WHEN THE GUT STOPS MOVING

GENERAL PRINCIPLES OF INTESTINAL OBSTRUCTION

- all intestinal obstructions involve a significant physical or functional impairment in the efficient movement of luminal contents distally along the intestines
- clinical signs may include lethargy, depression, inappetence or anorexia, vomiting, abdominal pain (colic), abdominal distension etc.

Proximal to Mid Small Intestinal Obstruction

- **blockage of the upper or mid small intestine → peracute to acute** clinical signs of obstruction
- → proximal accumulation of fluid (from ingesta, saliva and gastric, pancreatic, biliary and intestinal secretions) and gas (either swallowed or produced by gastrointestinal bacteria) → intestinal distension and decreased motility → luminal sequestration of water and electrolytes (**stagnant loop syndrome**)
- stasis may → overgrowth of luminal bacteria → stimulation of further secretion
- stagnant luminal contents may act osmotically → further distension by water derived from the circulation

- **rapid progression** leads to:

  - **vomiting** or **regurgitation** or **reflux** - may relieve abdominal pain temporarily but exacerbates fluid and electrolyte losses; can → oesophageal ulceration and/or aspiration pneumonia
  - **dehydration** or **hypovolaemic shock**
  - **hypochloraemia**
  - **hypokalaemia**
  - **metabolic alkalosis** - due to loss of hydrochloric acid in vomitus/reflux or, in ruminants, sequestration of abomasal hydrochloric acid in the rumen

- transmural pressure from intestinal distension may ultimately → compression of thin-walled mural veins and venules → ischaemic necrosis (venous infarction) +/- intestinal rupture → septic peritonitis

Distal Small and Large Intestinal Obstruction

- **obstruction of the ileum or large bowel → more gradual** development of proximal GI distension, with a **more chronic clinical course**
- **fluid, electrolyte and acid-base disturbances are less severe than in proximal to mid small intestinal obstructions** because vomiting/reflux/regurgitation may not occur or is less severe and luminal fluid proximal to the obstruction may be absorbed gradually over time
- may see dehydration, inappetence or anorexia, weight loss, abdominal distension and discomfort, **metabolic acidosis** (due to sequestration of intestinal bicarbonate) and straining to defaecate (**tenesmus**) or decreased frequency of defaecation due to constipation

- in **horses**, complete obstruction of the large bowel may → tympany of the caecum and colon due to gas production from bacterial fermentation → rupture → septic peritonitis

CONGENITAL MALFORMATIONS OF THE INTESTINES

Segmental Intestinal Aplasia or Atresia

- segmental anomalies (**aplasia** or **atresia**) of the intestine are **common** in domestic animals
- probably result from segmental ischaemia during early foetal life (e.g. during the period when coiled loops of intestine herniate through the umbilical ring and are then withdrawn into the abdominal cavity)
- may cause **stenosis** (incomplete occlusion of the lumen) or **atresia** (complete luminal occlusion)
- in atresia, the lumen may be occluded by a membrane or there may be two blind ends of bowel with or without an intervening cord of connective tissue and mesentery

- segmental anomalies → progressive abdominal distension due to accumulation of meconium and faeces proximal to the obstruction
- prenatal distension may be sufficient to cause dystocia (difficult parturition)

- **atresia ilei** - especially **calves** but also reported in foals, lambs, piglets and puppies

- **atresia coli** - the **most common intestinal segmental anomaly**
- especially **Holstein calves** (in which it is inherited as an autosomal recessive trait and especially involves the spiral colon) and **foals** (large or small colon)

- **atresia ani = imperforate anus**
- especially **pigs** (inherited) and **calves**
- can be caused by *in utero* vitamin A deficiency
- may be an isolated defect or associated with other congenital malformations (e.g. distal spinal column deformities such as sacral or coccygeal vertebral agenesis (complete failure of development), rectovaginal fistula, renal agenesis, polycystic kidneys and/or cryptorchidism (failure of descent of one or both testes into the scrotal sac))

Congenital Colonic Aganglionosis

- inherited as an autosomal recessive trait in **white foals** born to parents with multiple spots ("frame overo" pattern of coat colour)
- analogous to Hirschsprung's disease in humans
- absence of ganglia of the myenteric plexus of the distal ileum, caecum and colon → absence of peristaltic contractions → segmental stenosis with accumulation of meconium and gas proximal to the stenosis
- → colic and death usually within 48 hours of birth
- melanocytes are largely absent from the skin (melanoblasts and myenteric plexus ganglionic neurons are both derived embryologically from neural crest cells)

