miniVET guide

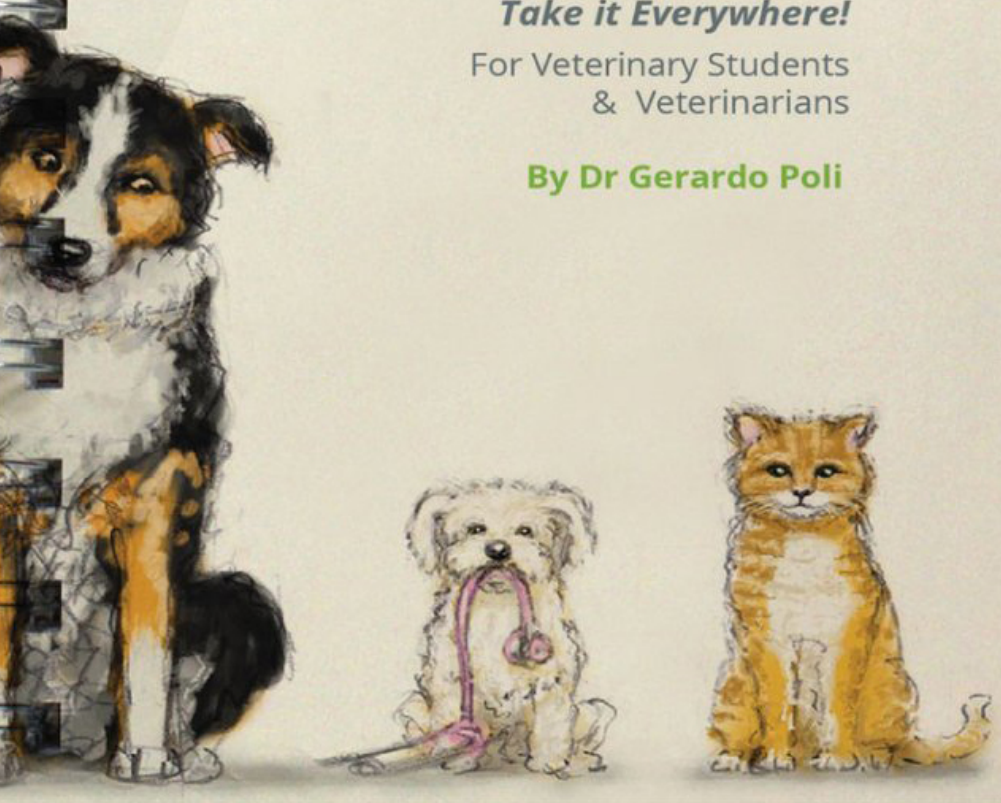
COMPANION ANIMAL MEDICINE

Portable and Concise ***VET*** Guide

Take it Everywhere!

For Veterinary Students
& Veterinarians

By Dr Gerardo Poli



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Parameter:	Dog:	Cat:
HR:	60-180 (pups 220)	140-220
RR:	10-30	25-40
Temp:	37.5-39.1	38.0-39.1
PCV:	37-55%	25-45%
TP:	52-80	57-80
BG:	4.3-6.7	4.2-8.0
USG:	>1.030	>1.035
Urine output:	1-2ml/kg/hr	1-2ml/kg/hr
Fluid rate:		
- Maintenance:	3ml/kg/hr	3ml/kg/hr
- Surgical:	6-10ml/kg/hr (depending on volume and hydration deficits)	6ml/kg/hr (depending on volume and hydration deficits)
- Shock:	Up to 60-90ml/kg/hr divided into boluses, titrated to effect	up to 55ml/kg/hr divided into boluses, titrate to effect

Disclaimer:

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Abbreviations:

- ✓ PO: Per os
- ✓ NPO: Nil per os
- ✓ SC: Subcutaneous
- ✓ IV: Intravenous
- ✓ IM: Intramuscular
- ✓ IT: Intratracheal
- ✓ SID: Once a day or every 24 hours
- ✓ BID: Twice a day or every 12 hours
- ✓ TID: Three times a day or every 8 hours
- ✓ QID: Four times a day or every 6 hours
- ✓ EOD: Every other day
- ✓ CRI: Continuous rate infusion
- ✓ BWt: Bodyweight
- ✓ CSx: Clinical signs
- ✓ GPEx: General physical examination
- ✓ Hx: History
- ✓ Dx: Diagnosis
- ✓ DDx: Differential diagnosis
- ✓ Tx: Treatment
- ✓ Px: Prognosis
- ✓ FNA: Fine needle aspirate
- ✓ CHF: Congestive heart failure
- ✓ DCM: Dilated cardiomyopathy
- ✓ HCM: Hypertrophic cardiomyopathy
- ✓ CVS: Cardiovascular system
- ✓ CRF: Chronic renal failure
- ✓ AKI: Acute kidney injury
- ✓ UOP: Urine output
- ✓ BG: Blood glucose
- ✓ BP: Blood pressure
- ✓ USG: Urine specific gravity
- ✓ PU/PD: Polyuria/Polydipsia
- ✓ VD: Ventrodorsal
- ✓ DV: Dorsoventral
- ✓ CBC: Complete blood count
- ✓ C&S: Culture and sensitivity
- ✓ FIV: Feline immunodeficiency virus
- ✓ FeLV: Feline leukaemia virus

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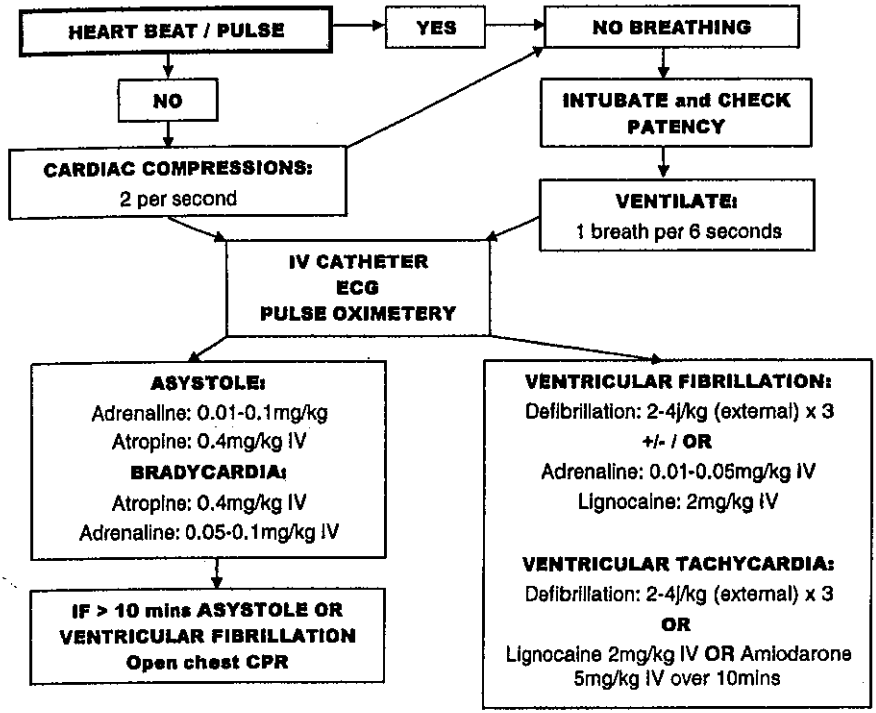
Cardiopulmonary Resuscitation



Drug:	Route:	Dose rate:	Dosage:
Adrenaline (1:1,000 = 1mg/ml) (NON-DILUTED)	IV/IT/IC IV/IM/SC	**CPR:** 0.01-0.1mg/kg (q 5mins) **Anaphylaxis:** 0.005mg/kg	0.1-1ml/10kg 0.05ml/10kg
Adrenaline (1:10,000) = 0.1mg/ml (1ml of 1:1000 in 9mls 0.9% NaCl)	IV/IT/IC IV/IM/SC	**CPR:** 0.01-0.1mg/kg (q 5mins) **Anaphylaxis:** 0.005mg/kg	1-10ml/10kg 0.5ml/10kg
Atropine (600µg/ml = 0.6mg/ml)	IV	0.04mg/kg	0.7ml/10kg
Lignocaine (HCl) 2% = 20mg/ml	IV slow	Dog: 2mg/kg repeat x 3-4 Cat: 0.5mg/kg repeat x 3	Dogs: 1ml/10kg Cat 0.1ml
Saline 0.9% ▪ Only if hypovolaemic	IV	Dog: 10-20ml/kg bolus IV Cat: 10ml/kg bolus IV	

- **Compressions:**
  - ✓ Small breed dogs and cats: Direct cardiac compressions over the heart
  - ✓ Keel chested dogs: Direct cardiac compressions over the heart, put towel under dependant side of chest to improve technique
  - ✓ Large breed dogs: Compression over the widest part of the chest, i.e. thoracic pump method
  - ✓ Barrel chested dogs: Direct cardiac compression but in dorsal recumbency
  - ✓ Must allow chest to expand between compressions
  - ✓ +/- Interposed abdominal counter pressure

- **Drugs:**
  - ✓ Can administer down the endotracheal tube but require a threefold dose increase
  - ✓ Best administered intravenously and flush through with a 20ml/kg crystalloid bolus to help drug delivery to the heart
- **Monitoring:**
  - ✓ Pulse oximeter: Assess heart rate and oxygen saturation (aim for >95%) indicates effectiveness of CPR
  - ✓ ECG: To assess electrical activity of the heart
  - ✓ Doppler: Place on eye and hear pulsations in the retinal arteries
  - ✓ End tidal CO_2 >15mmHg indicates reasonable circulation
- **Intra-thoracic cardiac massage:**
  - ✓ Clip
  - ✓ Aim for 7-8th rib
  - ✓ Cut cranially to rib avoid blood vessels and nerves
  - ✓ Reach under and massage heart
- **Complications:**
  - ✓ Short term:
    - ➢ Cardiac arrhythmias
    - ➢ Metabolic acidosis
    - ➢ Organ failure due to prolonged tissue hypoxia
    - ➢ If comatose: Variable prognosis, consider humane euthanasia if no improvement after 72 hours
  - ✓ Long term:
    - ➢ Personality change
    - ➢ Cortical blindness: Sight usually returns after few weeks

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**

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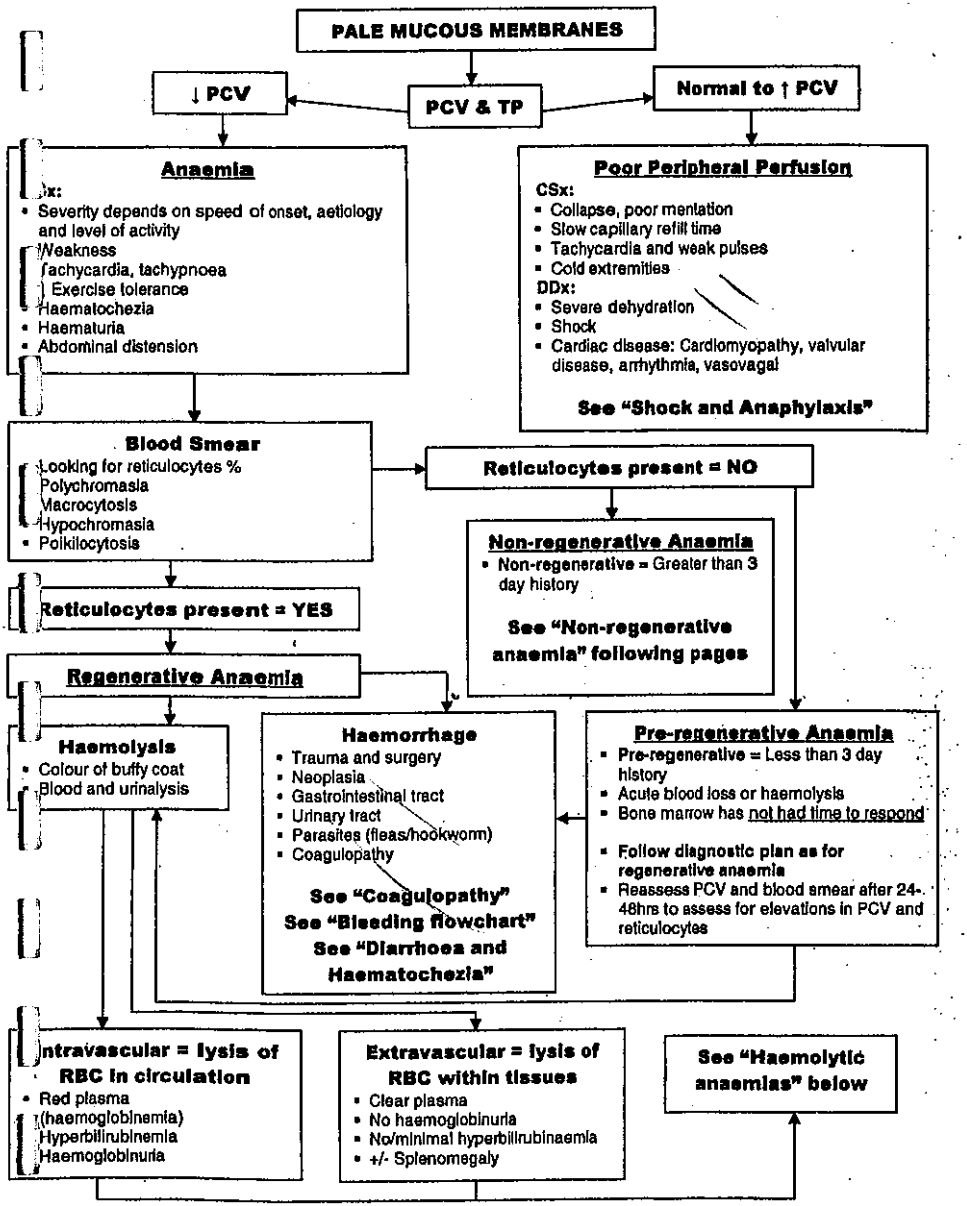
History:

- ✓ 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 e.g. renal disease, tumours, viral infections (FIV/FeLV)
- ✓ Recent administration or access to medications



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 e.g. *Mycoplasma, Babesia, Leptospria,* or viruses FeLV, FIP, FIV, *Ehrlichia 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

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Non-regenerative Anaemia

- **Features:**
  - ✓ Can appear non-regenerative if blood loss or haemolysis has only recently occurred i.e. <48-72 hours
  - ✓ Non-regenerative anaemia is 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

Biochemistry:
- 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:

ted 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

one marrow necrosis/fibrosis:
- Precursor cells are destroyed

Myelodysplasia:
- Defective precursor cells lead to abnormal maturation or cellular morphology
- Idiopathic or secondary to FIV, FeLV

one 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

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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 envenomation and other immune mediated diseases
- **Clinical signs:**
  - ✓ Pyrexia, anaemia, icterus (rarely seen in cats), weakness, tachycardia, tachypnoea, 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 antibodies 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
    - ➢ +/- Cyclosporine 5-10mg/kg PO divided BID
  - ✓ Gastric protectants:
    - ➢ H_2 antagonist, proton pump inhibitors and sucralfate 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

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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:
  - ➢ +/- Cyclosporine 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

- **Haemotropic mycoplasmas:**

Pathophysiology:

- ✓ Epierythrocytic parasite:
  - ➢ Dogs: *Mycoplasma haemocanis* (Europe) main species, transmitted by brown dog tick *Rhipicephalus sanguineus*. Splenectomised 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, tachypnoea, 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

- **Babesiosis:**

Pathophysiology:

- ✓ Intraerythrocytic protozoal parasite spread primarily by ticks but also by transplacental transmission and blood transfusions
- ✓ Several different species worldwide, the widest 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

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- **Clinical signs:**
  - ✓ Lethargy, weakness, pyrexia, anaemia, icterus, splenomegaly, tachycardia, tachypnoea
- **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

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Anaesthesia and Analgesia

- **This chapter covers:**
  - ✓ What to consider before anaesthesia
  - ✓ Types of premedicants, anaesthetic agents and analgesics and possible combinations
  - ✓ Local anaesthetics and blocks
  - ✓ Monitoring anaesthesia

Before anaesthesia:

- ✓ History: Current or prior health problems or drug administration
- ✓ General physical examination
- ✓ Diagnostics: Preferably at least PCV/TP, electrolytes, BUN and ALT, resting blood pressure
- ✓ Risk of anaesthesia: Based on history, physical examination, diagnostics and type of procedure
- ✓ Selection of premedication and anaesthetic agent
- ✓ Monitoring during anaesthesia

Premedication:

- **ADJUST DOSAGES ACCORDING TO PATIENT**

Drug and Mode of action:	Effect:	Concerns and Side effects:	Dose:
cepromazine: • Dopamine (D1 and D2) antagonist α1- adrenoreceptor antagonist agonist	• Anxiolytic • Sedation – less reliable unless combined with an opioid	• Cardiovascular disease: ➢ Vasodilation leads to ↓ blood pressure • Reduced doses in large dogs and Boxers	0.01-0.05mg/kg SC/IM/IV (lower range if IV or if Boxers or large breed dogs)
Medetomidine: • α2 adrenoreceptor agonist	• Sedative (potent) • Analgesic (potent)	• Cardiovascular effects: ➢ ↓ HR & cardiac output ➢ Oxygen delivery to central organs maintained • Avoid: Heart disease, liver, old and young, diabetic • Can cause vomiting	10-20µg/kg SC/IM 0.1-0.5µg/kg IV • Reverse with: Atipamezole 5-20µg/kg • Reverse bradycardia: Atropine 2.5-5µg/kg IV Ketamine 0.1-0.2mg/kg IV
lazepam: • ↑ GABA	• Anxiolytic • Anti-seizure • Muscle relaxant	• Minor effects on cardiovascular and respiratory systems	0.2-0.5mg/kg SC/IM/IV
ploids: Morphine & Methadone: ➢ µ-opioid receptor agonist Hydromorphone & Oxymorphone: ➢ µ-opioid receptor agonist • Buprenorphine: ➢ Partial µ-opioid agonist Butorphanol: ➢ Partial µ-opioid agonist/antagonist	• Sedation when combined • Oxymorphone more potent • Butorphanol - potent sedation but reduced analgesia	• Minor effects on cardiovascular system • Slight respiratory depression • Urinary retention • Morphine: Can cause vomiting, histamine	• Morphine & Methadone: 0.1-0.5mg/kg SC/IM/IV • Hydromorphone & Oxymorphone: Dogs: 0.1-0.4mg/kg SC/IM/IV Cats: 0.05-0.2mg/kg SC/IM/IV • Buprenorphine: Dogs/Cats: 5-20µg/kg SC/IM/IV • Butorphanol: 0.1-0.5mg/kg SC/IM/IV (>0.4mg/kg = potent sedation)

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- **Premedicant combinations:**

Combinations and Uses:	Effect:	Contraindication:	Doses:
Butorphanol:	▪ Potent sedation if given IV	▪ Good for CVS disease	0.1-0.3mg/kg IV
Benzodiazepine + opioid: ▪ Premedication ▪ Safe for debilitated patients	▪ Light sedation ▪ Not for cats (not effective) ▪ Butorphanol (↑ sedation)	▪ Good for CVS disease ▪ Young and ill = more potent sedation	▪ Benzodiazepine: 0.2-0.5mg/kg SC/IM/IV ▪ Methadone: 0.3mg/kg SC/IM/IV ▪ Butorphanol: 0.1-0.3mg/kg SC/IM/IV
Benzodiazepine + ketamine: Benzodiazepine reduce ketamine excitation ▪ Sedation ▪ Safe for moderately ill patients but not if HCM	▪ Potent sedation and analgesia ▪ Good for cats	▪ Beware HCM in cats – can cause myocardial ischaemia due to inotropic effects	Mix benzodiazepine with ketamine and titrate to effect IV ▪ Benzodiazepine: 0.2-0.3mg/kg ▪ Ketamine: 2mg/kg
Acepromazine + opioid: ▪ Premedication ▪ Not for moderately or severely ill	▪ Light sedation ▪ Butorphanol (↑ sedation) ▪ IV increases effect but reduce dose	▪ Not for CVS compromise (ACP)	▪ ACP: 0.03-0.05mg/kg SC/IM/IV (lower IV) ▪ Methadone: 0.3mg/kg SC/IM/IV ▪ Butorphanol: 0.1- 0.3mg/kg SC/IM/IV
Medetomidine + opioid: ▪ Premedication ▪ Sedation for invasive procedures ▪ **Only if healthy** ▪ Reverse after with Atipamezole	▪ Potent sedation and analgesia ▪ Butorphanol (↑ sedation)	▪ Not for CVS compromise or systemic disease	▪ Medetomidine: 10-20µg/kg IM ▪ Methadone: 0.3mg/kg ▪ Butorphanol: 0.1- 0.3mg/kg SC/IM/IV
Medetomidine + benzodiazepine: ▪ Sedation ▪ **Only if healthy** ▪ Reverse after with Atipamezole	▪ Potent sedation and analgesia	▪ Not for CVS compromise or systemic disease	▪ Medetomidine: 10-20µg/kg IM ▪ Benzodiazepine: 0.2-0.3mg/kg SC/IM/IV
Medetomidine + ketamine: Medetomidine reduces ketamine excitation ▪ Sedation for invasive procedures ▪ **Only if healthy** ▪ Reverse after with Atipamezole	▪ Potent – sedation and analgesia ▪ Can add opioid to reduce doses of both	▪ Not for CVS compromise or systemic disease ▪ If reverse medetomidine hyperexcitability will occur	▪ Medetomidine: 10-20µg/kg IM ▪ Ketamine: Dog: 5mg/kg IV Cat: 7mg/kg OV

