

# Satellite Article

## Strangulating obstructions of the small intestine

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### Introduction

Obstructions of the small intestine represent an important cause of surgical colic in the horse (Ducharme *et al.* 1983; Phillips and Walmsley 1993). Obstructions are classified as either nonstrangulating (simple obstructions; where the obstruction is not complicated initially by vascular compromise of the bowel) or strangulating. Strangulating obstructions are characterised by interruption of the intestinal blood supply with simultaneous blockage of the intestinal lumen (White *et al.* 1980). The strangulating obstruction produces intestinal oedema and congestion, intestinal distension and eventually gangrene or rupture (or both).

**This article describes the pathophysiology, clinical signs, diagnosis and common causes of strangulating obstructions of the small intestine.**

### Pathophysiology of strangulating obstructions of small intestine

The pathological effects of strangulating obstructions are caused by the effects of both physical obstruction of the bowel lumen and ischaemia of the bowel wall (Allen and Tyler 1990; White 1999). Physical obstruction of the small intestine prevents the passage of fluids from the upper alimentary tract reaching the absorptive surfaces of the large intestine. The fluid therefore becomes sequestered inside the intestinal lumen, which results in **hypovolaemia, decreased cardiac output and acid-base disturbances.**

The fluid, gas and ingesta that accumulate proximal to the obstruction **arise from several sources** including saliva, gastric secretions, swallowed air, pancreatic secretions, bile, gas from bacterial fermentation, ingested fluid and solids, and secretions from the intestinal mucosa (Allen *et al.* 1986). **Accumulation** of these substances in the intestinal lumen causes an increased intraluminal hydrostatic pressure, which influences microvascular circulation in the mucosa (Dabareiner *et al.* 1993). **Peristaltic waves diminish** and then cease altogether as the intestinal lumen is progressively filled,

leaving an atonic, rapidly distending tube (**Fig 1**). As the intraluminal hydrostatic pressure continues to rise, the absorption of water by the mucosa ceases and, instead, water begins to flow from the mucosa into the lumen (Snyder 1989). The increasing pressure and the expanding volume of fluid causes reflux into the stomach.

Vascular compromise of the strangulated segment of intestine may be venous, or venous and arterial, but the typical lesion causes venous occlusion before arterial occlusion with consequent venous congestion (Allen and Tyler 1990). Within minutes of strangulation occurring, the involved segment of bowel and its mesentery become deep red as the **veins and venules are distended with blood. If there is immediate concurrent arterial occlusion**, as in some cases of obstruction by a pedunculated lipoma for example, the intestine becomes cyanotic but not oedematous. More often, thicker walled arteries and arterioles resist compression for a while and continue to pump blood into the distended veins and venules. As the involved intestine is **engorged with blood**, vascular stasis develops and the segment becomes red/black in colour (**Fig 2**). Almost immediately, the vascular endothelium becomes more permeable and plasma diffuses into the tissue. Within a few hours, degeneration of vascular



**Fig 1:** Small intestinal strangulating obstruction showing a strangulated (distended and congested) loop of intestine lying adjacent to a loop of proximal bowel which is also distended. Loops of empty, distal small intestine are present in the foreground.

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**Fig 2: Congested and oedematous jejunum caused by strangulation due to a pedunculated lipoma.** Venous occlusion has resulted in engorgement of the strangulated segment of intestine with blood. Distended proximal intestine is also seen in the foreground.

endothelium becomes so extensive that blood pours out of the distended vessels into the tissues (venous infarction stage) and eventually into the lumen (Sullins *et al.* 1985).

**Eighty percent of the blood supply to the intestine goes to the mucosa.** The mucosal villi are extremely sensitive to hypoxia and, within minutes of oxygen deprivation, ultrastructural morphological changes are evident (White *et al.* 1980). The epithelial cells slough in sheets, starting at the tip of the villus and working towards the crypts. Within 4–5 h, the mucosal epithelium is completely necrotic. At the same time, the mesothelium of the serosa also sloughs and the fibrous layer becomes disrupted and oedematous (White 1999). By 6–7 h, the degenerative effects of hypoxia have extended through the external muscular layer.

