DISEASES OF THE PERITONEUM

GENERAL CONSIDERATIONS

- the **peritoneum** = the smooth, glistening, serosal membrane (**tunica serosa**) that lines the abdominal and pelvic portions of the peritoneal cavity
- covers the entire surface of **intra-peritoneal viscera** (e.g. intestines) up to where the mesentery attaches to these organs
- with **retroperitoneal viscera** (e.g. kidneys and adrenal glands), the peritoneum covers only the organ surface that faces the peritoneal cavity
- the **ligaments**, **mesenteries** and **greater** and **lesser omentum** of the peritoneal cavity are composed of double serosal membranes

- the peritoneum constitutes a very large surface area that is semi-permeable to water and small solutes

- in health, the cavity contains just enough serous (watery) fluid to keep the membranes moist to reduce friction
- in health, peritoneal fluid is in osmotic equilibrium with circulating blood but does not contain fibrinogen or other high molecular weight proteins and therefore does not clot
- peritoneal fluid is normally dispersed by respiratory movements and intestinal peristalsis

Routes of Drainage of Peritoneal Fluid

- the volume of peritoneal fluid is kept in check in health via an equilibrium between transudation of fluid out of capillary beds (at the arteriolar end) and reabsorption of fluid (at the venular end of capillary beds and via lymphatic drainage)

- most peritoneal fluid drains via small stomata (pores) into diaphragmatic lymphatics that form a large **plexus in the muscular part of the right ventral diaphragm** → a pleural plexus on the opposite side of the diaphragm → **right lymphatic duct** (via sternal lymph nodes) or to the **thoracic duct** (via mediastinal nodes) → eventual return to the right side of the heart

- a small proportion of peritoneal fluid drains through the omentum and abdominal viscera via lymphatics and lymph nodes to the thoracic duct

Mesothelial Cells

- a monolayer of flattened, elongate (squamous) mesothelial cells with surface microvilli
- line the peritoneal cavity (and the pleural cavity and pericardial sac)
- mesodermal in origin
- rest upon a layer of connective tissue (lamina propria serosae)
- produce a polysaccharide that acts as a low viscosity lubricant
- capable of mitotic division when stimulated and can regenerate rapidly

Response of the Peritoneum to Injury

- following mild irritation, mesothelial cells commonly undergo **hypertrophy** (an increase in size) and **hyperplasia** (an increase in number due to mitotic division) and appear cuboidal or columnar rather than squamous

- activated mesothelial cells produce a **plasminogen activator** → activation of plasminogen to plasmin → lysis of fibrin (and therefore protection against adhesion formation within damaged serosal cavities)
- activated mesothelial cells are also capable of **phagocytosis** (akin to macrophages)

- however, mesothelial cells are also **fragile** and many of them slough following peritoneal injury
- sloughing of mesothelial cells and degranulation of subserosal mast cells → release of inflammatory mediators → increased vascular permeability → exudation of fibrin and chemotaxis of neutrophils into the peritoneal cavity
- loss of mesothelium → reduced fibrinolysis → persistence of fibrin → potential for development of permanent fibrous adhesions (bridging scar tissue) within the cavity

- in uncomplicated peritoneal wounds, debris must first be removed by phagocytes (especially macrophages derived from circulating monocytes but also reactive mesothelial cells)
- the wound will heal by growth of **granulation tissue** (new fibrovascular tissue that grows from the underlying connective tissues into the fibrin) and by **reconstitution of the mesothelial layer**
- mesothelial cells can be replaced by mitotic division of surviving cells at the margin of the lesion
- new mesothelial cells can also differentiate from the subserosal mesenchyme to recover the peritoneal surface from below
- **repair of the mesothelial layer is therefore not influenced by the size of the defect**