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Anaesthesia:

- **Induction agents:**
  - ✓ Work out dose required for body weight then titrate to effect (give a ¼ at a time)
  - ✓ Always have more available

Drugs:	Effects:	Side Effects:	Dose:
Alfaxan CD RTU:	• Non-cumulative • Short duration of effect	• Excitement and twitching on induction and wake-up, premedication can reduce this side effect	Dogs: 1-2mg/kg Cats: 2-5mg/kg **IV titrated**
Etomidate:	• Sedative hypnotic • Minimal cardiovascular side effects	• Excitement and twitching on induction and wake-up, premedication can reduce this side effect • Long term CRI's can lead to suppressed adrenocortical function	Dogs: 0.5-3mg/kg Cats: 0.5-2mg/kg **IV titrated**
Ketamine:	• Analgesia • Sympathetic stimulation ➢ Blood pressure is maintained to hypertensive • Non-cumulative	• Protective reflexes maintained • Increased muscle tone MUST be used in combination with muscle relaxants: e.g. Medetomidine, diazepam • NOT for: ➢ Renal disease ➢ Hyperthyroidism ➢ Eye surgery ➢ Seizure animals ➢ Head trauma patients (increased intracranial pressure)	Combination with diazepam - mix and titrate IV: • Ketamine: 5-10mg/kg • Diazepam: 0.2-0.3mg/kg
Propofol:	• Non-cumulative • Short duration of effect • Anti-seizure	• Hypoventilation • Hypotension	2-10mg/kg IV titrated
Thiopentone:	• Anti-seizure • Minimal cardiovascular side effects	• Alkaline: Irritant if perivascular • Hypoventilation • Avoid: ➢ Young (<12 weeks) ➢ Greyhounds and sight hounds	10-20mg/kg IV titrated to effect

- **Maintenance:**
  - ✓ Either CRI or small boluses of induction agent:
    - ➢ Ensure that the agent used does not accumulate
  - ✓ Continue with inhalation anaesthetic:
    - ➢ Once maintained reduce anaesthetic gas to an appropriate level

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Anaesthetic machine setup:

- **Testing for leaks in the anaesthetic circuit:**
  1) **Oxygen supply:** Turn on the O_2 valve on the cylinder and check the gauge for how full it is
  2) **Oxygen flow metre:** Ensure that it can sustain a high constant flow by opening flow meter to maximum
  3) **Vaporiser:** Fill vaporiser to full will liquid anaesthetic
  4) **Carbon dioxide absorber:** Check the colour and the amount left
  5) **Assemble breathing circuit**
  6) **Assess for leaks:**
     - Occlude the end of the Y-piece
     - Close the spill valve and turn on oxygen flow
     - Fill reservoir bag until inflated
     - Turn off oxygen flow and wait to see (bag deflation) and hear for any leaks
  7) **Spill valve function:**
     - Open the spill valve, ensure that the reservoir bag deflates
  8) **One-way valves function:**
     - Fill the reservoir bag
     - **Inspiratory valve:** Squeeze the reservoir bag and watch to see the valve move/flutter
     - **Expiratory valve:** Blow into Y-piece and watch to see the valve move/flutter

Anaesthesia in patients with concurrent disease:

- **Diabetes Mellitus:**
  - ✓ No food after dinner (give normal insulin dose with dinner) – water ok
  - ✓ Give half morning dose of insulin with no food
  - ✓ Pre-operative blood glucose:
    - If low to normal (~12-15mmol/L), use a 2.5% glucose/dextrose solution
    - If ~15mmol/L, use a fluid that does not contain glucose (e.g. Hartmanns or 0.9% saline)
  - ✓ Intraoperative blood glucose:
    - Measure regularly (e.g. every 30 minutes) during surgery
    - If falls below 12mmol/L, use a 2.5% glucose/dextrose solution
    - If very high (~20mmol/L), administer a judicious dosage of regular (rapid-acting) insulin by SC or IM injection (use a dose between 0.2unit/kg)
  - ✓ Postoperative blood glucose:
    - Measure every hour for 2-3 hours after recovery
  - ✓ Feed as soon as safely possible after the surgery:
    - If eats give the remaining half of the insulin dose (otherwise proportional to how much is eating)
  - ✓ Avoid stopping insulin all together, even in animals that are inappetant:
    - If not eating reduce by 25-50% as must halt lipolysis as the same time avoid hypoglycaemia
  - ✓ Glycaemic control over the next 24-48 hours may not be as effective, usually seen as increased drinking
- **Hypoadrenocorticism:**
  - ✓ No food after dinner – water ok
  - ✓ In the morning give normal Fluronef dose and a higher dose of prednisolone than normal e.g. 0.5mg/kg instead of 0.2mg/kg, to provide enough corticosteroids to respond with the stress
  - ✓ The next morning give a slightly higher dose e.g. 0.3mg/kg then back to normal dose the morning after
  - ✓ Monitor electrolytes before and after the anaesthesia

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Monitoring anaesthesia:

- **Planes of anaesthesia:**

Light plane of anaesthesia:

- ✓ Slight muscle relaxation
- ✓ Swallowing
- ✓ Nystagmus, corneal and palpebral reflexes present
- ✓ Central eye position and dilated pupil

Surgical plane of anaesthesia:

- ✓ Good muscle relaxation
- ✓ No swallowing
- ✓ No nystagmus, palpebral reflexes, corneal reflex is maintained
- ✓ Ventral eye position
- ✓ Relaxed anal tone

- **Deep (dangerous) plane of anaesthesia:**
  - ✓ Profound muscle relaxation
  - ✓ All eye reflexes are lost
  - ✓ Central eye position and constricted pupils
  - ✓ No anal tone

Parameters to monitor during anaesthesia:

Muscle tone and reflexes:

- ✓ Limb and jaw tone
- ✓ Palpebral reflex
- ✓ Withdrawal reflexes and pain response

Mucous membranes:

- ✓ Colour and capillary refill time

Cardiovascular system:

- ✓ **Heart Rate:**

	Bradycardia:	Tachycardia:
• **Heart rate:**	• Larger dogs: <50-60bpm • Smaller dogs: <70-90bpm • Cats: <80-100bpm	• Dogs: >120-200 (depends on breed) • Cats: >200-240bpm
• **Causes:**	• Hypothermia, hypertension, drugs (opioids, α2-agonist), hyperkalaemia	• Sympathetic nervous system stimulation, hypotension, hyperthermia, drugs (atropine, ketamine), hypoxia

- ✓ **Pulse:** Rate, pulse pressure, timing with heart rate
- ✓ **Electrocardiogram:** Assess electrical activity of the heart, see **"Cardiovascular disease"**

- **Respiration:**
  - ✓ Should be regular and even
  - ✓ **Light anaesthesia:** Haphazard breathing pattern, tachypnoea or apnoea
  - ✓ **Deep anaesthesia:** Apnoea or very slow and shallow breathing pattern
  - ✓ **Excessive effort:** Airway obstruction or endotracheal tube is blocked/kinked
  - ✓ **No inspiratory efforts: Anaesthesia is too deep or light**
    - ➢ Check patient:
      - Assess vitals: Heart rate, pulses and mucus membrane colour
      - Assess depth of anaesthesia: Jaw and limb tone, reflexes
    - ➢ Stimulate breathing: Pressure and rubbing over costochondral junctions or nasal acupressure point
    - ➢ Check circuit:
      - Assess spill valve: Ensure it is open

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✓ **Adequacy of ventilation:** Best assessed via capnography or blood gas

Parameter:	CO_2 levels (mmHg):
• Ideal inspired CO_2 • Inspired CO_2	• 0mmHg • >0mmHg = problem with apparatus ➢ Exhausted soda lime ➢ Spill valve closed ➢ Large dead space
• Ideal expired CO_2	• 30-40mmHg
• Hyperventilating - Too much CO_2 expired	• <30-40mmHg
• Hypoventilation - Not enough CO_2 expired	• >50mmHg

- **Blood pressure:**
  - ✓ **Aim maintaining blood pressures:**
    - ➢ Systolic pressure: >100mmHg
    - ➢ Mean arterial pressure: >80mmHg
    - ➢ Renal perfusion requires: >60-70mmHg MAP
  - ✓ **Palpation:**
    - ➢ Digital arteries lost at systolic pressure <80mmHg
    - ➢ Carpal and dorsal pedal artery lost at systolic pressure <60mmHg
    - ➢ Femoral artery lost at systolic pressure <40mmHg

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Anaesthetic complications:

Complication:	Treatment:	Mode of action:	Dosage (mg/kg):
Bradycardia:	▪ Atropine ▪ Glycopyrrolate	▪ Vagolytic (↓ vagal tone) ▪ Vagolytic (↓ vagal tone)	▪ 0.02mg/kg IV ▪ 0.01 mg/kg IV
Tachycardia:	▪ Propranolol ▪ ↑ Analgesia OR anaesthesia	▪ β blocker (↓ sympathetic tone)	▪ 0.05-0.06mg/kg IV
Ventricular arrhythmias:	▪ Lignocaine 2% (20mg/ml) ▪ Procainamide	▪ Membrane stabiliser	▪ 2mg/kg IV (slow) then 20-80 µg/kg/min ▪ 0.5-2.0 IV then 20-40µg/kg/min
Hypotension: (BP ≈ CO & PR)	▪ Fluids: Titrated to effect ➢ Crystalloids ➢ Colloids ▪ Dopamine CRI	 ▪ 10-20ml/kg IV bolus ▪ 5ml/kg IV bolus ▪ α + β agonist = + inotrope (contraction strength)	 ▪ 3-5µg/kg/min IV
Hyperkalaemia: (bradycardia / atrial standstill)	▪ 0.9% saline fluids ▪ Insulin (regular) with dextrose (always) ▪ Ca+ gluconate 10%	 ▪ Protect heart	 ▪ Insulin: 0.25U/kg ▪ Dextrose: 2g/unit of insulin: = 4mL of 50%/U = 40mL of 5%/U ▪ 0.5-1.5ml/kg IV (slow over 20mins)
Hypocalcaemia:	▪ Ca+ gluconate 10%		▪ 0.5-1ml/kg IV (slow over 20mins)
Hypoglycaemia:	▪ Dextrose 5%		▪ 1-2ml/kg IV diluted
Broncho-constriction:	▪ Terbutaline		▪ 0.01mg/kg SC/IM
Excitement / delirium:	▪ Acepromazine ▪ Diazepam ▪ Midazolam		▪ 0.05-0.2mg/kg IV/IM ▪ 0.25-0.5mg/kg IV/IM ▪ 0.5-0.2mg/kg IV/IM

- **Receptors and their activity:**
  - ✓ α = ↑ Vasopressor activity
  - ✓ β1 = ↑ Myocardial contractility (inotrope) and ↑ heart rate (chronotrope)
  - ✓ β2 = Dilatation of veins, vessels in skeletal muscle, and bronchodilation

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Analgesia:

- **Levels of pain and management:**
  - ✓ **Mild pain:** Non-opioids or weak opioids +/- adjuvants
    - ➢ Oral single drug therapy
  - ✓ **Moderate pain:** Weak opioids + non-opioids +/- adjuvants
    - ➢ Start injectable single drug therapy
    - ➢ Continue multimodal therapy
  - ✓ **Severe pain** (all surgical patients)**:** Strong opioids +/- non-opioids +/- adjuvants
    - ➢ Multimodal injectable drug therapy
  - ✓ **MUST ensure:**
    - ➢ Nursing care: Keep warm, dry, gentle handling, hydrated, etc.
    - ➢ Fracture immobilisation

- **Types of drugs:**
  - ✓ **Adjuvants:**
    - ➢ Local blocks +/- epidurals
    - ➢ Muscle relaxants: Benzodiazepines
    - ➢ Sedatives/Tranquilisers: Acepromazine, benzodiazepines
  - ✓ **Non-opioids:**
    - ➢ NSAIDS: Not during surgery and only when hydrated and eating to reduce gastrointestinal and renal side effects
    - ➢ α2-adrenergic agonists: Medetomidine
    - ➢ Tramadol: Non-opioid μ-agonist
    - ➢ Ketamine infusions: Low dose via IV or SC infusion (0.1-0.6mg/kg/hr)
  - ✓ **Opioids:**
    - ➢ Weak: Codeine, buprenorphine, butorphanol
    - ➢ Strong: Morphine, methadone, fentanyl

Opioids:

- **Methods of administration:**
  - ✓ Bolus dose: Buprenorphine, morphine, methadone, fentanyl etc.
  - ✓ Titration: Fentanyl, morphine etc.
  - ✓ Constant rate infusion: Morphine, fentanyl etc. increase or decrease rate accordingly
  - ✓ Dermal patches: Fentanyl, buprenorphine

Drug:	Duration:	Note:
Morphine	3 - 6 hours	
Methadone	4 - 6 hours	
Fentanyl	20 minutes	
Hydromorphone	4 - 6 hours	
Oxymorphone	3 -4 hours	
Buprenorphine	3 - 12 hours	*Ceiling effect on pain relief*
Butorphanol	30 - 120 minutes	*Ceiling effect on pain relief*
Naloxone	45 - 60 minutes	*Less effective with partial agonists*

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- **Titration:**
  - ✓ Small IV, IM or SC doses then repeat examinations until patient appears to be comfortable:
    - ➢ Morphine and methadone - 0.1mg/kg repeated at 10-15min intervals

- **Continuous rate infusions:**
  - ✓ Anaesthesia:
    - ➢ During anaesthesia monitoring of anaesthesia is important as pain relief can significantly reduce anaesthetic gas requirements
    - ➢ Monitoring of expired CO_2: Reduce CRI, depth of anaesthesia or manually ventilate if CO_2 >50mmHg
    - ➢ Opioid CRI can cause significant sedation, urine and faecal retention, hypothermia and hypoventilation
  - ✓ Morphine (10mg/ml):
    - ➢ Dogs: 0.1–0.5mg/kg/hr and cats: 0.05–0.25mg/kg/hr
    - ➢ Give loading (0.5mg/kg) dose prior to starting infusion
    - ➢ CRI = 4 hour dose (0.5mg/kg) given over 4 hours
    - ➢ 90mg in 500ml of saline = 0.18mg/ml
    - ➢ Run at 0.7– ml/kg/hr = 0.12mg/kg/h = 0.48mg/kg/4h
  - ✓ Butorphanol (10mg/ml):
    - ➢ Dogs and cats: 0.1mg/kg/hr
    - ➢ Below doses are based on a 0.05mg/kg dose per hour:
      - 1 x maintenance = 1.4ml (14mg) in 1L bag
      - 2 x maintenance = 0.7ml (7mg) in 1L bag
      - OR add 20mg to 500mls of saline and run at 1-2mls/kg/hr (1ml = 0.05mg/kg/hr, 2ml = 0.1mg/kg/hr)
  - ✓ Fentanyl (0.05mg/ml):
    - ➢ Dogs and cats: 0.001–0.004 mg/kg/hr (1-5µg/kg/hr)
    - ➢ Dogs with severe pain up to 0.01mg/kg/hr (10µg/kg/hr)
    - ➢ Give 5µg/kg IV prior to starting infusion
    - ➢ Add 1ml per 10mls of saline or 50mls in 500mls of saline
    - ➢ Run at 1-2ml/kg/hr (1ml = 0.005mg/kg/hr, 2ml = 0.01mg/kg/hr)

When to turn CRI down...	When to turn CRI up...
• Hypoventilation: ➢ ↓ Respiratory rate or depth ➢ Cyanosis ➢ ↓ Oxygen saturation ➢ Hypercapnia	• Tachypnoea
• CNS depression: ➢ Unresponsiveness ➢ Sleep, dysphoria	• Vocalization / Agitation** • Unresponsiveness**
• CNS stimulation: ➢ Agitation**	• Abnormal posture • Reluctance to move
• Hypothermia	
• Bradycardia or tachycardia**	• Tachycardia/bradycardia**
• Hypotension	• Pale mucous membranes: ➢ Peripheral vasoconstriction

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- **Transdermal patches:**
  - ✓ Fentanyl dosed at 2–4µg/kg/hr, takes approximately 12 hours until has an effect
  - ✓ Lasts to 72 hours in dogs and up to 100 hours in cats
  - ✓ Fentanyl patches may not provide sufficient analgesia, must monitor patient
  - ✓ NOTE: Buprenorphine patches may be better than fentanyl in cats

Weight:	Dogs:	Cats:
3 – 5kg	12.5 µg/hr patch	≤ 12.5 µg/hr patch
5 – 12kg	25 µg/hr patch	
12 – 30kg	50 µg/hr patch	
20 – 30kg	75 µg/hr patch	
>30kg	100µg/hr patch	
Onset	12 – 24 hours	6 - 12 hours
Duration of action	72 hours	100 hours

  - ✓ **Application of patches:**
    - ➢ Avoid contact to your skin
    - ➢ Clip do not shave skin, DO NOT clean skin with surgical scrub or alcohol
    - ➢ Avoid raw or clipper rash skin
    - ➢ Hold the patch to the skin for 1 minute to help adhesive bond
    - ➢ Cover well to avoid unwanted removal
    - ➢ To reduce dose, don't cut the patch, lift patch and cover part with occlusive tape
    - ➢ Respiratory depression not a problem post-operatively
    - ➢ Monitor patient closely: Hyperthermia (heating mat!) OR patients with fever can increase absorption

- **Side effects of opioids:**
  - ✓ Respiratory depression (lose sensitivity to CO_2)
  - ✓ Vasodilation
  - ✓ ↓ Gut motility (constipation)
  - ✓ Nausea/vomiting (↑ with morphine if pain free)
  - ✓ Urine retention
  - ✓ ↑ Vagal tone
  - ✓ Dysphoria

- **Opioid toxicity:**
  - ✓ Naloxone:
    - ➢ µ opioid receptor antagonist
    - ➢ Dilute 0.1 - 0.25 ml of naloxone 0.4 mg/ml to 10 ml with 0.9% saline (dilute further for very small patient)
    - ➢ Titrate in 1 ml increments per minute until depression/dysphoria have abated
    - ➢ Should not lose analgesia
    - ➢ Duration of action: 45 minutes, may need to repeat every 45 minutes until opioid wears off
  - ✓ Butorphanol:
    - ➢ Due to its µ-opioid receptor antagonism, it partially antagonises a pure µ-opioid agonists effect

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Local anaesthetics:

- **Methods of administration:**
  - ✓ Intraoperative blocks
  - ✓ Dental blocks
  - ✓ Intrapleural
  - ✓ Into fracture site
  - ✓ Continuous rate infusion, ensure dose is appropriate

- **Types of local anaesthetics:**
  - ✓ Lignocaine 4mg/kg IV/IM/SC maximum dose
    - ➢ Up to 3 hours duration of effect, used intravenously for cardiac arrhythmias
  - ✓ Bupivacaine 2mg/kg/4hrs IM/SC maximum dose
    - ➢ Up to 10 hours duration of effect, **never** used intravenously, extremely cardiotoxic

- **Signs of toxicity:**
  - ✓ Central nervous system:
    - ➢ Mild sedation: Excitement (inhibitory pathways blocked) → Overall depression (all pathways blocked)
  - ✓ Cardiovascular system:
    - ➢ Low doses: Anti-arrhythmic, no cardiac depression
    - ➢ High doses: Sinus bradycardia, ↓ cardiac contractility
  - ✓ Respiratory system:
    - ➢ Central depression
    - ➢ Eventually, reduced contractility of muscles of respiration
  - ✓ Local tissue damage

- **Preventing toxicity:**
  - ✓ Calculate maximum allowable dose in mg for each patient/drug
  - ✓ Always draw back before injecting (every time the needle is shifted)
  - ✓ Check landmarks for accurate placement
  - ✓ Bupivacaine is highly cardiotoxic! -- NEVER IV and avoid intrapleural administration

- **Maxillary nerve block:**
  - ✓ Blocks palatine branches of maxillary nerve:
    - ➢ Maxilla, palate, upper teeth, nose and upper lip
  - ✓ Technique:
    - ➢ 25G 15mm needle bent 45°
    - ➢ Inject 0.25–0.75ml of lignocaine 2% or bupivacaine 0.5%
    - ➢ Insert needle 90° two thirds laterally between midline of palate and last molar
    - ➢ Walk needle off back of hard palate
    - ➢ Direct needle laterally 30°
    - ➢ Aspirate then inject

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- **Mandibuloalveolar block:**
  - ✓ Blocks inferior alveolar branch of mandibular nerve:
    - ➢ Blocks sensory to lower teeth and lower lip
  - ✓ Technique:
    - ➢ 25G 12mm needle
    - ➢ Inject 0.25-0.75ml of lignocaine 2% or bupivacaine 0.5%
    - ➢ Palpate the angular process of mandible
    - ➢ Locate centre of the "bight" on lower edge of mandible
    - ➢ Advance needle full depth on the medial side of the mandible pointing towards the eye

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Antimicrobial Selection

- **This chapter covers:**
  - ✓ Types of antimicrobial agents and their side effects
  - ✓ Disease processes and the empirically recommended antimicrobial agent

- **Selection of antimicrobials:**
  - ✓ Initial: Empirical selection
  - ✓ Definitive: Based on culture and sensitivity results