- a similar megacolon syndrome due to segmental colonic aganglionosis is reported in **pigs**
- **Clydesdale foals** in Australia and USA may develop megacolon by 4-9 months of age due to hypoplasia (incomplete development) of the myenteric plexus of the dorsal large colon, transverse colon and/or small colon

EXTRINSIC OBSTRUCTION

- mass lesions in adjacent structures may cause **external compression of the intestines**, resulting in some degree of luminal obstruction
- e.g. tumours or abscesses, foci of massive fat necrosis, fibrous adhesions etc.
- e.g. left dorsal displacement of the large colon in horses
- e.g. external compression of herniated intestinal segments by a hernial ring

Rectal Prolapse

- especially **pigs**, **sheep** and **cattle**
- may develop in any animal that has protracted straining to defaecate or urinate (e.g. colitis, constipation, urinary tract obstruction, prostatic enlargement etc.)
- may develop during straining at parturition or in animals during intense coughing episodes

- may occur as a herd outbreak in **pigs** exposed to **zearalenone**, an oestrogenic mycotoxin produced by *Fusarium* spp. of fungi → marked congestion and oedema of vulval and vaginal mucosa → straining → vaginal +/- rectal prolapse
- rectal prolapse in **sheep** may also result from consumption of **oestrogenic pastures**

- the prolapsed distal rectal segment becomes congested and oedematous (due to constriction of venous outflow by the tight anal sphincter)
- the everted mucosa may undergo venous infarction and ulceration
- the necrotic segment may eventually slough → rectal stricture

LUMINAL OBSTRUCTION

- **obturation = occlusion of the intestine by an intra-luminal mass**

Food

- e.g. impaction of the caecum or colon of **horses** by **fibrous feed**
- may be associated with water deprivation, poor dentition or an abrupt change in diet from soft lush feed to hay or chaff

- e.g. impaction of the colon of **horses** by **sand**
- e.g. impaction of the small intestine by **gravel**, **fibrous feed** or **large numbers of acorns or leaves**

Faeces

- e.g. **constipation** and **obstipation** (impaction) of the colon and rectum are common in **dogs** and **cats**
- e.g. in dogs secondary to prostatic enlargement or anal sac impaction
- e.g. following pelvic or spinal cord trauma or damage to autonomic nerves of the intestines
- may cause **megacolon**
- many cases of megacolon in domestic animals are idiopathic

Bezoars and Enteroliths

- **phytobezoars** - most often found in the colon of **horses**, often as incidental findings

- **trichobezoars** - occasionally found in the intestines of **dogs** and **cats**, often as incidental findings

- **enteroliths** - rare except in **horses**
- usually composed of magnesium ammonium phosphate (struvite) deposited in concentric lamellae around a nidus (foreign body or feed particle)
- usually smooth and spherical
- may be solitary or multiple
- large enteroliths may weigh up to 10 kg
- may be an incidental finding but can impact at the pelvic flexure of the large colon or in the transverse or small colon
- associated with diets rich in magnesium and phosphate (e.g. bran, grain, alfalfa) or with consumption of alkaline water

Parasites

- e.g. masses of small intestinal roundworms in pigs and foals
- e.g. masses of small intestinal tapeworms in sheep

Foreign Bodies

- especially **dogs** and **cats**
- **sharp foreign bodies** may perforate the intestine → septic peritonitis
- **blunt foreign bodies** (e.g. corn cobs) may become impacted and cause local pressure necrosis or act as a nidus for enterolith formation
- **linear foreign bodies** (e.g. string, strips of cloth, stockings, fishing line) can → pleating (plication) of the intestines (akin to a concertina) and eventually perforation if one end of the material becomes fixed (e.g. around the base of the tongue or at the gastric pylorus)

INTESTINAL HYPOXIA AND INFARCTION (VASCULAR ACCIDENTS)

- inadequate vascular perfusion of the intestines is a **common** problem, especially in **horses**

- in the **small intestine**, 5-10 minutes of hypoxia → sloughing of enterocytes from the tips of the villi and progressing towards the base of the villi (complete within 1-3 hours)
- crypt epithelial necrosis begins 2-4 hours after onset
- by 30-60 minutes post-onset, there is necrosis of serosal mesothelium → fibrin exudation
- smooth muscle necrosis does not begin until after 6 hours of hypoxia

