miniVET guide

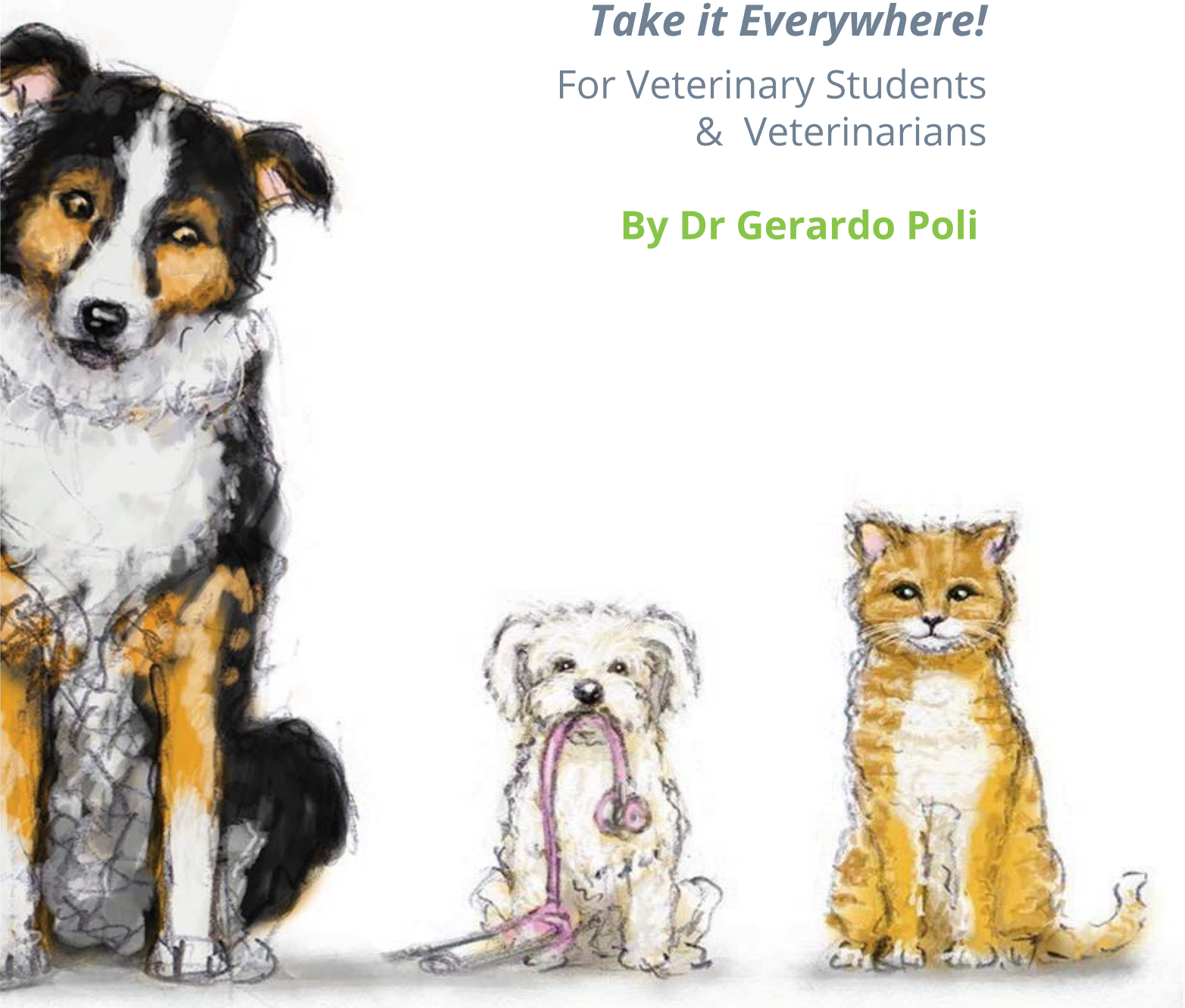
COMPANION ANIMAL MEDICINE

Portable and Concise ***VET*** Guide

Take it Everywhere!

For Veterinary Students
& Veterinarians

By Dr Gerardo Poli



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Anaemia and Pale Mucous Membranes

- **This chapter covers:**
  - ✓ Determining the severity of the anaemia
  - ✓ Assessing for regenerative response
  - ✓ Diagnostic pathway to **"Pale Mucous Membranes"**

- **Degree of anaemia:**
- Refer to **"Pale Mucous Membranes"** next page for diagnostic pathway

	Packed cell volume / Haematocrit:		
	Mild:	**Moderate:**	**Severe:**
Dogs:	30-35%	20-30%	<20%
Cats:	20-25%	20-15%	<15%

- **Reticulocytes:**
  - ✓ Immature non-nucleated red blood cells
  - ✓ Appear larger and lack central pallor
  - ✓ Characterised as macrocytic (high MCV), hypochromasia (low MCHC) and polychromasia (variation in colour between cells)
  - ✓ Quantify using "Corrected Reticulocyte Percentage" formula:

Observed reticulocyte % x (patient's HCT % / "normal" HCT %) = "Corrected reticulocyte %"

("Normal HCT"= 45% in dogs, 40% in cats)

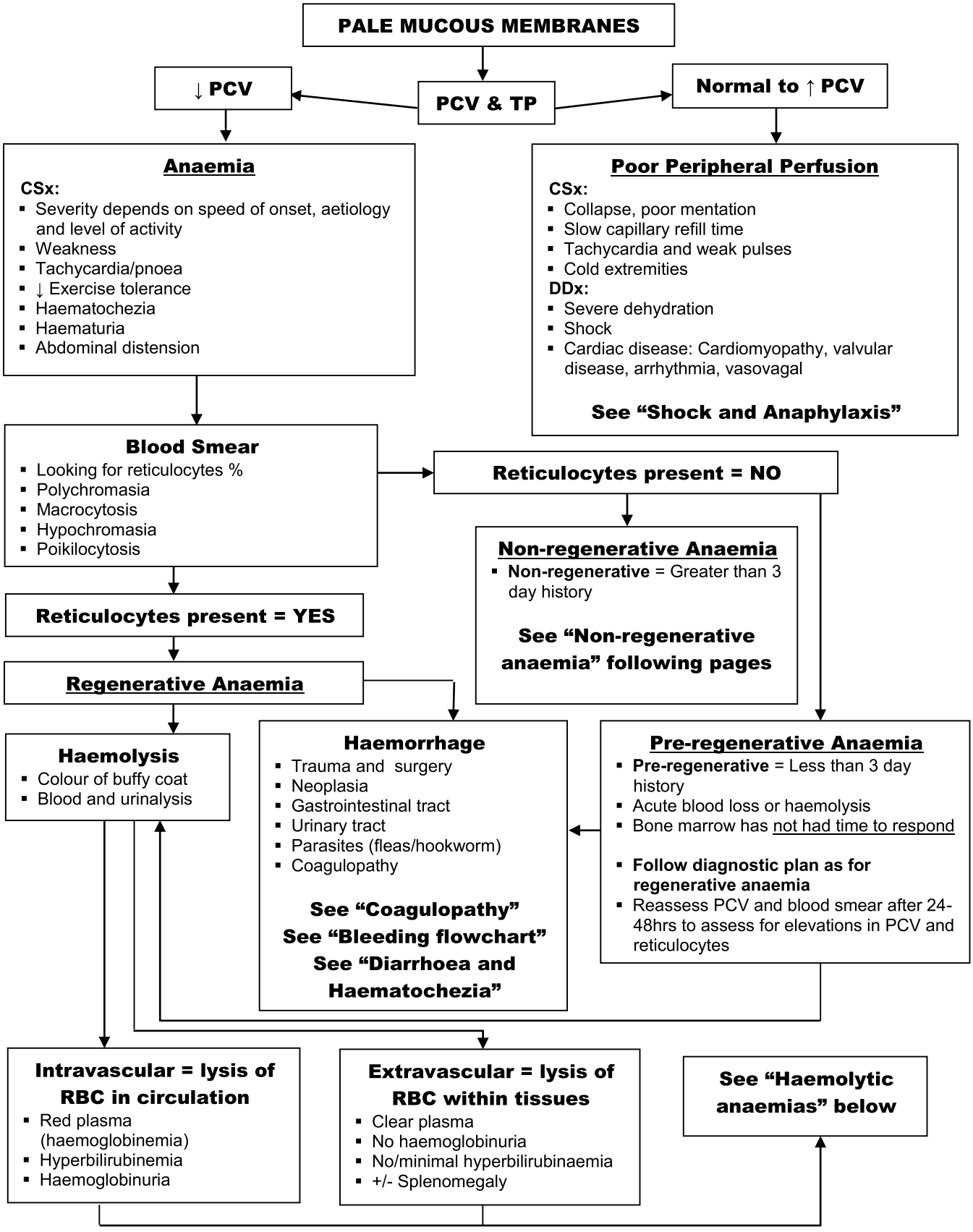
- **Regenerative anaemia:**
  - ✓ Increased number of reticulocytes in peripheral circulation
  - ✓ Bone marrow response takes 3 to 4 days, if no reticulocytes it could be **pre-regenerative**, assess history to help determine time frame
  - ✓ Cats: Two types of reticulocytes:
    - ➢ Aggregate type: Only count this type when assessing response to anaemia
    - ➢ Punctate type: Present in healthy cats, increased numbers if regenerative response been for up to 3 to 4 weeks

Degree:	**Dogs:**	**Cats:**
Mild:	1.5 – 4%	0.5 - 2%
Moderate:	5 – 20%	3 – 4 %
Marked:	>20%	> 4%
	% = number of reticulocytes per 100 RBC	

- **Refer to "Haematology", "Transfusion Therapy" and "Coagulopathy" for more information**

History:

- ✓ Assess history for duration of clinical signs
- ✓ Trauma, bleeding (faecal, urinary, integument, respiratory, abdominal, cardiac)
- ✓ Access to rodenticide, snakes and other anticoagulants
- ✓ Prior health issues eg. renal disease, tumours, viral infections (FIV/FeLV)
- ✓ Recent administration or access to medications