Antibiotics:		
Type:	**Examples:**	**Side effects:**
• Beta-lactams: ➢ Bactericidal	• Amoxicillin • Ampicillin • Amoxicillin-clavulanic acid • Cephalosporin • Ticarcillin	• Gastrointestinal upset ➢ Administer with food
• Aminoglycosides: ➢ Bactericidal	• Gentamicin • Amikacin	• Nephrotoxic • Ototoxic • Vestibular disease
• Fluoroquinolones: ➢ Bactericidal	• Enrofloxacin • Marbofloxacin	• Gastrointestinal upset ➢ Give on an empty stomach • Cartilage defects
• Nitroimidazoles: ➢ Bactericidal	• Metronidazole	• Neurological signs • Ataxia, tremors, vomiting, weakness
• Lincosamides: ➢ Bactericidal/static (dose dependant)	• Clindamycin	• Gastrointestinal upset • Oesophagitis
• Macrolides: ➢ Bactericidal/static (dose dependant)	• Erythromycin • Tylosin • Azithromycin	• Gastrointestinal upset • Hepatotoxic
• Tetracycline: ➢ Bacteriostatic	• Doxycycline • Oxytetracycline	• Dental staining • Oesophagitis • Oxytetracyclines: ➢ Fever in Cats ➢ Hepatotoxic ➢ Nephrotoxic
• Sulphonamides: ➢ Bacteriostatic	• Trimethoprim-sulpha	• Keratoconjunctivitis sicca • Haematological abnormalities • Gastrointestinal upset • Polyarthritis • Haemolytic anaemia • Large breeds e.g. Rottweiler /Dobermans: hypersensitivity, hepatopathy
Antifungals:		
• Macrolide antibiotics: ➢ Fungicidal	• Amphotericin B • Nystatin (topical)	• Nephrotoxic
• Griseofulvin: ➢ Fungistatic	• Griseofulvin	• Teratogenicity • Haematological abnormalities • Gastrointestinal upset

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▪ Imidazole: ➢ Fungicidal/static	▪ Enilconazole ▪ Clotrimazole ▪ Fluconazole ▪ Ketoconazole ▪ Itraconazole ▪ Miconazole	▪ Not in pregnant animals ▪ Gastrointestinal upset ▪ Hepatopathy

▪ **Body system:**

Skin/Soft tissue infections:		
▪ **Superficial pyoderma**	▪ *1st:* Cephalexin 22mg/kg BID ▪ *2nd:* Clindamycin 11mg/kg BID	▪ Minimum 3 weeks or 10 days after complete resolution
▪ **Deep pyoderma** *Diagnostic work-up:* ▪ Skin scrapings ▪ Tissue biopsies ▪ Assessment for predisposing disease	▪ *1st:* Amoxicillin-clavulanic acid 12.5-25mg/kg BID ▪ *2nd:* Fluoroquinolones 10mg/kg BID ▪ *Topical therapy:* 3% chlorhexidine shampoo/lotions	▪ At least 4 weeks, or 2 weeks after resolution
▪ **Abscessation**	▪ *1st:* Amoxicillin-clavulanic acid 12.5-25mg/kg BID ▪ *2nd:* Based on culture and sensitivity *Concurrent therapy:* ▪ Surgical debridement, lavage and drainage	▪ 2 weeks
▪ **Dermatophytes** *Diagnostic work-up:* ▪ Trichogram ▪ Woods lamp (*Microsporum canis*) ▪ Fungal culture of hair	▪ *Topical therapy:* Miconazole preparations ▪ *Systemic:* Griseofulvin OR ketoconazole OR itraconazole ▪ E.g. Itraconazole (5mg/kg SID for one week every, then one week break, then repeat one week. Repeat for three cycles) *Concurrent therapy:* ▪ Environmental decontamination	▪ Weeks
▪ ***Malassezia*** *Diagnostic work-up:* ▪ Swabs (ears/claw folds) ▪ Sticky tape impressions	▪ *Topical therapy:* Miconazole preparations (twice weekly) ▪ *Systemic:* Ketoconazole 5-10mg/kg BID	▪ 3 weeks
▪ **Mycobacterial** Draining sinus tracts *Diagnostic work-up:* ▪ Cytology ▪ Mycobacterial culture and sensitivity	▪ *M. smegmatis* ▪ *1st:* Doxycycline 5mg/kg BID + moxifloxacin 5mg/kg BID ▪ *Other mycobacteria* ▪ *1st:* Clarithromycin 15mg/kg BID + Moxifloxacin 5mg/kg BID ▪ *Nocardia* ▪ Trimethoprim-sulpha 15mg/kg BID + another based on culture and sensitivity	▪ Months

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Upper respiratory tract infections:		
▪ **Rhinitis (canine)**		
▪ **Acute rhinitis** *Diagnostic work-up:* ▪ Radiology ▪ Nasal flush ▪ Rhinoscopy ▪ Biopsy – cytology, histopathology, culture and sensitivity, LCAAT, PCR	▪ ***Primary rhinitis is not a clinical entity in dogs*** ▪ Require diagnostic work-up ▪ Doxycycline 5mg/kg BID	▪ 14 days
▪ **Chronic rhinitis:** ▪ Rarely primary bacterial disease, need investigation ▪ Lymphocytic plasmacytic rhinitis, foreign body, fungal, neoplasia, tooth root	▪ No empiric antimicrobial, require diagnostic work-up ▪ Selection is based on culture and sensitivity	
▪ **Fungal rhinitis:** ➢ Aspergillosis (nasal depigmentation	▪ *Higher success rates with topical:* ▪ *Topical:* Enilconazole **OR** clotrimazole ▪ *Systemic:* Itraconazole + amphotericin B	
▪ **Rhinitis (feline)**		
▪ **Acute and chronic rhinitis:** ▪ Commonly primary viral due to FHV and FCV but can be foreign body ▪ Latent FHV infections can flare up under stressful conditions ▪ Secondary bacterial infections *Diagnostic work-up:* ▪ Feline respiratory PCR panels ▪ If suspect foreign body, see "Canine acute rhinitis" ▪ "Chronic snufflers" ➢ Nasal biopsy: cytology, staining, culture and sensitivity, PCR ➢ Advanced imagery	▪ *1st*: Doxycycline 5mg/kg BID + famciclovir 30mg/kg BID (anti-herpes therapy) ▪ *2nd*: Based on culture and sensitivity ▪ Clindamycin 10mg/kg BID – if chondritis or osteomyelitis	▪ 14 days or 2-3 months if chronic
▪ **Fungal rhinitis:** ➢ Cryptococcal	▪ *After debridement* ▪ *Systemic:* Itraconazole **OR** fluconazole + amphotericin B	

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Tracheobronchitis: *Diagnostic work-up:* • Radiographs • BAL if severe - Airway cytology, staining, culture and sensitivity • Bronchoscopy	• Doxycycline 5mg/kg BID	• 14 days

Lower respiratory tract infections:		
• **Non-life threatening:** *Diagnostic work-up:* • Radiographs • If chronic or severe +/- BAL cytology, staining, culture and sensitivity, PCR for *Mycoplasma spp.* and *Bordetella spp.*	• 1st: Doxycycline 5mg/kg BID • 2nd: Amoxicillin-clavulanic acid 12.5-25mg/kg BID	• 3-4 weeks • If suspect *Mycoplasma* infection, then 6 weeks of doxycycline
• **Life threatening:** *Diagnostic work-up:* • Radiographs • BAL cytology, staining, culture and sensitivity	*Intravenous therapy:* • Amoxicillin 20mg/kg QID + metronidazole 10mg/kg BID + fluoroquinolones 5mg/kg SID *Concurrent therapy:* • IV fluid hydration • Nebulisation +/- gentamicin • Coupage • +/- Ventilation	
• **Pyothorax:** • Uncommon in dogs, concerned about foreign body migration (especially if *Actinomyces spp.* Cultured) *Diagnostic work-up:* • Radiographs • Thoracentesis: Cytology, staining, culture and sensitivity • Repeat radiographs after drainage to assess pulmonary structures • Advanced imagery • *Concurrent therapy:* ➢ Indwelling drainage, lavage	***Canine:*** *Intravenous therapy:* • Amoxicillin 20mg/kg TID **OR** cephalosporin 20mg/kg TID + metronidazole 10mg/kg BID + fluoroquinolones 5mg/kg SID • Change based on culture and sensitivity If branching or filamentous rods are present on staining then add: • Trimethoprim-sulpha 15mg/kg BID ***Feline:*** *Intravenous therapy:* • Amoxicillin 20mg/kg TID **OR** amoxicillin-clavulanic acid 12.5-25mg/kg BID + metronidazole 10mg/kg BID	• Initially IV, then oral therapy for 4-6 weeks

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Urinary tract infections:		
Cystitis: *Diagnostic work-up:* • Cystocentesis sample • Urinalysis and culture and sensitivity • See above	• 1st: Amoxicillin 15mg/kg TID **OR** amoxicillin clavulanic acid 25mg/kg BID (use **HIGH dose**) • 2nd: Change based on culture and sensitivity • Fluoroquinolones 5mg/kg SID **OR** trimethoprim-sulpha 15mg/kg BID	• 14 days
Pyelonephritis: *Diagnostic work-up:* • Cystocentesis sample • Urinalysis and culture and sensitivity • CBC/biochemistry • Ultrasound	*Intravenous therapy:* • Cephalosporin 20mg/kg TID + metronidazole 10mg/kg BID *Oral therapy:* • Oral based on culture and sensitivity start amoxicillin-clavulanic acid 12.5-25mg/kg BID	• Initially IV, then oral therapy • 4-6 week, avoid aminoglycosides
Prostatitis: *Diagnostic work-up:* • Cystocentesis sample • Urinalysis, culture and sensitivity • Haematology and biochemistry • Ultrasound • Prostatic aspirates/washes	• Trimethoprim-sulpha 15mg/kg BID **OR** Fluoroquinolones 5mg/kg SID *Concurrent therapy:* • Castration • +/- surgery	• 4 weeks

Reproductive tract infections:		
• **Metritis:**	• Amoxicillin clavulanic acid 12.5-25mg/kg BID	
Pyometron: *Diagnostic work-up:* • Culture and sensitivity	*Intravenous therapy:* • Amoxicillin 20mg/kg TID + metronidazole 10mg/kg BID + fluoroquinolones 5mg/kg SID *Concurrent therapy:* • Surgery	• Initially IV, then oral therapy for 2 weeks
Mastitis: • If nursing – use amoxicillin-clavulanic acid not enrofloxacin	*If stable:* • Amoxicillin clavulanic acid 12.5-25mg/kg BID *If unstable then intravenous therapy:* • Amoxicillin 20mg/kg TID + metronidazole 10mg/kg BID	

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Haemolymph Infections:		
▪ **Sepsis and bacteraemia** *Diagnostic work-up:* ▪ Blood cultures (repeated)	*If suspect overwhelming sepsis, then intravenous therapy:* ▪ Amoxicillin 20mg/kg TID + metronidazole 10mg/kg BID + fluoroquinolones 5mg/kg SID ▪ Adjust based on culture and sensitivity	
▪ ***Mycoplasma haemofelis*** See **"Anaemia and Pale Mucous Membranes"** *Diagnostic work-up:* ▪ Blood smears: In-house and external lab ▪ CBC/biochemistry ▪ PCR	▪ Doxycycline 5mg/kg BID	▪ 3 weeks
▪ **Endocarditis** *Diagnostic work-up:* ▪ Blood cultures (repeated) ▪ Cardiac ultrasound ▪ New diastolic murmur	*Intravenous therapy:* ▪ *1st:* Cephalosporin 20mg/kg TID (3rd generation) + fluoroquinolones 5mg/kg SID ▪ Adjust based on culture and sensitivity	▪ Weeks

Oral Infections:		
▪ **Periodontal disease & abscessation** ▪ Concurrent therapy: ➢ Remove tartar and plaque	***Canine:*** *After debridement and definitive treatment:* ▪ *1st:* Doxycycline 5mg/kg BID **OR** Clindamycin 5-11mg/kg BID *Second line:* ▪ *2nd:* Metronidazole 10mg/kg BID ***Feline:*** *After debridement and definitive treatment:* ▪ *1st:* Amoxicillin-clavulanic acid 12mg/kg BID ▪ *2nd:* Clindamycin 5-11mg/kg BID **OR** doxycycline 5mg/kg BID	▪ 3 days before then 14 days after
▪ **Stomatitis:** ▪ **Feline:** Suspected associated with FCV infection – ▪ **Candidiasis**	▪ *After debridement and definitive treatment:* ▪ *1st:* Doxycycline 5mg/kg BID **OR** clindamycin 5-11mg/kg BID **OR** metronidazole 10mg/kg BID ▪ *2nd:* Amoxicillin-clavulanic acid 12mg/kg BID *Concurrent therapy:* ▪ +/- Antivirals ▪ +/- Removal of teeth ▪ +/- Anti-inflammatories ▪ Fluconazole **OR** itraconazole	▪ 2 weeks

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Gastrointestinal infections:		
• Enteritis	• Metronidazole 10mg/kg BID	• 2 weeks
• Small intestinal bacterial overgrowth AKA antibiotic responsive diarrhoea	• Metronidazole 10mg/kg BID **OR** Tylosin 20mg/kg SID	• 2 weeks
• Giardiasis • See **"Diarrhoea"** for more information	• Fenbendazole 50mg/kg SID	• 5 days

Hepatobiliary infections:		
• **Cholangitis/hepatitis:** Reduce dosage *Diagnostic work-up:* • CBC/biochemistry • Ultrasound • Dynamic hepatic testing • Aspirates or biopsies: cytology, culture and sensitivity, histopathology	*Intravenous therapy:* • Ampicillin 20mg/kg TID + Metronidazole 7.5mg/kg BID + Fluoroquinolones 2.5mg/kg BID *Oral therapy:* • Amoxicillin-clavulanic acid 12mg/kg BID + Metronidazole 7.5mg/kg BID + Fluoroquinolones 2.5mg/kg BID	• Initially IV, then oral therapy
• **Hepatic encephalopathy:** *Diagnostic work-up:* • As above	*Intravenous and oral therapy:* • Metronidazole 7.5mg/kg BID **OR** Amoxicillin 20mg/kg BID	

Bone and joint infections:		
• **Osteomyelitis:** *Diagnostic work-up:* • Radiographs • Aspirates or biopsies – cytology, culture and sensitivity, histopathology	*Intravenous therapy:* • Cephalosporin 22mg/kg TID + fluoroquinolones 5mg/kg SID *Oral therapy:* • Amoxicillin-clavulanic acid 12.5mg/kg BID **OR** Fluoroquinolones 5mg/kg SID • Adjust based on culture and sensitivity	• 6-8 weeks
• **Fungal osteomyelitis:** ➢ Aspergillosis	• Fluconazole + amphotericin B	
• **Discospondylitis:** *Diagnostic work-up:* • Radiographs • Urine and blood cultures	*Intravenous therapy:* • Cephalosporin 22mg/kg TID + Fluoroquinolones 5mg/kg SID *Oral therapy:* • Amoxicillin clavulanic acid 12.5mg/kg BID **OR** • Fluoroquinolones 5mg/kg SID • Adjust based on culture and sensitivity	• Long term
• **Fungal discospondylitis:** ➢ Aspergillosis	• Itraconazole + amphotericin B	• Oral long term therapy based on serology and blood and urine cultures

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• **Septic arthritis:** *Diagnostic work-up:* • Radiographs • Joint tap – cytology, culture and sensitivity	*Injectable therapy:* • Cephalosporin 22mg/kg TID **OR** • Amoxicillin-clavulanic acid 12.5mg/kg BID • Adjust based on culture and sensitivity • Oral long term therapy based on serology and blood and urine cultures	• 2 weeks after resolution of clinical signs

Central nervous system infections: *Diagnostic work-up:* • CSF tap – Neurological PCR panel, cytology and culture and sensitivity		
• **Bacterial meningoencephalitis**	• Trimethoprim-sulpha 15mg/kg BID +/- amoxicillin clavulanic-acid 12.5mg/kg BID	Start injectable then continue with oral therapy
• **Protozoal meningoencephalitis** • See **"Parasitic Disease"**	• Trimethoprim-sulpha 15mg/kg BID **OR** clindamycin 5-11mg/kg BID	
• **Fungal infections:** ➢ Aspergillosis ➢ Cryptococcal	 • Itraconazole + amphotericin B • Itraconazole + amphotericin B	 • Long term • Long term
Ocular infections:		
• **Uveitis** • **Fungal infections:** ➢ Aspergillosis ➢ Cryptococcal	• Topical ocufloxacin q4hrs + Doxycycline 5mg/kg BID • Itraconazole + amphotericin B • Itraconazole + amphotericin B	 • Long term • Long term

Pyrexia:

- ***Dogs:*** Immune mediated disease, young dogs – parvovirus
- ***Cats:*** Commonly abscessations also upper respiratory tract viral infections (FHV, FCV), immune mediated disease is uncommon

- Assess for immune mediated disease (meningitis, polyarthritis), neoplasia, fungal disease, viral infections
- Rule out endocarditis, peritonitis, pneumonia, prostatitis, pyothorax, pyometra, abscesses, infectious arthritis

Diagnostic pathway:

- General physical examination - joint pain, neck and spinal pain, cardiac murmurs, uveitis, upper respiratory tract inflammation (cats), clip for abscessations (cats)
- Haematology, biochemistry, urinalysis, blood smear
- Collect blood and urine for culture and sensitivity, blood and joint fluid may require enrichment media
- Radiographs of chest, abdomen, spine, limbs
- Abdominal ultrasound
- +/- CSF and joint fluid analysis
- +/- PCR tests for parasites

AVOID anti-inflammatories – mask pyrexia

Mild illness:

- Amoxicillin clavulanic acid 25mg/kg BID use HIGH dose

If suspect overwhelming sepsis, then intravenous therapy:

- Amoxicillin 20mg/kg TID + metronidazole 10mg/kg BID + fluoroquinolones 5mg/kg SID

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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**

Albumin:

INCREASED:	DECREASED:
• Dehydration (↑ *PCV*, ↑ *TP*) • Artefact • Neoplasia	• ↓ 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 e.g. IV fluids (↓ *PCV*, ↓ *TP*) • ↓ Intake (*malnutrition*)

ALP: Alkaline phosphatase

Mainly produced by bile canalicular membranes, bones, kidneys

Different isoenzymes in osteoblasts, chondroblasts and hepatobiliary cells

CATS any increase is significant as normally has rapid clearance, indicates active inflammation

INCREASED:	DECREASED:
• 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	• Artefact

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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)

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Bilirubin:
Breakdown product of haemoglobin

INCREASED:	DECREASED:
Increased production (unconjugated): ➢ Excessive erythrocyte destruction (↓ *PCV*) ↓ Hepatic uptake/conjugation/excretion (unconjugated): ➢ Hepatocellular damage (↑ *ALT*) ➢ ↓ Functional mass, PSS (↓ *Alb*, ↓ *glucose*, ↓ *urea*, ↑ *bile acids*) Cholestasis (conjugated): ➢ Bile duct obstruct (↑ *ALP, GGT*, ↑ *bile acids*)	▪ Sepsis and hypertension ▪ Starvation and fever ▪ Artefact: Lipid, haemolysis

BUN: Blood urea nitrogen
Produced by liver from ammonia (breakdown product of protein metabolism)

INCREASED:	DECREASED:
▪ Azotaemia → ↓ removal of nitrogenous waste → ↓ renal excretion **Pre-renal (↑ USG):** ➢ Mild elevations: • Gastrointestinal bleeding (*normal creatinine*) • Protein catabolism (starvation, fever, sepsis, burns, steroids) ➢ Moderate elevations: • Low perfusion (↓ renal blood flow = ↑ BUN reabsorption • Dehydration, heart disease, haemorrhage, ischaemia (*+/-* ↑ *ALT*) • Also see ↑ creatinine, amylase, lipase, phosphorus ▪ **Renal (inappropriately dilute to isothenuric USG):** ➢ Renal damage (usually ↑ creatinine as well) **Post-renal (variable USG):** ➢ Rupture/obstructed ureter/bladder/urethra (*+/-* ↑ *K+*)	▪ ↓ Production: ➢ Hepatic insufficiency / PSS (↓ *Alb*, ↓ *glucose*, ↓ *urea*, ↑ *bile acids/bilirubin*) ➢ Low protein diet ▪ ↑ Urea excretion (*diuresis*): ➢ PU/PD (↓ *USG*) ➢ Diabetes insipidus (↓ *USG*) ➢ Renal failure (↓ *USG, +/- proteinuria*) ➢ Hyperadrenocorticism (↑ *ALP*, ↓ *USG*)