- both **fibroplasia** (proliferation of fibroblasts with subsequent collagen deposition) and **replacement of lost mesothelial cells** are well under way by 5-8 days after peritoneal injury
- if surface fibrin persists more than 3-4 days post-injury, granulation tissue bridges between the apposed viscera and between the viscera and the cavity wall → **fibrous adhesion formation**
- fibrous adhesions contract as collagen matures → tugging on tethered viscera → potential organ dysfunction
- however, post-inflammatory adhesions can also be beneficial in that they may permit walling off and localisation of inflammatory/infectious pockets (especially in cattle)

- **adhesion formation** - promoted by hypoxic injury, increasing severity of necrosis, sepsis (presence of infectious agents, especially bacteria) and presence of foreign material (e.g. sutures) (i.e. any factor that intensifies and/or protracts the inflammatory process)

POST MORTEM DECOMPOSITION

- after death, blood-stained watery (serosanguineous) fluid that does not clot accumulates in serosal cavities (peritoneum, pleura and pericardial sac)
- diffusion of haemoglobin from lysing red blood cells → red staining of the serosa and viscera
- diffusion of bilirubin from the gall bladder, bile ducts and/or duodenum → green or orange-yellow staining of the serosa and viscera
- sulfmethaemoglobin produced by interaction of haemoglobin and bacterial H_2S → blue-purple, grey or black discolouration of the serosa and viscera
- gas bubbles may develop subserosally over the viscera and in the adjacent mesenteries and ligaments

DISPLACEMENT AND HERNIATION OF VISCERA

- normal peritoneal structures can be responsible for persistent displacement of viscera
- e.g. the nephrosplenic ligament (that runs between the spleen and the left kidney) in horses can entrap the left dorsal and left ventral colon (**left dorsal displacement of the colon**)

Internal Herniation

- **internal peritoneal hernia = displacement of viscera (especially intestines) through a normal or abnormal hole (foramen) within the peritoneal cavity, without formation of a hernial sac**

- uncommon

Herniation through Natural Foramina

- mainly horses
- in **incarceration in the epiploic foramen**, part of the small intestine passes down into the omental bursa through the epiploic foramen → incarceration (entrapment) of the small intestine +/- rupture of the omental bursa

Omental and Mesenteric Hernias

- passage of intestine through tears in the greater or lesser omentum or mesentery
- the tears are usually traumatic in origin
- mesenteric tears most commonly involve the mesentery of the small intestine

Pelvic Hernia ("Gut-tie")

- young ruminants and rarely other species after castration
- excessive traction on the spermatic cord at castration → tearing of the peritoneal fold of the ductus deferens → herniation and incarceration of intestine between the ductus deferens and the lateral abdominal or pelvic wall

External Herniation

- external hernias typically consist of a **hernial sac** (formed as a pouch of parietal peritoneum +/- a covering of skin and soft tissues), a **hernial ring** (the opening in the abdominal wall) and the **hernial contents** (usually part of the omentum, a segment of intestine +/- other viscera)
- the hernial ring may be a natural opening in the abdominal wall (e.g. the vaginal ring at the inguinal canal) or an abnormal opening
- external hernias may be congenital (present at birth) or acquired (in post-natal life)

Ventral Hernia of the Abdominal Wall

- herniation through abdominal muscles into the subcutis or to the exterior
- e.g. inherited schistosomus reflexus (a complex malformation) in fetal calves
- e.g. spontaneous rupture of the abdominal wall in heavily pregnant females
- e.g. following blunt abdominal trauma, horn injuries or at sites of surgical scars or inflammation that weaken or perforate the abdominal muscles

- need to differentiate from ante mortem rupture of the pre-pubic tendon and, in bloated carcases, from post mortem rupture of the abdominal wall

Umbilical Hernia

- **common**, especially in pigs, foals, calves and puppies
- often congenital +/- inherited
- due to persistent patency of the umbilical ring
- incarceration of herniated intestines is uncommon

