was thick, oedematous and reactive with a layer of fibrin and polymorphs on the surface. In the jejunum and ileum the changes were similar with the addition of eosinophils

and occasional globule leukocytes to a slightly more intense mononuclear cell infiltrate. Calcium was present in the intima of submucosal arterioles.

Examination of the large intestines revealed some loss of surface epithelium with dilated capillaries, occasional macrophages and mucosal oedema. There was a mild to moderate mononuclear and eosinophil mucosal infiltrate which included large numbers of globule leukocytes but caused little mucosal distortion. There was variable overspill into an extremely oedematous submucosa. Large reactive lymphoid follicles were present in the mucosa, muscularis mucosa and submucosa. There was marked lymphocytolysis at the follicle centres. Many lymphocytes were present in the adjacent submucosa at these points. In the dorsal colon there was mild cuffing of deep submucosal blood vessels by lymphocytes, plasma cells, eosinophils and macrophages. Throughout the large intestines the serosa was thick, oedematous and reactive with much fibrin and many polymorphs on the surface.

In addition in the liver there was mild periportal fibrosis and biliary hyperplasia with a slight mononuclear cell infiltrate.

No significant abnormalities were detected in the lungs, stomach, spleen, pancreas or kidneys.

Case 15 (92E)

16Y F Pony

GRANULOMATOUS TYPHLOCOLITIS

ENTERITIS

Large Intestinal Ulceration

Hepatic Haemosiderosis

At necropsy there was progressive thickening of the wall of the small intestines distally. The large intestinal wall was also thickened but here the mucosa was ulcerated. The changes appeared to be centred on the ventral colon and were less marked in the caecum and reduced in severity progressively along the dorsal colon only to reappear in the small colon and rectum.

The intestinal lymph nodes were enlarged and oedematous

Microscopic examination of the small intestines revealed oedema of the mucosa and submucosa and a mild mononuclear cell mucosal infiltrate both of which increased in severity caudally. In some sections the villi were apparently of normal length with the expected villus crypt ratio in others the villi were stunted and broad, with frond-like tips. There was a moderate increase in intraepithelial lymphocytes throughout.

Examination of the large intestines revealed a marked eosinophil and mononuclear cell infiltrate composed of lymphocytes, plasma cells and macrophages. This had spilled over into the oedematous submucosa to form a

thick band below the muscularis mucosa. There was a very marked increase in the number of intraepithelial lymphocytes. Lymphoid nodules were present in the mucosa and submucosa. Large areas of mucosal ulceration were apparent. These did not have sharply defined edges but instead the mucosa gradually increased in thickness and was covered by attentuated epithelial cells.

Examination of the lymph nodes confirmed the oedema seen grossly. It was extremly marked "blowing" the congested node apart.

In the liver there was a large amount of haemosiderin in periportal macrophages. Haemosiderin deposition was also marked within the spleen.

Case 16 (62E)

19y F Thoroughbred X

GRANULOMATOUS COLITIS

ENTERITIS

Gastric Ulceration

Haemomelasma ilei

Splenic Microabscesses

Gross examination of this animal revealed only oedema of the small intestines which progressively worsened caudally to cause thickening of the ileum and also thickening of the wall of the small colon.

Microscopic examination of the ileum revealed short, club shaped villi with a marked increase in intraepithelial lymphocyte numbers and a moderate number of eosinophils. Clumps of lymphocytes and plasma cells were present at the base of the mucosa, where the increase in intraepithelial cells was most marked, and in the muscularis mucosa and submucosa.

Examination of the colon revealed mucosal and submucosal oedema. The mucosa was thickened due to a marked mononuclear cell infiltrate. This was composed predominantly of lymphocytes and plasma cells but many macrophages were present superficially, below the surface epithelium, or at the mucosal surface where the epithelium had been lost. There was overspill of the infiltrate into the submucosa, and a marked increase in intraepithelial lymphocytes and occasional eosinophils scattered throughout. Occasional dilated crypts and small lymphoid nodules were apparent.

Case 17 (2E)

7 Y Mn Thoroughbred

TYPHLOCOLITIS

Inflammatory Hydrocoele

Oesophageal Papilloma

At necropsy the animal was thin but not emaciated. Detailed examination of the gastrointestinal tract revealed a single oesophageal papilloma of less than 2mm diameter. The proximal ileum was dilated by apparently poorly digested, biphasic content, and the fluid drained away to leave fine fibrous material. The content in the remainder of the tract appeared

normal. There was slight thickening of the mucosa of the the distal ileum but the surface was of expected appearance. A small area of focal adhesions, less than 5cm in diameter, was present on the liver capsule, the cause of which was not apparent. Evidence of parasitism was limited to one 5mm eosinophilic nodule in the right caudal lung lobe and another in the ventral colon.

In addition there was bilateral hydrocoele with haemorrhage over the tunica albuginea and congestion of the local vessels. The fluid exudate was golden yellow, soft and jelly like and most marked on the left. No abnormalities were detected in the heart, liver, pancreas and kidneys.

Microscopic examination of the small intestines revealed moderate oedema of the submucosa and a mild eosinophil infiltrate which was much more marked in the jejunum where there was also a very mild mononuclear cell mucosal infiltrate. The ileal mucosa appeared thickened with long slender villi and oedema of the lamina propria.

Examination of the large intestines again revealed oedema of the submucosa and a mild eosinophil mucosal and submucosal infiltrate. The gut associated lymphoid tissue was reactive and obvious in all sections. The most notable finding however was the presence of a layer of large, pale histiocytic cells just below the surface epithelium. On closer inspection these were seen to be macrophages with pale granular cytoplasm. The granules were periodic acid schiff positive but Ziehl-Neelsen negative.

Examination of sections of testis confirmed the presence of a proteinaceous exudate and revealed superficial heamorrhage and granulation tissue.

Case 18 (80E)

6Y F Thoroughbred

ENTERITIS

PERITONITIS

Colonic Ulceration

At necropsy significant lesions were confined to the abdomen. There was an acute peritonitis with much fibrin coating the intestinal serosa. This had been superimposed upon a chronic peritonitis with abscessation. The cause of this was not apparent. The intestinal nodes were reactive. Opening the intestines revealed oedema of the small intestinal mucosa and ulceration of the colonic mucosa.

Microscopic examination of the intestines revealed a thick, oedematous, fibrous serosa covered by a layer of granulation tissue and fibrin. Polymorphs were present on the surface with a mononuclear infiltrate in the deeper layers.

Examination of the small intestines revealed a marked mononuclear cell mucosal infiltrate composed of lymphocytes and plasma cells. This had resulted in stunting and fusion of the villi and many had florid, frond-like tips which contained occasional macrophages with much cell debris. There was marked oedema of the submucosa and patchy oedema of the mucosa

with the lymphatic vessels plugged by lymphocytes and plasma cell. Lymphoid nodules were present in the mucosa, muscularis mucosa and submucosa and there was a moderate increase in intraepthelial lymphocyte numbers.

The only microscopic abnormalities in the large intestine were oedema of the submucosa and a moderate eosinophil infiltrate.

The mesenteric lymph nodes were reactive with lymphocytolysis at the follicle centres. The medullary sinuses were packed with polymorphs and lymphocytes and plasma cells had spilled out into the perinodal fat.

Case 19 (31E)

9Y Mn Thoroughbred

LYMPHOID HYPERPLASIA

Gastric Ulceration

Haemomelasma ilei

Splenic Microabscesses

At necropsy the small intestinal content was biphasic composed of grass and liquid. Areas of flattened mucosa and hyperaemia were present in the jejunum and there was shallow gastric ulceration.

In addition raised red lesions were present on the ileal serosa which resembled Haemomelasma ilei and there were a number of small fibrous tags on the liver.

Microscopic examination of the duodenum revealed oedema of the lamina propria and submucosa and occasional eosinophils. In the jejunum and, to an even greater extent, the ileum there were multiple large reactive lymphoid follicles in the mucosa, muscularis mucosa and submucosa. At the centre of these follicles there were large, pale cells with a high mitotic rate. When the follicles were present in the mucosa and muscularis mucosa there was flattening of the mucosa with loss of villi and a great many intraepithelial lymphocytes. Lymphocytes and plasma cells spilled out into the adjacent submucosa and were joined by eosinophils. Examination of the ileum confirmed that the serosal lesions were Haemomelasma ilei and composed of vascular fibrous tissue with much haemosiderin and some fresh haemorrhage.

Examination of the large intestines revealed oedema of the mucosa and submucosa. The gut associated lymphoid tissue was reactive. Occasional small clumps of mononuclear cells were present in the mucosa and superficial submucosa and there was a mild eosinophil infiltrate. Many protozoa were apparent both on the mucosal surface and within crypts.

Microabscesses were present in the spleen with a central area of necrosis haemorrhage and inflammation surrounded by a fibrous capsule. Much haemosiderin was present both within macrophages in the fibrous tissue and free within the parenchyma.