Ca+: Calcium

INCREASED:	DECREASED:
Neoplasia (parathyroid-like hormone) (↑ *Phos*, ↓ *PTH*) ➢ Lymphoma, anal gland adenocarcinoma ▪ Primary hyperparathyroidism (neoplasia) (↓ *Phos*, ↑ *PTH*) Secondary hyperparathyroidism: ➢ Renal disease (↑ *azotaemia*, ↑ *Phos*, ↑ *PTH*) ▪ Hyperalbuminaemia (↑ *PCV*, ↑ *Alb*, ↑ *TP*): ➢ Dehydration / haemoconcentration Bone disease (↑ *Phos*, *+/-* ↑ *ALP*): ➢ Multiple myeloma, leukaemia (*+/- lytic bone lesions*) ▪ Hypoadrenocorticism (*+/-* ↑ *K+*, ↓ *Na+*, *+/-* ↓ *glucose*) ▪ Vitamin D toxicity (↑ *Phos*, *+/- azotaemia*): ➢ Rodenticides (old types) (*coagulopathy*) Idiopathic (feline) – common cause ▪ **Clinical signs:** Weakness, PU/PD (toxic effect on kidneys and ↓ sensitivity to ADH), vomiting, diarrhoea, constipation, twitching, seizures See **"Fluid therapy"** for how to correct	▪ Due to hypoalbuminaemia (↓ *Alb*) ▪ Alkalosis (*Altered protein binding*) ▪ Milk fever ▪ Hypoparathyroidism/Parathyroidectomy ▪ Chronic renal failure (↓ *USG, +/-* ↑ *azotaemia*) ▪ Acute pancreatitis (*+/-* ↑ *lipase, amylase, vomiting*) ▪ Phosphate retention enema ▪ Oxalate poisoning ▪ Ethylene glycol (*Ca+ oxalate crystalluria*, ↑ *azotaemia, +/-* ↑ *K+*) ▪ Diet ▪ **Clinical signs:** Muscle tremors, fasciculation, seizures, ↑ temperature ▪ See **"Fluid therapy"** for how to correct

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Ionised Calcium: iCa+

Biologically active component (~50% of total serum calcium)

Hypoalbuminaemia → hypocalcaemia but no clinical signs if ionised calcium remains normal

INCREASED:	DECREASED:
▪ Acidosis (*altered protein binding*) ▪ Hypercalcaemia (**see above**) ▪ Can use formula to adjust for hypoalbuminaemia but direct measurement is more accurate ▪ See **"Fluid therapy"** for how to correct	▪ Alkalosis (*altered protein binding*) ▪ Severe hypocalcaemia (**see above**) ▪ See **"Fluid therapy"** for how to correct

Cholesterol:

INCREASED:	DECREASED:
▪ Hypothyroidism (↑ *ALP*) ▪ Hyperadrenocorticism (↑ *ALP*, ↓ *USG*) ▪ Corticosteroids ▪ Diabetes Mellitus (↑ *ALP*, ↑ *blood & urine glucose*) ▪ Cholestasis (↑ *ALP*, ↑ *GGT*, ↑ *bile acids*) ▪ Post-prandial ▪ Primary – rare	▪ Protein losing enteropathy (+/- vomiting and diarrhoea): ➢ Malabsorption ➢ Maldigestion ▪ Starvation ▪ Liver failure (+/- ↑ *liver enzymes*) ▪ Hypoadrenocorticism (+/- ↑ *K*, ↓ *Na+*, +/- ↓ *glucose*)

CK: Creatine Kinase

Produced in striated muscle

INCREASED:	DECREASED:
▪ Produced in striated muscle → organ-specific, muscle damage, nutritional, snake bite	

Creatinine:

Produced at a relatively standard rate by muscle metabolism

Used to evaluate kidney function as it filters creatinine from blood

INCREASED:	DECREASED:
▪ ↑ Muscle mass ▪ Pre-renal, renal, post-renal: ➢ See above **"BUN"**	▪ ↓ Muscle mass

Azotaemia	µmol/L	mg/dl
Mild		
- Dog	125-179	1.4 - 2
- Cat	140-249	1.6 - 2 8
Mod		
- Dog	180-439	2.1 - 5
- Cat	250-439	2.9 - 5
Severe		
- Dog	>440	>5
- Cat	>400	>5

GGT: Gamma-glutamyl transpeptidase

Bile duct epithelium

INCREASED:	DECREASED:
▪ Cholestasis (↑ *ALP*, ↑ *bilirubin & bile acids*) ▪ Drugs (corticosteroids, phenobarbitone) (↑ *ALP*)	

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Globulin:

INCREASED:	DECREASED:
▪ Dehydration *(↑ PCV, ↑ TP)* ▪ Chronic immune stimulation ▪ Neoplasia ▪ Inflammation *(↑ WBC)* ▪ Viral (FIV / FIP)	▪ ↓ Production: ➢ Liver failure *(↓ Alb)* ➢ ↓ Plasma cells and lymphocytes *(normal Alb)* ➢ Malabsorption *(↓ Alb)* ▪ ↑ Loss with albumin: ➢ Blood loss *(↓ PCV)* ➢ Burns ➢ PLE *(↓ Alb)* ▪ Dilution *(↓ PCV, ↓ TP)*

Glucose:

Measure of endocrine function of the pancreas

INCREASED:	DECREASED:
▪ Post-prandial ▪ Fear/excitation/stress ▪ Hyperadrenocorticism *(↑ ALP, ↓ USG)* ▪ Corticosteroids *(+/- ↑ ALP)* ▪ Ethylene glycol *(Ca+ oxalate crystalluria, ↑ azotaemia, +/- ↑ K+)* ▪ Progesterone ▪ Diabetes mellitus (type 1 and type 2) *(↑ ALP, ↑ urine glucose)* ▪ **Clinical signs:** Polydipsia, polyuria, depression, weight loss, obesity and polyphagia ▪ See **"Fluid therapy"** for how to correct	▪ **< 3 mmol/l → Hypoglycaemia → emergency** ▪ Starvation/malabsorption ▪ Hepatitis, hepatic insufficiency, portosystemic shunt, neoplasia *(↓ Alb, ↓ urea, +/- ↑ bile acids, ALP, ALT)* ▪ Pregnancy ▪ Heat stroke ▪ Severe hypothermia ▪ Hypoadrenocorticism *(+/- ↑ K+, ↓ Na+)* ▪ Acute severe illness – sepsis, multiple organ dysfunction syndrome, hepatic failure ▪ Hyperinsulinaemia – insulinoma, overdose of insulin ▪ Neonatal and toy breed hypoglycaemia ▪ **Clinical signs:** Trembling, weakness, ataxia, collapse/unconscious, blindness, seizures ▪ See **"Fluid therapy"** for how to correct

Lipase:

INCREASED:	DECREASED:
▪ Pancreatitis (>3 x ↑): ➢ ↑ Sensitivity c.f. amylase, but 30% can have normal levels ▪ Gastrointestinal disease (>3 x ↑) *(+/- vomiting and diarrhoea)* ▪ Renal disease (2-3 x ↑) *(↑ azotaemia)* ▪ Steroids/hyperadrenocorticism (up to 5x ↑) *(↑ ALP, ↓ USG)*	

Phos: Phosphate

INCREASED:	DECREASED:
▪ Secondary hyperparathyroidism: ➢ Renal disease *(↑ Ca+, azotaemia)* ➢ ↑ Nutritional hyperparathyroidism (↑↑ dietary P intake) ▪ Bone disease *(↑ Ca+, ↑ ALP)*	▪ Recent stress or excitement ▪ Primary hyperparathyroidism (neoplasia) *(↑ Ca+)* ▪ Insulin therapy or hyperinsulinaemia *(↓ glucose)* ▪ Milk fever and eclampsia *(↓ Ca+)* ▪ ↓ Dietary intake ▪ **Clinical signs:** Asymptomatic or see weakness, seizures, haemolytic anaemia

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Total Protein:	
INCREASED: ▪ Dehydration *(↑ PCV)* ▪ Hyperfibrinogenaemia due to inflammatory or neoplastic disease ▪ Hyperglobulinaemia due to infection, inflammation *(↑ globulin)*	**DECREASED:** ▪ Over hydration/dilution *(↓ PCV)* ▪ ↓ Production (liver) *(+/- ↓ urea, +/- ↑ bile acids, ALP, ALT)* ▪ ↑ Loss of albumin: ➢ Protein losing enteropathy *(+/- vomiting and diarrhoea, +/- hypoglobulinaemia)* ➢ Protein losing nephropathy *(proteinuria, no hypoglobulinaemia)* ➢ Haemorrhage ➢ Burns ▪ Acute tissue injury
Urea – see above "BUN":	

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Cardiovascular Disease

- **This chapter covers:**
  - ✓ Basic principles of a cardiovascular examination
  - ✓ Diagnostic tests
  - ✓ Principles of heart disease and commonly seen conditions
  - ✓ Treatment principles

Cardiovascular examination:

- Mucous membrane colour and capillary refill time
- Blood oxygen saturation (SPO_2)
- Cardiac auscultation, **see below**
- Femoral pulse pressure/tone:
  - ✓ Pulse pressure: Degree of collapsibility or resistance to compression
  - ✓ Rate should be in time with heartbeat
  - ✓ Pulse pressure variations:
    - ➢ Reduced pulse pressure: Due to reductions in systolic blood pressure due to reduced cardiac output e.g. hypovolemia or cardiogenic shock
    - ➢ Increased pulse pressure: Either due to reduced diastolic pressure or increased systolic pressure (e.g. Hyperdynamic shock due to sympathetic stimulation)
  - ✓ Pulse deficits:
    - ➢ Premature heart contraction, before adequate ventricular filling
  - ✓ Pulsus paradoxus:
    - ➢ Increased pulse pressure during inspiration, usually seen with pericardial effusion
- Jugular vein distension:
  - ✓ Distension or failure to collapse after holding off can indicate right side CHF
- Respiration: **See "Respiratory Disease"**
  - ✓ Inspiratory dyspnoea: Pleural disease (e.g. effusion), upper airway obstructive disease
  - ✓ Inspiratory/expiratory dyspnoea: Pulmonary disease (e.g. oedema due to left sided CHF), +/- lower airway obstructive disease

Cardiac auscultation:

- **Heart rate and rhythm:**
- Arrhythmias:
  - ✓ Dogs: Usually due to extracardiac causes (unless Doberman or Boxer or severe mitral valve disease)
  - ✓ Cats: Usually due to cardiac disease
- Gallop rhythms:
  - ✓ Sequence of three sounds – normal S1 and S2 with a S3 or S4:
    - ➢ S3 sound = indicates heart disease is present, seen with DCM, severe mitral valve disease or HCM
    - ➢ S4 sound = usually associated with HCM in cats (either idiopathic or secondary to hyperthyroidism or hypertension)

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- **Murmurs:**
- Intensity: Grade I - VI

Grade:	Description:
I	Very soft, **not immediately audible**, careful auscultation in a quiet environment
II	Soft murmur that is audible with careful auscultation
III	Moderate murmur **immediately audible** with auscultation
IV	Loud murmur without a thrill
V	Loud murmur with a **palpable thrill**
VI	Audible with stethoscope held slightly off chest wall

- **Location:**
  - ✓ Left or right, base or apex
- **Timing:**
  - ✓ S1 sound = Systolic = Atrioventricular valve closure = "LUBB"
  - ✓ S2 sound = Diastolic = Aortic and pulmonary valve closure = "DUBB"
- **Feline murmurs:**
  - ✓ 25-75% of heart murmurs are innocent
  - ✓ 50% of hypertrophic cardiomyopathy patients have no murmur
  - ✓ 50% of hypertrophic cardiomyopathy patients with congestive heart failure have no murmur
  - ✓ Gallop and/or irregular rhythms almost always indicates cardiac disease
- **Innocent murmurs:**
  - ✓ Young pets, disappear between 3-6 months of age
  - ✓ Systolic murmur, less than grade 3 or 4
  - ✓ No signs of congestive heart failure or syncope
  - ✓ Echocardiology if persistent

Diagnostics:

- **Blood Pressure:**

Blood Pressure:	Dog:	Cat:
Normal	120/80mmHg	110/70mmHg
Hypertension	>160/110mmHg Treat if systolic BP >170mmHg	>170/120mmHg Treat if systolic BP >180mmHg

- **Electrocardiogram interpretation:**
  - ✓ What is the heart rate?:
    - ➢ Is it a tachycardia or bradycardia?
  - ✓ Is the rhythm regular or irregular, i.e. are the R-R intervals even?
  - ✓ Assess the QRS complexes:
    - ➢ P wave followed by QRS
    - ➢ QRS preceded by P wave
    - ➢ Assess the QRS complex:
      - Normal then supraventricular
      - Wide and bizarre then ventricular
    - ➢ Assess the timing of ectopic (abnormal) beats:
      - If it occurs earlier than expected, then it is "premature":
        - o If greater than 3 in a row, then it is a tachycardia can be paroxysmal or sustained
      - If it occurs later than expected, then it is "escape"

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- **Other diagnostics:**
  - ✓ Biochemistry:
    - ➢ Assess for concurrent organ failure due to reduced cardiac output
  - ✓ Cardiac ultrasound
  - ✓ Radiographs – see "**Radiology**":
    - ➢ Typical features that may be seen with heart disease:
      - Cardiac silhouette or chamber enlargement:
        - o LHS enlargement common with valvular disease
        - o Variable chamber enlargement with dilated cardiomyopathy
        - o Globular heart, loss of chamber outline with pericardial effusion & dilated cardiomyopathy
      - Elevation of the trachea and compression of the main stem bronchi
      - +/- Congestion of pulmonary veins and interstitial/alveolar pattern due to pulmonary oedema with LHS congestive heart failure
      - +/- Dilated caudal vena cava, hepatomegaly, ascites and pleural/pericardial effusion with RHS congestive heart failure

Features of heart disease:

- **Types of heart failure:**
- Forward failure:
  - ✓ Reduced cardiac output: Exercise intolerance, weakness, syncope, pale mucous membranes, hypotension, reduced urine output
- Backward failure:
  - ✓ LHS backward failure: Congestion of the pulmonary veins causing pulmonary oedema
  - ✓ RHS backward failure: Congestion of vena cava causing pleural effusion, hepatomegaly, ascites

- **Canine heart disease:**
- Valvular endocardiosis disease (myxomatous valvular disease):
  - ✓ Most commonly small breeds
  - ✓ Murmur and commonly LHS congestive or backward heart failure, **see above**
  - ✓ Intensity of the murmur in large breed dogs with mitral valve disease does not always correlate with severity of the disease
- Dilated cardiomyopathy (DCM):
  - ✓ Commonly medium to large breeds >20kg
  - ✓ May hear heart murmur but arrhythmias are more common (ventricular premature complexes, ventricular tachycardia and atrial fibrillation)
  - ✓ Weak pulses, lethargy, syncope and both or either forms of congestive heart failure, **see above**
  - ✓ Boxers and Dobermans can develop secondary DCM from electrical disease
- Pericardial effusion:
  - ✓ More common in large breeds
  - ✓ Muffled heart sounds and weak pulses with pulsus paradoxus (increased pulse intensity with inspiration)
- Primary electrical disease:
  - ✓ Dobermans and Boxers are predisposed to a primary electrical disease that leads to DCM
  - ✓ Arrhythmia (ventricular ectopy, atrial fibrillation), collapse, sudden death

- **Feline heart disease:**
  - ✓ Most commonly caused by cardiomyopathy, rarely primary valvular disease
  - ✓ Hypertrophic cardiomyopathy is the most common cause of heart disease in cats (approx. 60%):
    - ➢ Idiopathic: Defect in myocardial sarcomeres, compensate with concentric hypertrophy
    - ➢ Secondary to hypertension: Chronic renal disease, hyperthyroidism etc.

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- ✓ Heart sounds:
  - ➢ 25-75% of heart murmurs are innocent
  - ➢ 50% of hypertrophic cardiomyopathy patients have no murmur
  - ➢ 50% of hypertrophic cardiomyopathy patients with congestive heart failure have no murmur
  - ➢ Gallop and/or irregular rhythms almost always indicates cardiac disease
- ✓ Presenting symptoms:
  - ➢ Dyspnoea due oedema (pulmonary or pleural) – usually triggered by stressful event
  - ➢ Limb paresis/paralysis from thromboembolism
  - ➢ Hypothermia
  - ➢ Tachycardia and arrhythmias

- **Common causes of murmurs in cats:**
  - ✓ Hypertrophic cardiomyopathy (HCM) (~50%) with systolic anterior motion (SAM) of the mitral valve:
    - ➢ Concentric hypertrophy leads to pathological thickened papillary muscles that pull mitral valve open
  - ✓ Dynamic Right Ventricular Outflow Tract Obstruction (~50%):
    - ➢ Non-pathological (< grade 5)
    - ➢ Mild obstruction with no haemodynamic consequences

Management of heart failure:

- **Myxomatous mitral valve degeneration:**
  - ✓ **Run a full blood test (complete blood count, biochemistry, electrolytes) to assess for concurrent organ damage**

- **Pulmonary oedema:**
  - ✓ **Acute:**
    - ➢ Oxygen therapy:
      - Mask (10L/min), cage, nasal (50-100ml/kg/line)
    - ➢ Furosemide 4-6mg/kg IV q2hrs until see reduction in respiratory rate and effort, then 2mg/kg IV q2-4hrs OR CRI at 0.6mg/kg/hr after an initial 0.6mg/kg IV (VERY potent can rapidly dehydrate):
      - Monitor respiratory rate, SPO_2, azotaemia and electrolytes
      - Continue high doses once see a 30% reduction in respiratory rate and effort reduce dose
      - Offer water once diuresis begins
      - Once pulmonary oedema has resolved then continue maintenance therapy 2-4mg/kg PO BID-TID, dose needs to be higher than previous maintenance dose
    - ➢ Pimobendan 0.25mg/kg PO BID
    - ➢ Sedation to improve breathing dynamics and reduce stress e.g. Butorphanol 0.1-0.2mg/kg IM
    - ➢ +/- Afterload reduction via vasodilation
      - Sodium nitroprusside CRI, start low and titrate
      - Must monitor invasive blood pressure, maintain MAP >70mmHg
    - ➢ Preload reduction via venodilation:
      - 2% nitroglycerin patch or ointment
  - ✓ **Chronic:**
    - ➢ Furosemide:
      - **Most important drug**
      - Monitor resting respiratory rate (aim for <25 breaths per minute whilst sleeping)
      - Start at 2mg/kg BID then increase to TID, then increase by 1mg/kg per dose until 4mg/kg TID
    - ➢ ACE inhibitors:
      - E.g. Benazepril 0.25-0.5mg/kg PO SID
      - NOT if azotaemic (especially if high creatinine) and must be well hydrated before starting

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- Pimobendan 0.25mg/kg PO BID
- Spirolactone 2mg/kg PO SID:
  - Use to counter aldosterone escape, as chronic use of ACE inhibitor will lead to aldosterone escape
- Dietary management: May help by avoiding high salt foods
- Monitoring:
  - Diary of resting respiratory rate (aim for <25 breaths per minute whilst sleeping)
  - Recheck for azotaemia and electrolytes 1 week after increasing diuretic dose or after starting ACE inhibitor then every 3 months
  - Stop ACE inhibitor if azotaemia is significant (especially creatinine)
  - If not responding perform chest radiographs to assess for pulmonary oedema, may require dose increase

✓ **Refractory:**
- Furosemide:
  - Increase furosemide dose **(see above)**
  - Addition of another diuretic e.g. Spirolactone at 2mg/kg PO SID up to 4mg/kg PO BID
- Increase ACE inhibitor and pimobendan to maximum doses
- +/- Antihypertensive: **See "Hypertension"**
  - Amlodipine 0.1mg/kg PO SID
- Dietary management: May help by avoiding high salt foods

✓ Complicated: e.g. **Uremic with congestive heart failure**
- Decide between diuretics or IV fluids: Treat the condition that is currently more life threatening

✓ Refractory coughing:
- Rule out pulmonary oedema
- Assess for main stem bronchial compression (lateral view):
  - Tramadol 2mg/kg PO BID-TID
  - Codeine 0.5mg/kg PO QID

- **Pulmonary hypertension:**

✓ Require ultrasound to diagnose

✓ Sildenafil

✓ Acute pulmonary hypertension, treat as per acute management of pulmonary oedema

- **Arrhythmias:**

✓ Supraventricular tachycardia, may also see atrial fibrillation but only in the most advanced stages of atrial disease

✓ Anti-arrhythmic therapy, see **"Management of arrhythmias"** following

- **Dilated cardiomyopathy:**

✓ **Run a full blood test (complete blood count, biochemistry, electrolytes) to assess for concurrent organ damage**

- **Preclinical:**

✓ ACE inhibitors e.g. Benazepril 0.5 mg/kg PO SID can delay onset

- **Pulmonary oedema:**

✓ **Acute:**
- Oxygen therapy:
  - Mask (10L/min), cage, nasal (50-100ml/kg/line)
- Furosemide 4-6mg/kg IV q2hrs until see reduction in respiratory rate and effort, then 2mg/kg IV q2-4hrs OR CRI at 0.6mg/kg/hr after an initial 0.6mg/kg IV (VERY potent can rapidly dehydrate):
  - Monitor respiratory rate, SPO_2, azotaemia and electrolytes
  - Continue high doses once see a 30% reduction in respiratory rate and effort reduce dose
  - Offer water once diuresis begins

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- Once pulmonary oedema has resolved then continue maintenance therapy 2-4mg/kg PO BID-TID, dose needs to be higher than previous maintenance dose
- ➢ Pimobendan 0.25mg/kg PO BID
- ➢ Sedation to improve breathing dynamics and reduce stress e.g. Butorphanol 0.1-0.2mg/kg IM
- ➢ +/- Afterload reduction via vasodilation
  - Sodium nitroprusside CRI, start low and titrate
  - Must monitor invasive blood pressure, maintain MAP >70mmHg
- ➢ Preload reduction via venodilation:
  - 2% nitroglycerin patch or ointment