Inguinal Hernia

- the deep (internal) inguinal ring remains patent in intact male animals but the tendency for herniation of viscera through it may be inherited
- a large inguinal ring may promote an **indirect inguinal hernia** (in which viscera herniate into the inguinal canal of the groin and lie between the internal inguinal ring and the external vaginal ring)
- in males, an indirect inguinal hernia may evolve into a **scrotal hernia**, with the herniated viscera passing through both the inguinal and vaginal rings to eventually lie within the tunica vaginalis of the scrotum
- may be congenital or acquired

- less common is a **direct** (or **false**) **inguinal hernia** in which herniated viscera pass **subcutaneously, outside the inguinal canal**
- especially horses
- may result from rupture of an originally indirect hernia or from rupture of the peritoneum and transverse fascia overlying the deep inguinal ring
- direct inguinal hernias are more serious than indirect ones as the herniated tissues are more likely to become fixed by adhesions and are more prone to strangulation (compromised blood supply/venous drainage → venous infarction)

Femoral Hernia

- develops as an outpouching of peritoneum through the femoral triangle
- contains omentum +/- small intestine

Perineal Hernia

- mainly **older male dogs** that are straining to urinate or defaecate (e.g. due to prostatic enlargement or severe constipation)
- usually unilateral but can be bilateral
- retroperitoneal pelvic fat bulges through a defect between the coccygeus medialis muscle and the anterior border of the anal sphincter
- may cause deviation of the rectum and caudal displacement of the prostate and urinary bladder into the pelvic cavity
- in some cases, the prostate and urinary bladder may be forced through ruptured perineal fascia

Diaphragmatic Hernia

- **common**
- more commonly **acquired** than congenital
- usually results from increased intra-abdominal pressure
- rarely causes strangulation of herniated gut

Congenital Pleuroperitoneal Diaphragmatic Hernia

- very rare
- most often seen in dogs (in which it can be inherited)
- usually associated with a defect in the muscle of the left dorsal diaphragm → herniation of a large mass of abdominal viscera into the thorax
- most small animals with this defect die perinatally but some large animals may survive and may have dyspnoea (difficult breathing) or abdominal pain

Congenital Peritoneopericardial Diaphragmatic Hernia

- usually triangular ventral defects in the diaphragm
- can be associated with cardiac anomalies, umbilical hernias or malformations of the sternum and costochondral junctions
- part of the liver, spleen, omentum and small intestine may herniate into the pericardial sac
- surprisingly, often clinically silent

Acquired Pleuroperitoneal Diaphragmatic Hernia

- the **most common type** of diaphragmatic hernia and usually caused by **external abdominal trauma**
- in **small animals**, the tear is usually in the muscular part of the diaphragm → herniation of especially liver and small intestine into the thorax
- surprisingly, may remain clinically silent (sometimes for years)
- in **horses**, the tear usually involves the tendinous part of the diaphragm → herniation of viscera into the thorax → usually abdominal pain rather than dyspnoea

ABDOMINAL TRAUMA

- **common** in domestic animals
- introduction of bacteria from the exterior or perforation of the gastrointestinal tract → **septic peritonitis**
- **blunt abdominal trauma** → bruising of viscera, rupture of hollow organs (e.g. stomach, urinary bladder, gall bladder) → septic or sterile peritonitis, avulsion of organs, internal or external herniation of viscera ± laceration of the capsule of solid organs (e.g. liver, spleen, kidneys)
- ± leakage of bile or urine into the peritoneal cavity
- +/- uterine rupture in pregnant animals
- pieces of spleen may implant ectopically and persist ("splenic satellites" or "daughter spleens")
- in massive trauma, the liver or spleen may be pulped → potentially fatal haemorrhage into the peritoneal cavity (**haemoperitoneum**)

ABNORMAL PERITONEAL CONTENTS

- abnormal peritoneal contents include foreign bodies, gut contents (ingesta), parasites, blood, urine, bile, excess non-inflammatory fluid (ascites) and inflammatory exudate (in peritonitis)