No abnormalities were detected in the pancreas, liver or kidneys.

Case 20 (112E)

30Y Mn Welsh Mountain Pony

DENTAL EXHAUSTION

CHRONIC ENTEROPATHY

Oral Ulceration

Cyathostomes

Hepatic Haemosiderosis

Renal Abscess

Cranial Mesenteric Arterial Aneurysm

The animal was in very poor bodily condition. The abdomen contained a moderate amount of yellow-orange fluid. Erosions were present inside the lips (one upper, one lower) and cheeks. The cheek teeth were extremely uneven, they were absent, worn to gum level or up to 3cm in height with erosions on the gums. The stomach was half full and contained dry fibrous food material. This appeared poorly masticated and contained lengths of hay up to 10cm long. The small intestines were macroscopically normal. The large intestinal content was fluid with some sand in the caecum and ventral colon. Multiple dark spots representing cyathostome larvae were present in the caecal mucosa. There was an aneurysm in the cranial mesenteric artery which approximately tripled its lumenal diameter but the intima was smooth with no evidence of active *Strongylus vulgaris* activity.

In addition the liver was slightly firm with fibrous scars and tags on the capsule. The right kidney cortex contained a 1cm abscess. The dorsal

surface of both lungs was covered by a tortuous network of small blood vessels. The mycardium of the septum and ventricles contained multiple, small areas of fibrosis.

Microscopic examination of the small intestines revealed only mild oedema of the submucosa and villus lamina propria. This extended to involve the crypt area in some places.

Examination of the large intestines again revealed oedema of the mucosa and submucosa with increased numbers of intraepithelial cells in all but the small colon. The presence of cyathostome larvae was confirmed but the number involved considered insignificant. There was no demonstrable mucosal inflammatory infiltrate.

A large amount of haemosiderin was present within the splenic and hepatic parenchyma and in periportal macrophrages.

INDEX OF REFERENCES

Acland, H.M., Gunson, D.E. and Gillette, D.M. 1983. Ulcerative duodenitis in foals. *Veterinary Pathology* **20** 653-661.

Adams, S.B. 1988. Recognition and management of ileus. *Veterinary Clinics of North America: Equine Practice* **4** (1), 91-104.

Allen, D., Swayne, D. and Belknap, T.K. 1989. Ganglionneuroma as a cause of small intestinal obstruction in the horse: a case report. *Cornell Veterinarian* **79** 133-141.

Allison, N. and Gillis, J.P. 1990. Enteric pythiosis in a horse. *Journal of the American Veterinary Medical Association* **196**, (3), 462-464.

Al-Mashat, R.R. and Taylor, D.J. 1986. Bacteria in enteric lesions of horses. *Veterinary Record* **118** 453-458.

Anderson, G.A., Mount, M.E., Vrins, A.A. and Ziemer, E.L. 1983. Fatal acorn poisoning in a horse: Pathologic findings and diagnostic considerations. *Journal of the American Veterinary Medical Association* **182** (10), 1105-1110.

Argenzio, R.A. 1975. Functions of the equine large intestine and their interrelationship in disease. *Cornell Veterinarian* **65** 303-330.

Arnold, J.S., Meagher, D.M. and Lohse, C.L. 1978. Rectal tears in the horse. *Journal of Equine Medicine and Surgery* 2 55-61.

Austen, R., Saperstein, G. and Leipold, H.W. 1977. Congenital defects in foals. *Journal of Equine Medicine and Surgery* 1 146-161.

Bailey, G.D. and Hutchins, D.R. 1987. Small colon intussusception in a mare managed with a diverting colostomy. *Australian Veterinary Journal* 64 (4), 114-115.

Baker, J.R. 1973. A case of generalised tuberculosis in a horse. *Veterinary Record* 93 105-106.

Baker, J.R. 1975. Diarrhoea in horses associated with tetracycline therapy. *Veterinary Annual* 15th Issue. Editors C.S.G. Grunsell and F.W.G. Hill. 178-180.

Baker, S.J. 1991. Equine gastroduodenal ulceration: a poorly understood disease. *Equine Veterinary Education* 3 (2), 66-22.

Baker, J.R. and Ellis, C.E. 1981a. A survey of post mortem findings in 480 horses 1958 to 1980: (1) Causes of Death. *Equine Veterinary Journal* 13 (1), 43-46.

Baker, J.R. and Ellis C.E. 1981b. A survey of post mortem findings in 480 horses 1958 to 1980: (2) Disease processes not directly related to the cause of death. *Equine Veterinary Journal* 13 (1), 47-50.

Baker, J.R. and Leyland, A. 1973. Diarrhoea in the horse associated with stress and tetracycline therapy. *Veterinary Record* **93** 583-584.

Baker, S.J. 1991. Equine gastroduodenal ulceration: A poorly understood disease. *Equine Veterinary Journal* **3** (2), 60-62.

Bancroft, J.D. and Stevens, A. 1990. *Theory and Practice of Histological Techniques*. **Third Edition. Editors** J.D. Bancroft and A. Stevens. Churchill Livingstone. Edinburgh.

Barclay, W.P., Foerner, J.J. and Phillips, T.N. 1980. Volvulus of the large colon in the horse. *Journal of the American Veterinary Medical Association* **177** (7), 629-630.

Barclay, W.P., Foerner, J.J., Phillips, T.M. and MacHarg, M.A. 1982. Primary gastric impaction in the horse. *Journal of the American Veterinary Medical Association* **181** (7), 682-683.

Barclay, W.P., McCracken, R.J., Phillips, T.N. and Foerner, J.J. 1987. Chronic nongranulomatous enteritis in seven horses. *Journal of the American Veterinary Medical Association* **190** (6), 684-686.

Barclay, W.P., Phillips, T.N. and Foerner, J.J. 1982. Intussusception associated with *Anoplocephala perfoliata* infection in five horses. *Journal of the American Veterinary Medical Association* **180** (7), 752-753.

Barker, I.K. and Van Dreumel, A.A. 1985. The Alimentary system. In: *Pathology of Domestic Animals*. Vol II. Third edition. Editors K.V.F. Jubb, P.C. Kennedy and N. Palmer. Academic Press Inc. San Diego. pp178

Barlow, R.M. 1969. Neuropathological observations in grass sickness of horses. *Journal of Comparative Pathology* 79 407-411.

Barth, A.D., Barber, S.M. and McKenzie, N.T. 1980. Pyloric stenosis in a foal. *Canadian Veterinary Journal* 21 234-236.

Baxter, G.M., Broome, T.E. and Moore, J.N. 1989. Abdominal adhesions after small intestinal surgery in the horse. *Veterinary Surgery* 18 (6), 409-414.

Becht, J.L. and Byars, T.D. 1986. Gastroduodenal ulceration in foals. *Equine Veterinary Journal* 18 (4), 307-312.

Becht, J.L. and McIlwraith, C.W. 1980. Jejunal displacement through the mesometrium in a pregnant mare. *Journal of the American Veterinary Medical Association* 177 (5), 436,

Becht, J.L. and Semrad, S.D. 1986. Gastrointestinal diseases of foals. *Compendium on Continuing Education* 8 (7), S367-S374.

Bennett, S.P. and Franco, D.A. 1969. Equine protozoan diarrhoea (equine intestinal trichomoniasis) at Trinidad racetracks. *Journal of the American Veterinary Medical Association* 154 58-60.

Beroza, G.A., Barclay, W.P., Phillips, T.N., Foerner, J.J. and Donawick, W.J. 1983. Cecal perforation and infection in three horses. *Journal of the American Veterinary Medical Association* **183** (7), 804-806.

Bester, R.C. and Coetzer, J.A.W. 1978. A chronic wasting syndrome in a horse associated with granulomatous enteritis. *Journal of the South African Veterinary Association* **49** (4), 351-353.

B.E.V.A. 1965. British Equine Veterinary Association survey of equine disease, 1962-63. *Veterinary Record* **77** (19), 528-538.

Blackwell, N.J. 1973. Colitis in equines associated with strongyle larvae. *Veterinary Record* **93** 401-402.

Bohanon, T.C. 1988. Duodenal impaction in a horse. *Journal of the American Veterinary Medical Association* **192** (3), 365-366.

Boles, C.L. and Kohn, C.W. 1977. Fibrous foreign body impaction colic in young horses. *Journal of the American Veterinary Medical Association* **171** (2), 193-195.

Bolton, J.R., Merritt, A.M., Cimprich, R.E., Ramberg, C.F. and Streett, W. 1976. Normal and abnormal xylose absorption in the horse. *Cornell Veterinarian* **66** 183-197.

Boulton, C.H. and Williamson, L. 1984. Cryptococcal granuloma associated with jejunal intussusception in a horse. *Equine Veterinary Journal* **16** (6), 548-551.