- hypoxia may cause initial smooth muscle hyperexcitability but thereafter see **paralytic ileus** (see below) → bowel stasis, accumulation of luminal fluid and gas, and luminal proliferation of anaerobic bacteria

- the **colon** is less sensitive to hypoxia than the small intestine, at least in the dog and horse
- in the colon, sloughing of surface enterocytes occurs within 1 hour of onset
- necrosis of colonic crypt epithelium is apparent by 3-4 hours post-onset

- **reperfusion injury** may follow restoration of blood flow and may → further damage in areas of subcritical hypoxia
- mediated by local generation of reactive oxygen species (free radicals) derived from the

intestinal mucosa and from neutrophils, complement activation, neutrophil invasion of reperfused tissues with local release of their proteolytic enzymes and pro-inflammatory cytokines (e.g. tumour necrosis factor-α and interleukin-1)

- **short term hypoxia** (maximal 3-4 hours) → survival of at least some cells at the base of the crypts → mitotic division → re-epithelialisation of the mucosal surface within 1-3 days
- **more protracted hypoxia** → full thickness mucosal necrosis with ulceration → repair by granulation tissue and re-epithelialisation from surviving epithelial cells at the margins of the infarct → loss of functional absorptive area and risk of stricture

- until re-epithelialisation occurs, animals are at risk of **hypovolaemia** (from intra-luminal leakage of blood and plasma proteins), **secondary invasion by bacteria (+/- fungi)** and/or **absorption of bacterial toxins into circulation**

- **protracted hypoxia** → full thickness infarction of the intestine → perforation → **septic peritonitis**

Venous Infarction

- **obstruction of venous outflow is the most common cause of intestinal infarction**
- affected segments of bowel become swollen and turgid due to severe mural congestion, oedema and haemorrhage → luminal distension by bloody fluid and gas → critical hypoxia → venous infarction → +/- rupture
- associated mesenteries are also congested and oedematous
- usually a sharp line of demarcation between affected and unaffected intestine

Intestinal Strangulation

- strangulation involves **extrinsic compression of venous outflow** from the intestine
- e.g. by pedunculated lipomas in horses
- e.g. by hernial rings
- e.g. in intestinal torsion, mesenteric torsion (intestinal volvulus) and intussusceptions

Intestinal Torsion

- **intestinal torsion = rotation of the intestine across its long axis**

- most common in the **caecum** of **cattle** and **horses**
- may be predisposed to by hypoplasia of the caecocolic fold
- clinical signs of obstruction are associated with **360º torsion of the caecum**

- in **ruminants**, **caecal dilation** is usually associated with a sudden dietary change to a high concentrate ration → increased VFA production (especially butyric acid) → caecal atony and distension with watery ingesta → clockwise or anti-clockwise **torsion** → venous infarction
- especially cattle within 2 months of parturition
- may also occur in late stages of pregnancy or secondary to ileus (intestinal paralysis)

- in **horses**, **caecal and colonic tympany** usually develop after a sudden feed change to a high concentrate diet → ↑ VFA production → ↓ pH → fermentation switches to produce poorly absorbed acids (butyric and lactic acids) → hypomotility and osmotic drag of fluid into the large bowel lumen

- may lead to **torsion of the caecum** or **volvulus of the large colon** (see below)
- → severe abdominal distension → respiratory compression, impaired venous return to the right heart, circulatory collapse and metabolic acidosis

Mesenteric Torsion (Intestinal Volvulus)

- **mesenteric torsion (= intestinal volvulus) = rotation of the intestine on its mesenteric axis**

- **rapidly fatal**

- common in suckling or artificially reared **lambs** and **calves** due to guzzling of feed → gas formation → volvulus of most of the small intestine

- common in **pigs** due to gluttonous consumption of highly fermentable feed → gas production in the colon → usually anti-clockwise mesenteric torsion involving the small and sometimes large intestine

- in **dogs**, small intestinal volvulus can be associated with ingestion of a large volume of food or because there are bulky intestinal contents due to exocrine pancreatic insufficiency

- in **horses**, may involve segments of **small intestine**
- horses are also predisposed to **volvulus of the large colon** due to its lack of mesenteric anchorage and hence enhanced mobility; tympany of the large colon also predisposes to volvulus (see grain overload above)
- the ventral colon may rotate dorsolaterally or dorsomedially relative to the dorsal colon
- dorsomedial rotation of the left colon is most common → kinking at the caecocolic fold or at the sternal and diaphragmatic flexures
- clinical signs of obstruction are associated with colonic rotations of **at least 180^{o}**