**As soon as the mucosal barrier is damaged,** Gram-negative bacteria and endotoxins permeate the *lamina propria* and submucosa. Early in the development of the ischaemic lesions, the bacteria and endotoxins readily gain entry to the circulation via viable tissue adjacent to the lesion (Moore *et al.* 1981). In addition, by 6 h or possibly earlier, as the muscularis degenerates, bacteria and toxins leak through the serosa into the peritoneal cavity, from which they are readily absorbed. **Release of endotoxin into the general circulation** results in damage to endothelial cells and platelets (Moore and Barton 1998). Platelets are immediately stimulated and release the vasoconstrictor substances **thromboxane** and **serotonin**. Damage to the endothelium increases vascular permeability, **prostacyclin** is released and neutrophils are stimulated, especially in the lungs and sites of intestinal injury. The **endotoxic shock** is dose-related, and is more severe and rapidly developing the greater the length of bowel involved and the severity of vascular occlusion.

**The clinical picture is acute** with severe pain which is continuous and shows no, or only temporary, response to analgesics. Heart rate increases progressively and pulse quality deteriorates. Mucous membranes become congested and capillary refill time increases. Packed cell volume (PCV) and total plasma protein (TPP) rise progressively and respiratory



**Fig 3: Strangulation of a single loop of jejunum by a pedunculated lipoma.**



**Fig 4: A mesenteric lipoma arising close to the mesenteric border of the intestine which had resulted in recurrent, intermittent simple obstruction of the intestine.**

rate increases in response to the developing metabolic acidosis. At first, the **peritoneal fluid** is slightly serosanguinous with a mild increase in protein and leucocytes. As the strangulation process continues, all these substances increase dramatically and the fluid becomes flocculent and turbid. **Toxic neutrophils** indicate leakage of toxins and bacteria. The clinical course is rapid and **most horses with an untreated strangulation obstruction of small intestine die within 24–36 h** of the onset of disease from irreversible septic shock and marked vascular collapse.

The **cause of pain** in small intestinal obstruction is related to ischaemia, the increase in intraluminal pressure, and the resultant increase in bowel wall tension and increased pull of the viscera on the mesentery. The **intensity of pain** is proportional to the rapidity with which the intestinal tension develops and to the magnitude of the tension (Allen *et al.* 1986). The magnitude of the increase in intraluminal hydrostatic pressure in intestine proximal to the obstruction appears to have a direct effect on the **prognosis for survival**. In one study of horses with small intestinal obstruction, horses which lived following surgery had a **significantly lower** ( $P < 0.001$ ) **mean intraluminal hydrostatic pressure** (6.3 cmH<sub>2</sub>O) compared to horses that died (15.0 cmH<sub>2</sub>O) (Allen *et al.* 1986).

Circulating endotoxin, coupled with sequestration of large volumes of fluid into the intestinal lumen, results in cardiovascular compromise and low splanchnic blood flow. This leads to anoxic damage to the villi, starting with necrosis of the villus tip (White *et al.* 1980). This is an important cause of continued degeneration of the intestinal wall that can occur in segments of bowel left in the abdomen after resection and anastomosis procedures have been performed (Meschter *et al.* 1986).

**Reperfusion injury** might also be important in causing damage to the mucosa, although its importance in the small intestine is uncertain at this time. During intestinal ischaemia, reductions in blood flow lead to alterations in normal cellular physiology, including depletion of energy reserves and oxygen (Fantone 1990). After correction of the intestinal lesion and restoration of blood flow, additional injury (reperfusion injury) can develop as a result of the liberation of accumulated inflammatory products and oxygen-derived free radicals (Moore *et al.* 1995). This secondary bowel injury can lead to ileus, fluid secretion and even infarction after surgery (White 1999).

## Clinical signs and diagnosis

### Pain

Colic is a consistent feature of small intestinal disease and varies in intensity with the nature of the lesion and the length of time it has been present (White 1990; Edwards and Proudman 2001). **Strangulation obstruction** leads to severe unrelenting pain which persists for several hours until the ischaemic segment of gut becomes necrotic. As the affected segment of bowel becomes necrotic, the pain may abate and the horse becomes increasingly depressed.

### Rectal examination

Sequestration of fluid oral to the obstruction quickly leads to distension of the intestine that **can be identified by rectal palpation** (Kopt 1987; White 1990) or **transabdominal ultrasound** (Klohn *et al.* 1996). In horses presented early (<4 h), careful palpation over a period of several minutes may be necessary before one or 2 distended loops are identified; whereas multiple, tightly distended loops filling the caudal abdomen and extending to the pelvic inlet are present in horses in which the obstruction has been in existence for more than 12 h (Mueller 2002a). Proper restraint of the horse is essential when performing a rectal examination, to prevent injury to the examiner or the horse. In horses with severe pain, and in fractious or nervous horses, the use of short-acting sedatives (such as xylazine) and/or a nose twitch should be considered (Mueller 2002b). Straining by the horse can be reduced if necessary by administering 50–60 ml 2% lignocaine (lidocaine) *per rectum*, or by i.v. administration of hyoscine-N-butylbromide.