Anaemia and Pale Mucous Membranes

Haemolytic anaemias:

- **Immune mediated haemolytic anaemia (IMHA):**
  - ✓ See below under **"Specific conditions"**
- **Drugs/Toxins:**
  - ✓ Bacterial toxins, rodenticide, snake bite (+/- clinical signs of lower motor neuron paresis/paralysis, or haemoglobinuria)
- **Oxidative injury:**
  - ✓ Heinz body formation and eccentrocytes
  - ✓ Onion/garlic ingestion, acetaminophen, heavy metal
- **Haemolytic transfusion reactions:**
  - ✓ Donor RBC's are lysed by host alloantibodies
  - ✓ Immediate or delayed (1 to 2 weeks), see **"Transfusion Therapy"** for more information
- **Microangiopathic anaemia:**
  - ✓ Physical destruction of RBC as they pass through disorganised blood vessels (e.g. tumour):
    - ➢ Haemangiosarcoma, DIC, haemolytic uraemic syndrome, heartworm disease
  - ✓ Schistocytes formation
- **Infectious haemolytic anaemia:**
  - ✓ Direct infection and damage to RBC's by infectious organisms eg. *Mycoplasma, Babesia*, *Leptospria*, or viruses FeLV, FIP, FIV, *Ehrilichia canis*, *Bartonellosis, Cytauxzoon felis*
  - ✓ Indirect damage to RBC's via antibodies directed against infectious organism
  - ✓ See below under **"Specific conditions"**
- **Neonatal isoerythrolysis:**
  - ✓ Neonate RBC lysed by dam antibodies, can be absorbed from colostrum
  - ✓ In cat it can be naturally occurring, dogs require sensitization

- **Diagnostic tests:**
  - ✓ Biochemistry, haematology and blood smear:
    - ➢ IMHA: Spherocytes
    - ➢ Microangiopathic anaemia: Schistocytes
    - ➢ Infectious haemolytic anaemia: *Babesia*, *Mycoplasma haemofelis*
    - ➢ Oxidative damage: Heinz bodies and eccentrocytes
  - ✓ Slide agglutination:
    - ➢ IMHA: Agglutination
  - ✓ Coomb's test:
    - ➢ IMHA, neonatal isoerythrolysis, haemolytic transfusion reactions
  - ✓ Blood typing or cross matching:
    - ➢ Between dam and puppy, donor and recipient
    - ➢ Neonatal Isoerythrolysis, haemolytic transfusion reactions
  - ✓ Ultrasound and radiography:
    - ➢ Microangiopathic anaemia (neoplasia), IMHA (neoplasia)
  - ✓ Blood culture and sensitivity:
    - ➢ Infectious haemolytic anaemia
  - ✓ PCR/Serology:
    - ➢ *Mycoplasma haemofelis, Babesia, Cytauxzoon felis (USA)*
  - ✓ Snake venom detection tests

Non-regenerative Anaemia

- **Features:**
  - ✓ Can appear non-regenerative if blood loss or haemolysis has only recently occurred ie. <48-72 hours
  - ✓ Non-regenerative anaemia are not as common in dogs as they are in cats
  - ✓ Typically chronic process with no clinical signs of anaemia due to compensation

>48-72 hours = Non-regenerative:

- Bone marrow has not responded

Either:

Bone marrow pathology OR

Non-bone marrow pathology

Haematology and blood smear:

- Assess RBC features:
  - Typically normocytic/normochromic
  - If microcytic/hypochromic – iron deficiency
  - If macrocytic - could be **FIV, FeLV**
- Assess WBC features:
  - If pancytopenia can primary bone marrow pathology or toxicities or infections affecting bone marrow

Biochemisty:

- Assess for non-bone marrow pathology

Bone marrow biopsy:

- Assess for bone marrow pathology
- Presence of abnormal cells
- Reduction of cells lines
- Can see pancytopenia

Non-bone marrow pathology:

Anaemia of chronic disease:

- Secondary to prolonged inflammation, infection, neoplasia, liver disease

Chronic renal disease:

- Reduce EPO production and uremic damage to RBC

Hypothyroidism/Hypoadrenocorticism:

- Reduced stimulation of EPO production

Iron deficiency:

- Typically microcytic and hypochromic
- Fleas, hookworms

Toxicity:

- Can see pancytopenia
- Drugs/metals:
  - Chemotherapy, phenobarbitone, methimazole, lead, chloramphenicol, trimethoprim-sulfa
- Hormones:
  - Oestrogen toxicity or sertoli cell tumour

Infections:

- Can see pancytopenia
- Viral (FIV and FeLV), can be macrocytic
- Parasitic (*Babesia, Leishmania*)
- Bacteria (*Mycoplasma, Ehrlichiosis*)

Immune mediated haemolytic anaemia:

- Immune destruction of RBC precursors in bone marrow

Bone marrow pathology:

Red blood cell aplasia:

- Destruction of only RBC precursors
- Secondary to idiopathic, immune, drugs and toxins

Aplastic anaemia:

- All cell line precursors are reduced = pancytopenia
- Idiopathic or secondary to immune, drugs and toxins, parvovirus, FeLV

Bone marrow necrosis/fibrosis:

- Precursor cells are destroyed

Myelodysplasia:

- Defective precursor cells leads to abnormal maturation or cellular morphology
- Idiopathic or secondary to FIV, FeLV

Bone marrow tumour:

- Precursor cells destroyed by neoplastic cells
- Primary: See large numbers of immature cells of the same cell line
- Metastatic: See cell types not normally seen in bone marrow

Specific conditions:

- **Immune mediated haemolytic anaemia:**
- **Pathophysiology:**
  - ✓ Immune response against RBC antigens, due to a breakdown in immunotolerance to own RBC antigens
  - ✓ IgG and IgM and complement binding
  - ✓ Observe autoagglutination, seen as red blood cells clustered like bundles of grapes
  - ✓ Can cause intravascular or extravascular haemolysis, with extravascular haemolysis more frequent. The spleen is the main site for extravascular haemolysis.
  - ✓ Intravascular haemolysis caused by complement binding
  - ✓ Cats: Overall IMHA is rare, but more commonly secondary compared to primary
- **Causes:**
  - ✓ Primary:
    - ➢ Dogs more commonly primary compared to secondary
    - ➢ Idiopathic (70% of cases in dogs)
  - ✓ Secondary (triggered by cross-reaction with foreign antigens):
    - ➢ Cats more commonly secondary compared to primary – must assess for feline viral diseases and blood borne parasites
    - ➢ Drugs, neoplasia, infections (any infection, perform feline virus testing), snake envonmation and other immune mediated diseases
- **Clinical signs:**
  - ✓ Pyrexia, anaemia, icterus (rarely seen in cats), weakness, tachycardia/pnoea, splenomegaly, respiratory distress
- **Diagnostics:**
  - ✓ Saline agglutination test:
    - ➢ Must confirm agglutination (grapes) and rule out rouleaux (stack of coins)
    - ➢ To assess for IgM and IgG antibodies
  - ✓ Haematology and blood smear:
    - ➢ Spherocytes and autoagglutination, polychromasia, neutrophilia, high MCV (regeneration)
    - ➢ If decreased thrombocytes, consider Evan's syndrome
    - ➢ Feline RBCs are smaller and lack central pallor complicating the assessment of spherocytes
  - ✓ Hyperbilirubinaemia, high ALT
  - ✓ Perform Coombs test if no agglutination, Coombs test detects antibiodies and complement on the surface of erythrocytes
  - ✓ Full medical workup to assess for underlying cause, include feline virus testing and blood parasites
- **Treatment:**
  - ✓ Primary IMHA is more difficult and takes longer to treat compared to secondary IMHA
  - ✓ Blood transfusion if acute reduction in PCV <20 or chronic drop <15 or transfusion triggers, see **"Transfusion Therapy"**
  - ✓ Immunosuppressive agents:
    - ➢ Dexamethasone 0.5mg/kg SC, then 12 hours later start prednisolone 2mg/kg/day PO
    - ➢ +/- Azathioprine 2mg/kg PO SID until remission then 0.5mg/kg PO EOD, monitor for bone marrow suppression and hepatoxicity, also very toxic in cats
    - ➢ +/- Cyclosporin 5-10mg/kg PO divided BID
  - ✓ Gastric protectants:
    - ➢ H_2 antagonist, proton pump inhibitors and sulcralfate 0.5-1gm PO TID
  - ✓ Anti-thrombotic agents:
    - ➢ Thromboembolic disease is a common complication
    - ➢ Aspirin 0.5mg/kg PO SID **or** dalteparin100IU/kg SC TID
    - ➢ Clopidogrel 20mg/cat PO SID, dogs: 2-4mg/kg PO SID

- **Monitoring:**
  - ✓ Serial haematology panels, PCV and blood smears, ideally daily until PCV is stable (>23-30%) and no spherocytes
  - ✓ Repeat diagnostics at least weekly until anaemia resolves
  - ✓ Once stable for a couple weeks, then consider tapering of the immunosuppressive medications with weekly monitoring.
  - ✓ Reduce prednisolone dose by 25% every 2 – 4 weeks if the PCV remains stable
  - ✓ If PCV is still dropping or if spherocytes are still present after six weeks despite stable PCV keep the same prednisolone dose and add in another agent, and continue monitoring as above:
    - +/- Cyclosporin 5-10mg/kg PO divided BID
    - +/- Azathioprine 2mg/kg PO SID until remission then 0.5mg/kg PO EOD, monitor for bone marrow suppression and hepatoxicity, also very toxic in cats
    - When PCV and blood smears are normal/stable, reduce doses and frequency of the second agent then the prednisolone after