Foreign Bodies

- uncommon
- usually parasites or foreign bodies entering via penetrating wounds

Ingesta

- **common in horses and cattle**; uncommon to rare in other species
- perforation of the gastrointestinal tract may result from intestinal impaction, torsion, volvulus, strangulation, intussusception, ulceration, heavy parasitism, trauma etc.
- usually easy to find the perforation/rupture site in the acute phase but may be difficult once peritonitis and adhesions develop

- therefore, need to check **predilection sites for perforation**:

  - any devitalised segments of intestine
  - segments of impacted or tympanic bowel
  - stomach, caecum and rectum in horses
  - abomasum in cows

- **differential diagnosis - post mortem rupture of viscera** - ingesta tends to be localised close to the rupture site, without haemorrhage at the margins of the tear and without evidence of peritonitis

Parasites

- most parasites in the peritoneal cavity of animals are migrating to another site or are in an aberrant location
- irritation and inflammation of the peritoneum during transit → residual lesions of fibrosis
- e.g. **immature liver fluke** - can cause peritonitis in sheep and cattle
- e.g. ***Stephanurus dentatus*** (a nematode that typically migrates in pigs from the liver to the kidneys)
- e.g. ***Strongylus edentatus*** **and** ***S. equinus*** (large strongyle nematode larvae) - may cause fibrous tag formation over the hepatic capsule and diaphragm in horses
- e.g. ***Dioctophyma renale*** (giant kidney worm) - dogs

- the peritoneal cavity can be the normal habitat for some parasites
- e.g. **cysticercus -** the intermediate (metacestode) stage of taeniid tapeworms
- appears as a fluid-filled, thin-walled cyst into which the head and neck of a solitary tapeworm larva is invaginated
- e.g. cysticerci of the long-necked bladder tapeworm, *Taenia hydatigena* (the adult tapeworm is in the intestinal tract of dogs)
- especially common in the peritoneal cavity of sheep but also seen in cattle, pigs and occasionally other species
- usually non-pathogenic

Haemoperitoneum

- **haemoperitoneum = blood in the peritoneal cavity**

- may be unclotted or partially or completely clotted
- may or may not be fatal (depends on volume and rate of haemorrhage)

- extravasated blood can be rapidly removed via diaphragmatic lymphatics and returned to the circulation

- **causes of haemoperitoneum** include:

  - **trauma** - especially to the liver, spleen or kidneys - **the most common cause in small animals**
  - a bleeding splenic or hepatic haemangiosarcoma (a malignant neoplasm of vascular endoethelium) - especially dogs
  - rupture of any friable intra-abdominal or retroperitoneal neoplasm
  - spontaneous rupture of an enlarged spleen or liver
  - rodenticide anticoagulant poisoning and other coagulopathies
  - manual ablation of the corpus luteum in cows
  - laceration of the uterus or rupture of a uterine artery at parturition in cows and mares

Ascites (Hydroperitoneum)

- **ascites = accumulation of excess non-inflammatory fluid (transudate or modified transudate) in the peritoneal cavity**

- the fluid is usually distributed diffusely throughout the cavity (c.f. inflammatory exudate in peritonitis can be localised)
- usually watery, colourless to yellow or lightly blood-stained fluid that contains exfoliated **mesothelial cells** and **small numbers of leukocytes** (nucleated cell counts < 7 x 10^9/L)
- serosal membrane usually remains smooth and glistening
- if the ascites is chronic (of several weeks' duration), the serosa may appear cloudy/milky due to subserosal collagen deposition
- excess fluid → pressure on abdominal viscera +/- interference with diaphragmatic and lung movement during respiration

Reduced Removal of Peritoneal Fluid

- due to **obstruction of lymphatic drainage**
- e.g. peritoneal carcinomatosis – carcinomas (malignant epithelial neoplasms) arising within the peritoneal cavity tend to implant most extensively on the serosa over the right ventral diaphragm in the area of the lymphatic stomata
- obstruction of lymphatic flow cranial to the diaphragm may also cause ascites, e.g. neoplasia involving the cranial mediastinal and sternal lymph nodes