Breider, M.A., Kiely, R.G. and Edwards, J.F. 1985. Chronic eosinophilic pancreatitis and ulcerative colitis in a horse. *Journal of the American Veterinary Medical Association* **186** (8), 809-811.

Brown, C.C. and Roberts E.D. 1988. Intestinal pythiosis in a horse. *Australian Veterinary Journal* **65** (3), 88-89.

Brown, C.M., Taylor, R.F. and Slanker, M.R. 1987. Sudden and unexpected death in adult horses. *Compendium Equine* **9** (1), 78-85.

Browning, G.F., Chalmers, R.M., Snodgrass, D.R., Batt, R.M., Hart, C.A., Ormarod, S.E., Leadon, D., Stoneham, S.J. and Rossdale, P.D. 1991. The prevalence of enteric pathogens in diarrhoeic Thoroughbred foals in Britain and Ireland. *Equine Veterinary Journal* **23** (6), 405-409.

Buergelt, C.D., Green, S.L., Mayhew, I.G., Wilson, J.H. and Merritt, A.M. 1988. Avian mycobacteriosis in three horses. *Cornell Veterinarian* **78** 365-380.

Burns, G.A., Karcher, L.F. and Cummings, J.F. 1990. Equine myenteric ganglionitis: A case of chronic intestinal pseudo-obstruction. *Cornell Veterinarian* **80** 53-63.

Calmette, A. 1923. *Tubercle Bacillus Infections*. Williams and Wilkins, Baltimore.

Campbell, M.L., Colahan, P.C., Brown, M.P., Granstedt, M.E. and Peyton, L.C. 1984. Cecal impaction in the horse. *Journal of the American Veterinary Medical Association* **184** (8), 950-952.

Chaffin, M.K., Fuenteablat, C.I., Schumacher, J., Welch, R.D. and Edwards, J.F. 1992. Idiopathic muscular hypertrophy of the equine small intestine: 11 cases (1980-1991). *Equine Veterinary Journal* 24 (5), 372-378.

Chiejina, S.N. and Mason, J.A. 1977. Immature stages of *Trichonema spp.* as a cause of diarrhoea in adults horses in spring. *Veterinary Record* **100** 360-361.

Cho D.-Y. and Taylor, H.W. 1986. Blind-end atresia coli in two foals. *Cornell Veterinarian* **76** 11-15.

Church, S., Baker, J.R. and May, S.A. 1986. Gastric retention associated with aquired pyloric stenosis in a gelding. *Equine Veterinary Journal* **18** (4), 332-334.

Cimprich, R.E. 1974. Equine granulomatous enteritis. *Veterinary Pathology* **11** 535-547.

Cimprich, R.E. 1981. Granulomatous enteritis of horses: Clinical and postmortem findings. *The Compendium on Continuing Education* 3 (11), S437-S440.

Clark, E.S., Morris, D.D., Allen, D. and Tyler, D.E. 1988. Lymphocytic enteritis in a filly. *Journal of the American Veterinary Medical Association* 193 (10), 1281-1283.

Clayton, H.M. 1978. Ascariasis in foals. *Veterinary Record* 102 553-556.

Clayton, H.M., Duncan, J.L. and Dargie, J.D. 1980. Pathophysiological changes associated with *Parascaris equorum* infection in the foal. *Equine Veterinary Journal* 12 (1), 23-25.

Clem, M.F., DeBowes, R.M. and Leipold, H.W. 1987. Rectal leiomyosarcoma in a horse. *Journal of the American Veterinary Medical Association* 191 (2), 229-230.

Cline, J.M., Schlafer, D.W., Callihan, D.R., Vanderwall, D. and Drazek, F.J. 1991. Abortion and granulomatous colitis due to *Mycobacterium avium complex* infection in a horse. *Veterinary Pathology* 28 89-91.

Coleman, G.T. and Sutton, R.H. 1993. Grey horse melanoma - Is it a true neoplasm? *New Zealand Veterinary Journal* 41 (1), 43-44.

Collier, M.A. and Trent, A.M. 1983. Jejunal intussusception associated with leiomyoma in an aged horse. *Journal of the American Veterinary Medical Association* **182** (8), 819-821.

Collins, L.G. and Tyler, D.E. 1984. Phenylbutazone toxicosis in the horse: A clinical study. *Journal of the American Veterinary Medical Association* **184** (6), 699-706.

Collins, L.G. and Tyler, D.E. 1985. Experimentally induced phenylbutazone toxicosis in ponies: Description of the Syndrome and its prevention with synthetic prostaglandin E_2. *American Journal of Veterinary Research* **46** (8), 1605-1615.

Conboy, H.S. and Powers, R.D. 1971. Equine Malignant lymphoma. *Journal of the American Veterinary Medical Association* **159** (1), 53-54.

Conner, M.E. and Darlington, R.W. 1980. Rotavirus infection in foals. *American Journal of Veterinary Research* **41** (10), 1699-1703.

Cook, W.R. 1973. Diarrhoea in the horse associated with stress and tetracycline therapy. *Veterinary Record* **93** 15-16.

Cordes, D.O., Perry, B.D. Rikihisa, Y. and Chickering, W.R. 1986. Enterocolitis caused by *Ehrlichia* sp. in the horse (Potomac Horse Fever). *Veterinary Pathology* 23 471-477.

Cosgrove, J.S., Sheeran, J.J. and Sainty, T.J. 1986. Intussusception associated with infection with *Anoplocephala perfoliata* in a two year old Thoroughbred. *Irish Veterinary Journal* **40** 35-36.

Cotchin, E. 1960. Tumours of farm animals: A review of tumours examined at the Royal Veterinary College, London during 1950-1960. *Veterinary Record* **72** 816-822.

Cotchin, E. 1977. A general survey of tumours in the horse. *Equine Veterinary Journal* **9** (1), 16-21.

Cotchin, E. and Baker-Smith, J. 1975. Tumours in horses encountered in an abattoir survey. *Veterinary Record* **97** 339.

Cotran, R.S., Kumar, V. and Robbins, S.L. 1989. Diseases of Immunity In: *Robbins Pathologic Basis of Disease* 4th Edn., Eds. R.S. Cotran, V. Kumar and S.L. Robbins. W.B. Saunders Co. Philadelphia. pp 176-177.

Crawley, G.R. 1985. Lymphosarcoma resulting in diarrhoea, weight loss, and gastrointestinal ulcerations. *Veterinary Medicine*, 66-69.

Crowe, M.V. and Swerczek, T.W. 1985. Equine congential defects. *American Journal of Veterinary Research* **46** 353-358.

Cudd, T.A. and Pauly, T.H. 1987. Necrotizing enterocolitis in two equine neonates. *Compendium Equine* **9** (1), 88-92, 96.

Dade, A.W., Lickfeldt, W.E. and McAllister, H.A. 1973. Granulomatous colitis in a horse with histoplasmosis. *Veterinary Medicine/Small Animal Clinician*, 279-281.

Dart, A.J., Snyder, J.R. and Harmon, F.A. 1992a. Microvascular circulation of the descending colon in horses. *American Journal of Veterinary Research* 53 (6), 1001-1006.

Dart, A.J., Snyder, J.R., Julian, D. and Hinds, D.M. 1991. Microvascular circulation of the cecum in horses. *American Journal of Veterinary Research* 52 (9), 1545-1550.

Dart, A.J., Snyder, J.R., Pascoe, J.R., Farver, T.B. and Galuppo, L.D. 1992b. Abnormal conditions of the equine descending (small) colon: 102 cases (1979-1989). *Journal of the American Veterinary Medical Association* 200 (7), 971-978.

Davies, J.V. 1983. Ischaemic necrosis of the jejunum of a horse caused by a penetrating foreign body. *Equine Veterinary Journal* 15 (1), 66-68.

Davies, J.V. and Gerring, E.L. 1985. Effect of experimental vascular occlusion on small intestinal motility in ponies. *Equine Veterinary Journal* 17 (3), 219-224.

Davis, L.E. 1987. Adverse drug reactions in the horse. *Veterinary Clinics of North America: Equine Practice. Clinical Pharmacology* 3 (1), 153-180.

De Boom, H.P.A. 1975. Functional anatomy and nervous control of the equine alimentary tract. *Journal of the South African Veterinary Association* 46 (1), 5-11.

Di Pietro, J.A., Boero, M. and Ely, R.W. 1983. Abdominal abscess associated with *Parascaris equorum* infection in a foal. *Journal of the American Veterinary Medical Association* 182 (9), 991-992.

Dobson, H. and Lopez, A. 1981. Intestinal obstruction and gastric rupture involving a penetrating foreign body. *Equine Veterinary Journal* 13 (3), 204-205.

Ducharme, N.G. and Fubini, S.L. 1983. Gastrointestinal complications associated with the use of atropine in horses. *Journal of the American Veterinary Medical Association* 182 (3), 229-231.