- **<u>Haemotropic mycoplasmas:</u>**
- **Pathophysiology:**
  - ✓ Epierythrocytic parasite:
    - Dogs: *Mycoplasma haemocanis* (Europe) main species, transmitted by brown dog tick *Rhipicephalus sanguineus*. Splenectomized or immunocompromised dogs are greater risk of developing severe anaemias
    - Cats: *Mycoplasma haemofelis* main species that causes clinical signs. Transmitted most commonly by fleas, but also by fights. Developing of clinical signs is usually due to concurrent illness or immunocompromised
  - ✓ Leads to destruction of red blood cells by the immune system, leading to extravascular haemolysis in typically in the spleen
  - ✓ Can have a carrier state where non-clinical infections can occur and cause transient parasiteaemia
  - ✓ Reoccurrences are common
- **Clinical signs:**
  - ✓ Pyrexia, anaemia, icterus, weakness, tachycardia/pnoea, splenomegaly
- **Diagnostics:**
  - ✓ Haematology and blood smear:
    - Regenerative anaemia with reticulocytes, spherocytes, polychromasia
    - +/- Parasites on RBC surface – cocci or rod shaped, single or chains around the surface
  - ✓ Biochemistry:
    - +/- Icterus
  - ✓ PCR at local laboratory
  - ✓ Other: Feline virus testing due to high risk of concurrent viral infection
- **Treatment:**
  - ✓ Doxycycline 5-10mg/kg PO SID or enrofloxacin 5mg/kg PO SID for 3 weeks
  - ✓ Corticosteroids at immunosuppressive doses:
    - Dexamethasone 0.5mg/kg SC/IV, then 12 hours later start prednisolone 2mg/kg/day PO divided
  - ✓ Blood transfusion if becomes anaemic

- **<u>Babesiosis:</u>**
- **Pathophysiology:**
  - ✓ Intraerythrocytic protozoal parasite spread primarily by ticks but also by transplacental transmission and blood transfusions
  - ✓ Severel different species worldwide, the most wide spread is *Babesia canis* and *Babesia gibsoni*
  - ✓ Leads to immune mediated haemolytic anaemia and thromobocytopenia
  - ✓ Can have a carrier state where non-clinical infections can occur and cause transient parasiteaemia
  - ✓ Reoccurrences are common

- **Clinical signs:**
  - ✓ Lethargy, weakness, pyrexia, anaemia, icterus, splenomegaly, tachycardia/pnoea
- **Diagnostics:**
  - ✓ Haematology and blood smear:
    - ➢ Regenerative anaemia with reticulocytes, spherocytes, polychromasia
    - ➢ Thrombocytopenia – can present primarily for severe thromobocytopenia without anaemia
    - ➢ +/- Microorganisms in RBC's on capillary blood smear analysis
  - ✓ Biochemistry:
    - ➢ +/- Icterus
  - ✓ Serology: Babesia antibody titres
  - ✓ PCR assay: Can identify species
- **Treatment:**
  - ✓ Full clearance of the organism and prevention of relapse requires combination therapy
  - ✓ *Babesia canis:*
    - ➢ Imidocarb dipropionate 6.6mg/kg IM, repeat in 2 weeks **and** Diminazene aceturate 4-7mg/kg IM once
  - ✓ *Babesia gibsoni:*
    - ➢ Imidocarb dipropionate 6.6mg/kg IM, repeat in 2 weeks **and** Diminazene aceturate 4-7mg/kg IM once **and** clindamycin 15-25mg/kg PO BID for 2 weeks
    - ➢ Alternative regime
      - Atovaquone (Mepron) 13.5 mg/kg PO TID (with fatty meal) **and** Azithromycin 10 mg/kg PO SID for 10 days
  - ✓ Immunosuppressive therapy:
    - ➢ Not indicated initially, can be used if not responding to anti-protozoal therapy and there is continuing destruction of red blood cells and platelets
    - ➢ Prednisolone 2mg/kg/day PO divided and tapering
  - ✓ Blood transfusion if becomes anaemic

Biochemistry

- **This chapter covers:**
  - ✓ The differentials for increases and decreases seen in a biochemistry panel
  - ✓ What other changes may be seen with the different differentials
  - ✓ **See also:**
    - ➢ **Hepatobiliary Disease, Pancreatic Disease, Renal Disease, Endocrine Disease**

<table>
<tr><th colspan="2">Albumin:</th></tr>
<tr><td>INCREASED:
▪ Dehydration (↑ PCV, ↑ TP)
▪ Artefact
▪ Neoplasia</td><td>DECREASED:
▪ ↓ Production:
➢ Liver disease (+/- ↑ liver enzymes, may not if chronic, ↓ urea)
➢ PSS (↓ Alb, ↓ glucose, ↓ urea)
▪ ↑ Loss:
➢ Protein losing enteropathy (+/- vomiting and diarrhoea)
➢ Protein losing nephropathy (Proteinuria, +/- azotaemia, no ↓ globulins)
➢ Haemorrhage
➢ Burns
▪ Dilution eg. IV fluids (↓ PCV, ↓ TP)
▪ ↓ Intake (malnutrition)</td></tr>
<tr><td colspan="2">ALP: Alkaline phosphatase
Produced by canalicular membranes
Different isoenzymes in osteoblasts, chondroblast and hepatobiliary cells
CATS any increase is significant as normally has rapid clearance, indicates active inflammation</td></tr>
<tr><td>INCREASED:
▪ Liver damage (↑ ALT)
▪ Liver disease, that can cause ↑ ALP only:
➢ Hyperadrenocorticism
➢ Idiopathic vacuolar hepatopathy
➢ Hepatic neoplasia
➢ Nodular hyperplasia
➢ Drug induction
▪ ↑ Cortisol (hyperadrenocorticism, chronic stress, corticosteroids – cats no cortisol isoenzyme)
▪ Diabetes mellitus (↑ blood and urine glucose)
▪ Cholestasis (↑ bilirubin, bile acids, ↑ GGT)
▪ Bone disease (lysis and hyperparathyroidism) (↑ Ca+, Phos)
▪ Young growing animals (osteoblasts)
▪ Hyperparathyroidism (↑ Ca+, Phos)
▪ Hyperthyroidism (↑ ALT)
▪ Hypothyroidism (↑ Cholesterol)
▪ Carcinomas and mammary gland tumours</td><td>DECREASED:
▪ Artefact</td></tr>
</table>

ALT: Alanine aminotransferase
Produced by hepatocytes
Also other cells renal, muscle, pancreatic cells

INCREASED:	DECREASED:
▪ **Hepatocyte damage** (major source) ▪ **Liver-specific enzyme:** ➢ Hypoxic damage, inflammation/infection, neoplasia, toxic (↑ *ALP,* ↑ *AST*) ➢ Drugs (phenobarbitone) (↑ *ALP*) ➢ Diabetes mellitus (↑ *blood and urine glucose*) ➢ Hyperadrenocorticism (↑ *ALP,* ↓ *USG*) ➢ Hypertension (↑ *blood pressure, +/- proteinuria*) ➢ FeLV (cats) ➢ Trauma (cats) ▪ **Other sources:** ➢ Renal cells (*+/- azotaemia*) ➢ Cardiac muscle (damage), skeletal muscle (damage) (↑ *CK, +/-* ↑ *AST*) ➢ Pancreas (*+/-* ↑ *amylase, lipase*)	▪ Reduced liver mass ▪ Puppies due to immaturity

Ammonia:

INCREASED:	DECREASED:
▪ Liver failure (↓ uptake) (cirrhosis and PSS*:* (↓ *Alb,* ↓ *glucose,* ↓ *urea,* ↑ *bile acids, ammonium biurate crystals* (*PSS*)) ▪ Haemolysis (↑ *bilirubin,* ↓ *PCV*)	

Amylase:
Non-specific, produced by many abdominal pathologies

INCREASED:	DECREASED:
▪ Up to 3 – 4 x ↑ - Acute necrotising pancreatitis, flare-ups of chronic pancreatitis or obstruction of pancreatic ducts ▪ Renal failure (2-3 x ↑) (↑ *azotaemia*) ▪ Liver disease (↑ *ALT*)	

AST: Aspartate aminotransferase
Produce by hepatocytes, muscles

INCREASED:	DECREASED:
▪ **Non liver-specific enzyme:** ▪ Non-specific liver damage (↑ *ALT*) ▪ Muscle inflammation or necrosis (↑ *CK*) ▪ Haemolysis (*+/-* ↓ *PCV,* ↑ *bilirubin*)	▪ Cephalosporin use

Bile Acids:
Don't need to measure if ↑ bilirubin, but may see increases before ↑ bilirubin
Pre and post-prandial bile acids – used to assess hepatocellular function and enterohepatic function

INCREASED:	DECREASED:
▪ ↓ Liver function or functional mass (↓ bile acid recycling): ➢ Chronic hepatitis/Hepatic cirrhosis: (↓ *Alb,* ↓ *glucose,* ↓ *urea,*↑ *bilirubin*) ➢ Neoplasm (*+/-* ↑ *ALT, ALP, GGT*) ▪ Cholestasis (obstructing overflow) (↑ *ALP, GGT*) ▪ PSS (bypass liver recycling) (↓ *Alb,* ↓ *glucose,* ↓*urea*)	▪ Small intestinal malabsorption (↓ absorption)

Effusions

- **This chapter covers:**
  - ✓ How to collect and store samples
  - ✓ Interpretation of the samples
  - ✓ Common differentials

- **Sample collection:**
  - ✓ Collect sample into a EDTA, serum or sterile tube
  - ✓ Make smear and stain → microscope:
    - ➢ Inflammatory, neoplastic, non-inflammatory/neoplastic, bacteria other
  - ✓ Assess:
    - ➢ PCV/TP – compare to blood
    - ➢ Glucose – compare to blood glucose
  - ✓ Send away for culture and cytology (smears)

- **Type of effusion and features:**
  - ✓ Note: If sample is turbid, spin it down in a PCV tube to get a more accurate protein concentration