The presence of distended small intestine in horses with colic is, in the great majority of cases, an **indication for**

**surgical intervention**, which will identify a physical obstruction. Possible exceptions include horses suspected of having anterior enteritis or grass sickness.

Specific lesions of the small intestine are identified infrequently by rectal palpation, because the long mesentery allows the intestine to occupy any part of the abdomen and much of it may therefore be out of reach. In stallions with inguinal hernia, palpation in the region of the inguinal rings usually evokes a pain response as well as allowing identification of distended loops of intestine or a strand of mesentery entering one internal inguinal ring.

### Cardiovascular changes

Increases in heart rate, PCV and TPP occur in response to developing endotoxaemia and/or hypovolaemia (White 1990; Edwards and Proudman 2001). Mucous membranes provide a useful 'quick index' of cardiovascular compromise, as they soon become congested with endotoxaemic shock. Capillary refill time is extended with advancing hypovolaemia and severely shocked patients develop **purple mucous membranes** indicating hypoperfusion of peripheral tissues. These changes have been shown to correlate well with prognosis (Parry *et al.* 1983; Rakestraw 2002).

### Nasogastric intubation

Nasogastric intubation may reveal gastric reflux depending on the location of the obstruction and the length of time it has been in existence. Generally, more than 2 l of fluid with a pH >5 is considered abnormal. Significant gastric reflux can occur with either strangulating or nonstrangulating obstructions, and its presence is not necessarily an indication to perform surgery.

### Peritoneal fluid

Examination of peritoneal fluid is helpful in distinguishing between **simple** and **strangulation obstruction**, but not between **small** and **large intestinal problems**. Since abdominocentesis is an invasive procedure, and because of the small risk of traumatising grossly distended small intestine, resulting in leakage of intestinal contents, **paracentesis should be avoided** in horses where evidence based on other findings, particularly rectal examination, indicates the need for surgery (Edwards and Proudman 2001). Although the evaluation of peritoneal fluid can be extremely useful in aiding the decision to perform surgery, both false positive and false negative results are sometimes obtained. The presence of normal peritoneal fluid does not necessarily rule out the presence of intestinal strangulation, since the strangulated loop of intestine may be isolated from the peritoneal cavity (e.g. intussusception, inguinal hernia and diaphragmatic hernia). Discoloured peritoneal fluid (suggestive of intestinal strangulation) may be present in horses with anterior enteritis or nonstrangulating intestinal infarction.

### **Abdominal auscultation**

Monitoring intestinal motility is done by identification of intestinal sounds and evidence of faecal transit. Auscultation is not a good indicator of small intestinal activity and sounds of large colon and caecal motility may be present without any small intestinal activity. However, although sounds may be reduced temporarily in many cases of colic, a **persisting absence of gut sounds is expected in strangulating obstructions.**

### **Transabdominal ultrasonography**

Transabdominal ultrasonography (with a 5 MHz linear or 2.5 MHz sector scanner) can be particularly useful in detecting small intestinal disease. One study showed that the accuracy of abdominal ultrasonography in diagnosing small intestinal strangulating obstructions surpassed that of rectal palpation (Klohn *et al.* 1996). Oedematous, distended and immotile loops of small intestine are visualised in such cases. **Abnormal bowel is most commonly identified in the lower flank region and the ventral abdomen.**

### **Common causes of small intestinal strangulating obstructions**

#### ***Pedunculated lipomas***

Strangulation by pedunculated lipomas (**Figs 2 and 3**) is one of the commonest causes of strangulating obstruction of the small intestine (Blikslager *et al.* 1992; Edwards and Proudman 1994). **It has been estimated** that between 1.0 and 2.6% of all horses with colic, between 0.25 and 7.8% of surgical colic cases and between 0.5 and 17% of all horses with colic that undergo surgery because of small intestinal lesions have strangulation or obstruction of small intestine by a mesenteric lipoma (Freeman and Schaeffer 2001). Lipomas are benign fatty masses that originate in the mesentery of older horses, particularly pony geldings. In a recent study, the mean age of horses affected by strangulation by a lipoma was 19.2 years (Freeman and Schaeffer 2001).