Duncan, J.L. 1985a. Internal parasites of the horse and their control. *Equine Veterinary Journal* 17 (2), 79-82.

Duncan, J.L. 1985b. The hidden handicappers. *Equine Veterinary Journal* 17 (3), 164-165.

Duncan, J.L. and Pirie, H.M. 1975. The pathogenesis of single experimental infections with *Strongylus vulgaris in foals*. *Research in Veterinary Science* **18** 82-93.

Dyson, S. 1983. Review of 30 cases of peritonitis in the horse. *Equine Veterinary Journal* **15** (1), 25-30.

Dyson, S. and Orsini, J. 1983. Intussusception of the large colon in a horse. *Journal of the Veterinary Medical Association* **182** (7), 720.

Edens, L.M., Taylor, D.D., Murray, M.J., Spurlock, G.H. and Anver, M.R. 1992. Intestinal myxosarcoma in a Thoroughbred mare. *Cornell Veterinarian* **82** 163-167.

Edwards, G.B. 1981. Obstruction of the ileum in the horse: A report of 27 clinical cases. *Equine Veterinary Journal* 13 (3), 158-166.

Embertson, R.M., Colahan, P.T., Brown, M.P., Peyton, L.C., Schneider, R.K. and Granstedt, M.E. 1985. Ileal impaction in the horse. *Journal of the American Veterinary Medical Association* **186** (6), 570-572.

English, P.B. and Roberts M.C. 1983. Adverse reactions to antimicrobial agents in the horse. *American Research Communications* 7 207-210.

Estes, R. and Lyall, W. 1979. Congential atresia of the colon: A review and report of four cases in the horse. *Journal of Equine Medicine and Surgery* 3 495-498.

Feldman, W.H. 1932. *Neoplasms of Domestic Animals*. W.B. Saunders, Philadelphia. pp96-103.

Foerner, J.J. 1982. Diseases of the large intestine. Differential diagnosis and surgical management. *Veterinary Clinics of North America: Large Animal Practice* 4 (1), 138-146.

Ford, T.S., Freeman, D.E., Ross, M.W., Richardson, D.W., Martin, B.B. and Madison, J.B. 1990. Ileocecal intussusception in horses: 26 cases (1981-1988). *Journal of the American Veterinary Medical Association* 196 (1), 121-125

Freeman, D.E., Cimprich, R.E., Richardson, D.W., Gentile, D.G., Orsini, J.A., Tulleners, E.P. and Fetrow, J.P. 1988. Early mucosal healing and chronic changes in pony jejunum after various types of strangulation obstruction. *American Journal of Veterinary Research* 49 (6), 810-818.

Freeman, D.E., Koch, D.B. and Boles, C.L. 1979. Mesodiverticular bands as a cause of small intestinal strangulation and volvulus in the horse. *Journal of the American Veterinary Medical Association* 175 (10), 1089-1094.

French, D.D. and Chapman, M.R. 1992. Tapeworms of the equine gastrointestinal tract. *The Compendium* 14 (5), 655-661.

Furie, W.S. 1983. Persistent cloaca and atresia ani in a foal. *Equine Practice* 5 (1), 30-33.

Gajadhar, A.A., Caron, J.P. and Allen, R.J. 1985. Cryptosporidiosis in two foals. *Canadian Veterinary Journal* 26 132-134.

Gay, C.C. and Lording, P.M. 1980. Peritonitis in horses associated with *Actinobacillus equuli*. *Australian Veterinary Journal* 56 296-300.

Getty, R. 1975. *Sisson and Grossman's The Anatomy of the domestic animals*. Volume I. Fifth Edition. Ed. R. Grossman. W.B. Saunders Co. Philadelphia. pp 454-497.

Gibbons, D.F. 1980. Equine Salmonellosis: A review. *Veterinary Record* 106 356-359.

Gibson, K.T. and Alders, R.C. 1987. Eosinophilic enterocolitis and dermatitis in two horses. *Equine Veterinary Journal* 19 (3), 247-252.

Gibson, J.A., Hill, M.W.M. and Huber, M.J. 1983. Cryptosporidiosis in Arabian foals with severe combined immunodeficiency. *Australian Veterinary Jornal* 60 (12), 378-379.

Giles, C.J., Urquhart, K.A. and Longstaffe, J.A. 1985. Larval cyathostomiasis (immature trichonema-induced enteropathy): A report of 15 clinical cases. *Equine Veterinary Journal* 17 (3), 196-201.

Goetz, T.E. and Coffman, J.R. 1984. Ulcerative colitis and protein losing enteropathy associated with intestinal salmonellosis and histoplasmosis in a horse. *Equine Veterinary Journal* **16** (5), 439-441.

Grant, B.D. and Tennant, B. 1973. Volvulus associated with Meckel's diverticulum in the horse. *Journal of the American Veterinary Medical Association* **162** (7), 550-551.

Greatorex, J.C. 1975. Diarrhoea in horses associated with ulceration of the colon and caecum resulting from *S vulgaris* larval migration. *Veterinary Record* **97** 221-225.

Green, P. and Tong, J.M.J. 1988. Small intestinal obstruction associated with wood chewing in two horses. *Veterinary Record* **123** 196-198.

Greet, T.R.C. 1992. Ileal intussusception in 16 young Thoroughbreds. *Equine Veterinary Journal* 24 (2), 81-83.

Gregory, M.W., Longstaff, J.A. and Giles, C.J. 1986. Tissue-invading ciliates associated with chronic colitis in a horse. *Journal of Comparative Pathology* **96** 109-114.

Griffiths, A.S. 1936. Naturally acquired tuberculosis in various animals. Some unusual cases. *Journal of Hygiene (Cambridge)* **36** 156-168.

Hackett, R.P. 1983. Nonstrangulated colonic displacement in horses. *Journal of the American Veterinary Medical Association* **182** (3), 235-240.

Hackett, N.S. and Hackett, R.P. 1989. Chronic ileiocecal intussusception in horses. *Cornell Veterinarian* **79** 353-361.

Hanes, G.E. and Robertson, J.T. 1983. Leiomyoma of the small intestine in a horse. *Journal of the American Veterinary Medical Association* **182** (12), 1398.

Harbour, D.A. 1985. Infectious diarrhoea in foals. *Equine Veterinary Journal* **17** (4), 262-264.

Harmon, B.G., Ruoff, W.W. and Huey, R. 1986. Cyathostome colitis and typhlitis in a filly. *Compendium of Continuing Education for the Practicing Veterinarian* **8** (6), S301-S306.

Harrison, I.W. 1988. Equine large intestinal volvulus. A review of 124 cases. *Veterinary Surgery* **17** (2), 77-81.

Harrison, I.W. 1989. Cecal torsion in a horse as a consequence of cecocolic fold hypoplasia. *Cornell Veterinarian* **79** 315-317.

Hattel, A.L., Drake, T.R., Anderholm, B.J. and McAllister, E.S. 1991. Pulmonary aspergillosis associated with acute enteritis in a horse. *Journal of the American Veterinary Medical Association* **199** (5), 589-590.

Hayes, M.H. 1889. *Veterinary Notes for Horse-owners*. Fourth edition pp 315-316.

Head, K.W. 1976. Tumours of the lower alimentary tract. *Bulletin of the World Health Organisation* **53** 167-186.

Hibbs, C.M., Johnson, D.R., Reynolds, K. and Harrington, R. 1977. *Clostridium sordellii* isolated from foals. *Veterinary Medicine and Small Animal Clinician* 256-258.

Hilbert, B.J., Little, C.B., Bolton, J.R. and McGill, C.A. 1987. Caecal overload and rupture in the horse. *Australian Veterinary Journal* **64** (3), 85-86.

Hillyer, M.H. and Mair, T.S. 1992. Multisystemic eosinophilic epitheliotropic disease in a horse: attempted treatment with hydroxyurea and dexamethasone. *Veterinary Record* **130** 392-395.

Hodgson, D.R. and Allen, J.R. 1982. Granulomatous enteritis in a Thoroughbred horse. *New Zealand Veterinary Journal* **30** 180-182.

Honnas, C.M., Snyder, J.R., Olander, H.J. and Wheat, J.D. 1987. Small intestinal adenocarcinoma in a horse. *Journal of the American Veterinary Medical Association* **191** (7), 845-846.

Hooper, R.N., 1989. Small intestinal strangulation caused by Meckel's diverticulum in a horse. *Journal of the American Veterinary Medical Association* **194** (7), 943-944.

Howard-Martin, M., Morton, R.J., Qualls, C.W. and MacAllister, C.G. 1986. Clostridium perfringens type C enterotoxemia in a newborn foal. *Journal of the American Veterinary Medical Association* **189** (5), 564-565.

Hultgren, B.D. 1982. Ileocolonic aganglionosis in white progeny of overo spotted horses. *Journal of the American Veterinary Medical Association* **180** (3), 289-292.