✓ **Chronic:**

➢ Treat as for myxomatous mitral valve disease

- **Ascites:**
  - ✓ Abdominocentesis:
    - ➢ Repeated abdominocentesis can cause electrolyte abnormalities and hypoproteinaemia
  - ✓ Diuretics:
    - ➢ Spirolactone 1-2mg/kg PO BID (as a diuretic and to counter aldosterone escape)
    - ➢ Furosemide 1-2mg/kg PO SID-TID
  - ✓ ACE inhibitor e.g. Benazepril 0.25 - 0.5mg/kg PO SID
- **Arrhythmias:**
  - ✓ Ventricular tachyarrhythmia and atrial fibrillation
  - ✓ Anti-arrhythmic therapy, see **"Management of arrhythmias"** following

- **Feline hypertrophic cardiomyopathy:**
  - ✓ **Run a full blood tests (complete blood count, biochemistry, electrolytes) to assess for concurrent organ damage also thyroid testing**
  - ✓ NOTE: Cats are prone to dehydration – beware pre-renal azotemia and hypokalaemia

- **Pulmonary oedema:**
  - ✓ **Acute:**
    - ➢ Oxygen therapy:
      - Mask (10L/min), cage, nasal (50-100ml/kg/line)
    - ➢ Diuretics:
      - Furosemide 2-4mg/kg IV q1hr to stabilize, reduce to 1-2mg/kg q2-4hrs when respiratory rate and effort reduces, then maintenance 1mg/kg SID for maintenance
      - Feline doses are significantly lower than canine doses
    - ➢ +/- Afterload reduction via vasodilation
      - Nitroglycerine ointment (¼ inch topically applied q6-8hr)
  - ✓ **Chronic:**
    - ➢ Furosemide 1-2mg/kg PO BID
    - ➢ ACE inhibitor e.g. Benazepril 0.25 - 0.5mg/kg PO SID
- **Arrhythmias:**
  - ✓ Supraventricular tachycardia
  - ✓ Anti-arrhythmic therapy, see **"Management of arrhythmias"** following
- **Pleural effusion:**
  - ✓ Oxygen therapy
  - ✓ Thoracentesis
  - ✓ Diuretics:
    - ➢ Furosemide 2mg/kg IV q1hr to stabilize, reduce to 1-2mg/kg q2-4hrs when respiratory rate and effort reduces, then maintenance 1mg/kg SID for maintenance
    - ➢ ACE inhibitor e.g. Benazepril 0.25 - 0.5mg/kg PO SID

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- **Anti-thrombotic therapy:**
  - ✓ See **"Aortic thromboembolism"** next
- **Dietary management:**
  - ✓ Avoid high salt foods
  - ✓ Moderate salt restriction (e.g. kidney diets) only for animals on high diuretic doses

Associated cardiovascular conditions:

- **Aortic thromboembolism:**
- **Pathophysiology:**
  - ✓ Common complication of feline heart disease
  - ✓ Requires injury to the myocardial endothelium + hypercoagulability + blood stasis (e.g. swirling of blood in an enlarged left atrium) → thrombus formation → embolism → small clot to right arm OR large clot to pelvis (70%) → **PAIN + PARALYSIS + PULSELESSNESS**
- **Clinical signs:**
  - ✓ Sudden and severe pain
  - ✓ Paralysis: Hind or forelimb
  - ✓ Loss of pulses
  - ✓ Cold limbs and pale pads
  - ✓ Dyspnoea +/- cyanosis
  - ✓ Gallop rhythm and/or heart murmur
- **Diagnosis:**
  - ✓ Clinical signs
  - ✓ Echocardiology and ultrasound (of clot)
  - ✓ Radiographs: Chest to assess for pulmonary oedema
  - ✓ Biochemistry and blood gas: Assess renal function, electrolytes and acid-base status
- **Prognosis:**
  - ✓ Improved if upper thigh control, normal mental activity and breathing, body temperature and anal tone
  - ✓ Guarded if concurrent CHF
- **Management:**
  - ✓ Immediate:
    - ➢ Pain relief e.g. Pure or partial opioid receptor agonist
    - ➢ If in CHF diuretics and anti-arrhythmic (if indicated)
    - ➢ If not in CHF IV fluids and correction of electrolyte derangements
  - ✓ Thrombolytics:
    - ➢ No longer readily available, e.g. Streptokinase (must be within 6 hours and has associated risks i.e. coagulopathy)
  - ✓ Anti-thrombotic:
    - ➢ Does not lyse clot but help prevent formation of more:
      - Deltaprine 100IU/kg SC BID or aspirin 10mg/kg PO q48-72 hours
  - ✓ Reperfusion injury (6 – 24 hours):
    - ➢ Monitor intensively for hyperkalaemia, metabolic acidosis, renal failure, arrhythmia
  - ✓ Long term:
    - ➢ Treatment of underlying cardiac disease
    - ➢ Prevent re-embolisation:
      - Clopidogrel 20mg/cat PO SID or aspirin 10mg/kg PO q48-72 hours
    - ➢ Physical therapy

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- **Systemic hypertension:**
- **Causes:**
  - ✓ Stress (artefact), hyperthyroidism (occasionally), hyperadrenocorticism, chronic renal disease, diabetes mellitus, medication (corticosteroids, NSAIDs, mineralocorticoids), idiopathic, pheochromocytomas
- **Target organ damage:**
  - ✓ Can result from sustained hypertension
    - ➢ Moderate risk = systolic >160mmHg
    - ➢ Severe risk = systolic >180mmHg
  - ✓ Organs affected:
    - ➢ Eyes: Retinal detachment, haemorrhage
    - ➢ Cardiac: Systolic murmurs, gallop rhythms, HCM
    - ➢ Kidneys: Azotaemia and proteinuria (higher the proteinuria the worse the prognosis)
    - ➢ Epistaxis
    - ➢ CNS: Seizures, altered mentation, lethargy, head tilt, nystagmus, ataxia, vestibular signs etc.
- **Diagnosis:**
  - ✓ Blood pressure measurements – invasive arterial blood pressure measurements are best
    - ➢ Repeated measurements over the day, least stressful environment is best
  - ✓ Biochemistry, urinalysis (UP:C), fundic examinations and echocardiology, thyroid hormone testing, ACTH stimulation tests/LDDST test
- **Management:**
  - ✓ Aim to reduce blood pressure <150mmHg to reduce risk of end-organ damage
  - ✓ Prognosis has been linked with proteinuria, if no reduction in UP:C then no improvement in prognosis:
    - ➢ ACE inhibitor:
      - Benazepril at 0.5 - 1.0mg/kg PO SID
      - Only mild reduction in blood pressure approximately 5-15mmHg
      - Can combine with calcium channel blocker treatment (amlodipine)
    - ➢ Amlodipine:
      - Cat: 0.7-1.2mg/cat/day, dog 0.1mg/kg
      - Average reduction approximately 40-50mmHg
      - Must monitor electrolytes as can get hypokalaemia
  - ✓ Monitoring:
    - ➢ Re-evaluate blood pressures one week after starting or altering dosage
    - ➢ If severe hypertension and progressive neurological signs, then every 1 to 2 days
    - ➢ Repeat biochemistry, urinalysis, UP:C, fundic exams to monitor target end organ damage

- **Pericardial effusions:**
- **Pathophysiology:**
  - ✓ Common in dogs especially middle age to older, rare in cats
  - ✓ Intrapericardial pressure rises to equal or greater than normal cardiac filling pressure
  - ✓ The faster the rate of fluid accumulation the less fluid required to result in tamponade
  - ✓ Tamponade limits right ventricular filling, when severe also limits left ventricular filling resulting in reduced cardiac output and blood pressure
- **Clinical signs:**
  - ✓ Signs are due to reduced cardiac output
  - ✓ Weakness and lethargy
  - ✓ Muffled heart sounds, tachycardia, jugular distension, poor pulses +/- pulsus paradoxus
  - ✓ Tachypnoea and dyspnoea
  - ✓ Chronic cases can lead to hepatomegaly and ascites
- **Causes:**
  - ✓ >70% neoplastic: Haemangiosarcoma, malignant mesothelioma and heart base tumours
  - ✓ Other: Include idiopathic, traumatic, rupture left atrium, coagulopathy

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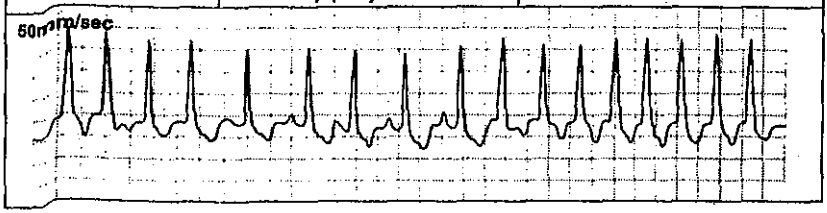
Diagnosis:
- ✓ Echocardiography: Free fluid between epicardium and parietal pericardium
- ✓ Radiographs: Large cardiac silhouette
- ✓ Electrocardiogram: Sinus tachycardia with electrical alternans or reduced QRS complex
- ✓ Other: Biochemistry and abdominal ultrasound

Treatment:
- ✓ Emergency pericardiocentesis to reduce tamponade
- ✓ +/- Treatment of the underlying disease

Abnormalities of heart rate or conduction:

Clinical signs:
- ✓ Both bradycardia and tachycardia cause the same clinical signs
- ✓ Weakness, syncope, pale mucous membranes, hypotension and reduced urine output

Tachycardia:	Cause/clinical signs:	ECG:
Sinus tachycardia: • Dogs: >180bpm • Puppies: >220bpm • Cat: >220bpm	• Increased SA node depolarisation due to extra-cardiac factors: ➢ Increased sympathetic activity ➢ Hypokalaemia ➢ Anaemia ➢ Hyperthyroidism	• Normal P-QRS-T but faster than normal
Supraventricular tachycardia (SVT): (image below) • A series of premature supraventricular beats	• Depolarisations originating from above the ventricles but NOT from the SA node: ➢ Atrial ectopic foci ➢ Cardiomyopathy causing atrial enlargement ➢ Valvular disease ➢ Drugs (anesthetics) • Heart rate >160bpm • No CSx but if HR > 300bpm can cause episodic weakness/syncope • Can develop dilated cardiomyopathy if sustained	• Usually normal P-QRS-T but faster than normal • P waves may be hidden in the preceding T wave



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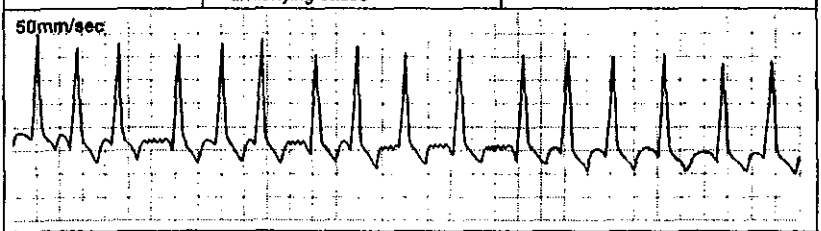
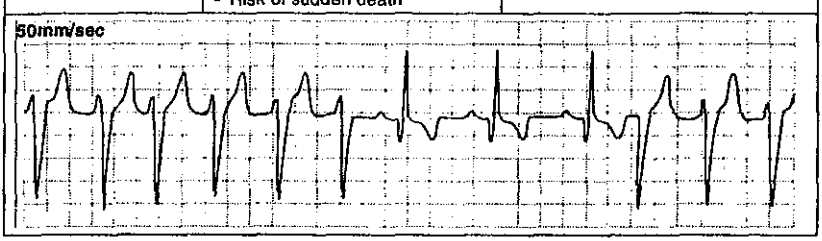
Atrial fibrillation: (image below)	• Rapid and irregular depolarisations originating in the atria (not SA node) • Usually due to profound cardiac disease and cardiac remodeling i.e. DCM, severe mitral valve disease, Irish wolfhounds (primary), cats (poor prognostic indicator) • CSx usually due to the underlying cause	• No P waves • Irregular RR intervals • Narrow supraventricular QRS complexes • Baseline moves up and down



Ventricular Tachycardia: (image below) • A series of ventricular premature complexes (VPC) • VPC are a risk factor for sudden death in Boxers, Dobermans	• Depolarisations originating from the ventricles: ➢ Cardiomyopathy ➢ Hypovolemia ➢ Hypoxemic states (anaemia, GDV, hypoxia) ➢ Hypokalaemia ➢ Hypomagnesaemia ➢ Neoplasia/SIRS ➢ Metabolic acidosis • Can be asymptomatic but can cause weakness/collapse • Risk of sudden death	• **Wide bizarre QRS complexes** • P waves not coincide with QRS complexes • Due to a series of ventricular premature complexes



Bradycardia:	Cause/clinical signs:	ECG:
Sinus bradycardia: • Dogs: <70bpm • Cat: <100bpm	• Reduced depolarisation from the SA node due to extra-cardiac factors: ➢ Increased parasympathetic activity (respiratory disease, GIT disease, CNS disease, ocular disease) ➢ Hypothyroidism ➢ Hypothermia ➢ Hyperkalaemia ➢ Hypoglycaemia	• Normal complexes but the rate is slower than normal

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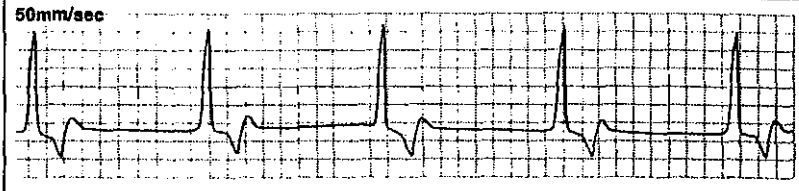
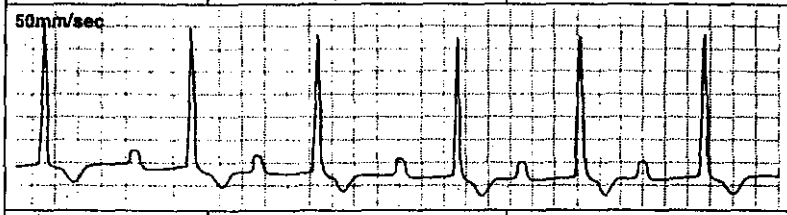
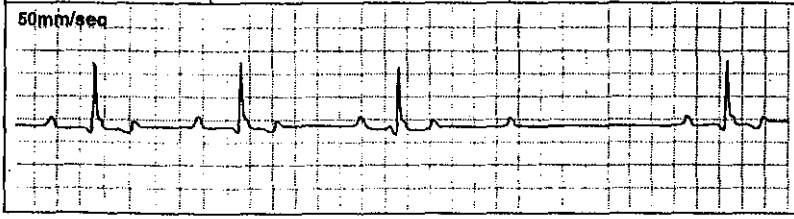
Atrial standstill: (image below)	▪ Disease of the atrial muscle or atrial myocytes are not stimulated by the SA node: ➢ Atrial muscle damage ➢ Hyperkalaemia (>8.5mEq/L) ▪ Normal heart sounds heard ▪ Heart rate usually <60 bpm ▪ Usually lethargy, sometimes syncope	▪ No P waves ▪ QRS complexes are normal ▪ QRS complexes can be wide if caused by hyperkalaemia



1st degree AV block: (image below)	▪ **Delay** in conduction through AV node: ➢ Disease of AV node ➢ High resting parasympathetic tone (see **Sinus Bradycardia**) ▪ Usually no clinical signs and does not require treatment	▪ All P waves are conducted ▪ Prolonged PR interval ▪ P and QRS-T complex are normal



2nd degree AV block: (image below)	▪ **Occasional failure** of conduction through AV node ▪ Disease of the AV node or high parasympathetic tone (see **Sinus Bradycardia**) ▪ Treatment if CSx of exercise intolerance, weakness, syncope ▪ Mobitz 2: ➢ Can potentially lead to 3rd degree AV block	▪ Only some P waves are conducted ▪ i.e. only some P waves are followed with a QRS-T complex ▪ Mobitz Type 1: ➢ PR interval gets longer prior a P wave not followed by a QRS ▪ Mobitz Type 2: ➢ PR interval remains consistent



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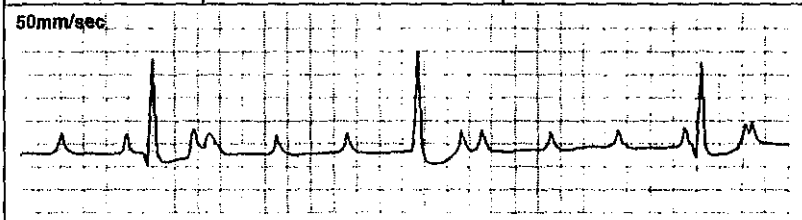
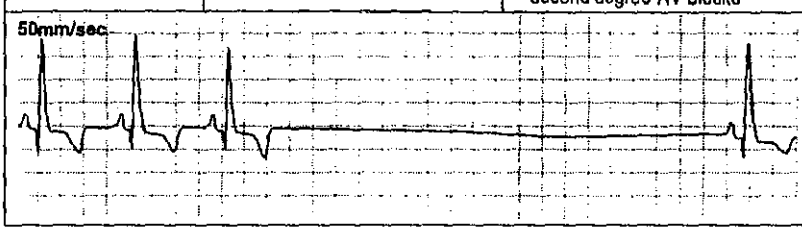
3rd degree AV block: (image below) ▪ Dog ventricular rate: 40-65bpm ▪ Cat ventricular rate: 90-120bpm	▪ **Persistent** failure of conduction through AV node ▪ AV node pathology ▪ Treat if see weakness, collapse	▪ Complete disassociation between P waves and QRS-T ▪ P waves are faster and do not coincide with the QRS-T complexes ▪ A ventricular focus becomes the pacemaker



Sick sinus syndrome: (image below)	▪ Disease of the SA node that can affect the entire cardiac conduction system ▪ See weakness and collapse	▪ Sinus bradycardia and sinus arrest ▪ +/- Supraventricular tachycardia ▪ Also see combinations of first and second degree AV blocks



- **Management of arrhythmias:**
- **Arrhythmias:**
  - ✓ **Must rule out extracardiac causes of arrhythmias**
  - ✓ **Dogs likely to be non-cardiac related, cats likely to cardiac related**

- **Tachycardia:**
  - ✓ Supraventricular tachycardia:
    - ➢ Acute stabilisation:
      - Diltiazem 0.05mg/kg IV over 10mins OR
      - Procainamide 2mg/kg IV repeated up to a max of 15mg/kg
    - ➢ Long term:
      - Diltiazem, atenolol or propranolol, sotalol
      - Beware with β-blockers and calcium channel blockers with myocardial failure as they are negative inotropes
  - ✓ Atrial fibrillation:
    - ➢ Digoxin (first choice) or diltiazem, can use a combination of both
  - ✓ Ventricular premature complexes:
    - ➢ Only treat if:
      - Symptoms of reduced cardiac output e.g. Weakness, collapse, hypotension, reduced urine output, pale mucous membranes and slow capillary refill time
      - If heart rate is continuously >180bpm

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- Multiform QRS complexes (QRS complexes of varying size), indicates multifocal disease
- R on T rhythm (R wave is lost in the preceding T wave)
- Ruled out extra-cardiac causes of VPC's

✓ Ventricular Tachycardia:

➢ Acute stabilisation:

- Slow IV bolus of lignocaine 2mg/kg can repeat every 10 minutes for a max dose of 6mg/kg. If responds, then start lignocaine CRI at 40-80µg/kg/min
- Do not try to stop all VPC's and monitor for lignocaine toxicity
- If not responding to lignocaine, procainamide 2mg/kg IV repeated up to a max of 15mg/kg

➢ Long term:

- Sotalol or sotalol and mexiletine combination

Bradycardia:

✓ Rule out hyperkalaemia and causes of high vagal tone such as severe respiratory, gastrointestinal, ocular or central nervous system disease, hypothyroidism

✓ Atropine trial: Eliminates vagal tone

➢ If positive response (heart rate increases by 100% or >150bpm) then bradycardia is due to excessive vagal tone, can repeat if partial response

➢ If still no response then atrial standstill, if increase in P waves but not QRS then pathological AV blocks ($2^{nd}/3^{rd}$ degree), these usually require pacemaker implantation

✓ Medical response is usually temporary and ultimately pacemaker implantation is necessary:

➢ Sympathetic comimetics:

- Terbutaline or clenbuterol

➢ Parasympatholytics:

- Propantheline bromide (most commonly used)

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Coagulopathy

- **This chapter covers:**
  - ✓ Basic overview of haemostasis and basic diagnostics tests
  - ✓ Information on common causes of coagulopathies and treatment principles

- **Pathophysiology:**
  - ✓ Coagulation is a complex interaction between cells, platelets and coagulation proteins, it is divided into primary and secondary haemostasis to simplify the understanding of the diagnostic process.