Effusion:	Protein Concentration (g/l):	Total Nucleated Cell Count:
Transudate	< 25 (<1.010)	< 1.5 x 10^9
▪ Formed by **passive** process – low oncotic pressure ▪ Fluid is clear to pale straw coloured ▪ Can have low numbers of mesothelial and inflammatory cells, macrophages and neutrophils		
Modified transudate:	25~50 (1.010~1.030)	1 – 5 x 10^9
▪ More **chronic** process – increased hydrostatic pressure or increased capillary/lymphatic permeability ▪ Fluid is yellowish, +/- blood tinged, slightly turbid ▪ High protein concentration compared to transudate ▪ Can have low numbers of mesothelial and inflammatory cells, macrophages and neutrophils		
Exudate:	> 30 (>1.018)	> 5 x 10^9
▪ Due to inflammatory process, leading to compromise of blood vessel integrity ▪ Fluid is turbid to cloudy, yellow, white, red ➢ **Non-septic:** • Non-degenerate neutrophils and activated mesothelial cells predominate • Non-infectious cause ➢ **Septic:** • Degenerate neutrophils predominate: Nuclear swelling and pale staining • Intracellular or extracelluar microorganisms • Culture and sensitivity: Aerobic and anaerobic • Abdominal fluid [glucose] < serum [glucose] • Abdominal fluid [lactate] > serum [lactate]		
Chyle:	Variable protein concentration	
▪ Opaque to pink ▪ Rupture or obstruction of lymphatic flow (neoplasia, traumatic, idiopathic) or secondary to heart failure (especially in cats) ▪ Pseudocyle (usually formed by lymphoma) ▪ Fluid [TAG] > serum [TAG] ▪ Large number of lymphocytes and other inflammatory cells		

Radiology

Systematic review of abdominal radiographs:

- **The ability to visualise detail within the abdominal cavity depends on:**
  - ✓ Intra-abdominal fat (falciform ligament, greater omentum, mesentery and retroperitoneum)
  - ✓ Animals conformation, particularly deep chested breeds influences organ visibility
- **Positioning/Coning/Exposure:**
- **Serosal detail:**
  - ✓ How well you see the serosal surfaces of organs
  - ✓ Can lose detail separately in peritoneal space as it is separate

1). Liver:
- Cranial abdomen → look at edge of liver falciform fat can obscure edge appear to disappear
- Can protrude past costal arch
- Enlarged if displaces the gastric axis (should be verticle on lateral view) or displaces the stomach caudally and to the left (VD)

2). Kidneys:
- In retroperitoneal space → can lose detail separate to peritoneal space
- Size: 2.5-3 x length of L2 on VD
- Left is lower and may not see the cranial pole of the right as within the liver
- If unsure about patency of urinary tract perform an excretory urogram

3). Spleen:
- Head of spleen on VD, behind the fundus of the stomach and cranial to LHS kidney
- Tail seen on lateral view variable location and size
- Almost never see tail of spleen in a cat

4). Urinary bladder:
- Varied size, can extend up to the level of the umbilicus

5). Prostate/uterus:
- Prostate: At the neck of the bladder if enlarged drops into the caudal abdomen
  - Normal size <75% height between pubis and ventral sacrum or <1.5 x length of L2
- Uterus: Sits between the colon and the urinary bladder if enlarged

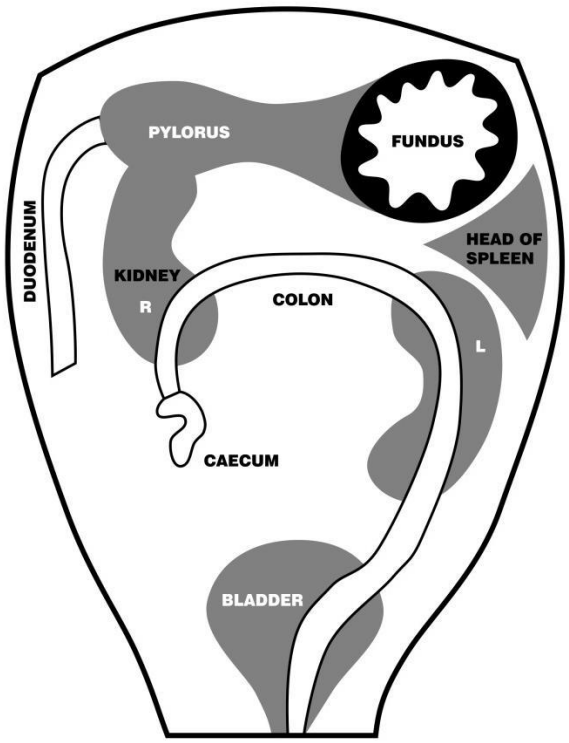
6). Stomach:
- Gastric axis: Use to indicate size of the liver – should be vertical on the lateral view

7). Duodenum:
- Lateral view: Runs in the midline from the pylorus of the stomach caudally
- VD view: Runs along the right side of the abdomen caudally from the pylorus of the stomach

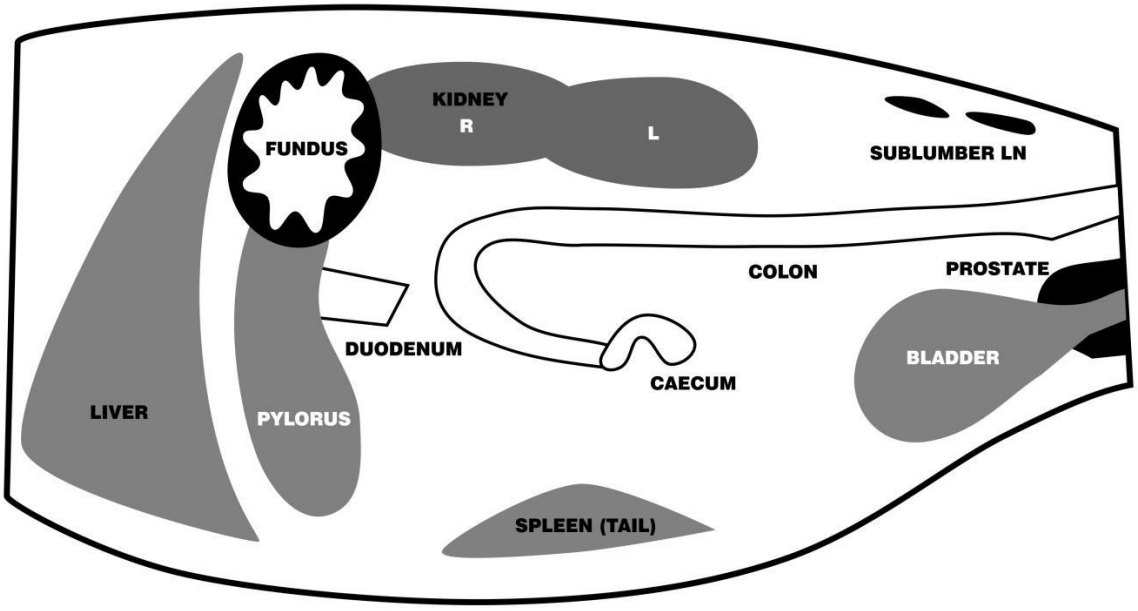
8). Small intestine:
- Once the other abdominal structures are identified, the small intestine is everything else
- Dilation: Compare width of small intestine to the height of L2 in dogs, and L4 in cats
- Can't judge thickness of wall (fluid looks same as soft tissue)

9). Large intestine:
- "Question mark" shaped, displaced, enlarged
- Caecum: Pocket of gas at the start of the ascending colon, usually in the RHS mid-abdominal region

10). Sublumbar lymph nodes:
- When enlarged, soft tissue ventral to the lumbar spine, displacing colon ventrally



- **Abdominal cavity – VD view**



- **Abdominal cavity – Lateral view**

Abdominal cavity:

- Assessment of the gastrointestinal tract, unless obvious, usually requires contrast studies or ultrasound

- **Serosal detail:**
  - ✓ Increased: Obese animals
  - ✓ Decreased: Emaciated or young animals (brown fat), free abdominal fluid (urine, blood, pus), peritonitis and inflammation (bowel rupture, bile, urine, pancreatitis, pyometron)

- **Oesophagus:** See below "Thoracic cavity"

- **Stomach:**
  - ✓ Take a LHS lateral view to assess the pylorus
  - ✓ Gastric axis on lateral view, can be used to assess liver size
- Pathology:
  - ✓ Abnormal position:
    - ➢ Cranially: Microhepatica, diaphragmatic hernia
    - ➢ Caudally: Hepatomegaly, neoplasia
  - ✓ Dilation:
    - ➢ Outflow obstructions:
      - Intraluminal: Foreign bodies, strictures, intussusceptions
      - Intramural: Neoplasia (dog: adenocarcinoma, cat: lymphosarcoma), hypertrophic
    - ➢ Air: Aerophagia (panting)or outflow obstruction
    - ➢ Ingesta: Over engorgement or outflow obstruction
    - ➢ Gastric dilation and volvulus:
      - Likely secondary to gastric motility disorder
      - Dilation leads to volvulus – seen as pylorus moving dorsally (LAT), cranially and to the left (VD)
      - Secondary small intestinal ileus and oesophageal dilation
      - Small intestines are dorsal and on top of the stomach (LAT)
      - "Double bubble"
  - ✓ Foreign bodies

- **Small intestine:**
  - ✓ Distension: Greater than the widest part of the L2 for dogs and L4 for cats
- Pathology:
  - ✓ Abnormal position:
    - ➢ Displacement due to organomegaly
  - ✓ Localised dilation:
    - ➢ Foreign body, neoplasia (dog: adenocarcinoma, cat: lymphosarcoma), intussusception, strictures, hernias, torsions
  - ✓ Generalised dilation:
    - ➢ Severe enteritis (parvovirus), ileus (pain, hypoxia - shock), electrolyte derangements (↓ K+, ↓ Ca+), pancreatitis, neurological, also causes of "localised dilation" above
  - ✓ Intestinal plication:
    - ➢ Concertina like gathering of intestines, usually with dispersed small pockets of gas, can indicate linear foreign bodies
    - ➢ Obese animals can have apparent clumping of the small intestine due to excessive abdominal fat reducing intra-abdominal space