Humphrey, M., Watson, D.A., Edwards, H.G. and Wood, C.M. 1984. Lymphosarcoma in a horse. *Equine Veterinary Journal* **16** (6), 547-548.

Huskamp, B. and Kopf, N. 1983. Right dorsal displacement of the large colon in the horse. *Equine Practice* **5** (1), 20-29.

Imrie, H. and Jacobs, D.E. 1987. Prevalence of horse tapeworm in north London and Hertfordshire. *Veterinary Record* **120** 304.

Innes, J.R.M. 1949. Tuberculosis in the horse. *The British Veterinary Journal* **105** 373-383.

Jasko, D.J. and Roth, L. 1984. Granulomatous colitis associated with small strongyle larvae in a horse. *Journal of the American Veterinary Medical Association* **185** (5), 553-554.

Johnson, J.A., Prescott, J.F. and Markham, R.J.F. 1983. The pathology of experimental *Corynebacterium equi* infection in foals following intragastric challenge. *Veterinary Pathology* **20** 450-459.

Johnston, J.K. and Morris, D.D. 1987. Comparison of duodenitis/proximal jejunitis and small intestinal obstruction in horses: 68 cases (1977-1985). *Journal of the American Veterinary Medical Association* **191** (7), 849-854.

Jones, R.L., Adney, W.S., Alexander, A.F., Shideler, R.K. and Traub-Dargatz, J.L. 1988. Hemorrhagic necrotising enterocolitis associated with *Clostridium difficile* infection in four foals. *Journal of the American Medical Association* **193** (1), 76-79.

Jones, T.C. and Hunt, R.D. 1983. The muscoloskeletal system. In: *Veterinary Pathology*. Fifth Edition. Lea and Febgiger. Philadelphia. pp 1174.

Jubb, K.V.F., Kennedy, P.C. and Palmer, N. 1991. The pancreas. In: *Pathology of Domestic Animals*. Vol II. Fourth Edition. Eds. K.V.F. Jubb, P.C. Kennedy and N. Palmer. Academic Press Inc. San Diego. pp 319.

Karcher, L.F., Dill, S.G., Anderson, W.I. and King, J.J. 1990. Right dorsal colitis. *Journal of Veterinary Internal Medicine* 4 247-253.

Kiper, M.L., MacAllister, C. and Qualls, C. 1988. Hematochezia attributable to cranial mesenteric arterial aneurysm with connecting tracts to cecum and ileum in a horse. *Journal of the American Veterinary Medical Association* 193 (10), 1278-1280.

Kirkpatrick, C.E. and Shand, D.L. 1985. Giardiasis in a horse. *Journal of the American Veterinary Medical Association* **187** (2), 163-164.

Kirsner, J.B. 1976. In *Gastroenterology*. Vol II. Third Edition. Editor H.L. Bockus. W.B. Saunders and Co., Philadelphia p521.

Kobluk, C.N. and Smith, D.F. 1988. Intramural hematoma in the jejunum of a mare. *Journal of the American Veterinary Medical Association* **192** (3), 379-380.

Larsen, A.B., Moon, H.W. and Merkal, R.S. 1972. Susceptibility of horses to *Mycobacterium paratuberculosis*. *American Journal of Veterinary Research* **33** (11), 2185-2189.

Laufenstein-Duffy, H. 1969. Equine intestinal trichomoniasis. *Journal of the American Veterinary Medical Association* 155 (12), 1835-1840.

Lilley, C.W. and Beeman, G.M. 1981. Gastric dilatation associated with acute necrotising pancreatitis. *Equine Practice* 3 (6), 8-15.

Lindberg, R. 1984. Pathology of equine granulomatous enteritis. *Journal of Comparative Pathology* 94 233-247.

Lindberg, R. and Karlsson, L. 1985. Topography and enterocyte morphology of the small bowel mucosal surface in equine granulomatous enteritis. *Journal of Comparative Pathology* 94 65-78.

Lindberg, R. and Persson, S. 1979. Eosinofil granulomatos enterit hos hast. *Allmant Veterinarmote*. Kompendium. Sveriges Veterinarforbund, Stockholm, pp 147-150.

Lindberg, R., Persson, S.G.B., Jones, B., Thoren-Tolling, K. and Ederoth, M. 1985. Clinical and pathophysiological features of granulomatous enteritis and eosinophilic granulomatosis in the horse. *Zentralblatt Fur Veterinarmedizin. Reihe A. (Berlin)* 33, 526-539.

Lindsay, W.A., Confer, A.W. and Ochoa, R. 1981. Ileal smooth muscle hypertrophy and rupture in a horse. *Equine Veterinary Journal* 13 (1), 66-67.

Livesay, M.A., Hulland, T.J. and Yorich, J.V. 1986. Colic in two horses associated with smooth muscle intestinal tumours. *Equine Veterinary Journal* **18** (4), 334-337.

Livesay, M.A. and Keller, S.D. 1986. Segmental ischemic necrosis following mesocolic rupture in postparturient mares. *Compendium Equine* **8** (10), 763-768.

Lloyd, K., Hintz, H.F., Wheat, J.D. and Schryver, H.F. 1987. Enteroliths in horses. *Cornell Veterinarian* **77** 172-186.

Loeb, W.F., McKenzie, L.D. and Hoffsis, G.F. 1972. The carbohydrate digestion - absorption test in the horse. Technic and normal values. *Cornell Veterinarian* **62** 524-531.

Love, S. 1992. Parasite-associated equine diarrhoea. *The Compendium* **14** (5), 642-649.

Love, S., Mair, T.S. and Hillyer, M.H. 1992. Chronic diarrhoea in adult horses: A review of 51 referred cases. *Veterinary Record* **130** 217-219.

Luke, D. 1958. Tuberculosis in the horse, pig, sheep and goat. *Veterinary Record* **70** (26), 529-236.

Lyons, E.T., Drudge, J.H. and Tolliver, S.C. 1988. Natural infection with *Eimeria leukarti*: Prevalence of oocysts in feces of horse foals on several

farms in Kentucky during 1986. *American Journal of Veterinary Research* 49 (1), 96-98.

Lyons, E.T., Drudge, J.H., Tolliver, S.C., Swerczek, T.W. and Crowe, M.W. 1984. Prevalence of *Anoplocephala perfoliata* and lesions of *Drashia megastoma* in Thoroughbreds in Kentucky at necropsy. *American Journal of Veterinary Research* 45 (5), 996-999.

Lyons, E.T., Tolliver, S.C., Drudge, J.H., Swerczek, T.W. and Crowe, M.W. 1983. Parasites in Kentucky Thoroughbreds at necropsy: Emphasis on stomach worms and tapeworms. *American Journal of Veterinary Research* 44 (5), 839-844.

Lyons, E.T., Tolliver, S.C., Drudge, J.H., Swerczek, T.W. and Crowe, M.W. 1987. Common internal parasites found in the stomach, large intestine, and cranial mesenteric artery of Thoroughbreds in Kentucky at necropsy (1985-1986). *American Journal of Veterinary Research* 48 (2), 268-273.

MacAllister, C.G., Mosier, D., Qualls, C. and Cowell, R.L. 1990. Lymphocytic-plasmacytic enteritis in two horses. *Journal of the American Veterinary Medical Association* 196 (12), 1995-1998.

MacKay, R.J., French, T.W., Nguyen, H.T. and Mayhew, I.G. 1983. Effects of large doses of phenylbutazone administration. *American Journal of Veterinary Research* 44 (5), 774-780.

Mair, T.S., Davies, E.V. and Lucke, V.M. 1992. Small colon intussusception associated with an intralumenal leiomyoma in a pony. *Veterinary Record* **130** 403-404.

Mair, T.S., Hillyer, M.H., Taylor, F.G.R. and Pearson, G.R. 1991. Small intestinal malabsorption in the horse: an assessment of the specificity of the oral glucose tolerance test. *Equine Veterinary Journal* **23** (5), 344-346.

Mair, T.S. and Lucke, V.M. 1992. Chyloperitoneum associated with torsion of the large colon in a horse. *Veterinary Record* **131** 421.

Mair, T.S., Taylor, F.G.R. and Brown, P.J. 1990a. Leiomyosarcoma of the duodenum in two horses. *Journal of Comparative Pathology* **102** 119-123.

Mair, T.S., Taylor, F.G.R., Harbour, D.A. and Pearson, G.R. 1990b. Concurrent cryptosporidium and coromavirus infections in an Arabian foal with combined immunodeficiency syndrome. *Veterinary Record* **126** 127-130.

Marsh, M.N. 1990. Grains of truth: evolutionary changes in small intestinal mucosa in response to environmental antigen challenge. *Gut* **31** 111-114.

Mason, R.W. and King, S.J. 1971. *Eimeria leuckarti* in the horse. *Australian Veterinary Journal* 47 460.