Primary haemostasis: **"Problems with platelet plug formation"**	
Clinical signs:	**Causes:**
▪ Ecchymosis and **petechiae** ▪ See on skin and mucous membranes ▪ Hyphema ▪ Epistaxis ▪ Haematuria and melena	▪ **Thrombocytopenia – reduced platelet numbers:** ➢ Reduced platelet numbers (<30x10^9/L) ▪ ***Increased consumption or destruction:*** ➢ Disseminated intravascular coagulation, severe haemorrhage, immune mediated thrombocytopenia (primary or secondary) ▪ ***Decreased production:*** ➢ Drug induced: Estrogen, antibiotics (TMS), chemotherapy, antifungals ➢ Primary bone marrow disease: • Infections: FeLV, FIV, FIP, Parvo/distemper virus, fungal, Rickettsial, *Ehrlichia* • Neoplasia, myelofibrosis
	▪ **Thrombocytopathia – abnormal platelet function:** ➢ Normal platelet numbers (>30x10^9/L) and **increased BMBT** ▪ ***Hereditary:*** ➢ Von Willebrand's disease (e.g. Dobermans, German Shepherd) ▪ ***Acquired:*** ➢ Systemic lupus erythematosus, hepatic and renal disease, hypothyroidism, viral (FeLV), myeloproliferative disease, neoplasia ➢ Drugs e.g. aspirin, ibuprofen
	▪ **Vessel wall defects:** ➢ Very uncommon: vasculitis, hyperadrenocorticism
Secondary haemostasis: **"Problems with activation of clotting factors"**	
Clinical signs:	**Causes:**
▪ Severe bleeding internally or externally ▪ Cavity bleeding and haematomas ▪ Bleeds deep into subcutaneous tissues, body cavities, joints ▪ Epistaxis and haemoptysis	▪ ***Hereditary:*** Usually single factor defects ➢ Individual factor deficiency, Devon Rex (Vitamin K dependant factors), haemophilia A/B
	▪ ***Acquired:*** Usually multiple defects ➢ **Reduce production:** • Hepatic disease e.g. necrosis, cirrhosis, portosystemic shunts • Vitamin K disorders (II, VII, IX, X) e.g. rodenticide toxicity, bowel disease, chronic antibiotics ➢ **Activation and consumption:** • Snake bite • Disseminated intravascular coagulation ➢ **Dilution of factors:** • Blood loss replaced with fluids deficient in clotting factors (crystalloids)

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Diagnostics tests:		
• **Severity of blood loss:**	• **PCV/TP and assessment for internal bleeding** ➢ Significant blood loss can result in a secondary haemostasis disorder	
• **Primary haemostasis:** • Ecchymosis and **petechiae** • See on skin and mucous membranes • Hyphema • Epistaxis • Haematuria and melena	• **Haematology**	• Assess platelet number, +/- platelet width • Clumping can artificially reduce platelet counts
	• **Blood smear**	• Assess platelet number: ➢ If clumping, then enough platelets for coagulation ➢ Usually no clinical signs of bleeding if platelet numbers are greater than $30x10^9/L$ ➢ Minimum number of platelets per high powered field: • 1 platelet on a smear = $15x10^9/L$ • >5 for dogs, >7 for cats ➢ **If low, then still assess secondary haemostasis** • Assess platelet morphology: ➢ Large platelets indicate regenerative response • Also look at RBC morphology: ➢ Schistocytes/spherocytes
	• **Buccal mucosal bleeding time (BMBT)**	• If normal platelet numbers BMBT tests platelet function and vessel wall function: ➢ Dogs: 2 – 4 minutes ➢ Cats: 1 – 2.5 minutes • If normal platelet count but prolonged BMBT then indicates thrombocytopathia or vessel wall function • Next step is vWF testing • BMBT will be prolonged with thrombocytopenia
	• **von Willibrand Factor**	• vWF involved in adhesion of platelets at the site of vascular injury • Perform if adequate platelet numbers with a prolonged BMBT • If vWF is normal, then specific platelet tests are required
• **Secondary haemostasis:** • Severe bleeding internally or externally • Cavity bleeding and haematomas • Bleeds deep into subcutaneous tissues, body cavities, joints • Epistaxis and haemoptysis	• *Intrinsic pathway:* **ACT, APTT** (↑ time if <30% factors remain) • *Extrinsic pathway:* **PT** (↑ time if <30% factors remain) • *Common pathway:* **ACT, APTT, PT, TT** • **IF APTT, PT, TT are all prolonged then MULTI-FACTOR DEFICIENCY**	
	• **Activated clotting time** • Intrinsic and common pathway	• Dogs: 90-120 seconds at body temp • Cats: 60-80 seconds at body temp • Prolonged in severe thrombocytopenia • Variations can exist depending on method
	• **Activated partial thromboplastin time** • Intrinsic and common pathway	• Factors VIII (haemophilia A), IX (haemophilia B), XI and XII • Not affected by platelet numbers • Prolonged with normal PT most commonly: ➢ Factor VIII (haemophilia A) ➢ Factor IX (haemophilia B)
	• **Prothrombin time** • Extrinsic and common pathway	• Factors I, II, V, VII and X • Prolonged with normal APTT most commonly: ➢ Most commonly early Vit K antagonism

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	• **Prolongation of both APPT and PT (and ACT)**	• **MULTI-FACTOR DEFICIENCY** • Most commonly: ➢ Advanced rodenticide toxicoses (Vit K dependant factors (II, VII, IX, X) ➢ Liver failure ➢ Disseminated intravascular coagulation ➢ Snake envenomation ➢ Heparin overdose

Common causes of coagulopathy:

- **Immune mediated thrombocytopenia:**
- **Pathophysiology:**
  - ✓ Immune mediated destruction of platelets, autoantibodies bind to the surface resulting in destruction by mononuclear phagocytic system in the spleen
  - ✓ Female middle aged dogs most common signalment
  - ✓ Causes:
    - ➢ Primary (idiopathic): Most common cause in dogs
    - ➢ Secondary (loss of immunotolerance): Neoplasia, medications/vaccinations, infections (*Leptospirosis, Ehrlichia, Babesia*, FIV, FeLV, FIP, fungal etc.), immune mediated disease (IMHA)
- **Clinical signs:**
  - ✓ Petechiae and ecchymosis, hyphema, melena, haematuria
  - ✓ +/- Pale mucous membranes
  - ✓ +/- Pyrexia
  - ✓ Lethargy
- **Diagnosis:**
  - ✓ CBC and blood smear:
    - ➢ +/- Anaemia due to blood loss or concurrent IMHA (Evan's syndrome)
    - ➢ Reduced numbers of platelets (<30x10^9/L), +/- macroplatelets, blood smear (<3 platelets per hpf)
    - ➢ Neutrophilia
    - ➢ Concurrent non-regenerative anaemia, neutropenia, assess for bone marrow disease
  - ✓ If confirm thrombocytopenia, perform diagnostics to assess for a primary underlying disease
    - ➢ Biochemistry, urinalysis, ultrasound and radiographs, testing targeted at infectious agents
- **Treatment:**
  - ✓ Confinement to reduce risk of bleeding
  - ✓ Start immunosuppressive agents:
    - ➢ Dexamethasone 0.5mg/kg SC/IV, then 12 hours later start prednisolone 2-4mg/kg PO divided BID
    - ➢ Taper slowly over months, once platelet numbers have stabilised
    - ➢ +/- Azathioprine 2mg/kg PO SID, then 0.5mg/kg PO EOD, monitor for bone marrow suppression and hepatoxicity, also very toxic in cats (0.3mg/kg PO SID)
    - ➢ +/- Cyclosporine 5-10mg/kg PO divided BID, cats 5mg/kg PO SID
  - ✓ Fresh whole blood transfusion:
    - ➢ RBC for anaemia
    - ➢ Platelets are only viable for 6 hours after collection
  - ✓ +/- Start gastric protectants:
    - ➢ H_2 antagonist, proton pump inhibitors and sucralfate 0.5-1gm PO TID
  - ✓ Treatment of underlying disease if identified
    - ➢ +/- Anti-thrombotic agents: Not routine, thromboembolic disease not as common as with IMHA
- **Monitoring:**
  - ✓ Platelet counts monitored daily until >15x10^9/L, can take up to 2 weeks
  - ✓ Once normal, taper immunosuppressive medications over 4 – 6 months, 20% reduction every 2 weeks

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- **Von Willebrand disease:**
- **Pathophysiology:**
  - ✓ Inherited deficiency of vWF, leading to a failure of platelet binding to sites of vascular injury
  - ✓ Common in German Shepherd and Doberman
  - ✓ Severe cases of vWD can see spontaneous bleeding before 12 months old
- **Clinical signs:**
  - ✓ Can be spontaneous or post-trauma/surgery
  - ✓ Anywhere from no petechiae to bleeding from multiple sites and oozing from blood vessels
- **Diagnosis:**
  - ✓ Prolonged BMBT
  - ✓ Usually normal platelet counts
  - ✓ Normal PT, APTT
  - ✓ Confirmatory test: vWF antigen assay (ELISA)
- **Treatment:**
  - ✓ Haemostasis (pressure bandage)
  - ✓ Fresh frozen plasma or full blood transfusion to replace vWF:
    - ➢ See **"Transfusion Therapy"** can administer a bolus of 10ml/kg of fresh frozen plasma, then repeated until clotting times are normal or bleeding has stopped
    - ➢ Used to stop bleeding or if a surgical procedure is anticipated

Rodenticide toxicity: :

- **Pathophysiology:**
  - ✓ Reduction in vitamin K due to an inhibition of vitamin K epoxide reductase, leading to a reduction in vitamin K dependant clotting factors (II, VII, IX, X)
  - ✓ Clinical signs of toxicity usually occur between 8 to 72 hours post-ingestion
  - ✓ History of ingestions or use around the house
- **Clinical signs:**
  - ✓ Haemorrhagic tendencies from multiple sites
- **Diagnosis:**
  - ✓ Always take ACT, PT and APTT, PCV/TP before initiating therapy to get a baseline
  - ✓ If recent ingestion (<4hrs):
    - ➢ Induce vomiting, +/- gastric lavage and activated charcoal (refer to **"Toxicology"** for vomiting agents)
    - ➢ +/- Commence vitamin K prophylaxis for 2 to 4 weeks depending on the type of rodenticide ingested (see vitamin K therapy below)
  - ✓ If unsure about ingestion OR ingested and potentially already absorbed;
    - ➢ Perform ACT, PT and APTT:
      - Will see an increase in PT before APTT but not for 24-48 hours
      - Factor VII has the shortest half-life therefore PT will be prolonged first
  - ✓ Interpretation:
    - ➢ If normal PT and APTT and **less** than 48 hours after ingestion:
      - Perform PT and APTT again in 24-48hrs and do not start vitamin K but monitor for signs of bleeding and keep animal quiet, can be life threatening if coagulopathy develops
      - OR begin Vitamin K therapy as prophylaxis, minimal side effects and safer than treating a coagulopathy if one develops
    - ➢ If normal and **longer** than 48 hours after ingestion:
      - Likely not require treatment, confine and monitor closely, repeat ACT, PT and APTT 24 hours later
    - ➢ If prolonged ACT, PT or APTT:
      - Begin treatment

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- **Treatment:**
  - ✓ Vitamin K therapy:
    - ➢ ACT should improve within 24 hours
    - ➢ Start with a vitamin K injection at 5mg/kg SC
    - ➢ Follow with oral therapy at 2.5mg/kg BID PO for the duration of the toxins effect, oral absorption is more effective, and should be given with a small fatty meal
      - Warfarin 14 days
      - Bromodiolone 21 days
      - Brodifacoum 30 days
      - Unknown treat for 30 days
  - ✓ Fresh frozen plasma or whole blood transfusion:
    - ➢ Given if clinical evidence of bleeding or high risk
    - ➢ See **"Transfusion Therapy"**, administer fresh frozen plasma 10ml/kg, then repeated until clotting times are normal or bleeding has stopped
- **Monitoring:**
  - ✓ Perform ACT or PT and APTT (better) and PCV/TP, 48-72 hours after stopping vitamin K therapy

- **<u>Disseminated intravascular coagulation (DIC):</u>**
- **Pathophysiology:**
  - ✓ Wide spread activation of the coagulation system causing intravascular coagulation and the formation of microthrombi
  - ✓ Release of pro-inflammatory mediators triggered by severe inflammation e.g. endotoxaemia, sepsis, pancreatitis, neoplasia, crush injuries result in activation of the coagulation system, this overwhelms anti-inflammatory mediators, triggers activation of fibrinolytic pathways, leading to consumption of platelets and clotting factors
- **Clinical signs:**
  - ✓ Haemorrhagic tendencies from multiple sites, haemolytic anaemia
  - ✓ Clinical signs of underlying disease
- **Diagnosis:**
  - ✓ Disease causing severe inflammation/infection
  - ✓ Evidence of bleeding
  - ✓ Thrombocytopenia (or decreasing numbers)
  - ✓ ↑ PT, APTT and later ↑ ACT
  - ✓ ↑ D-dimer assay
- **Treatment:**
  - ✓ Treatment of underlying disease process is essential, otherwise will not remove the stimulus for DIC
  - ✓ Supportive therapy and administration of blood products or fresh frozen plasma if coagulopathic
  - ✓ If significant clinical haemorrhage fresh frozen plasma at 10ml/kg boluses until reduced haemorrhage, normalised ACT then reduce to 2ml/kg/hr as a maintenance
  - ✓ If mild bleeding and underlying cause is being treated can administer fresh frozen plasma 10ml/kg as needed
  - ✓ If no bleeding but coagulopathic and need to perform surgery administer fresh frozen plasma 10ml/kg

- **<u>Snake envenomation:</u>**
- **Pathophysiology:**
  - ✓ Can cause acute coagulopathy due to enzymatic interference with coagulation cascade, can see prolongation of ACT relatively quickly after initial bite
- **Clinical signs:**
  - ✓ Clinical signs of coagulopathy, but can see lower motor neuron paralysis, haemolysis or haematuria
  - ✓ Snake venom detection kit identifies what type of venom is present and therefore what antivenin is required, not what type of snake bit them

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- **Treatment:**
  - ✓ Antivenom:
    - ➢ Deactivation of venom is important before administration of fresh frozen plasma
    - ➢ Snake venom detection kits:
      - Repeated after each vial of antivenom to identify if venom is still present, if negative and a coagulopathy is still present then plasma is indicated to replace clotting factors
  - ✓ Fresh frozen plasma:
    - ➢ See **"Transfusion Therapy"** can administer a bolus of 10ml/kg, then repeated until clotting times are normal or bleeding has stopped

- **<u>Ehrlichiosis and Rocky mountain spotted fever:</u>**
- **Pathophysiology:**
  - ✓ Tick borne diseases of similar clinical signs and management:
    - ➢ Ehrlichiosis caused by *Ehrlichia canis*, seen North America but also Africa, Europe
    - ➢ Rocky Mountain Spotted Fever caused by *Rickettsia rickettsia*, seen in North and South America
  - ✓ Ehrlichiosis:
    - ➢ Three stages of disease:
      - Acute: 2-4 weeks if untreated. Multiplication and spread through lymph nodes, liver and spleen. Can result in organomegaly, thrombocytopenia and bleeding, also vasculitis which can lead to neurological signs and polyarthropathies
      - Sub-clinical: Lasts months to years if untreated in the acute phase. Persistent mild thrombocytopenia as the organism evades immune system.
      - Chronic: Development of bone marrow suppression and myelodysplasia.
    - ➢ Rocky Mountain Spotted Fever:
      - Invasion into the vascular endothelium causing a vasculitis. This leads to peripheral oedema and organ swelling
      - Formation of anti-platelet antibodies leads to the development of thrombocytopenia
- **Clinical signs:**
  - ✓ Lethargy, anorexia, weight loss, pyrexia, lymphadenomegaly and organomegaly
  - ✓ Bleeding: Pale mucous membranes, petechiae, hyphaemia and epistaxis
  - ✓ Anterior uveitis and other ocular pathology
  - ✓ Neurological signs e.g: seizures, ataxia, vestibular signs
  - ✓ Polyarthritis: Shifting lameness, joint pain
- **Diagnosis:**
  - ✓ Thrombocytopenia, anaemia to pancytopenia
  - ✓ Hyperglobulinaemia, hypoalbuminaemia
  - ✓ Non-specific: Liver enzyme elevation
  - ✓ Ehrlichiosis:
    - ➢ Antibody testing via immunofluorescence: Antibodies present within 1 to 2 weeks
    - ➢ ELISA assay: Antibodies to the organism
    - ➢ PCR assay: Can identify the presence of the organism and the species
  - ✓ Rocky Mountain Spotted Fever:
    - ➢ Immunofluorescence of skin biopsies or positive nested PCR
    - ➢ Serology: Some cross-reactivity with different rickettsia species. Paired titres 3-4 weeks apart with an increase over that time frame.
- **Treatment:**
  - ✓ Both: Doxycycline 10mg/kg PO SID for 28 days
  - ✓ Ehrlichiosis: Alternative option is Imidocarb dipropionate 5mg/kg IM repeated in 2 weeks
  - ✓ Immunosuppressive therapy if severe thrombocytopenia:
    - ➢ Prednisolone 1-2mg/kg/day PO tapering
  - ✓ General supportive therapy +/- blood transfusions

Coagulopathy

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Constipation and Tenesmus

- **This chapter covers:**
  - ✓ Terminology
  - ✓ Clinical signs and differentials for constipation and tenesmus
  - ✓ Basic diagnostic pathway
  - ✓ Treatment of constipation

- **Terminology:**
  - ✓ Constipation: Difficulty in defecating with the retention of faeces in the colon/rectum
  - ✓ Tenesmus: Straining to defecate
  - ✓ Dyschezia: Difficult defecation
  - ✓ Haematochezia: Blood in faeces
  - ✓ Obstipation: Severe prolonged constipation refractory to control

- **Clinical signs:**
  - ✓ Tenesmus, dyschezia, haematochezia
  - ✓ Non-productive or minimal amounts of faeces passed

Differentials for tenesmus:	
• Constipation: **See below** • Anal gland disorders: See **"Rectal and Perineal Disease"** • Urinary tract disorders: See **"Urinary Tract Disease"** • Rectal and perineal disease: See **"Rectal and Perineal Disease"** • Prostatic disease: See **"Prostatic Disease"**	
Differentials for constipation:	
• **Gastrointestinal Disease:** • Foreign body • Rectal neoplasia and polyps • Idiopathic (cats) – motility dysfunction • Rectal strictures	• **Extra-gastrointestinal Disease:** • Dietary – bones • Dehydration • Drugs – opioids, diuretics, antacids, antihistamine • Electrolyte abnormalities: ➢ Hypercalcaemia, hypokalaemia • Narrowing or compression of the colon: ➢ Prostatic disease ➢ Rectal/perineal disease ➢ Sublumbar lymph node enlargement ➢ Pelvic canal narrowing (trauma, neoplasia, malunion) • Hypothyroidism • Neurological: ➢ Spinal cord disease ➢ Cauda equine syndrome ➢ Dysautonomia • Aversion to litter tray (cats)

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- **Diagnostics:**
  - ✓ Watching defecation behaviour
  - ✓ Bladder palpation, especially in cats to rule out obstructive disease
  - ✓ Rectal palpation (anal glands, rectum, prostate)
  - ✓ Neurological examination
  - ✓ Haematology, biochemistry and electrolytes
  - ✓ Radiographs:
    - ➢ Confirm constipation, assess prostatic size, position of colon (narrowing)
    - ➢ Megacolon: Colon is >1.5 x width of L7
  - ✓ Ultrasound of prostate/sublumbar lymph nodes
  - ✓ +/- Colonoscopy to assess for strictures and intraluminal masses

- **Feline constipation:**
  - ✓ Can be a result of or lead to megacolon
  - ✓ Megacolon is a syndrome of severe irreversible dilation and hypomotility of the colon
  - ✓ Two forms of megacolon:
    - ➢ Dilation (idiopathic):
      - Most common form of megacolon approximately 70% of cases
      - Due to end stage dysfunction of the colon typically seen in older cats
      - Likely due to impaired smooth muscle function, but possibly due to electrolytes abnormalities
      - Rule out obstructive lesions
    - ➢ Hypertrophy:
      - Secondary to outflow obstruction: Pelvic narrowing, rectal strictures, tumours, foreign bodies

Treatment:

All cases:
- ✓ Need to correct underlying dehydration and electrolyte imbalances
- ✓ Correction of underlying disease process

Mild:
- ✓ Can trial faecal softeners/ laxatives and increased dietary fibre
  - ➢ High-fibre diets:
    - Non-absorbable and hydrophilic e.g. Hills r/d ®, Psyllium husk 1-4 teaspoon PO SID-BID (e.g. Metamucil ®)
  - ➢ Laxatives:
    - Only in well hydrated patients that are eating and drinking
    - Emollients: Facilitate mixing of fat and water in colon, detergent action e.g. coloxyl 50
    - Lubricants: Coat faeces to decrease water absorption in colon e.g. Laxatone gel
    - Osmotic agents: Non-absorbable sugars e.g. Lactulose 0.5ml/kg PO BID-TID, polyethylene glycol 3350, ¼ teaspoon PO BID
    - Stimulant agents: Create strong peristaltic action e.g. Senokot, Biscodyl
  - ➢ Micro-enemas:
    - Warm water or normal saline with KY lubricant 1:1
    - Dioctyl sodium sulfosuccinate 5-10 mL/small dog or cat; 10-20 ml/medium sized dog and 20-30 ml/large dog
    - Microlax enema
    - Lactulose enema 5ml/kg (cat) or 10ml/kg (dog) of a 3 parts lactulose and 7 parts water

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- **Severe:**
  - ✓ Correction of fluid and electrolyte imbalances
  - ✓ Enema:
    - ➢ Typically, under general anaesthesia
    - ➢ Commence antibiotic coverage
    - ➢ Manual evacuation of faeces via colonic irrigation
    - ➢ Can use warm tap water or isotonic saline
    - ➢ Can breakdown faeces via abdominal massage or blunt instruments
    - ➢ May require multiple sessions