Overproduction of Peritoneal Fluid

- ascites will only result if the normally high lymphatic drainage capacity of the peritoneal cavity is exceeded by the rate of fluid production

- ascites may result from **increased permeability of blood vessels** within the peritoneal cavity
- e.g. clostridial intoxications, endotoxaemias, acute renal failure in pigs and ruminants, and vitamin E deficiency in pigs

- ascites may result from **hypoalbuminaemia**
- albumin is the most important circulating protein responsible for osmotic (oncotic) retention of water in the bloodstream
- serum [albumin] < 10-15 g/L → decreased plasma osmotic/oncotic pressure → inability to retain

water in circulation → transudation into body cavities, subcutis and other interstitial tissues

- ascites may result from **increased hydrostatic pressure within veins, venules and capillaries** within the cavity → water is forced out of circulation
- e.g. cirrhosis → portal hypertension (increased pressure in the portal vein) → increased pressure in subsidiary veins and capillary beds of the splanchnic viscera and mesenteries) → ascites
- e.g. right-sided congestive heart failure → hepatic congestion → increased pressure in the hepatic sinusoids → increased hepatic lymph formation → ascites (with the excess fluid pouring out of the liver)

PERITONITIS

- **peritonitis = inflammation of the peritoneum, with accumulation of exudate in the cavity**

- the inflammatory fluid (exudate) that accumulates in the peritoneal cavity typically has **a high protein concentration and nucleated cell count** because of increased vascular permeability and chemotaxis of leukocytes

- peritonitis is **common in large animals**, less so in small animals

- **primary peritonitis** usually results from **haematogenous localisation of infectious agents** to the cavity
- most cases of peritonitis arise **secondary to extension of an inflammatory or necrotising process involving an intra-peritoneal organ**

Chemical Peritonitis

- e.g. intraperitoneal injections of therapeutics, including antibiotics
- e.g. talc and starch granules from surgical gloves → mild granulomatous peritonitis
- e.g. barium sulphate → potentially fatal haemorrhagic peritonitis
- e.g. urine (uroperitoneum)
- e.g. chylous ascites (due to leakage of chyle from a ruptured cysterna chyli or other lymphatic channel) → mild granulomatous peritonitis
- e.g. bile peritonitis following rupture of the extra-hepatic biliary tree (may evolve into a septic peritonitis due to the detergent action of bile salts)
- e.g. pancreatic necrosis → local release of activated pancreatic digestive enzymes → enzymatic necrosis of peripancreatic fat

Uroperitoneum

- urine may accumulate in the peritoneal cavity due to rupture of the urinary tract at the level of the kidneys, ureters, bladder or urethra
- depending on the cause, see progressive signs of chemical or bacterial peritonitis within 8-24 hours of rupture → death within several days if untreated, due to retention of toxic waste products
- depending on the cause of rupture, the peritoneal fluid may be a transudate, modified transudate or septic or non-septic exudate
- the rupture site may be small and difficult to identify

Bacterial Peritonitis

- potential **routes of entry of bacteria** to the cavity include:

  - **perforation of the gastrointestinal tract** (the **most common cause** in domestic animals)
  - penetrating wounds of the abdominal wall with introduction of bacteria from the external environment
  - rupture of infected segments of the extra-hepatic biliary tree, uterus or urinary bladder
  - seepage through devitalised gastrointestinal or uterine segments prior to rupture
  - proximally through the oviducts in salpingitis (inflammation of the oviducts)
  - haematogenously
  - as an extension from an infected intra-peritoneal organ
  - as an extension from an infected umbilicus in neonatal animals.