Mason, T.A. 1978. Strangulation of the rectum of a horse by the pedicle of a mesenteric lipoma. *Equine Veterinary Journal* 10 (4), 269.

Mayberry, J.F., Rhodes, J. and Heatley, R.V. 1980. Infections which cause ileocolic disease in animals: Are they relevant to Crohn's disease. *Gastroenterology* **78** 1080-1084.

McCabe, L., Griffin, L.D., Kinzer, A., Chandler, M., Beckwith, J.B. and McCabe, R.B. 1990. Overo lethal white foal sydrome: Equine model of aganglionic megacolon (Hirschsprung disease). *American Journal of Medical Genetics* **36** 336-340.

McChesney, A.E., England, J.J. and Rich, L.J. 1973. Adenoviral infection of foals. *Journal of the American Veterinary Medical Association* **162** (7), 545-549.

McConnell, S., Katada, M., Fiske, R.A. and Martens, J.G. 1982. Equine lymphosarcoma diagnosed as equine infectious anaemia in a young horse. *Equine Veterinary Journal* 14 (2), 160-162.

McQueary, C.A., Worley, D.E. and Catlin, J.E. 1977. Observations on the life cycle and prevalence of *Eimeria leukarti* in horses in Montana. *American Journal of Veterinary Research* **38** 1673-1674.

Merritt, A.M., Cimprich, R.E. and Beech, J. 1976. Granulomatous enteritis in nine horses. *Journal of the American Veterinary Medical Association* **169** (6), 603-609.

Meschter, C.L., Craig, D. and Hackett, R. 1991. Histopathological and ultrastructural changes in simulated large colonic torsion and reperfusion in ponies. *Equine Veterinary Journal* **23** (6), 426-433.

Meschter, C.L., Gilbert, M., Krook, L., Maylin, G. and Corradino, R. 1990. The effects of phenylbutazone on the intestinal mucosa of the horse: a morphological, ultrastructural and biochemical study. *Equine Veterinary Journal* **22** (4), 255-263.

Meschter, C.L., Maylin, G.A. and Krook, L. 1984. Vascular pathology in phenylbutazone intoxicated horses. *Cornell Veterinarian* **74** 282-297.

Meschter, C.L., Tyler, D.E., White, N.A. and Moore, J. 1986. Histologic findings in the gastrointestinal tract of horses with colic. *American Journal of Veterinary Research* **47** (3), 598-606.

Meuten, D.J., Butler, D.G., Thomson, G.W. and Lumsden, J.H. 1978. Chronic enteritis associated with malabsorption and protein-losing enteropathy in the horse. *Journal of the American Veterinary Medical Association* **172** (3), 326-333.

Mfitilodze, M.W. and Hutchinson, G.W. 1989. Prevalence and intensity of non-strongyle intestinal parasites of horses in Northern Queensland. *Australian Veterinary Journal* **66** (1), 23-26.

Milne, D.W., Tarr, M.J., Lochner, F.K., McAllister, E.S., Muir, W.W. and Skarda, R.T. 1977. Left dorsal displacement of the colon in the horse. *Journal of Equine Medicine and Surgery* (1), 47-52.

Milne, E.M., Pogson, D.M. and Doxey, D.L. 1990. Secondary gastric impaction associated with ragwort poisoning in three ponies. *Veterinary Record* **126** 502-504.

Milne, E.M., Pogson, D.M., Else, R.W. and Rowland, A.C. 1989. Caecal intussusception in two ponies. *Veterinary Record* **125** 148-150.

Misdorp, W. 1967. Tumours in large domestic animals in the Netherlands. *Journal of Comparative Pathology* **77** 211-216

Morris, D.D., Vaala, W.E. and Sartin, E. 1982. Protein-losing enteropathy in a yearling filly with subclinical disseminated intravascular coagulation and autoimmune haemolytic disease. *The Compendium on Continuing Education* 4 (12), S542-S546.

Mulville, P. 1991. Equine monocytic ehrlichiosis (Potomac horse fever): a review. *Equine Veterinary Journal* **23** (6), 400-404.

Murray, M.J., Parker, G.A. and White, N.A. 1988. Megacolon with myenteric hypoganglionosis in a foal. *Journal of the American Veterinary Medical Association* 7 (1), 917-919.

Myers, L.L., Shoop, D.S. and Byars, T.D. 1987. Diarrhoea associated with enterotoxigenic *Bacteriodes fragilis* in foals. *American Journal of Veterinary Research* 48 (11), 1565-1567.

Neufield, J.L. 1973a. Lymphosarcoma in the horse: A review. *Canadian Veterinary Journal* 14 (6), 129-135.

Neufield, J.L. 1973b. Lymphosarcoma in a mare and review of cases at the Ontario Veterinary College. *Canadian Veterinary Journal* 14 (7), 149-153.

Nickel, R., Schummer, A. and Seiferle, E. 1979. The alimentary canal of the horse. In: *The Viscera of Domestic Mammals*. Second revised edition. Editors A. Schummer, R. Nickel and W.O. Sack. Vertag Paul Parey, Berlin, pp 180-197.

Niilo, L. and Chalmers, G.A. 1982. Hemorrhagic enterotoxemia caused by *Clostridium perfringens* type C in a foal. *Canadian Veterinary Journal* 23 299-301.

Obel, A-L. 1955. Studies on grass disease. The morphological picture with special reference to the vegetative nervous system. *Journal of Comparative Pathology* 65 334-346.

Ochoa, R. and Kern, S.R. 1980. The effects of *Clostridium perfringens* type A enterotoxin in shetland ponies - Clinical, morphologic and clinicopathologic changes. *Veterinary Pathology* **17** 738-747.

Oliver, D.F., Jenkins, C.T. and Walding, P. 1977. Duodenum rupture in a nine-month-old colt due to *Anoplocephala magna*. *Veterinary Record* **101** 80.

Olsen, S.N. 1992. Squamous cell carcinoma of the equine stomach: a report of five cases. *Veterinary Record* **131** 170-173.

Orsini, J.A., Orsini, P.G., Sepesy, L., Acland, H. and Gillette, D. 1988. Intestinal carcinoid in a mare: An etiologic consideration for chronic colic in horses. *Journal of the American Veterinary Medical Association* **193** (1), 87-88.

Owen, Rh.apRh., Jagger, D.W. and Quan-Taylor, R. 1988. Prevalence of *Anoplocephala perfoliata* in horses and ponies in Clwyd, Powys and adjacent English marches. *Veterinary Record* **123** 562-563.

Owen, Rh.apRh., Jagger, D.W. and Quan-Taylor, R. 1989. Caecal intussusceptions in horses and the significance of *Anoplocephala perfoliata*. *Veterinary Record* **124** 34-37.

Parker, J.E., Fubini, S.L. and Todhunter, R.J. 1989. Retrospective evaluation of repeat celiotomy in 53 horses with acute gastrointestinal disease. *Veterinary Surgery* 18 (6), 424-431.

Parks, A.H., Doran, R.E., White, N.A., Allen, D. and Baxter, G.M. 1989. Ileal impaction in the horse: 75 cases. *Cornell Veterinarian* 79 83-91.

Parks, A.H., Wyn-Jones, G., Cox, J.E. and Newsholme, J. 1986. Partial obstruction of the small colon associated with an abdominal testicular teratoma in a foal. *Equine Veterinary Journal* 18 (4), 342-343.

Pascoe, P.J. 1982. Colic in a mare caused by a colonic neurofibroma. *Canadian Veterinary Journal* 23 24-27.

Pascoe, R.R. and Summers, P.M. 1981. Clinical survey of tumours and tumour-like lesions in horses in south east Queensland. *Equine Veterinary Journal* 13 (4), 235-239.

Pasquini, C.J., Reddy, V.K. and Ratzlaff, M. 1978. *Atlas of Equine Anatomy*. Sudz Publishing, Albion, pp141-153.

Pass, D.A. and Bolton, J.R. 1982. Chronic eosinophilic gastroenteritis in the horse. *Veterinary Pathology* 19 486-496.

Pass, D.A., Bolton, J.R. and Mills, J.N. 1984. Basophilic enterocolitis in a horse. *Veterinary Pathology* 21 362-364.

Pearson, E.G., Hedstrom, O.R., Sonn, R. and Wedam, J. 1986. Hemorrhagic enteritis caused by *Clostridium perfringens* type C in a foal. *Journal of the American Veterinary Medical Association* **188** (11), 1309-1310.

Pearson, H. and Waterman, A.E. 1986. Submucosal haematoma as a cause of obstruction of the small colon in the horse: A review of four cases. *Equine Veterinary Journal* **18** (4), 340-341.

Platt, H. 1982. Sudden and unexpected deaths in horses: A review of 69 cases. *British Veterinary Journal* **138** 417-429.

Platt, H. 1983. Caecal rupture in parturient mares. *Journal of Comparative Pathology* **93** 343-346.