As the mass enlarges, it draws out a pedicle of mesentery which allows the mass to move freely within the abdominal cavity. In cases where the mass arises close to the intestine, the pedicle may act as an extraluminal obstruction, compressing the intestine and causing a simple, often intermittent, obstruction (**Fig 4**). Lipomas with longer pedicles that originate further away from the intestine are more likely to become involved in strangulation of the small intestine; for unknown reasons, in some horses, such lipomas wrap themselves around the intestine, causing strangulation. Occasionally, signs of colic in a horse are reported to have commenced during or immediately after exercise; in such cases, it is hypothesised that the lipoma becomes wrapped around the gut as it swings around in the abdomen of the rapidly moving horse. Variable lengths of intestine may be involved, from 20 cm to 15 m. **Treatment** involves cutting the pedicle of the lipoma, followed by resection of the strangulated intestine.

### **Epiploic foramen entrapment**

The epiploic foramen is located in the right dorsal abdomen and is bordered by 2 organs (the liver and the pancreas) and by 2 large veins (the *vena cava* and the hepatic portal vein). In most horses, the foramen is a slit-like opening (approximately 4 cm long in a 450 kg horse) that may be partially entered by one or 2 of the surgeon's fingers at laparotomy (Freeman 1997). The foramen marks the entry to the omental bursa.

**Small intestine** (usually ileum) may enter this foramen and become obstructed (Turner *et al.* 1984; Vasey 1988; Engelbert *et al.* 1993; Vachon and Fischer 1995). Occasionally, the horse suffers only simple obstruction of small intestine. More commonly, strangulating obstruction of the ileum and jejunum is involved. Intestine most commonly passes from left to right through the epiploic foramen (**Fig 5**) to lie in the right side of the abdomen above the duodenum (Vachon and Fischer 1995; Freeman 1997). **This type of small intestinal obstruction is characterised by severe pain, consistent with ischaemic bowel that has not undergone complete necrosis.** Occasionally, horses with strangulation of small intestine in the epiploic foramen may present with signs of only mild abdominal pain (Vachon and Fischer 1995).

Surgical correction is not straightforward due to the inaccessibility of the foramen and the delicacy of the structures forming its margins. **Reduction** of the herniated intestine must be performed **with utmost care** to avoid the possibility of fatal tearing of the *vena cava* or portal vein (Vachon and Fischer 1995).

#### ***Volvulus***

Volvulus is an uncommon disease of the small intestine that results from intestine rotating about its mesentery for 360° or more, resulting in strangulation (Robertson 1990). The intestine may become twisted into a distinct spiral, or the loops of intestine may become entwined into a knot (*volvulus nodosus*) (**Fig 6**). Volvulus may occur as a **primary displacement** or **secondary** to a pre-existing lesion, such as inguinal hernia, mesodiverticular band or Meckel's diverticulum (Grant and Tennant 1973; Freeman *et al.* 1979; Moll *et al.* 1991). The length of intestine involved is variable, ranging from less than 1 m to most of the small intestine. Cases are characterised by extreme pain initially, followed by a decreasing level of pain as the intestine becomes necrotic. At this stage, endotoxaemia develops rapidly. Foals and yearlings seem to be particularly susceptible to primary volvulus, but other risk factors predisposing to the condition are unknown. Treatment involves correction of the twist and resection of ischaemic bowel. Correction can be particularly difficult in cases of *volvulus nodosus*.

#### ***Internal herniation***

Small intestine can become incarcerated and undergo strangulation within a normal or pathological opening in the peritoneal cavity. In addition to the epiploic foramen, **other**



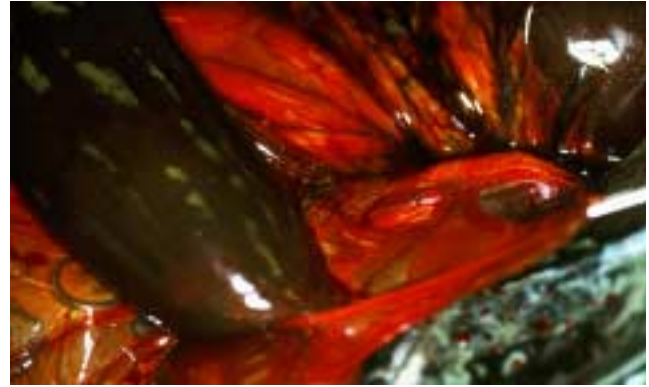
**Fig 5:** Left to right herniation and strangulation of small intestine through the epiploic foramen (post mortem appearance).