- **Recurrent constipation or megacolon:**
  - ✓ Correction of underlying cause if possible
  - ✓ Medical:
    - ➢ Combination of laxatives, high fibre or low residue diets and prokinetic agents:
      - Laxatives:
        - o Lactulose 0.5ml/kg PO BID-TID
        - o Polyethylene glycol 3350, ¼ teaspoon PO BID
      - Dietary management:
        - o Can try low residue diets if high fibre diets do not work
        - o Encourage water drinking
        - o Wet food rather than dry food
      - Prokinetic agents:
        - o Cisapride 0.1-0.5 mg/kg PO BID, 30 minutes before a meal, stimulates entire gastrointestinal tract, may cause vomiting and diarrhoea
        - o Ranitidine 2mg/kg PO BID, variable effect, can be combined with cisapride
  - ✓ Surgical:
    - ➢ If non-responsive to medical management then surgical assessment for subtotal colectomy or correction of pelvic abnormalities, only if no megacolon exists

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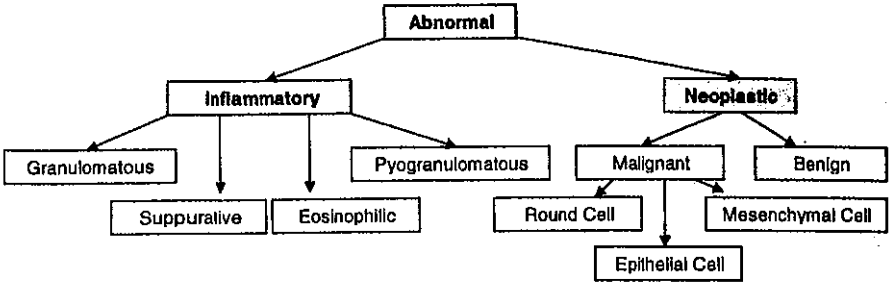
Cytology

- **This chapter covers:**
  - ✓ The basic approach to evaluating a smear
  - ✓ Guide to identifying signs of neoplasia and classification of the neoplasia
  - ✓ Guide to determining between different types of inflammation
  - ✓ Basic interpretation of lymph node cytology

- General approach to evaluation of the cytology smear:



IF ANY FEATURES OF A NEOPLASTIC PROCESS THEN IT IS NEOPLASTIC

- **Criteria of malignancy:**
  - ✓ When more than 4 criteria are present then usually indicates malignancy

Criteria:	Description:
Cellular:	
Anisocytosis	Variation in the sizes of cells
Macrocytosis	Increases in the size of cells
Pleomorphism	Variation in size and shape of a particular cell 'type'
Hyperchromasia	Increased amount of pigmentation/staining within the cell
Nuclear:	
Increased N/C ratio	Increases in the size of the nuclei compared to the cytoplasm
Macrokaryosis	Increases in the size of the nuclei
Multinucleation	Multiple nuclei are present
Anisokaryosis	Variation in the sizes of nuclei
Nuclear moulding	The nucleus of one cell is moulded and distorted by the nucleus of another cell
Macronucleoli	Increases in the size of the nucleoli (> than size of RBC)
Anisonucleoliosis	Variation in the size and shape of the nucleoli
Coarse chromatin pattern	The pattern of the chromatin pattern looks thicker/rougher
Increased mitotic figures	Mitotic figures are present (usually do not see)
Abnormal mitotic figures	Arrangement of the mitotic figures is abnormal, normally aligned like a clock face

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- **Classification of neoplasia:**

Epithelial tumours:	
▪ "adenoma", "carcinoma", "adenocarcinoma" ▪ Cells are large ▪ Round or square shaped ▪ Easily aspirated - get large clusters or rafts ▪ May be glandular in origin – cytoplasmic vacuoles	
Mesenchymal tumours (Spindle cell):	
▪ "oma", "sarcoma" ▪ Cells are small/medium sized ▪ Spindle shape – more than one tapered end ▪ Difficult to aspirate – only get small number of cells	
Round cell tumours:	
▪ Cells are small/medium sized ▪ Round shape ▪ Easily aspirated – get large numbers of cells	
Characteristics of different round cell tumours:	
Lymphosarcoma:	▪ Large immature lymphocytic cells - larger than neutrophils ▪ Cytoplasm – minimal amount of basophilic (blue staining) ▪ Nuclei are large and round shaped ▪ Nucleoli are prominent
Mast cell tumour:	▪ Cytoplasm – moderate amount, with distinct purple granules ▪ Granules can be poorly granulated if they are highly malignant ▪ Nuclei large round to ovoid
Histiocytoma:	▪ Very large cells ▪ Cytoplasm – large amount of pale basophilic (blue staining) ▪ Nuclei large are displaced to the sides and can have more than one ▪ Can appear highly malignant – mitoses
Plasmacytoma:	▪ Nuclei are displaced to the sides "peri-nuclear clear zone" and have a coarse chromatin pattern ▪ Cytoplasm – moderate amount of pale basophilic (blue staining) ▪ Typically act benign
Melanoma:	▪ Look like anything ▪ Cytoplasm – black pigment ▪ Mostly behave benign BUT if from nail bed or mouth = malignant

- **Types of inflammation:**
  - ✓ **If any evidence of neoplasia = NEOPLASTIC**

Type:	**Characteristics:**
Suppurative:	▪ Acute inflammation → >85% neutrophils, bacteria
Granulomatous:	▪ Chronic inflammation → mostly macrophages +/- giant cell
Pyogranulomatous:	▪ Mix of neutrophils and macrophages ▪ Foreign body reaction, furunculosis, fungi
Eosinophilic:	▪ >10% eosinophils ▪ Allergic, fungal, parasites

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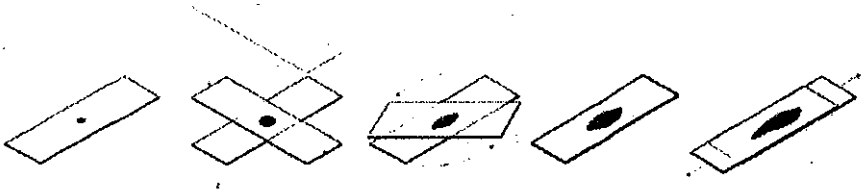
- **Lymph node cytology:**

Normal Lymph Node:	
▪ Mixed cell types present ▪ >75% small mature lymphocytes ▪ <25% medium to large lymphocytes/blasts, neutrophils, plasma cells, macrophages	
Enlarged Lymph Node:	
Reactive lymph node hyperplasia:	▪ Mixed cell population ▪ BUT increased number of macrophages, plasma cells, immature lymphocytes ▪ **Small lymphocytes predominate cell type**
Lymphadenitis: *Inflammation of the lymph node*	▪ Increased number of other cells types such as neutrophils, macrophages and eosinophils
Lymphosarcoma:	▪ **Homogeneous cell population** ▪ **Large immature cells predominate >50%** ▪ +/- Malignant criteria
Metastatic neoplasia:	▪ Presence of abnormal cells within the lymph node ▪ Especially with carcinomas, melanomas, mast cell tumours, bone marrow neoplasia

- Preparation of cytology specimen:



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Dental Disease

- **This chapter covers:**
  - ✓ The basic anatomy of the tooth and the "normal bite"
  - ✓ Basic information on dental disease
  - ✓ Indications and methods or tooth removal

- **Tooth structure:**
  - ✓ Enamel:
    - ➢ Formed at 8 weeks, after which no more is made, it wears away
  - ✓ Dentine:
    - ➢ Bulk of tooth, thousands of tubes
  - ✓ Pulp: Nerves; blood vessels etc.
  - ✓ Gum/bone/periodontal ligament → supports tooth
  - ✓ Sulcus: Measure for charts:
    - ➢ Dog: 3 mm normal (all sizes)
    - ➢ Cat: Should not be able to measure anything (0.5mm) is pathological
  - ✓ Cementum
  - ✓ Crown: Above gum
  - ✓ Root: Below gum; 2 x size of crown

- **Normal bite: 3 Elements of Normal Bite**
  - ✓ Incisor scissor bite:
    - ➢ Upper incisors just in front of lowers and make contact (back edge of uppers):
      - Ledge inside upper incisors for bottom teeth to fit in
  - ✓ Canine position:
    - ➢ Lower canine between upper I3 and C AND no contact with either:
      - Evenly spaced between both – no pressure
  - ✓ Premolars interdigitate (if no interdigitation then abnormal):
    - ➢ Most important – tip of 1 in valley of other 2 in opposite arcade
    - ➢ Premolars should interdigitate with lower premolars first
    - ➢ L PM1 – U PM1 – L PM2 – U PM2 ...
  - ✓ Need all 3 correct for normal bite
- **Malocclusions:**
  - ✓ Related to genetics of jaw growth (stops at 7-8 months):
    - ➢ *Overshot bite* → Top jaw outside lower jaw
    - ➢ *Undershot bite* → Top jaw within lower

- **Dental formulae:**

	Dog:	Cat:
Adult:	• 42 teeth = I3/3; C1/1; PM4/4; M2/3	• 30 teeth = I3/3; C1/1; PM 3/2; M 1/1 • Upper: PM 2,3,4 → no upper PM1 • Lower: PM 3 and 4 → no PM 1 or 2
8 weeks old:	• 28 teeth = i3/3; c1/1; p3/3; m0/0 • All baby teeth (lose them at 3 months); no adult teeth • 6 weeks – NOT ALL BABY TEETH ARE PRESENT	• 26 teeth = i3/3; c1/1; p3/2; m0/0

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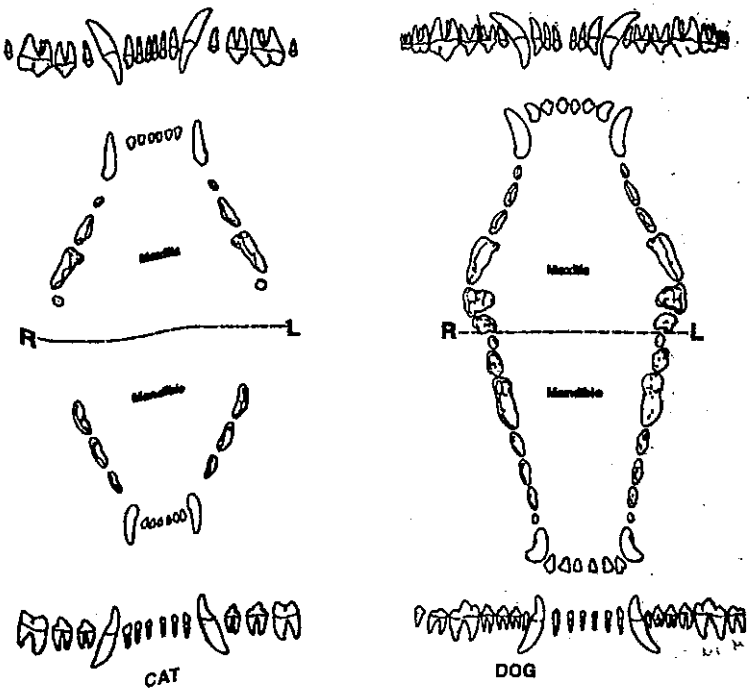

CAT DOG

- **Periodontal disease:**
  - ✓ Definition:
    - ➢ Periodontal Disease = disease of the supporting structures of tooth:
      - Gingiva; periodontal ligament; alveolar bone
  - ✓ Stages:
    - ➢ Gingivitis:
      - Reversible inflammation of marginal gingival
      - Plaque in sulcus → body reacts with WBCs → oedema/swelling/inflammation of gums
      - Precursor to periodontitis, so must treat gingivitis
    - ➢ Periodontitis:
      - Irreversible inflammation of gingiva and supra-alveolar tissue with loss of connective tissue attachment and bone (i.e. loss of periodontal support) – deep pocket
      - Need to lose 80% support before tooth loose
      - Can treat but not reversible
      - If not loose, scale under gums BUT need to get it all out (gingival flap)
    - ➢ Advanced periodontitis:
      - Heavy calculus build up
      - Calculus = calcified plaque; mostly above gum; cosmetic – irrelevant to disease (except shows lax control) → need to look under gum
      - Tooth mobility with supporting bone loss >50%

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- ✓ Aetiology:
  - ➢ Plaque:
    - Adheres to all surfaces of teeth – layers of bacteria; saliva; food particles → mineralized (with calcium from saliva) to form calculus
    - Anaerobic bacteria release cytokines which leach into tissue, this stimulates osteoclasts (chronic inflammation) leading to thinning bone of socket
    - Need mechanical removal
- ✓ Home care advice:
  - ➢ Prevention better than cure
  - ➢ Brush teeth (daily) = gold standard – finger brush; paste
    - Outside teeth and inside lower teeth (hot spots)
    - Not need to do inside cat's teeth (rough tongue)
  - ➢ Chew toys or bone substitutes
  - ➢ Bones – bigger the better (cow femur) – not cut longitudinally as can break teeth
  - ➢ Dental diets
  - ➢ Toothpaste – not use adult human paste (use baby paste with low fluoride)

- **<u>Indications for extraction:</u>**
  - ✓ Retained deciduous teeth (especially canines):
    - ➢ Hard to get out as brittle
    - ➢ Roots still significant (like permanent teeth)
    - ➢ Closes off entry for permanent teeth (will drift back to right position if get them early) and not fit with other jaw
  - ✓ Supernumerary teeth:
    - ➢ May or may not need to remove → how is bite?
  - ✓ Periodontal disease
  - ✓ Malocclusion:
    - ➢ For base narrow canines, can manually distract canines with thumbs as the adults emerge
    - ➢ Do couple mins a day as when playing with pet
    - ➢ Tipping of the tooth but not recommended until >8 months old
  - ✓ Malpositioned teeth
  - ✓ Broken teeth:
    - ➢ Dark spot = reparative dentine or hole = exposed pulp/nerve
    - ➢ Nerve/pulp exposed to bacteria WILL abscess (100%), must remove
    - ➢ Extraction or root canal therapy (replace internal tooth material with artificial material)
  - ✓ Oral neoplasia: See below
  - ✓ Teeth in fracture lines
  - ✓ Interceptive orthodontics:
    - ➢ e.g. Canine interlocked with incisor (undershot) → remove

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- **Extraction technique:**

Incisors:	▪ Single rooted tooth ▪ Cut epithelial attachment (root tip pick) and break down periodontal ligament (PDL) ▪ Hold position of elevator for 20-60 sec (fatigue ligament) → keep going around until get root tip pick to sit at 90°; repeat with bigger instrument ▪ Remove using extraction forceps – only when loose ▪ Ensure the root is intact, tip is smooth and round: if sharp/jagged, left some behind!
Canines:	▪ Surgical extraction only ▪ Remove gingival collar – so can suture fresh tissue ▪ Mucoperiosteal flap with wider base – lift periosteum so can reattach to bone ▪ Burr away bone ▪ Elevate as normal ▪ Remove upper canines (curved) by rotating root tip outwards (towards tip of nose) ▪ Suture closed
Multi-rooted Teeth:	▪ Section into single-rooted fragments ▪ Need to know position of furcations ▪ Remove each fragment as single-rooted ▪ Suture if needed

- **Pain relief:**
  - ✓ Regional nerve blocks: Give before extraction – see **"Anaesthesia and Analgesia":**
    - ➢ Maxillary block
    - ➢ Mandibular block
  - ✓ Systemic pain relief:
    - ➢ Opioids, NSAID's

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Dermatology

- **This chapter covers:**
  - ✓ The basic approach to common skin lesions
  - ✓ Diagnostic tests available
  - ✓ Guide to treatment of commonly seen conditions

- **Common skin lesions:**
  - ✓ **Pruritus**
  - ✓ **Alopecia**
  - ✓ **Scale, Crusting and Depigmentation**
  - ✓ **Nodules**

- **Pruritic dermatitis:**

- **Pruritic dog:** Flea allergy dermatitis and atopic dermatitis (90%)
- **Pruritic cat:** Flea allergy dermatitis (60%), food allergy dermatitis (fish protein) (25%)

Allergic:	• **Flea allergy dermatitis (FAD):** ➢ Pruritus: Dorsal lumbosacral, base of tail, inguinal and axillar region, ventrum • **Atopic dermatitis:** ➢ Pruritus: Axillae, ventrum, inguinal, perineum, face, feet, ears • **Food allergy dermatitis:** ➢ Pruritus: Axillae, ventrum, inguinal, perineum, face, feet, ears • **Mosquito bite hypersensitivity:** ➢ Pruritus: Ulcerative crusting on nose, pinnae • **Contact dermatitis:** ➢ Pruritus: Ventral regions - Axillae, ventrum, inguinal, perineum, chin, feet
Parasitic:	• **Flea allergy dermatitis (FAD):** ➢ Pruritus: Dorsal lumbosacral, base of tail, inguinal and axillar region, ventrum • **Demodecosis:** ➢ Localised: Face and forelegs ➢ Generalised: Anywhere over body • **Sarcoptes mange (Scabies):** ➢ Face, ears, elbows, ventrum, hocks
Bacterial:	• **Surface pyoderma:** ➢ Pyotraumatic dermatitis (hot spot) – usually due to an underlying allergic disease ➢ Skin fold pyoderma: Lip and facial folds, any skin fold, around vulva • **Superficial pyoderma:** ➢ Epidermal collarettes and target lesions also papular-pustular lesions • **Deep pyoderma:** ➢ Furunculosis (deep skin boils) • **Abscess/cellulitis** • **Atypical:** ➢ German shepherd pyoderma cellulitis
Fungal:	• **Malassezia dermatitis:** ➢ Pruritus: Ventral areas, ear and feet, perianal area • **Dermatophytosis:** ➢ Pruritus: Face, ears and feet

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- **Alopecia:**

Pruritic alopecia:		
• Due to secondary trauma – scratching and rubbing ➢ **See "Pruritic Dermatitis" above for differentials**		
Non-pruritic alopecia:		
• **Immunological:** **RARE**	• **Neoplastic:** ➢ Due to paraneoplastic syndrome – hormone secreting	• **Hormonal:** ➢ Bilateral alopecia ➢ Friction areas ➢ Hyperpigmentation
• **Drug eruption** • **Juvenile cellulitis** (Puppy strangles) • **Pemphigus foliaceus** • **Lupus erythematosus**	• **Sertoli cell tumour** • **Granulosa cell tumours**	• **Hypothyroidism:** ➢ Dull poor coat, poor regrowth ➢ Hyperpigmentation ➢ Recurrent skin infections • **Hyperadrenocorticism:** ➢ Alopecia with thin skin, comedones, hyperpigmentation +/- calcinosis cutis • **Hyperestrogenism:** ➢ Due to cystic ovaries, granulosa cell tumours, sertoli cell tumours ➢ Hyperpigmentation, linear preputial dermatitis (male), enlarged nipples ➢ Bone marrow suppression, aplastic anaemia

- **Scale, Crusting and Depigmentation:**

Pruritic scaling and crusts:	
• **See "Pruritic Dermatitis" above for differentials**	
Non-pruritic, painful with system signs of illness:	
• **Lupus Erythematosus:**	• **Pemphigus group:**
• **Systemic:** ➢ Ulceration, depigmentation, scale crusting and alopecia ➢ Systemic clinical signs • **Discoid:** ➢ Scale and crust and depigmentation ➢ Nose only ➢ Due to sunlight activation of anti-DNA antibodies	• **Pemphigus foliaceus:** ➢ Pustules, superficial erosions, scale and crusts ➢ Facial (bridge of nose, muzzle, periocular, pinnae), footpads, scrotum • **Pemphigus erythematosus:** ➢ Crusting, scale, vesicles and pustules ➢ Face, ears and dorsum of nose

- **Nodules:**

Single nodule:	**Multiple nodules:**	**Nodule with drainage tracts:**
• Neoplastic • Dermatophyte kerion • Abscess • Sterile inflammatory	• Neoplastic • Severe infection • Sterile inflammatory e.g. Urticaria • Mycobacterial infections	• Severe bacterial infections • Fungal infection • Foreign bodies • Abscesses • Mycobacterial infections

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Diagnostics tests:

- Flea comb
- Skin swab:
  - ✓ Recurrent skin infections and deep pustular lesions
  - ✓ Samples from the deepest parts of lesions or from aspirates of pustular lesions to reduce surface bacterial contamination
- Skin scrape:
  - ✓ Superficial: Scabies and other superficial mites
  - ✓ Deep: Demodex mites
- Sticky tape impression and stain: Bacteria, yeast and dermatophytes
- Hair pluck (Trichogram):
  - ✓ Microscopic examination: Dermatophytes and demodex
  - ✓ Fungal culture: Dermatophytes
    - ➢ If using in-house culture mediums, then must confirm fungal growth under microscope via sticky tape impression looking for macroconidia
- Woods lamp: Dermatophytes (only occasionally)
- Biopsy:
  - ✓ Punch (4, 6, 8mm) or wedge:
    - ➢ If superficial lesions no surgical preparation can disrupt histopathology
    - ➢ If en-bloc resections or tissue cultures, then surgical preparation is required
  - ✓ Send to histopathology, +/- culture
- Blood tests:
  - ✓ Total T4
  - ✓ Baseline hormones (oestradiol, progesterone, testosterone)
- Food trial (if not seasonal): Food allergy
  - ✓ Elimination diet 6-12 weeks – e.g. Hills Z/D®, kangaroo and sweet potato etc.
- Allergy testing (after food trial): Atopy
  - ✓ Combination of both IgE serological and intradermal skin testing
  - ✓ Previous corticosteroid exposure:
    - ➢ Injectable corticosteroid: Wait 6-8 weeks after (6 months for depomedrol)
    - ➢ Oral corticosteroid: Wait 4 weeks after
    - ➢ Oral anti-histamines and topical steroids (including eye and ears preparations): Wait 2 weeks after