Systematic review of thoracic radiographs:

- **Views:**
  - ✓ Lateral views allows visualisation of the **non-dependent** lung field

TAKE ALL THORACIC RADIOGRAPHS ON PEAK INSPIRATION

1). Thoracic wall:
- Subcutaneous structures: Assess soft tissue and fat planes outside of thorax
- Abdominal structures on the edge of the radiograph (and also inside the abdomen)
- Skeletal structures: Assess ribs, vertebrae and sternum

2). Diaphragm:
- Is it intact? Can the entire line of the diaphragm be identified on both lateral and VD

3). Pleural space:
- Do the lungs and pulmonary structures extend to the walls? If not is it fluid or gas

4). Mediastinal structures:
- **Trachea:** Heads towards the base of the heart on the lateral view, heads to the right side on VD
- **Cranial mediastinum**(ventral to trachea)**:** Contains CVC, LN and major branches of aorta
  - Lateral: Band of soft tissue ventral to trachea
  - DV/VD: Abnormal if >2 x width of a thoracic vertebral body
- **Oesophagus:** Not visible under normal conditions
- **Aorta:**
  - Lateral: Courses dorsally
  - DV/VD: Courses from cardiac silhouette to diaphragm on left (not always)
- **Caudal vena cava:**
  - Lateral: Courses from caudal border of cardiac silhouette to the diaphragm
  - DV/VD: Courses from caudal border of cardiac silhouette to diaphragm on RHS (not always)
- **Lymph nodes:** 3 groups only seen if enlarged
  - Cranial mediastinal LN: In cranial mediastinum
  - Tracheobronchial LN: Located at bifurcation of trachea into the main stem bronchi
  - Sternal LN: Located dorsal to the 2nd sternebrae on the lateral projection
    - Indicates cranial abdominal disease not thoracic disease

5). Heart:
- **Size:**
  - Round on both projections: Pericardial effusions, dilated cardiomyopathy, PDA/septal defects
  - Enlargement in certain areas: Specific chamber enlargement, **see next page**

6). Pulmonary parenchyma/vasculature: See below for **"Lung patterns"**
- **Air filled structures:**
- **Lung patterns:**
  - Alveolar
  - Interstitial:
    - Unstructured
    - Structured
    - Miliary unstructured – small multiple lesions but not fully discrete
  - Bronchial pattern
  - Vascular pattern

Thoracic cavity:

- **Pulmonary parenchyma/vasculature:**
- **Capsulated air filled structures:**
  - ✓ Thin outline: Bullae, cysts, abscess
  - ✓ Thickened irregular border: Neoplasia, abscess, granulomas

- **Vascular pattern:**
- Features:
  - ✓ Veins are central (VD) and ventral (LAT)
  - ✓ To assess for enlargement of the pulmonary arteries and veins, compare with the diameter of:
    - ➢ 9th rib on VD (up to 1.5 x diameter in cats is normal)
    - ➢ 4th rib on LAT

View:	Pulmonary artery:	Pulmonary vein:
Lat:	Dorsal	Ventral
DV:	Lateral	Medial

- Differentials:
  - ✓ Enlarged pulmonary veins:
    - ➢ Over hydration, pulmonary congestion, pulmonary hyperperfusion (shunts)
  - ✓ Enlarged pulmonary arteries:
    - ➢ Pulmonary hypertension, heartworm disease, pulmonary thromboembolism
  - ✓ Both enlarged:
    - ➢ Shunts, over hydation, severe LHS heart failure
  - ✓ Both reduced:
    - ➢ Shock states, dehydration, RHS heart failure

- **Bronchial pattern:**
- Features:
  - ✓ Abnormally defined bronchial walls, seen as “donuts” (end on bronchi) or “tram tracks” (side on bronchi):
    - ➢ Old age change
    - ➢ Bronchial disease: Chronic bronchitis, allergic, asthma, eosinophilic bronchopneumopathy
    - ➢ Mineralisation: Hyperadrenocorticism

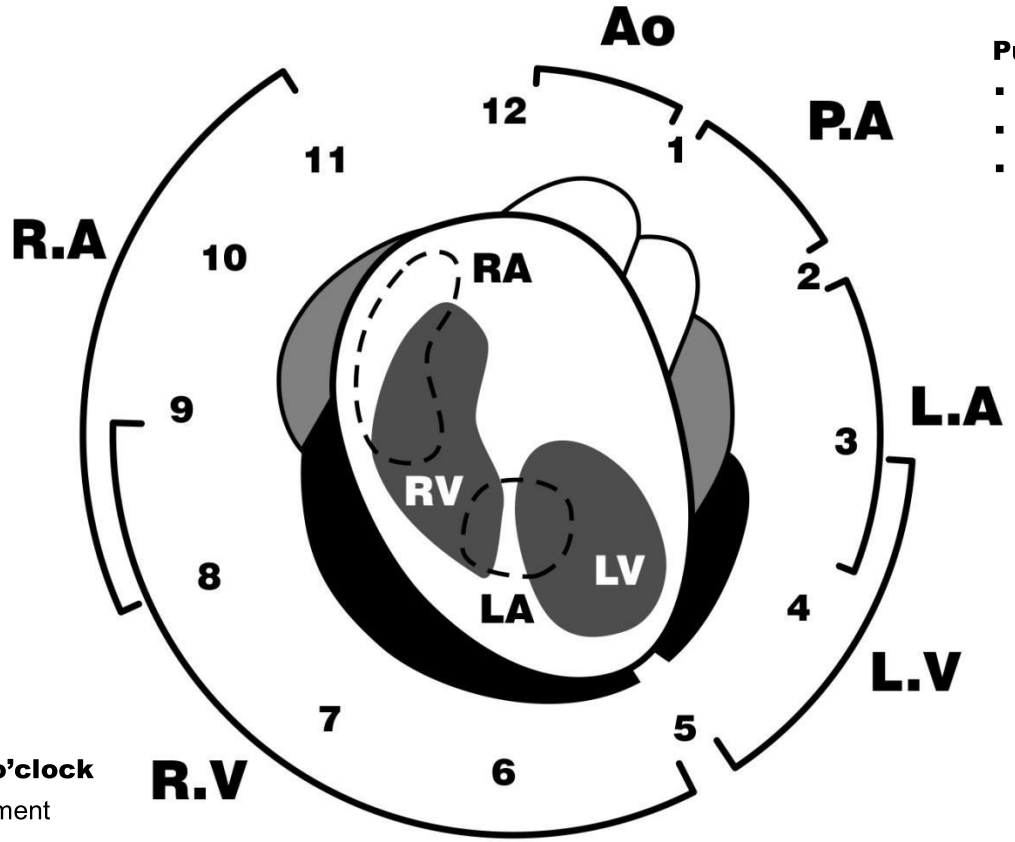
- **Alveolar pattern:**
- Features:
  - ✓ Pulmonary infiltration with fluid/soft tissue
  - ✓ “Fluffy” ill-defined regions of increased opacity
  - ✓ Can be lobar in distribution
  - ✓ Enhanced visualisation of airways, air bronchograms
  - ✓ Loss of visualisation of pulmonary vasculature
- Pattern of distribution:
  - ✓ Congestive heart failure: Dogs: begins hilar, cats: can look like anything
  - ✓ Pneumonia: Typically ventral or dependant side if aspirated
  - ✓ Caudal lobes: Neurogenic, post-obstructive
- Differentials:
  - ✓ Pneumonia: Infectious, aspiration, allergic
  - ✓ Pulmonary oedema: CHF, smoke, drowning, post-obstructive, seizures, head trauma, electrocution
  - ✓ Haemorrhage: Pulmonary contusions (traumatic), coagulopathic
  - ✓ Neoplasia
  - ✓ Atelectasis: Anaesthesia and bronchiectasis

Global enlargement
- Dilated cardiomyopathy
- Pericardial effusion:
  - Congestive heart failure
  - Right atrial tumour
  - Heart base tumour
  - Benign pericarditis
  - Cracked left atrium

Aortic arch = 12 – 1 o'clock
- Patent ductus arteriosus
- Aortic stenosis
- Tetralogy of Fallot
- Persistent right aortic arch

Pulmonary artery = 1 – 2 o'clock
- Pulmonic stenosis
- PDA
- Pulmonary hypertension

Right atrium = 8 – 11 o'clock
- Heartworm
- Pulmonic stenosis
- Right atrial tumour