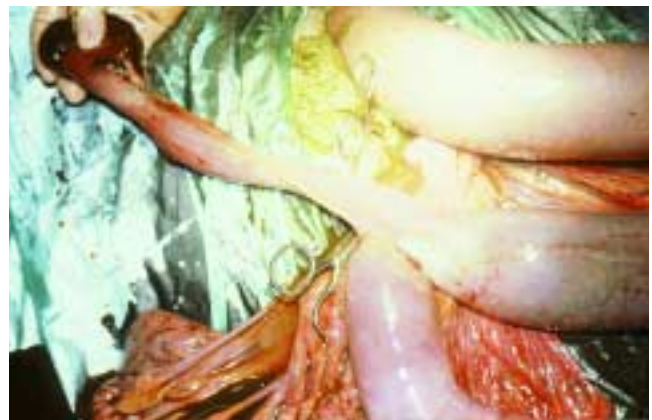
**Fig 6:** Strangulating obstruction due to volvulus nodosus. The intestine has become entwined into a knot.

**potential openings** include the nephrosplenic space, and defects or tears in the mesentery, caecocolic fold, omentum, gastrosplenic ligament, broad ligament and lateral ligament of the bladder (Tennant *et al.* 1975; Robertson 1990; Gayle *et al.* 2001). The gastrosplenic ligament is a broad attachment between the left part of the greater curvature of the stomach and the hilus of the spleen. Small intestine can pass through tears in the ligament, from caudal to cranial, resulting in strangulated loops of intestine lying lateral to the stomach and cranio-lateral to the spleen (Yovich *et al.* 1985).

Intra-abdominal foraminae may be formed by fibrous bands, adhesions and congenital anomalies. A mesodiverticular band develops from a vitelline artery and associated mesentery that fails to atrophy during early embryonic development. The band is usually found in the distal jejunum and extends from one side of the mesentery to the antimesenteric surface, thereby creating a triangular space. Intestine may become entrapped in a rent that forms in this space (**Fig 7**), and this may be complicated further by secondary volvulus (Freeman *et al.* 1979). Meckel's diverticulum is a remnant of the vitelline duct; it forms a blind extension from the antimesenteric surface of the distal



**Fig 7:** Incarceration and strangulation of small intestine through a mesodiverticular band.



**Fig 8:** Example of Meckel's diverticulum that resulted in strangulating obstruction of small intestine. Meckel's diverticulum is a blind extension from the antimesenteric surface of the distal jejunum or ileum.

jejunum or ileum. Occasionally, a fibrous band runs from the apex of Meckel's diverticulum to the umbilicus (the vitello-umbilical band). Meckel's diverticulum can become impacted and may undergo necrosis and rupture. It can also entangle and strangulate the small intestine (Hooper 1989) (**Fig 8**), or form an axis for *volvulus nodosus* (Grant and Tennant 1973).

### Adhesions

All horses that have undergone prior laparotomy are at risk of having intra-abdominal adhesions from surgery. Several studies have recognised an increased risk of colic in horses that have undergone previous colic surgery and it is suggested that intestinal obstruction due to adhesions is one explanation for this. **Adhesions are especially common following small intestinal surgery.** In one study, 22% of 172 horses that had surgery for small intestinal obstruction developed abdominal adhesions that required additional surgery or euthanasia (Baxter *et al.* 1989). Other studies have reported lower rates of adhesion formation following small intestinal surgery (Southwood and Baxter 1997). Many adhesions are asymptomatic. It is only when these adhesions create a hole



**Fig 9:** Oedema and discolouration of scrotal skin due to a scrotal rupture.



**Fig 10:** Herniation and strangulation of small intestine through a scrotal hernia in a stallion.

small enough for intestine to become entrapped that problems may occur. Adhesions may also cause simple obstruction of the small intestine by extraluminal compression of the bowel or by 'kinking' the bowel in such a way that flow of ingesta is obstructed.

**Adhesions may arise in horses that have never undergone abdominal surgery.** Intra-abdominal abscesses, parasite migration and peritonitis (possibly following castration) are all potential causes of peritoneal inflammation that may give rise to adhesions. Diagnosis of adhesions is almost impossible in the absence of clinical signs of intestinal obstruction. One area that is amenable to investigation is the ventral midline, where adhesions between viscera and the healed midline incision may be imaged **ultrasonographically**. The dorsal abdomen can also be evaluated for adhesions by **laparoscopy** in the standing patient.