Viral Peritonitis

- e.g. **feline infectious peritonitis (FIP)** caused by a mutant or recombinant strain of feline enteric coronavirus
- FIP virus causes a virulent systemic infection that is usually fatal over weeks to months
- viral antigen-antibody complexes → phagocytosis by macrophages → deposition in walls of blood vessels → fixation of complement + chemotaxis of neutrophils → vasculitis + perivascular pyogranuloma formation → increased vascular permeability → peritoneal +/- pleural effusion, with a typically high protein concentration

Consequences of Peritonitis

- in the first few hours of generalised peritonitis, may see intestinal hypermotility
- ultimately see **paralytic ileus** (paralysis of gastrointestinal motility), mediated by the autonomic nervous system
- ileus is advantageous in that it limits distribution of peritoneal exudate
- however, ileus allows fibrinous exudate to mat intestinal loops together → increased risk of permanent fibrous adhesions
- gas accumulates and fluids and electrolytes become sequestered within the hypomotile intestines

- absorption of bacterial endotoxins or exotoxins from the peritoneal cavity into the circulation → increased vascular permeability, shock, death from toxaemia etc.
- infectious agents may also spread from the peritoneal cavity via lymphatics → pleuritis or bacteraemia/septicaemia

- if treated early and appropriately, generalised peritonitis may:

  - resolve completely with no residual scarring
  - resolve with residual fine or coarse adhesions
  - evolve into chronic active localised peritonitis with adhesions; the localised foci may subsequently resolve or act as a source of new infection

- in **horses**, the omentum is small with limited capacity to wall off contaminated areas
- peritonitis in horses most commonly results from perforation or rupture of the stomach or

intestine, and is usually acute, generalised and fatal

- in **cattle**, the omentum may wall off septic foci within the cavity; the foci may evolve into abscesses that most commonly contain *Trueperella (Arcanobacterium) pyogenes* (a pyogenic or pus-inducing bacterium)
- e.g. localised fibrinosuppurative to suppurative peritonitis arising from **hardware disease** (traumatic reticuloperitonitis)

INTRA- AND RETROPERITONEAL FAT NECROSIS

- necrotic fat appears a brigher white than normal fat
- often firmer than normal due to secondary inflammation and fibrosis
- often gritty due to dystrophic mineralisation
- when necrosis is of recent onset (acute to subacute phase), see a red margin due to inflammatory hyperaemia (active inflammation → vasodilation of arterioles → increased blood flow into affected capillary beds)

Enzymatic Fat Necrosis

- due to release of proteolytic and lipolytic enzymes from exocrine acinar cells in pancreatic necrosis
- may only involve peripancreatic fat or may be widely scattered throughout the peritoneal cavity +/- pleural cavity

Focal or Multifocal Necrosis

- may be numerous small and widespread lesions or just localised
- common in **sheep** and sometimes seen in other species
- especially very fat animals
- pathogenesis unknown; may be triggered by local hypoxia of fat (e.g. due to pressure on large fat pads by adjacent viscera) or may be initiated by intracellular abnormalities in lipolysis during fat mobilisation

"Yellow Fat Disease" (Nutritional Steatitis)

- occurs in many species but not in ruminants
- may involve adipose tissue anywhere in the body
- caused by diets rich in polyunsaturated fats and low in vitamin E (anti-oxidant) → failure to scavenge reactive oxygen species (free radicals) → peroxidation of cell membrane phospholipids → cell degeneration/necrosis → secondary inflammation and fibrosis
- affected fat may appears grossly yellow due to accumulation of ceroid lipofuscin pigment
- in **cats**, the condition is usually recognised clinically as "nutritional panniculitis" involving the subcutaneous inguinal and ventral abdominal fat pads but can involve peritoneal and retroperitoneal fat stores