Left auricle = 2 - 4 o'clock
- Secondary to mitral endocardiosis
- VSD

Left ventricle = 3 – 5 o'clock
- Left ventricular enlargement

Right ventricle = 5 – 9 o'clock
- Right ventricular enlargement

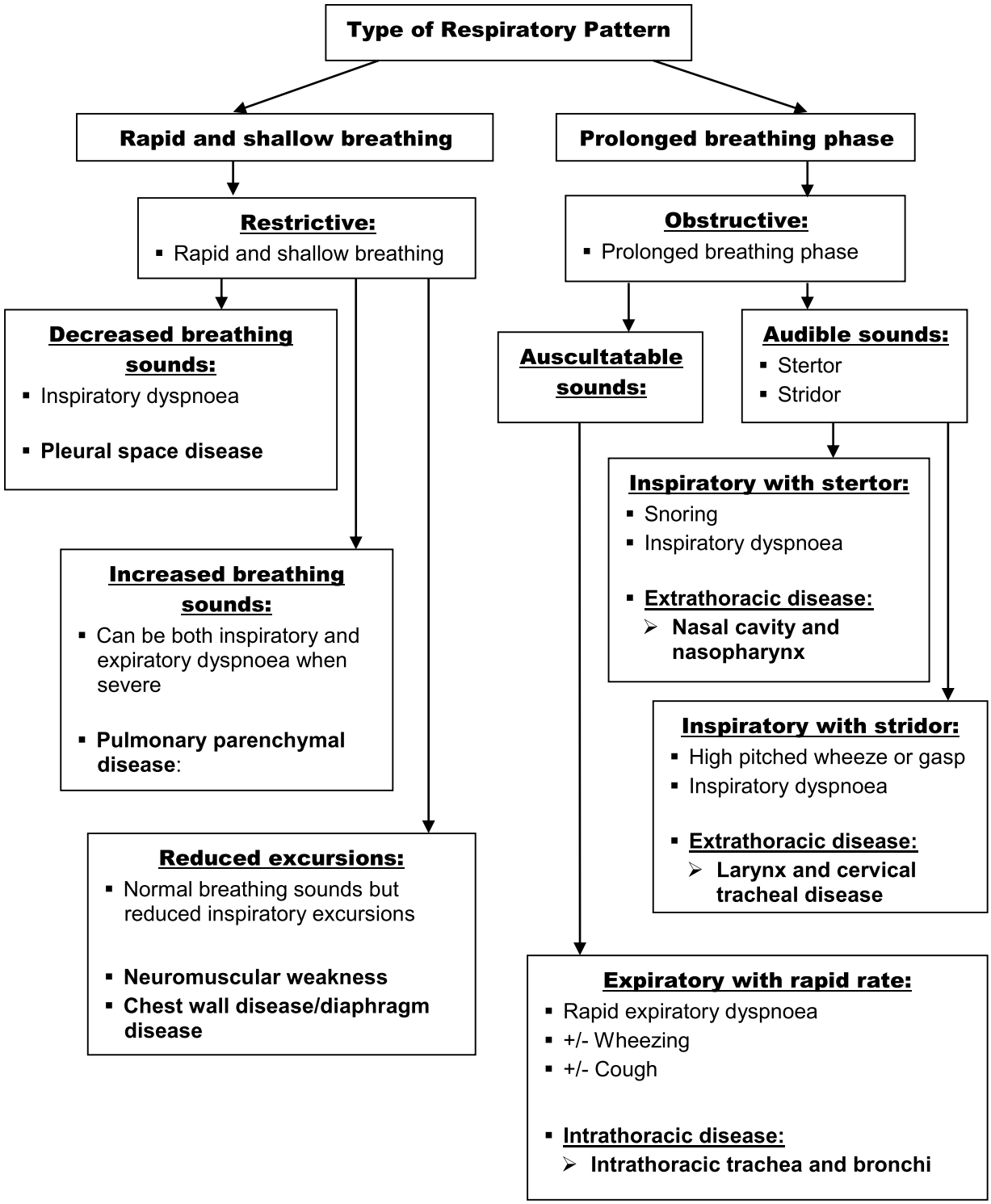
- **Dorsoventral (or ventrodorsal) view of the thoracic cavity**

Respiratory Disease

- **This chapter covers:**
  - ✓ Differentiation between respiratory patterns to help localise disease process
  - ✓ General diagnostic principles
  - ✓ Commonly seen respiratory disease: Features, clinical signs, diagnostic and treatment principles
- See **"Nasal and Nasopharyngeal Disease"** for upper respiratory tract disease

- **Dyspnoea:**
- **Presentation:**
  - ✓ **Dogs:** Sitting or standing (unable to lay down) with neck extended and open mouth breathing
  - ✓ **Cats:** Sternal recumbency with elbows abducted and abdominal effort to assist with inspiration
  - ✓ Characterised according to:
    - ➢ Phase: Inspiratory or expiratory
    - ➢ Type of accompanying noise: Stridor, stertor, wheeze, crackles
    - ➢ Respiratory rate
    - ➢ Pattern of excursion: Restrictive versus obstructive
    - ➢ Heart rate: Sinus arrhythmia usually indicates primary respiratory disease not cardiac disease
- **History:**
  - ✓ Duration and severity
  - ✓ Coughing, sneezing, tachypnoea, nasal discharge
  - ✓ Recent medications
- **Diagnostics:**
  - ✓ Auscultation of chest and heart, try to assess for heart murmurs, gallop rhythms, arrhythmias etc.
  - ✓ SPO_2
  - ✓ Blood gas:
    - ➢ Best if arterial blood sample
    - ➢ Assess pulmonary function, degree of oxygenation and adequacy of ventilation
  - ✓ Imagery:
    - ➢ Radiographs: 3 views, inspiratory views
    - ➢ Fluoroscopy: If suspecting dynamic airway disease
    - ➢ Ultrasound: If lesion is near the chest wall/mediastinal
  - ✓ Scoping:
    - ➢ Treacheobronchoscope: To visualise airways and to collect fluid samples
  - ✓ Airway fluid sampling:
    - ➢ Bronchoalveolar lavage (best performed with scoping) and transtracheal wash
    - ➢ Cytology, culture, PCR
  - ✓ Fine needle aspirates and swabs:
    - ➢ Cytology, culture, PCR

- **Emergency assessment and stabilisation:**
  - ✓ Assessment of respiratory pattern
  - ✓ Auscultation of chest and heart, try to assess for heart murmurs, gallop rhythms, arrhythmias etc.
  - ✓ SPO_2
  - ✓ Oxygen therapy: Severely dyspnoeic patients may require caged oxygen therapy to settle
  - ✓ Sedation:
    - ➢ Butorphanol 0.1-0.3mg/kg IV/IM
    - ➢ Acepromazine 0.01-0.05mg/kg IV/IM/SC (lower dose IV/IM) if certain that respiratory distress is not due to cardiac disease
  - ✓ IV catheter placement
  - ✓ +/- Cooling, emergency intubation and ventilation


Type of Respiratory Pattern
Rapid and shallow breathing
Prolonged breathing phase
Restrictive:
Rapid and shallow breathing
Obstructive:
Prolonged breathing phase
Decreased breathing sounds:
Inspiratory dyspnoea
Pleural space disease
Auscultatable sounds:
Audible sounds:
Stertor
Stridor
Inspiratory with stertor:
Snoring
Inspiratory dyspnoea
Extrathoracic disease:
Nasal cavity and nasopharynx
Increased breathing sounds:
Can be both inspiratory and expiratory dyspnoea when severe
Pulmonary parenchymal disease:
Inspiratory with stridor:
High pitched wheeze or gasp
Inspiratory dyspnoea
Extrathoracic disease:
Larynx and cervical tracheal disease
Reduced excursions:
Normal breathing sounds but reduced inspiratory excursions
Neuromuscular weakness
Chest wall disease/diaphragm disease
Expiratory with rapid rate:
Rapid expiratory dyspnoea
+/- Wheezing
+/- Cough
Intrathoracic disease:
Intrathoracic trachea and bronchi

Transfusion Therapy

- **This chapter covers**:
  - ✓ The types of blood products and their indications
  - ✓ Collection and cross matching
  - ✓ Administration of blood products and rates
  - ✓ Transfusion reactions, clinical signs and how to investigate and treatment

Blood products and indications:

- **Types of blood products:**

Types:	Aims:	Indications:
Whole blood (<8 hours old)	▪ Increase oxygen carrying capacity ▪ Clotting factors (all) ▪ Plasma protein ▪ Platelets: Blood must not be refrigerated and therefore must be transfused immediately after collection	▪ Anaemia ▪ Thrombocytopenia ▪ Coagulopathies
Packed red cells (>8 hours old)	▪ Increase oxygen carrying capacity	▪ Anaemia
Stored whole blood (<21 days old)	▪ Increase oxygen carrying capacity ▪ Stable clotting factors (Vitamin K dependent) ▪ Plasma proteins	▪ Anaemia ▪ Coagulopathy: ➢ Anti-coagulant toxicity (rodenticide)
Frozen fresh plasma (frozen <6 hours after collection, <3 months old)	▪ Clotting factors – stable and unstable ▪ Plasma proteins	▪ No anaemia ▪ Coagulopathy: ➢ Disseminated intravascular coagulation ➢ Hepatic disease ➢ Anti-coagulant toxicity (rodenticide and snake envenomation) ➢ vWD and hemophilia ▪ Hypoalbuminemia ▪ Colloid support
Fresh plasma (<6 hours old)	▪ Clotting factors – stable and unstable ▪ Plasma proteins ▪ Platelets	▪ Same as FFF ▪ Thrombocytopenia ▪ Colloid support
Stored or frozen plasma (frozen >6 hours after collection OR frozen plasma >3 months old)	▪ Stable clotting factors (Vitamin K dependent) ▪ Plasma proteins	▪ No anaemia ▪ Coagulopathy: ➢ Anti-coagulant toxicity (rodenticide) ▪ Hypoalbuminaemia ▪ Colloid support

- **Indications for blood products:**
  - ✓ **Red blood cells:**
    - PCV <15% or when rapidly drops <20% (15% in cats)
    - PCV <25% and need to do surgery or anaesthesia
    - Clinical signs of anaemia:
      - Exercise intolerance, tachycardia, tachypnoea, dyspnoea, weakness, hypotension, syncope and stupor
  - ✓ **Plasma:**
    - Indicated for coagulopathies or improve coagulation function prior to surgery
    - If significant clinical haemorrhage:
      - Fresh frozen plasma can be given in 10ml/kg boluses, repeated until clotting time improves
      - Or 10ml/kg/hr until reduced haemorrhage, normalised ACT then reduce to 2-3ml/kg/hr as a maintenance, up to 50ml/kg/day
    - If mild bleeding and underlying cause is being treated:
      - Fresh frozen plasma can be given in 5-10ml/kg boluses, then continued at 2-3ml/kg/hr as a maintenance
    - If no bleeding but need to perform surgery:
      - Can administer fresh frozen plasma as a bolus 10ml/kg
  - ✓ **Albumin:**
    - Low oncotic pressure and critically ill patients
    - Note: >10ml/kg of plasma is required to increase albumin by 1g/L, providing nutrition is a more efficient. Artificial colloids can be used to increase oncotic pressure, but its use in the prevention or oedema states has come under question, see **"Fluid Therapy"**
  - ✓ **Platelets:**
    - Thrombocytopenia
    - NOTE: Platelet transfusions not typically administered for thrombocytopenia (wait for regenerative response). 20ml/kg of fresh whole blood increases platelets by <40 x 10^9/L, vasopressin can be used to stimulate bone marrow to release platelets but variable response.