Intestinal Intussusception

- **intussusception = telescoping of one segment of intestine into the lumen of a more distal segment**

- most common in **young animals**
- can be predisposed to by foreign bodies, large parasite burdens, enteritis, previous intestinal surgery or intramural tumours or abscesses but often idiopathic
- can also be an incidental agonal (terminal) or post mortem event (due to ongoing peristaltic contractions); in these cases, there is no vascular compromise of the affected bowel segment

- most often seen in **dogs** - especially **ileocolic intussusception**
- also common in **lambs**, **calves** and **young horses** – may involve the **small intestine**, **colon** or **caecum** (e.g. caecal inversion into itself, or intussusception of the caecum into the right ventral colon)

- the length of the intussusception is limited by mesenteric tension to approximately 10-12 cm in small animals and 20-30 cm in large animals

- tension on and compression of mesenteric veins → congestion and oedematous swelling of the telescoped segment (the **intussusceptum**) → fibrin and neutrophilic exudation → adhesion (rendering it surgically irreducible) → ultimately venous infarction → possible sloughing

Arterial Infarction

- the intestines have a **parallel arterial blood supply**, with many anastomoses close to the mesenteric border
- occlusion of the smaller arterial vessels rarely causes infarction
- occlusion of a larger mesenteric artery near its origin from the abdominal aorta → infarction

- most often seen in **horses** as a consequence of **arterial thromboembolism** from thrombi in the root of the cranial mesenteric artery caused by ***Strongylus vulgaris*** **larvae**
- especially associated with secondary bacterial infection with softening of the thrombus
- the emboli often incompletely occlude small caecocolic branches of the mesenteric artery, often without infarction due to the presence of sufficient collateral circulation but may cause intermittent colic due to transient slow flow of arterial blood

- may occasionally see arterial thromboembolism and infarction of the intestines in bacteraemias or septicaemias (e.g. *Pasteurella* septicaemia in lambs, *Histophilus somni* bacteraemia in cattle)

Other Causes of Intestinal Hypoxia and Ischaemic Necrosis

- the following conditions may cause intestinal hypoxia with or without progression to infarction:

  - **sudden occlusion of the portal vein** (or a major mesenteric vein draining into the portal vein) → sudden death from venous infarction of the gut

  - **portal hypertension** - prolonged increase in pressure in the portal vein (e.g. cirrhosis) with chronic passive congestion of the intestines

  - **shock** - with pooling of venous blood in the splanchnic viscera - especially dogs

  - **disseminated intravascular coagulation** - with microthombosis of intestinal vessels

  - **non-steroidal ant-inflammatory drugs** (**NSAID**)
    - especially phenylbutazone (PBZ, "bute")
    - may cause oral, gastric and/or intestinal (especially **right dorsal colonic**) mucosal ulceration in horses
    - possibly a direct effect of PBZ on small blood vessels → microthrombosis
    - inhibition of prostaglandin synthesis may also → vasoconstriction and decreased mucosal blood flow

  - **hypoxia at the periphery of the circulatory field of the caudal mesenteric artery**
    - may predispose to rectal perforation in horses and rectal stricture in pigs

INTESTINAL STENOSIS

- **stenosis = narrowing or stricture of the lumen due to thickening of the intestinal wall**

- may be partial or complete, congenital or acquired
- e.g. mural abscesses or granulomas, haematomas, tumours, scarring following mucosal ulceration

Reparative Fibrosis

- intestinal stenosis at any level can result from previous mucosal ulceration and subsequent fibrosis (scarring)
- **pigs** that survive colonic/rectal ulceration caused by *Salmonella typhimurium* infection may develop **rectal stricture** due to reparative fibrosis

Intestinal Smooth Muscle Hypertrophy

- some degree of smooth muscle hypertrophy is expected to develop proximal to chronic partial obstructions of the small or large intestine

- prominent smooth muscle hypertrophy of the **distal ileum** is common in **horses** and **pigs**
- **often idiopathic** but may develop secondary to physical or functional obstruction of the ileocaecal valve
- sometimes associated in horses with ileocaecal valve parasitism by tapeworms (*Anoplocephala* spp.)
- **usually incidental** but may cause mild chronic colic in horses
- in some animals, can lead to formation of diverticula, impaction and/or rupture
- **diverticula** are saccular outpouchings of mucosa into the subjacent smooth muscle and subserosal layers of the intestine; they can become filled with ingesta and may ultimately rupture → septic peritonitis