Platt, H. 1986. Chronic inflammatory and lymphoproliferative lesions of the equine small intestine. *Journal of Comparative Pathology* **96** 671-684.

Platt, H. 1987. Alimentary lymphomas in the horse. *Journal of Comparative Pathology* **97** 1-10.

Prescott, J.F., Staempfli, H.R. Barker, I.K., Bettoni, R. and Delany, K. 1988. A method for reproducing fatal idiopathic colitis (colitis X) in ponies and isolation of a clostridium as a possible agent. *Equine Veterinary Journal* **20** (6), 417-420.

Priester, W.A. 1973. Skin tumours in domestic animals. Data from 12 United States and Canadian Colleges of Veterinary Medicine. *Journal of the National Cancer Institute* 50 457-466.

Priester, W.A., Mantel, N. 1971. Occurrence of tumours in domestic animals. Data from 12 United States and Canadian Colleges of Veterinary Medicine. *Journal of the National Cancer Institute* 47 1333-1344.

Proudman, C.J. 1991. A two year, prospective survey of equine colic in general practice. *Equine Veterinary Journal* 24 (2), 90-93.

Proudman, C.J. and Edwards, G.B. 1992. Diaphragmatic diverticulum (hernia) in a horse. *Equine Veterinary Journal* 24 (3), 244-246.

Ragle, C.A., Meagher, D.M., Lacroix, C.A. and Honnas, C.M. 1989. Surgical treatment of sand colic. Results in 40 horses. *Veterinary Surgery* 18 (1), 48-51.

Raisbeck, M.F., Holt, G.R. and Osweiler, G.D. 1981. Lincomycin-associated colitis in horses. *Journal of the American Veterinary Medical Association* 179 (4), 362-363.

Ramey, D.W. and Reinerston, E.L. 1984. Strangulating lipoma and small intestinal leiomyoma in a horse: A case report. *Equine Veterinary Science* 4 (2), 88-89.

Rebhun, W.C. and Bertone, A. 1984. Equine lymphosarcoma. *Journal of the American Veterinary Medical Association* **184** (6), 720-721.

Rebhun, W.C., Dill, S.G. and Power, H.T. 1982. Gastric ulcers in foals. *Journal of the American Veterinary Medical Association* **180** (4), 404-407.

Reef, V.B., Dyson, S.S. and Beech, J. 1984. Lymphosarcoma and associated immune-mediated hemolytic anaemia and thrombocytopenia in horses. *Journal of the American Veterinary Medical Association* **184** (3), 313-317.

Reid, S.W.J. and Howie, F. 1992. Factors associated with neoplastic disease in the horse. *Equine Veterinary Education* 4 (2), 66-68.

Rider, J.R. 1932. Diverticula of the small intestine. *Veterinary Record* **12** 1202-1206.

Roberts, M.C. 1983. Protein-losing enteropathy in the horse. *The Compendium on Continuing Education* **5** (10), S550-S556.

Roberts, M.C. 1985. Malabsorption syndromes in the horse. *The Compendium on Continuing Education* **7** (11), S637-S647.

Roberts, M.C. 1990. Acute equine colitis: experimental and clinical perspectives. *The Veterinary Annual*. 30th Issue. Editors C.S.G. Grunsell and M.E. Raw. Wright, London Pp 1-11.

Roberts, M.C. and Kelly, W.R. 1980. Granulomatous enteritis in a young standardbred mare. *Australian Veterinary Journal* 56 230-233.

Roberts, M.C. and Pinsent, P.J.N. 1975. Malabsorption in the horse associated with alimentary lymphosarcoma. *Equine Veterinary Journal* 7 (3), 166-172.

Robertson, J.T. 1982. Conditions of the stomach and small intestine. Differential diagnosis and management. *Veterinary Clinics of North America: Large Animal Practice* 4 (1), 105-127.

Robertson, J.T. and Tate, L.P. 1982. Resection of intussuscepted large colon in a horse. *Journal of the American Veterinary Medical Association* 181 (9), 927-928.

Rollins, J.B. and Clement, T. 1979. Observations on Incidence of Equine Colic in a Private Practice. *Equine Practice* 1 (5), 39-42.

Rooney, J.R. 1965. Volvulus, strangulation and intussusception in the horse. *Cornell Veterinarian* 55 644-653.

Ross, M.W., Martin, B.B. and Donawick, W.J. 1985. Cecal performation in the horse. *Journal of the American Veterinary Medical Association* 187 (3), 249-253.

Ross, M.W., Stephens, P.R. and Reimer, J.M. 1988. Small colon intussusception in a broodmare. *Journal of the Americal Veterinary Medical Association* **192** (3), 372-374.

Rous, R.C. 1975. Mesenteric thrombosis. *Journal of the South African Veterinary Association* **46** (1), 79-80.

Rubin, A. and Isaacson, P.G. 1990. Florid reactive lymphoid hyperplasia of the terminal ileum in adults: a condition bearing a close resemblance to low-grade malignant lymphoma. *Histopathology* 17 19-26.

Rumbaugh, G.E., Smith, B.P. and Carlson, G.P. 1978. Internal abdominal abscesses in the horse: A study of 25 cases. *Journal of the American Veterinary Medical Association* **172** (3), 304-309.

Sack, W.O. 1992. *Rooney's guide to the dissection of the horse.* Sixth edition. Veterinary Textbooks. Ithaca. pp33-47.

Sanders-Shamis, M. 1985. Perirectal abscesses in six horses. *Journal of the American Veterinary Medical Association* **187** (5), 499-500.

Sanford, S.E. 1989. Multisystemic eosinophilic epitheliotropic disease in a horse. *Canadian Veterinary Journal* **30** 253-254.

Schneider, J.E., Kennedy, G.A. and Leipold, H.W. 1979. Muscular hypertrophy of the small intestine in a horse. *Journal of Equine Medicine and Surgery* 3 226-228.

Schneider, R.K., Milne, D.W. and Kohn, C.W. 1982. Acquired inguinal hernia in the horse: A review of 27 cases. *Journal of the American Veterinary Medical Association* **180** (3), 317-320.

Schuh, J.C.L., Ross, C. and Meschter, C. 1988. Concurrent mercuric blister and dimethyl sulphoxide (DMSO) application as a cause of mercury toxicity in two horses. *Equine Veterinary Journal* 20 (1), 68-71.

Scott, E.A. and Todhunter, R. 1985. Chronic intestinal intussusception in two horses. *Journal of the American Veterinary Medical Association* **186** (4), 383.

Sellers, A.F. and Lowe, J.E. 1986. Review of large intestinal motility and mechanisms of impaction in the horse. *Equine Veterinary Journal* **18** (4), 261-263.

Sellers, A.F., Lowe, J.E., Rendano, V.T. and Drost, C.J. 1982. The reservoir function of the equine cecum and ventral large colon - its relation to chronic non-surgical obstructive disease with colic. *Cornell Veterinarian* 72 233-241.

Semrad, S.D. and Moore, J.N. 1983. Invagination of the caecal apex in a foal. *Equine Veterinary Journal* **15** (15), 62-63.

Shokry, M. and Lotfi, M.M. 1984. Malignant perianal melanoma in a horse. *Modern Veterinary Practice* **65** (3), 226.

Sims, L.D., Tzipori, S., Hazard, G.H. and Carroll, C.L. 1985. Haemorrhagic necrotising enteritis in foals associated with *Clostridium perfringens*. *Australian Veterinary Journal* **62** (6), 194-196.

Slocombe, R.F. and Slauson, D.O. 1988. Invasive pulmonary aspergillosis of horses: an association with acute enteritis. *Veterinary Pathology* **25** 277-281.

Slone, D.E., Humburg, J.M., Jagar, J.E. and Powers, R.D. 1982. Noniatrogenic rectal tears in three horses. *Journal of the American Veterinary Medical Association* **180** (7), 750-751.

Smith, B.P. 1981a. Equine salmonellosis a contemporary view. *Equine Veterinary Journal* **13** (3), 147-151.

Smith, B.P. 1981b. Salmonella infections in horses. *The Compendium* **3** (1), 16-25.

Snow, D.H., Bogan, J.A., Douglas, T.A. and Thompson, H. 1979. Phenylbutazone toxicity in ponies. *Veterinary Record* **105** 26-30.

Snow, D.H., Douglas, T.A., Thompson, H., Parkins, J.J. and Holmes, P.H. 1981. Phenylbutazone toxicosis in equidae: a biochemical and pathophysiologic study. *American Journal of Veterinary Research* 42 (10), 1754-1759.

Snyder, J.R. 1989. The pathophysiology of intestinal damage: effects of lumenal distention and ischemia. *Veterinary Clinics of North America: Equine Practice* 5 (2), 247-270.

Snyder, J.R., Olander, H.J., Pascoe, J.R., Holland, M. and Kurpershoek, C.J. 1988. Morphologic alterations observed during experimental ischemia of the equine large colon. *American Journal of Veterinary Research*. 49 (6), 801-809.