**Fig 11:** Ileo-ileal intussusception. The recipient section of bowel (the intussusciens) is distended by the invaginated bowel (the intussusceptum).



**Fig 12:** Jejuno-jejunal intussusception. Tension on the mesentery of the intussusceptum causes the bowel to spiral into a corkscrew configuration.

### **Diaphragmatic hernia**

Herniation of small intestine through a full thickness defect in the diaphragm usually results in strangulating obstruction of the affected piece of bowel. There appear to be different types of diaphragmatic defect, some **congenital** in nature while others appear to be **traumatic** rents (Hance *et al.* 1991). This latter type may be associated with a recent episode of trauma causing a sudden increase in intra-abdominal pressure, e.g. severe exercise, dystocia, a fall over an obstacle or becoming straddled on a gate or fence. The lesion is usually located in the tendinous portion of the diaphragm.

The presence of large intestinal herniation through a diaphragmatic defect usually leads to **signs of respiratory distress**. Small intestinal herniation invariably results in **colic** and **signs consistent with a strangulating obstruction** (Pearson *et al.* 1977; Bristol 1986). Rectal examination may give the impression of an 'empty' abdomen. Diagnosis of this condition can be frustrated by the absence of changes to the peritoneal fluid, which is due to ischaemic bowel being sequestered in the pleural cavity. **Confirmation of the diagnosis may be achieved by thoracic radiography and ultrasonography.**

### Inguinal/scrotal hernia

Herniation of small intestine through the inguinal canal into the vaginal tunic is not uncommonly observed in newborn foals. This **congenital form** of inguinal herniation rarely results in intestinal obstruction and usually resolves spontaneously as the foal develops (by age 3–6 months). However, strangulation can occur if the intestine herniates through a tear in the vaginal tunic distal to the vaginal ring. Escape of peritoneal fluid into the subcutaneous tissues results in pitting swelling and discolouration of the scrotal and preputial areas (**Fig 9**).

**Acquired inguinal hernia** in the stallion is a more problematical condition, but fortunately rare in most breeds. Most acquired inguinal hernias are strangulated and require immediate surgical intervention. A congenital predisposition to acquired inguinal hernia has been reported in the North American Standardbred (Sembrat 1975). The condition may occur after strenuous activity (e.g. mating) resulting in increased intra-abdominal pressure. This forces intestine into the vaginal tunic and small intestinal obstruction occurs (Schneider *et al.* 1982). As oedema and swelling of the obstructed bowel develop in the confined space of the vaginal tunic, the testicular and intestinal blood vessels become obstructed, causing ischaemic damage to herniated small intestine and the testis (**Fig 10**). Scrotal herniation should always be considered in cases of acute colic in entire males. Ultrasonography of the scrotum can be helpful in diagnosis.

### Umbilical hernia

Strangulation of small intestine in an umbilical hernia is rare. Often, only a portion of the wall of a loop of small intestine (usually jejunum or ileum) becomes incarcerated (Richter's hernia or parietal hernia). **Typical clinical signs** include swelling, heat, firmness and pain around the hernia (Markel *et al.* 1987). Ultrasonography can be helpful in evaluating the contents of an umbilical hernia if incarceration is suspected.

### Intussusceptions

Intussusceptions occur when intestine 'telescopes' into an adjacent segment of intestine. The recipient section of bowel is termed the *intussusciens* and the invaginated bowel the *intussusceptum*. The amount of intestine involved is variable; it can be as little as a few centimetres or as much as 3–5 m. Short intussusceptions, typically ileo-ileal (**Fig 11**) in young horses, may present as **recurrent colic cases** due to intermittent partial obstruction. Jejunal and ileocaecal intussusceptions (**Fig 12**) generally involve longer segments of intestine and cause complete obstruction. As oedema and tension increases at the point of invagination, strangulation of the bowel can occur.

Suspected predisposing factors include segmental motility differences caused by enteritis, heavy ascarid burdens, *Anoplocephala perfoliata* infestation, mesenteric arteritis and abrupt dietary changes. Ileocaecal intussusceptions are the most common form of small intestinal intussusceptions

encountered, and are commonest in young horses age less than 3 years (Freeman 1997). These intussusceptions can present as either an **acute obstruction** or a **chronic problem** (Ford *et al.* 1990). Jejunal intussusceptions are rarer and are more likely to present as acute colic, although chronic signs are occasionally seen (Gift *et al.* 1993). A luminal or intramural mass (such as leiomyomas and granulomas) is sometimes associated with these lesions.

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