Neoplasia

- **uncommon** in domestic animals (**apart from lymphoma**)
- **malignant tumours are more common in the intestines than benign tumours**

Benign Epithelial Masses

- so-called **colorectal polyps** are common in the distal rectum (within 10 cm of the anorectal junction) in middle-aged and older **dogs** → tenesmus and haematochezia
- may be sessile (broad-based) or pedunculated mass of 1-3 cm diameter
- often ulcerated → bleeding
- most are **adenomas** (i.e. **benign epithelial tumours**) and surgical excision is curative
- malignant transformation (e.g. epithelial anaplasia (poor differentiation) and invasion into the lamina propria and/or submucosa) may be histologically apparent in some polyps, especially those greater than 1 cm in diameter; such lesions may recur post-surgically if incompletely excised

- in other domestic animal species, **adenomatous hyperplastic polyps** (i.e. not true tumours) may develop in the intestinal mucosa at any level
- most are incidental findings but larger protruding polyps may cause partial intestinal obstruction
- hyperplastic polyps may develop in the mucosa of the small intestine of lambs and calves with chronic coccidiosis

Intestinal Adenocarcinoma

- a **malignant tumour** of **intestinal epithelial origin**

- usually grossly grey-white and firm
- annular tumours infiltrating the full thickness of the intestine → stenosis

- a few may be papillary and project into the bowel lumen
- typically composed of invasive cords and acini of mucin-producing carcinoma cells and usually scirrhous (firm) due to reparative fibrosis in sites of invasion
- some may have a gross glassy appearance due to copious mucin production by the tumour cells

- metastasise widely via lymphatics to regional lymph nodes +/- liver, spleen and lungs
- may also implant widely over the peritoneum (→ ascites due to diaphragmatic lymphatic obstruction)

- common in **sheep** in New Zealand and south-eastern Australia (especially in breeds used for fat lamb production), United Kingdom, Norway and Iceland
- high prevalence may reflect exposure to bracken fern or other carcinogenic agents
- in New Zealand, occurrence is associated with heavy use of certain fertilizers and/or ingestion of *Cynosaurus cristatus* (crested dogstail grass)
- usually located in the mid to lower small intestine

- in Scotland, **cattle** exposed to bracken fern and bovine papillomaviruses → **intestinal adenoma** (a benign tumour of intestinal epithelial origin) or **adenocarcinoma** development
- affected animals may have concurrent squamous papilloma or squamous cell carcinoma of the oesophagus or reticulum/rumen or papilloma, carcinoma or haemangioma of the urinary bladder

- relatively common in older **cats** (especially Siamese) in the ileum, jejunum or, less often, at the ileocaecal junction

- uncommon in older **dogs**
- boxers, collies, poodles and German shepherd dogs appear to be predisposed
- may involve the proximal small intestine or (more often) the colon/rectum
- ulcerated tumours commonly bleed → anaemia

Lymphoma

- a **malignant tumour of lymphocytes**

- most common in **cats** but can arise in any domestic animal species
- may arise as a **primary tumou**r in the intestines **or** be **part of multicentric lymphoma**
- may be segmental or diffusely involve the gastrointestinal tract
- often concurrent neoplastic involvement of regional lymph nodes +/- liver
- may appear grossly as soft, cream-white, protruberant mucosal/submucosal nodules but lamina proprial and submucosal infiltrates are often grossly inapparent or cause only subtle thickening of the affected segment of bowel

- **cats** - often segmental and especially involving the jejunum and ileum
    - **epitheliotropic T cell lymphoma** - old cats; slowly progressive
    - **large cell (lymphoblastic) B cell lymphoma** - any age; rapidly progressive
    - **large granular lymphoma** - composed of either T cells or natural killer cells containing large cytoplasmic granules; rapidly progressive with widespread metastasis +/- leukaemia

- **dogs** - especially small intestine
    - most are **epitheliotropic T cell lymphomas**
    - commonly → enteric protein loss → hypoproteinaemia

- **horses** - especially small intestine of young adults
    - usually **B cell lymphoma**
    - malabsorption and enteric protein loss → loss of condition +/- intermittent colic and diarrhoea