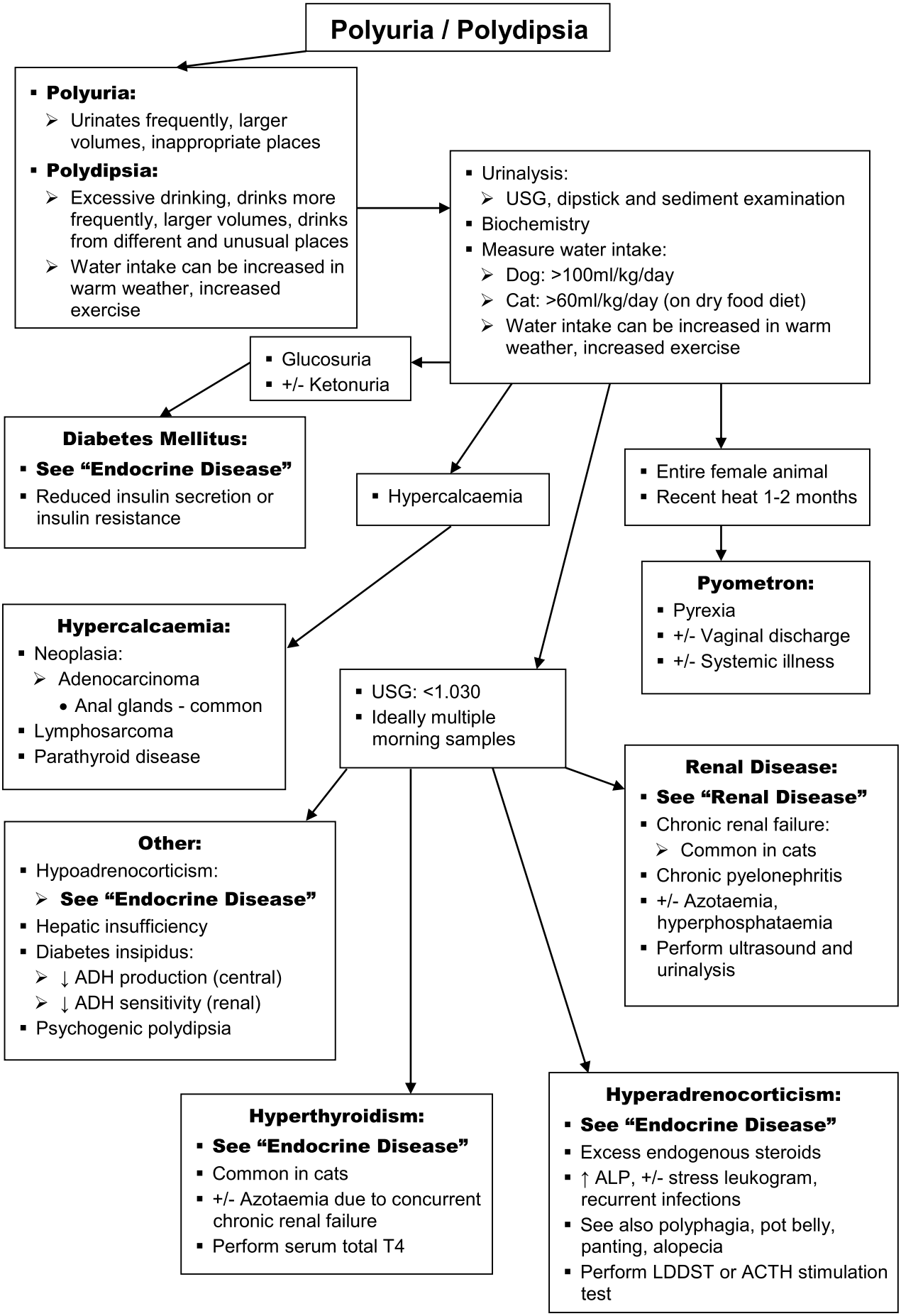
- **Blood types:**
  - ✓ Dogs (> 13 types):
    - Dogs can have more than one blood type
    - No natural alloantibodies, so if never received blood transfusion before cross matching should not be necessary
    - Antibodies form after 5 - 7 days, if second transfusion done after 5 days MUST cross match
  - ✓ Cats (A, B, AB and other):
    - **Blood typing and cross match**
    - Type A cats:
      - Most common antigen type
      - Have low levels of naturally occurring anti-B antibodies, if given type B blood, see a delayed reaction
    - Type B cats:
      - Have high amounts of anti-A antibodies, if given type A blood, see acute severe reaction
      - Typically Persians, Himalayans, British shorthairs, Devon, 25% of DSH
    - Type AB cats: Can have anyone's blood but can't donate

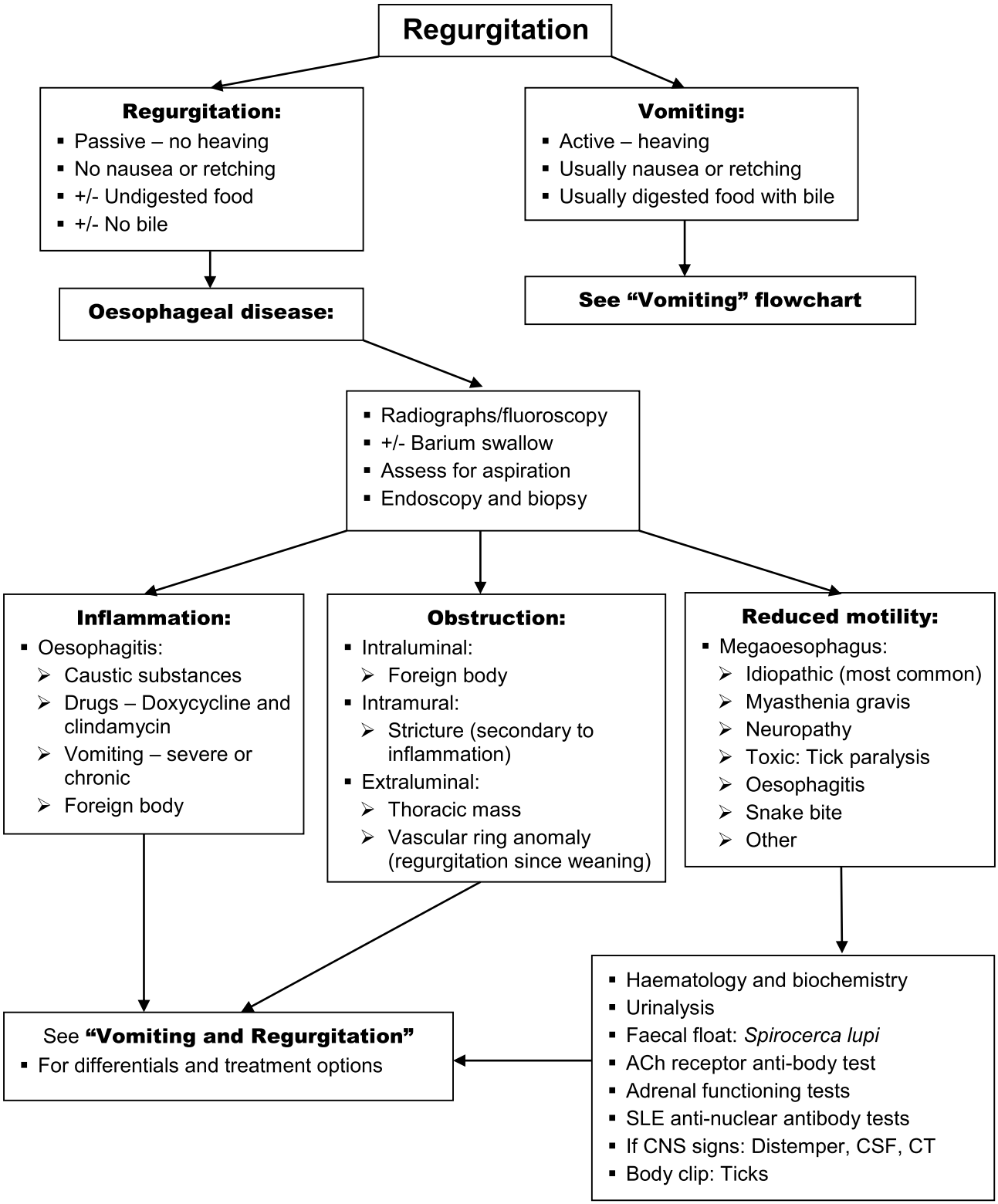
Collection and administration:

- **Donor selection:**
  - ✓ Dogs: Body weight >25Kg, PCV >35%, vaccinated, never received blood before, negative for heartworm and other infectious diseases
  - ✓ Cats: Body weight >5kg, PCV >35%, vaccinated and indoor, never received blood before, negative for FeLV, FIV, *mycoplasma felis* and toxoplasma

- **Collection from donor:**
  - ✓ Heavily sedate or anaesthetise donor
  - ✓ Place a jugular catheter, extension set (not in cats)
  - ✓ Can collect up to 10% of an animal's 'blood volume' (blood volume = 60ml/kg in Cats, 90ml/kg in Dogs)
  - ✓ Mix blood with 7:1 ratio with anti-coagulant, ie. blood:anticoagulant = 7ml:1ml
  - ✓ As blood is collecting in bag the blood must be mixed