Snyder, J.R., Pascoe, J.R., Olander, H.J., Hinds, D.M., Young, R. and Tyler, W.S. 1990. Vascular injury associated with naturally occurring strangulating obstructions of the equine large colon. *Veterinary Surgery* 19 (6), 446-455.

Snyder, J.R., Pascoe, J.R. Olander, H.J., Spier, S.J., Meagher, D.M. and Bleifer, D.R. 1989a. Strangulating volvulus of the ascending colon in horses. *Journal of the American Veterinary Medical Association* 195 (6), 757-764.

Snyder, J.R., Tyler, W.S., Pascoe, J.R., Olander, H.J., Bleifer, D.R., Hinds, D.M. and Neves, J.W. 1989b. Microvascular circulation of the

ascending colon in horses. *American Journal of Veterinary Research* **50** (12), 2075-2083.

Snyder, S.P., England, J.J. and McChesney, A.E. 1978. Cryptosporidiosis in immunodeficient Arabian foals. *Veterinary Pathology* **15** 12-17.

Specht, T.E. and Colahan, P.T. 1988. Surgical treatment of sand colic in equids: 48 cases (1978-1985). *Journal of the American Veterinary Medical Association* **193** (12), 1560-1564.

Spiers, V.C., Hilbert, B.J. and Blood, D.C. 1979. Dorsal displacement of the left ventral and dorsal colon in two horses. *Australian Veterinary Journal* **55** 542-544.

Spiers, V.C., Van Veenendaal, J.C., Christie, B.A., Lavelle, R.B., Gay, C.C. 1981. Obstruction of the small colon by intramural haematoma in three horses. *Australian Veterinary Journal* **57** 88-90.

Spencer, J., Diss, T.C. and Isaacson, P.G. 1990. A study of the properties of a low-grade mucosal B-cell lymphoma using a monoclonal antibody specific for the tumour immunoglobulin. *Journal of Pathology* **160** 231-238.

Sprinkle, F.P., Swerczek, T.W. and Crowe, M.W. 1984. Meckel's diverticulum in the horse. *Journal of Equine Veterinary Science* **4** 175-176.

Steckel, R.R. and Nugent, M.A. 1983. Parietal hernia in a horse. *Journal of the American Veterinary Medical Association* **182** (8), 818-819.

Stolfus, T.A. 1980. Breeding trauma in a miniature Appaloosa mare. *Veterinary Medicine and Small Animal Clinician* **75** 264.

Sullins, K.E., Stashak, T.S. and Mero, K.N. 1985. Pathologic changes associated with induced small intestinal strangulation obstruction and nonstrangulating infarction in horses. *American Journal of Veterinary Research* **46** (4), 913-916.

Sweeney, H.J. 1991. Gastroduodenal ulceration in foals. *Equine Veterinary Journal* **3** (2), 80-85.

Sweeney, R.W., Sweeney, C.R., Sack, J. and Lichtensteiger, C.A. 1986. Chronic granulomatous bowel disease in three sibling horses. *Journal of the American Veterinary Medical Association* **188** (10), 1192-1194.

Tennant, B., Keirn, D.R., White, K.K., Bentinck-Smith, J. and King, J.M. 1982. Six cases of squamous cell carcinoma of the stomach of the horse. *Equine Veterinary Journal* **14** (3), 238-243.

Todhunter, R.J., Erb, H.N. and Roth, L. 1986. Gastric rupture in horses: A review of 54 cases. *Equine Veterinary Journal* **18** (4), 288-293.

Traub, J.L., Gallina, A.M., Grant, B.D., Reed, S.M., Gavin, P.R. and Paulsen, L.M. 1983. Phenylbutazone toxicosis in the foal. *American Journal of Veterinary Research* 44 (8), 1410-1417.

Tulleners, E.P., Richardson, D.W. and Reid, B.V. 1985. Vaginal evisceration of the small intestine in three mares. *Journal of the American Veterinary Medical Association* **186** (4), 385-387.

Turner, T.A., Adams, S.B. and White, N.A. 1984. Small intestine incarceration through the epiploic foramen of the horse. *Journal of the American Veterinary Medical Association* **184** (6), 731-734.

Turner, T.A. and Fessler, J.F. 1980. Rectal Prolapse in the horse. *Journal of the American Veterinary Medical Association* **177** (10), 1028-1032.

Tzipori, S. and Campbell, I. 1981. Prevalence of *Cryptosporidium* antibodies in 10 animal species. *Journal of Clinical Micorbiology* 14 455-456.

Umemara, T., Ohishi, H., Ikemoto, Y., Satoh, H. and Fujimoto, Y. 1982. Histopathology of colitis X in the horse. *Japanese Journal of Veterinary Science* 44 717-724.

Urquhart, K. 1981. Diarrhoea in foals. *In Practice*. January, 22-29.

Uzal, F.A., Robles, C.A. and Olaechea, F.V. 1992. Histopathological changes in the coeliacomesenteric ganglia of horses with 'mal seco', a grass sickness-like syndrome, in Argentina. *Veterinary Record* **130** 244-246.

Van den Hoven, R. and Franken, P. 1983. Clinical aspects of lymphosarcoma in the horse: A clinical report of 16 cases. *Equine Veterinary Journal* **15** (1), 49-53.

Van der Gaag, I. and Tibboel, D. 1980. Intestinal atresia and stenosis in animals: A report of 34 cases. *Veterinary Pathology* **17** 565-574.

Van der Velden, M.A. 1989. Concurrent presence of mesenteric hernia and jejunal intussusception in a horse. *Veterinary Record* **125** 605.

Van der Velden, M.A. and Rutgers, L.J.E. 1990. Visceral prolapse after castration in the horse: A review of 18 cases. *Equine Veterinary Journal* **22** (1), 9-12.

Van Wuijckhuise-Sjouke, L.A. 1984. Three cases of obstruction of the small colon by a foreign body. *The Veterinary Quarterly* **6** (1), 31-36.

Vasey, J.R. 1988. Incarceration of the small intestine by the epiploic formen in fifteen horses. *Canadian Veterinary Journal* **29** 378-382.

Weaver, A.D. 1987. Masive ileal diverticulum: an uncommon anomoly. *Veterinary Medicine and Small Animal Clinician.* 82 73-74.

Wenkoff, M.S. 1973. *Salmonella typhimurium* septicaemia in foals. *Canadian Veterinary Journal* 14 (11), 284-287.

Wheeldon, E.B. and Greig, W.A. 1977. *Globidium leuckarti* infection in a horse with diarrhoea. *Veterinary Record* **100** 102-103.

White, N.A. 1981. Intestinal infarction associated with mesenteric vascular thrombotic disease in the horse. *Journal of the American Veterinary Medical Association* **178** (3), 259-262.

White, N.A. 1990. *The Equine Acute Abdomen.* Editor N.A. White. Lea and Febiger, Philadelphia.

White, N.A., Moore, J.N. and Trim, C.M. 1980. Mucosal alterations in experimentally induced small intestinal strangulation obstruction in ponies. *American Journal of Veterinary Research* 41 (2), 193-198.

White, N.A., Tyler, D.E., Blackwell, R.B. and Allen, D. 1987. Hemorrhagic fibrinonecrotic duodenitis - proximal jejunitis in horses: 20 cases (1977-1984). *Journal of the American Veterinary Medical Association* **190** (3), 311-315.

Whitlock, R.H. 1990. Feed additives and contaminants as a cause of equine disease. *Veterinary Clinics of North America: Equine Practice* **6** (2), 467-678.

Wilkie, J.S.N., Yager, J.A. Nation, P.N., Clarke, E.G., Townsend, H.G.G. and Baird, J.D. 1985. Chronic eosinophilic dermatitis: A manifestation of a multisystemic, eosinophilic, epitheliotropic disease in five horses. *Veterinary Pathology* **22** 297-305.

Wilson, D.A., Foreman, J.H., Boero, M.J., Didier, P.J. and Lerner, D.J. 1989. Small-colon rupture attirubutable to granulosa cell tumour in a mare. *Journal of the American Veterinary Medical Association* **194** (5), 681-682.

Wilson, D.G., Wilson, W.D. and Reinertson, E.L. 1983. Intussusception of the left colon in a horse. *Journal of the American Veterinary Medical Association* **183** (4), 464-465.

Wilson, R.G., Sutton, R.H., Groendyk, S. and Seawright, A.A. 1985. Alimentary lymphosarcoma in a horse with cutaneous manifestations. *Equine Veterinary Journal* **17** (2), 148-150.

Wimberly, H.C., Andrews, E.J. and Haschek, W.M. 1977. Diaphragmatic hernias in the horse: A review of the literature and an analysis of six additional cases. *Journal of the American Veterinary Medical Association* **170** (12), 1404-1407.