Plasma Cell Tumour

- a **benign or malignant tumour of plasma cells** (differentiated B lymphocytes)
- uncommon
- mainly seen in older **dogs** arising in the submucosa of the distal colon and rectum where they can bleed and/or cause large bowel diarrhoea
- composed of solid packets of pleomorphic plasma cells
- may contain amyloid deposits
- most are discrete tumours that are amenable to surgical excision
- a small proportion are locally invasive and may spread to regional lymph nodes and spleen

Mast Cell Tumour

- a **malignant tumour of intestinal mast cells**
- uncommon in aged **dogs** and seen occasionally **cats**
- arise in the mucosa of the stomach or, less often, the colon
- larger masses may ulcerate → haemorrhage
- composed of infiltrative sheets of mucosal mast cells (which contain few cytoplasmic granules)
- may metastasise to regional lymph nodes +/- liver and spleen

Carcinoid Tumour

- a **malignant tumour of intestinal mucosal enteroendocrine cells**
- rare
- most reported cases have been in older **dogs**, involving the duodenum, colon or rectum (rarely the stomach or distal small intestine)
- firm, lobulated, dark red to cream-coloured mass
- locally invasive and may metastasise to regional lymph nodes and liver

Intestinal Mesenchymal Tumours

- subtypes include benign and malignant smooth muscle tumours (**leiomyoma** and **leiomyosarcoma** respectively), benign and malignant tumours of fibrocytes/fibroblasts **(fibroma** and **fibrosarcoma** respectively) and **gastrointestinal stromal tumours** (derived from the pacemaker interstitial cells of Cajal)

- most often seen in **dogs**
- may be asymptomatic
- often appear as a well circumscribed, firm, cream-white mass bulging beneath an intact mucosa
- larger tumours and especially malignancies may → mucosal ulceration (+/- secondary bacterial infection with abscessation), perforation or obstruction
- malignant mesenchymal tumours are also locally invasive and may metastasise to at least the local lymph nodes (but early surgical excision can be curative)
- some GI stromal tumours of the large intestine of dogs have been reported to produce erythropoietin (→ erythrocytosis) or insulin-like growth factors (→ paraneoplastic hypoglycaemia)

FUNCTIONAL OBSTRUCTION

Paralytic (Adynamic) Ileus

- a **common** condition characterised by lack of normal smooth muscle tone and peristaltic movements of the intestines and/or stomach
- may be segmental or generalised

- often transient following abdominal surgery due to mild peritoneal irritation
- may also be caused by peritonitis, severe gastrointestinal pain, shock, toxaemia or electrolyte imbalances (e.g. hypocalcaemia, hypokalaemia), tetanus, heavy metal toxicity, renal failure etc.

- mediated by **sympathetic nerve reflexes** → continual tonic discharge by inhibitory neurons of the myenteric plexus → inhibition of contraction of circular smooth muscle → inhibition of peristalsis → functional intestinal obstruction (**pseudo-obstruction**) → flaccidity and distension of the affected segment by retained fluid, gas and ingesta
- clinical signs may include abdominal distension, anorexia, absence of bowel sounds and vomiting or reflux
- small intestinal ileus in horses may → gastric distension and eventual rupture

Some Other Causes of Functional Obstruction

Equine Dysautonomia (Grass Sickness)

- affects horses (especially 3-6 year-olds at pasture) in the UK and western Europe
- can cause repetitive bouts of colic over weeks to months but often acute disease with severe abdominal distension, skeletal muscle tremors, ptyalism (excessive salivation) and collapse due to gastric reflux and hypovolaemic shock
- may see large bowel impaction with dry ingesta, excess fluid in small intestine and stomach +/- gastric rupture
- high mortality rate
- neuronal degeneration (chromatolysis) and necrosis may be detectable in autonomic ganglia → decreased cholinergic responses → intestinal hypomotility
- aetiology uncertain but strong evidence implicates *Clostridium botulinum* type C neurotoxin

Feline Dysautonomia (Key-Gaskell Syndrome)

- unknown aetiology
- most cases reported in the UK and Europe
- neuronal chromatolysis and necrosis in multiple sympathetic and parasympathetic autonomic ganglia, ventral spinal grey matter, cranial nerve ganglia (III, V, VII and XII) and dorsal root ganglia
- variable clinical signs - e.g. depression, anorexia, constipation +/- megacolon, megaoesophagus (→ regurgitation +/- aspiration pneumonia), decreased lacrimation and salivation, urinary incontinence, pupillary dilation with delayed or absent pupillary light reflex, bradycardia etc.