- **Cross matching "in-house":**
- Used to determine the compatibility of the donor blood to the recipient
- Recommended for:
  - ✓ Whole blood and packed red blood cell transfusions
  - ✓ All cats
  - ✓ Dogs that have received a blood transfusion before (or 4 days after their first transfusion)
- Equipment needed:
  - ✓ 2 x 3ml syringes and needles
  - ✓ 2 x 5ml syringes and needles
  - ✓ 4 EDTA tubes
  - ✓ Slides
  - ✓ Saline 0.9%
- Method:
  - ✓ Collect 2mls of blood from the donor and recipient and store in two EDTA tubes labelled "donor" and "recipient"
  - ✓ Place EDTA tubes in centrifuge 1000g for 5 minutes to separate the plasma from the red blood cells or leave to stand
  - ✓ Collect the plasma and place in EDTA tubes again labelled "donor" and "recipient"
  - ✓ Collect 0.2mls of packed red blood cells and dilute down with 4.8mls of 0.9% saline – label the syringes "donor" and "recipient"
  - ✓ Label the slides as below:
    - ➢ Donor control: Determines if the cross match is viable
    - ➢ Recipient control: Determines if the cross match is viable
    - ➢ Minor cross match: Determines if the donor has antibodies to the recipient red blood cells
    - ➢ Major cross match: Determines if the recipient has antibodies to the donor red blood cells
  - ✓ Proceed as following:
    - ➢ Donor control: Place 1 drop of "donor plasma" and "donor RBC" on a slide, gently mix
    - ➢ Recipient control: Place 1 drop of "recipient plasma" and "recipient RBC" on a slide, gently mix
    - ➢ Minor cross match: Place 1 drop of "donor plasma" and "recipient RBC" on a slide, gently mix
    - ➢ Major cross match: Place 1 drop of "recipient plasma" and "donor RBC" on a slide, gently mix
- Interpretation:
  - ✓ Gently mix with a clean needle
  - ✓ Agglutination:
    - ➢ Seen as clumping of the red blood cells, "bundles of grapes"
    - ➢ Should occur within 5 minutes
    - ➢ If it occurs then add a drop of saline, if it dissipates then it was not agglutinated
  - ✓ Haemolysis


Polyuria / Polydipsia
Polyuria:
Urinates frequently, larger volumes, inappropriate places
Polydipsia:
Excessive drinking, drinks more frequently, larger volumes, drinks from different and unusual places
Water intake can be increased in warm weather, increased exercise
Urinalysis:
USG, dipstick and sediment examination
Biochemistry
Measure water intake:
Dog: >100ml/kg/day
Cat: >60ml/kg/day (on dry food diet)
Water intake can be increased in warm weather, increased exercise
Glucosuria
+/- Ketonuria
Diabetes Mellitus:
See "Endocrine Disease"
Reduced insulin secretion or insulin resistance
Hypercalcaemia
Entire female animal
Recent heat 1-2 months
Pyometron:
Pyrexia
+/- Vaginal discharge
+/- Systemic illness
Hypercalcaemia:
Neoplasia:
Adenocarcinoma
Anal glands - common
Lymphosarcoma
Parathyroid disease
USG: <1.030
Ideally multiple morning samples
Renal Disease:
See "Renal Disease"
Chronic renal failure:
Common in cats
Chronic pyelonephritis
+/- Azotaemia, hyperphosphataemia
Perform ultrasound and urinalysis
Other:
Hypoadrenocorticism:
See "Endocrine Disease"
Hepatic insufficiency
Diabetes insipidus:
↓ ADH production (central)
↓ ADH sensitivity (renal)
Psychogenic polydipsia
Hyperthyroidism:
See "Endocrine Disease"
Common in cats
+/- Azotaemia due to concurrent chronic renal failure
Perform serum total T4
Hyperadrenocorticism:
See "Endocrine Disease"
Excess endogenous steroids
↑ ALP, +/- stress leukogram, recurrent infections
See also polyphagia, pot belly, panting, alopecia
Perform LDDST or ACTH stimulation test


Regurgitation
Regurgitation:
Passive – no heaving
No nausea or retching
+/- Undigested food
+/- No bile
Vomiting:
Active – heaving
Usually nausea or retching
Usually digested food with bile
See "Vomiting" flowchart
Oesophageal disease:
Radiographs/fluoroscopy
+/- Barium swallow
Assess for aspiration
Endoscopy and biopsy
Inflammation:
Oesophagitis:
Caustic substances
Drugs – Doxycycline and clindamycin
Vomiting – severe or chronic
Foreign body
Obstruction:
Intraluminal:
Foreign body
Intramural:
Stricture (secondary to inflammation)
Extraluminal:
Thoracic mass
Vascular ring anomaly (regurgitation since weaning)
Reduced motility:
Megaoesophagus:
Idiopathic (most common)
Myasthenia gravis
Neuropathy
Toxic: Tick paralysis
Oesophagitis
Snake bite
Other
See "Vomiting and Regurgitation"
For differentials and treatment options
Haematology and biochemistry
Urinalysis
Faecal float: Spirocerca lupi
ACh receptor anti-body test
Adrenal functioning tests
SLE anti-nuclear antibody tests
If CNS signs: Distemper, CSF, CT
Body clip: Ticks

Antibiotics

Drug (*Trade Name*):	Action/Effect:	Indications:	Dosage:	Side Effects/Comments:
Amoxycillin-clavulanic acid *(Amoxyclav; Clavulox; Noroclav)*	Penicillins Bactericidal **Gram +ve** Gram -ve **Anaerobes** Not Pseudomonas	**Skin** / otitis externa **Bone** (less well) **URT**/pneumonia **Urogenital tract** (not ♂) Soft tissue **GI disease** (HGE) Pancreatitis; PSS; **pyometra**; mastitis	**Dogs:** 12.5-25mg/kg PO BID 10-20mg/kg SC/PO BID 1ml/20kg SC SID **Cats:** 62.5mg/cat PO BID	Do not give IV NOT – CSF; eye; bone; milk; abscess
Azithromycin	Macrolide Bactericidal Gram +ve Anaerobes	*Chlamydophila felis* Respiratory tract		Temporary clearance of organism Hepatotoxic NOT – CSF
Cephalexin (*Rilexine*)	Cephalosporin Bactericidal **Gram +ve** Gram –ve (some) Not anaerobes	**Skin** **Bone** Respiratory tract/pneum **Urogenital tract** (not ♂) Mastitis	22mg/kg BID	NOT – CSF; eye; milk; prostate Can get IMHA
Cephazolin/Cephalothin *(Keflin; Cefzol)*	2nd/3rd gen **Gram –ve**	Pre and post-surgery	10-30mg/kg IV/ SC TID	Administer slow IV (can cause anaphylaxis) Make 1g up to 9.6ml with water for injection
Chloramphenicol *(Chloropt)*	Bacteriostatic Gram +ve Gram –ve (some) Anaerobes	Eye (topical) FHV Crosses BBB (CNS) Bone/most tissues	2-3 times day	Also *Chlamydia; Mycoplasma; Rickettsia* Suppresses bone marrow Not for cats or young animals
Clindamycin *(Antirobe)*	Lincosamide Bacteriostatic **Gram +ve** **Anaerobes**	**Bone** / **cartilage** Consolidated **lungs** Rhinitis Skin/prostate/placenta	5.5-11mg/kg BID	Also Toxoplasma; Mycoplasma See GIT problems NOT – CSF
Doxycycline (*Vibravet*)	Tetracycline Bacteriostatic **Gram +ve** Gram –ve (some)	**Respiratory tract** Abscess Conjunctivitis Feline herpes Urogenital tract (including prostate) Bone; eye; CSF; sinus	Loading dose of 5mg/kg, then 2.5mg/kg q 12 hours for 2 doses, then maintenance dose 2.5mg/kg q 24 hrs	Also Bordetella; Mycoplasma; *Chlamydophila* May stain teeth in young dogs GIT upsets; oesophagitis; hepatotoxic; nephrotoxic (not Doxy)

Gastrointestinal Drugs

Drug (*Trade Name*):	Action/Effect:	Indications:	Dosage:	Side Effects/Comments:
Activated charcoal *(Carbasorb)*	↓ absorption of toxins	Intoxication	Granules:1-4g/kg PO Suspension: 5ml/kg PO	Care regarding aspiration pneumonia; administer via NGT
Apomorphine	Dopamine agonist Emetic agent	Intoxication e.g. rat bait	0.03mg/kg IV (dogs) 0.1mg/kg SC 0.25mg conjunctiva	Care regarding respiratory depression Can give metoclopramide to antagonise GIT effects NOT for cats
Carafate *(Sucralfate)*	Binds to and protects ulcerated/inflamed GIT	GI ulcers Chronic vomiting	0.25-1g PO TID	Administer as slurry; care regarding aspiration Administer antacids 1 hours after carafate
Cimetidine	H2 receptor antagonist (anti-histamine)	GI ulcers Chronic vomiting	10mg/kg QID-TID IV/IM/PO Renal failure: 2.5-5mg/kg BID PO	
Cisapride	Prokinetic – entire GIT Serotonin 5-HT4 receptor antagonist (increase Ach)	Gastric reflux Ileus Constipation	Cats: 2.5 – 5mg PO BID-TID	Beware when used with anti-fungals, erythromycin
Dolasetron *(Anzemet)*	Anti-emetic (central effects) Not a prokinetic	Use where other drugs failed	0.6-1.0mg/kg IV, PO SID-QID	Caution if dysrhythmias or severe electrolyte abnormalities – can affect electrical conduction in heart
Lactulose *(Duphalac)*	Stool softener	Constipation Hepatic encephalopathy	1ml/4.5kg PO TID to effect Dog: 0.5ml/kg TID Cat: 1ml/cat TID	Excessive use, can lead to fluid and electrolyte losses
Omeprazole *(Nexium)*	Proton pump inhibitor (↓ acid release)	Gastric ulcers Oesophagitis	0.5-1.0mg/kg IV, PO SID	Slow IV 24 hours for effect
Metoclopramide *(Metomide)*	Anti-emetic ↑ intestinal movement/tone (prokinetic) Anti-dopamine	Nausea Vomiting (CRTZ) Ileus/hypomotility	CRI: 1-2mg/kg/day 0.2-0.5mg/kg TID IV, IM, PO, SC	SC or IM/slow IV dose only last 2-4 hours Overdose → excitement, distress, diarrhoea Reverse with diphenhydramine 1mg/kg IV Do not use if intestinal bleeding or obstruction ↑ oesophageal sphincter tone; relax pyloric sphincter