Massive Fat Necrosis in Cattle

- especially **Channel Island breeds** (Jerseys and Guernseys) and especially **fat cattle**
- often an incidental finding but can be fatal, e.g. if the intestines or a ureter is obstructed
- firm masses of necrotic fat can be mistaken for a fetus, neoplasm or other mass on rectal

palpation
- may involve omental, mesenteric or retroperitoneal fat and may sometimes also involve intermuscular or subcutaneous fat stores
- small nodules to large solid masses
- **pathogenesis** - uncertain; possibly involves production in the rumen of high concentrations of saturated fatty acids that form long chain compounds that are solid at normal body temperature
- in deer, similar lesions have been associated with ingestion of endophyte-infested tall fescue pastures

PERITONEAL NEOPLASIA

Lipoma

- the **most common tumour of the peritoneal connective tissues**
- in **horses**, usually arises from the mesenteries and may reach enormous size
- in horses, tends to be pedunculated and may → acute intestinal strangulation
- in **dogs**, tends to arise in the omentum and settle on the abdominal floor; usually not pedunculated

Mesothelioma

- arises from the serosal mesothelium of the peritoneum, pleura or pericardium and may involve all three sites or be restricted to one cavity
- virtually all are **malignant** but tend to implant rather than metastasise
- most often diagnosed in cattle and dogs but rare occurrence
- may be present congenitally or develop early in life in calves
- peritoneal mesotheliomas usually cause ascites
- in humans, mesotheliomas are associated with exposure to asbestos fibres; an association with asbestos exposure has been documented in only a minority of canine cases
- typically appear as multiple, firm nodules or as villous projections from a thickened serosa; can grossly mimic peritoneal carcinomatosis
- a few mesotheliomas appear grossly as firm, fibrous plaques that may mimic chronic granulomatous (macrophage-rich) peritonitis (e.g. tuberculosis in cattle)

Secondary Neoplasms

- mainly arise by direct implantation of malignant cells on the serosal membrane rather than by metastasis via lymphatics or blood vessels
- **carcinomas (malignant epithelial tumours) more often implant on serosal membranes than do sarcomas (malignant mesenchymal tumours)**
- often referred to as **peritoneal carcinomatosis**
- secondary carcinomas may be very scirrhous (firm due to host fibrous tissue deposition in and around them) and, if accompanied by ascites, may resemble chronic peritonitis but there is **usually a relative or complete absence of adhesions**
- e.g. carcinomas arising from the ovaries, bile ducts, exocrine pancreas, stomach, intestines, urinary bladder or prostate

THE RETROPERITONEUM

- **retroperitoneum = the loose fibrofatty connective tissue immediately external to the peritoneal cavity**

- the largest area of the retroperitoneum is dorsal and extends from the diaphragm to the anus
- the retroperitoneum communicates cranially with the mediastinum and retropleura
- the retroperitoneum communicates ventrally with the potential space between the double serosal layers of the mesenteries, omentum and peritoneal ligaments
- there is no barrier to fluid movement within the retroperitoneal space, at least in dogs

Retroperitonitis

- may arise from penetrating wounds, migrating grass awns or other foreign bodies or from bacterial infections involving the abdominal or pelvic viscera or the mesenteric root
- suppurative retroperitonitis in dogs may evolve into a **fluctuant abscess or draining fistula in the flank** by following fascial planes
- retroperitonitis may extend to involve vertebrae (as periostitis or osteomyelitis)
- usually involves mixed bacterial flora

Retroperitoneal Masses and Neoplasms

- e.g. haematomas (palpable masses of clotted blood) or pockets of urine following trauma to the caudal abdomen or pelvis
- retroperitoneal haemorrhage in neonatal calves may be an important clue to vertebral fracture during assisted calving
- **primary neoplasms** - e.g. lipoma, fibrosarcoma, lymphoma, osteosarcoma, renal carcinoma, adrenal tumours
- **metastatic neoplasms** - e.g. carcinomas arising in the urinary bladder, prostate, colon/rectum, anal glands or perianal glands may metastasise to the retroperitoneal sublumbar lymph nodes