Treatment of specific diseases:

- **Bacterial skin infections:**
  - ✓ **Usually secondary and therefore must treat the predisposing disease process**
  - ✓ **Pyotraumatic dermatitis (hot spots):**
    - ➢ Features:
      - Acute onset superficial bacterial infection
      - Secondary to fleas, atopy, food allergy
    - ➢ Treatment:
      - Clip and clean (soapy chlorhexidine) – allows air exposure and proper treatment and monitoring
      - Cephalexin 22mg/kg PO BID for 20 days if secondary bacterial infection
      - Chlorhexidine shampoos
      - Topical antibiotic and steroids preparations
  - ✓ **Skin fold pyoderma:**
    - ➢ Features:
      - Usually lip and facial folds, but any skin fold e.g. vulva
      - Treatment as for pyotraumatic dermatitis
      - +/- Treatment for secondary malassezia infection

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- ✓ **Superficial pyoderma/folliculitis:**
  - ➢ Features:
    - Subcorneal collarette and target lesions
    - Folliculitis: Erythematous papular, pustular lesions
  - ➢ Treatment:
    - Cephalexin 22mg/kg PO BID for 20 days if secondary bacterial infection
    - Chlorhexidine shampoos
    - Prednisolone as required
- ✓ **Deep pyoderma:**
  - ➢ Features:
    - Single or multiple boils
  - ➢ Treatment:
    - Require biopsies and samples sent for culture and sensitivity
    - Antibiotics based on culture and sensitivity, for at least 1 week after resolution of lesions (could take up to 12 weeks)

- **Acral lick dermatitis (Lick granuloma):**
  - ✓ Features:
    - ➢ Focal thickened ulcerative lesion
    - ➢ Typically, on dorsal aspects of lower fore and hindlimbs
    - ➢ Primarily psychological or secondary to infections (bacterial, fungal), localised trauma/arthritis
    - ➢ Aetiological focal lesions e.g. Pyoderma, wounds, could have underlying arthritis/pain
  - ✓ Treatment:
    - ➢ Depends on underlying cause
    - ➢ Prevention of access: E-collars
    - ➢ Antibiotics depends on biopsy and culture results, duration of course until lesion resolves
    - ➢ Psychotropic drugs: Fluoxetine 1mg/kg PO SID
    - ➢ +/- Naloxone: Theory is that licking releases an opioid type compound that is addictive
- **Flea allergy dermatitis:**
  - ✓ Features:
    - ➢ Pruritic regions: Dorsal lumbosacral, base of tail, inguinal and axillar region, ventrum
  - ✓ Treatment:
    - ➢ Eradicate fleas (treat all pets): Integrated flea control important
    - ➢ Insect growth regulator and adulticides used concurrently combined with environmental flea control, see **"Parasitic Disease"**
      - E.g. Monthly spot-ons/tablet given fortnightly, flea bombs, fence off, spray shady dirt areas, etc.
    - ➢ Short course of prednisolone 0.5mg/kg PO BID tapering off
    - ➢ Cephalexin 22mg/kg PO BID for 20 days if secondary bacterial infection
    - ➢ Appropriate shampoo: Chlorhexidine (bacterial), miconazole (fungal), oatmeal
    - ➢ Coat to stop itching and reduce secondary trauma
  - ✓ Treatment failure:
    - ➢ Could be a combination of the following factors: Not treating all pets, incorrect use/application of anti-flea products, frequent swimming/bathing, introduction of a new source of fleas (new pets), poor environmental control
- **Atopic dermatitis:**
  - ✓ Features:
    - ➢ Suspect primarily Type 1 hypersensitivity to "inhaled" allergens combined with skin barrier defects
    - ➢ Depends on underlying cause, typically seasonal but can be year around
    - ➢ Age of onset 1-3 years
    - ➢ Pruritic regions: Generalised pruritus but typically axillae, ventrum, inguinal, perineum, face, feet, ears
    - ➢ Diagnosis based on elimination of all other causes of pruritus – parasites, infections (bacterial/fungal) food allergy

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- IgE serological and intradermal skin testing is used to identify which allergens to include in avoidance and desensitization therapy but not to confirm diagnosis

✓ Treatment:
- Implement strategies to avoid exposure to allergens identified by IgE serological and intradermal skin testing
- Treat secondary infections:
  - Cephalexin 22mg/kg PO BID for 20 days if secondary bacterial infection
  - Chlorhexidine shampoos
  - See **"Malassezia dermatitis"** for treatment of concurrent fungal infections
- Anti-inflammatory therapy:
  - Corticosteroid topical creams and sprays for localised therapy
  - Prednisolone:
    - Short term: 0.5mg/kg PO BID tapering dose
    - Long term: Intermittent low dose (e.g. 0.25 – 0.5mg/kg PO twice a week)
  - Cyclosporine:
    - 5mg/kg PO SID until at least 50% reduction in severity then taper
    - Therapy is lifelong: 1/3 require SID, 1/3 require EOD, 1/3 once-twice/week
- Improve barrier function:
  - Omega 3/6 fatty acids: Fish oil 100mg/kg PO SID
  - Shampoos/lotions: Oatmeal, Alpha Keri bath oil (10ml in 1-2L)
  - Commercial diets high in essential fatty acids
- Allergen desensitization therapy aka Allergen-specific immunotherapy:
  - 1/3 do really good, 1/3 better, 1/3 do not respond
  - Can take longer than 6 months to see effects

- **Puppies/kittens with allergic skin disease:**

✓ Features:
- Allergy testing: Not generally recommended for dogs under 12 months old as allergy profile may change after 12 months

✓ Treatment:
- Corticosteroids: Try to use sparingly – topical better than systemic
- Omega 3/6 oils: Fish oil 100mg/kg PO SID, oil sachets
- Anti-histamines
- Cyclosporine: Generally, not recommended for dogs under 6 months
- Dietary trial: Hypoallergenic diets are generally too low in protein for growth, can supplement with novel protein and balanced calcium powder (e.g. 500gm of meat needs approximately 1 x teaspoon of balanced calcium powder – MUST check with dietician)

- **Contact allergy:**

✓ Features:
- Type 4 hypersensitivity, typically non seasonal occurrence
- Pruritic regions: Ventral regions - axillae, ventrum, inguinal, perineum, chin, feet

✓ Treatment:
- Prevention of contact to with allergic source for a minimum of 2 weeks to determine relationship
- Skin suits or boots
- Topical corticosteroids
- Cephalexin 22mg/kg PO BID for 20 days if secondary bacterial infection
- Prednisolone 0.5mg/kg PO BID tapering dose if extensive

- **Food allergy:**

✓ Features:
- Type1 and 3 hypersensitivities, requires at least several months of exposure to the protein
- Can be a cause of allergic skin disease in dogs younger 6 months old
- Uncommon, non-seasonal pruritus

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- Pruritic regions: Axillae, ventrum, inguinal, perineum, face, feet, ears
- Don't always have to have gastrointestinal signs (vomiting and diarrhoea)
- Poorly responsive to steroids

✓ Treatment:

- Elimination diet for a minimum of 6 weeks but up to 10 weeks – e.g. Hills Z/D®, kangaroo and sweet potato etc.
- Confirm diagnosis by rechallenge with a single proteins source for at least 2 weeks
- Repeat this process for all major protein types, one protein source at a time
- Treat secondary bacterial infections, Cephalexin 22mg/kg PO BID if indicated
- Long term management with a diet that does not contain the offending protein

▪ **Urticaria and angioedema:**

✓ Features:

- Clinical manifestation of a cutaneous hypersensitivity reaction
- Angioedema is usually seen as swelling of the face
- Urticaria seen as raised wheals can be localised with only few lesions or generalised with hundreds of lesions all over the body
- Usually caused by vaccine or drug administration, insect bites, ingested allergens (proteins/food additives), contact with plants
- Can also be an early indication of other diseases – bacterial infections, mites (*demodex*), neoplasia (mast cells)

✓ Treatment:

- If mild and not showing systemic signs of anaphylaxis then treat as below, but if showing signs of systemic involvement then i.e. Pale mucous membranes, collapse, vomiting and diarrhoea, breathing difficulty then see **"Shock and Anaphylaxis"**
- Corticosteroids:
  - Single injection is usually enough, dexamethasone 0.2mg/kg SC/IM or prednisolone sodium succinate 2-10mg/kg IV if severe
- Anti-histamines:
  - Single injection diphenhydramine 2mg/kg IM or chlorpheniramine 0.5mg/kg IM
  - Can send home with a short course of oral anti-histamines if severe for 5 days

▪ **Dermatophytosis:**

✓ Features:

- Fungal species: *Microsporum, Trichophyton*
- Pruritic region: Face, ears, feet or generalised

✓ Treatment:

- Best if use a combination of topical, systemic and environmental treatments:
- Topical:
  - Miconazole shampoo SID on spots or bath twice a week
  - Clipping coats will remove infected hairs that act as fomites and improve efficacy of topical therapy
  - Must use with systemic therapy
- Systemic:
  - Griseofulvin 50mg/kg/day PO divided (micronized) or 15mg/kg/day PO divided (ultramicronized) with a fatty meal
    - Not with FIV positive, pregnant or young animals
  - Itraconazole 5-10mg/kg PO SID
- Environmental:
  - Main source of re-infection
  - Clean fomites and bedding in 1:10 bleach solution
  - Vacuuming and mopping (1:100 bleach)

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- **Malassezia dermatitis:**
  - ✓ Features:
    - ➢ Secondary to predisposing factors include allergy, keratinising defects and hormonal abnormalities
    - ➢ Pruritic regions: Ventral areas, ear and feet, perianal, periocular, perioral areas
  - ✓ Treatment:
    - ➢ Miconazole shampoos SID,on spots or bath twice a week
    - ➢ Systemic infections:
      - First line: Ketoconazole 5-10mg/kg/day PO SID for dogs. Itraconazole 5mg/kg PO SID for 4 weeks for cats
      - Second line: Itraconazole 10mg/kg PO SID for dogs. Fluconazole 2.5-10mg/kg PO SID for cats
    - ➢ Concurrent bacterial infection: Cephalexin 22mg/kg PO BID
- **Insect/arachnid:**
  - ✓ Features:
    - ➢ Mosquito: Ulcerative crusting on nose and pinnae
    - ➢ Seasonal
  - ✓ Treatment:
    - ➢ Keep inside for 2 weeks
    - ➢ Anti-histamine: Chlorpheniramine 4mg/kg PO BID
    - ➢ Corticosteroids: Dexamethasone 0.2mg/kg SC +/- prednisolone 0.5mg/kg PO BID tapering dose
    - ➢ Insect repellents: E.g. Advantix® **(not in cats)**
- **Mites – Demodex:**
  - ✓ Features:
    - ➢ Localised: Usually <1 year old, focal lesions affecting face and forelegs
    - ➢ Generalised: Entire body area or > 5 focal lesions
    - ➢ If puppy/young (3 – 18 months) and low grade and localised can treat with topical product e.g. Advocate® and re-assess, majority resolve without treatment
    - ➢ If adult then MUST investigate for underlying causes for generalised adult onset demodecosis as can indicate underlying immunosuppressive disease e.g. Hyperadrenocorticism, hypothyroid, lymphoma
  - ✓ Treatment:
    - ➢ Amitraz (0.025-0.05%) washes:
      - Weekly washes (use half strength for <5kg)
      - Clipping and combined use with antibacterial shampoos (to remove crusts) to improve efficacy
      - Side effects: Sedation, hypotension and sudden death
      - ANTI-DOTE: Antisedan ® (atipamezole) or Reversine ® (yohimbine)
    - ➢ Ivermectin (off-label):
      - Dose at 300-600µg/kg PO daily, start low end at 50µg/kg and slowly increase dose to 300µg/kg by 50µg/kg every couple of days
      - BEWARE toxicity can occur <600µg for any breed but Collie related breeds are more susceptible. Recommended to test for ABCB1-1 gene mutation before use
    - ➢ Retesting: Repeat skin scrapes at 4 weeks, discontinue treatment after 2 negative skin scrapes a fortnight apart
    - ➢ Concurrent pyoderma MUST be treated - systemic antibiotics are indicated for at least 4 weeks, can also use antimicrobial shampoos (e.g. chlorhexidine)
    - ➢ Avoid prednisolone even if receiving treatment for mites – use antibiotics to resolve concurrent infection to reduce clinical signs
- **Mites – Sarcoptes:**
  - ✓ Features:
    - ➢ Highly infectious
    - ➢ Pruritic regions: Face, ears, elbows, ventrum, hocks
  - ✓ Treatment:
    - ➢ Selamectin 6mg/kg (off label):

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- Applied topically every fortnight for 3 treatments
- Amitraz (0.025-0.05%) washes:
  - Weekly washes (use half strength for <5kg)
  - Clipping and combined use with antibacterial shampoos (to remove crusts) to improve efficacy
  - Side effects: Sedation, hypotension and sudden death
  - ANTI-DOTE: Antisedan ® (atipamezole) or Reversine ® (yohimbine)
- Ivermectin at 200µg/kg once then 200µg/kg PO twice per week for 4 weeks
  - Beware toxicity can occur at any dose in any breed but Collie related breeds are more susceptible. Recommended to test for ABCB1-1 gene mutation before use
- Must treat all animal that are in-contact even if asymptomatic
- Must clean/change all animal bedding and possible fomites

Immune-mediated (pemphigus, lupus):

✓ Features:
- Systemic lupus:
  - Ulceration, depigmentation, scale crusting and alopecia
  - Systemic clinical signs
- Discoid lupus:
  - Scale and crust and depigmentation
  - Nose only
  - Due to sunlight activation of anti-DNA antibodies
- Pemphigus foliaceus:
  - Pustules, superficial erosions, scale and crusts
  - Facial (bridge of nose, muzzle, periocular, pinnae), footpads, scrotum
- Pemphigus erythematosus:
  - Crusting, scale, vesicles and pustules
  - Face, ears and dorsum of nose

✓ Treatment:
- Immunosuppressive doses prednisolone 1mg/kg PO BID until resolution, then tapering 20% every 2 weeks while monitoring for relapse
- Cephalexin 22mg/kg PO BID for 20 days if secondary bacterial infection
- Long term control can use Azathioprine 2mg/kg PO SID until remission then 0.5mg/kg PO EOD, monitor for bone marrow suppression and hepatoxicity

Solar dermatitis:

✓ Treatment:
- Excise dysplastic skin
- Topical steroids
- 30+ sunscreen

Neoplasia:

✓ Treatment:
- Surgical excision +/- chemotherapy

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Dermatology – Feline

- **This chapter covers:**
  - ✓ The basic approach to common skin lesions seen in cats
  - ✓ General diagnostic pathway

- **Pruritic Dermatitis:**
  - ✓ Diagnostic challenge: Cats are closet scratchers and lickers – difficult to determine if have been licking and scratching themselves or not
  - ✓ **Causes of pruritus:** Flea allergy dermatitis (60%), Fish protein allergy (30%), Atopy, feline eosinophilic granuloma complex, mites (10%)

- **Common clinical presentations:**
  - ✓ **Feline eosinophilic granuloma complex**
  - ✓ **Bilaterally symmetrical alopecia**
  - ✓ **Head and neck pruritus**
  - ✓ **Feline miliary dermatitis**
  - ✓ **Nodular lesions**

- **Diagnostics:**
  - ✓ See "**Dermatology**" for diagnostics tests:

Feline eosinophilic granuloma complex:

- **Features:**
  - ➢ All responsive to corticosteroid treatment +/- pruritic

Indolent ulcers (Eosinophilic ulcer):

- ➢ On lip from constant licking due to allergy

Eosinophilic plaques:

- ➢ Single/multiple red round/oval lesions +/- ulcerated from constant licking due to allergy
- ➢ Abdomen, groin, interdigital area and inner thigh

Eosinophilic plaques:

- ➢ Linear granuloma

- **Regions:**
  - ➢ Abdomen
  - ➢ Inguinal
  - ➢ Axilla
  - ➢ Interdigital spaces
- **Differentials:**
  - ➢ Neoplastic skin diseases
  - ➢ Granulomas

- **Causes:**
  - ➢ Flea allergy dermatitis
  - ➢ Food allergy
  - ➢ Feline atopy
  - ➢ Mosquito bite hypersensitivity

- **Diagnostic pathway:**
  - ➢ Histology of lesions: Eosinophilic plaque
  - ➢ Control predisposing factors:-Bacteria/parasites, manage with anti-parasitic and antibiotics
  - ➢ Start on corticosteroids
  - ➢ Institute flea control program: Patient and all other pets, environment
  - ➢ If unresponsive to corticosteroids and flea control program
  - ➢ Institute food allergy trial (8 – 10 weeks):
    - Complete response: Diagnosis = food allergy → confirm with re-challenge diet one every 2 weeks to identify which proteins it is caused by
    - Partial or no response: Not food allergy therefore another allergy e.g. feline atopy or idiopathic eosinophilic granuloma complex

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Bilaterally symmetrical alopecia:

- **Manifestations:**
  - Hair loss on all flanks with main clinical sign is pruritus
- **Differentials:**
  - Psycogenic alopecia (pruritus unresponsive to corticosteroids)
  - Neurodermatitis (pruritus unresponsive to corticosteroids)
  - Idiopathic feline symmetrical alopecia
- **Causes:**
  - Flea allergy dermatitis
  - Food allergy
  - Feline atopy
  - Mosquito bite hypersensitivity
  - Dermatophytosis
  - Psychogenic alopecia – pruritus despite corticosteroid therapy
- **Diagnostic pathway:**
  - Perform a skin scrape, hair pluck, woods lamp +/- culture – rule out dermatophytosis
  - Follow diagnostic procedure for Eosinophilic granuloma complex
  - Rule out hypersensitivities as it is the most common cause of bilateral symmetrical alopecia

Head and neck pruritus:

- **Manifestations:**
  - Mild alopecia and erythema to erosive, ulcerated or crusted lesions on
- **Regions:**
  - Forehead
  - Periauricular areas
  - Pinnae
  - Head and neck
- **Causes:**
  - Parasites:
    - Notoedric mange (*Notoedres cati*)
    - Otodectic mange (*Otodectes cynotis*)
    - Demodecosis (*Demodex gatoi/cati*)
  - Allergies: FAD, food allergy, atopy, mosquito bite hypersensitivity
  - Infections: Dermatophytosis
  - Autoimmune: Pemphigus foliaceus
  - Psychogenic disorders
- **Diagnostic pathway:**
  - History and general physical examination: Fleas, flea dirt, food history
  - Perform a skin scrape (deep and superficial): Mites, see "Dermatology" for treatment
  - Hair pluck and skin scrape, woods lamp and culture: Dermatophytosis, see "Dermatology" for treatment
  - Follow diagnostic procedure for eosinophilic granuloma complex

Feline miliary dermatitis:

- **Manifestations:**
  - Eruption of crusting papular lesions
- **Regions:**
  - Back, lumbar, hindlimbs and neck → leading to alopecia, excoriation, crusts and pruritus
- **Causes:**
  - Allergies: Flea allergy dermatitis, food allergy, atopy
  - Parasites:
    - Notoedric mange (*Notoedres cati*)
    - Otodectic mange (*Otodectes cynotis*)
    - Demodecosis (*Demodex gatoi/cati*)
    - Cheyletiellosis (*Cheyletiella blakei*)
  - Infections: Dermatophytosis, pyoderma
  - Nutritional disorders: Essential fatty acid deficiency
  - Idiopathic disorders
  - Neoplasia – Mast cell tumours
- **Diagnostic pathway:**
  - History and general physical examination: Fleas, flea dirt, food history
  - Perform a skin scrape (deep and superficial): Mites, see "Dermatology" for treatment
  - Hair pluck, skin scrape, woods lamp and culture: Dermatophytosis, see "Dermatology" for treatment
  - Sticky tape impressions: Pyoderma, mites, see "Dermatology" for treatment
  - Follow diagnostic procedure for eosinophilic granuloma complex

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Nodular lesions:		
Single nodule:	**Multiple nodules:**	**Nodule with drainage tracts:**
▪ Neoplastic ▪ Sterile inflammatory ▪ Abscess	▪ Neoplastic ▪ Severe infection ▪ Sterile inflammatory ▪ Mycobacterial	▪ Abscess (most common) ▪ Severe bacterial infection ▪ Fungal infection ▪ Foreign bodies ▪ Mycobacterial
▪ **Diagnostic pathway:** ➢ History and general physical examination: ➢ Biopsy of lesion and send for histopathology and culture and sensitivity (include mycobacterial)		

Causes:

- **Allergies:**
  - ✓ Flea allergy dermatitis:
    - ➢ Hypersensitivity to flea saliva
    - ➢ Any age/sex
    - ➢ Moderate-severe pruritus
    - ➢ Responsive to corticosteroids and flea control program
    - ➢ **Diagnosis:** Clinical signs, fleas and flea dirt, response to corticosteroids and flea control
  - ✓ Food allergy:
    - ➢ Immunological reaction to food proteins
    - ➢ Any age/sex but Burmese and Siamese - predisposed
    - ➢ Mild to severe pruritus, +/- gastrointestinal signs (vomiting and diarrhoea)
    - ➢ Variable response to corticosteroids
    - ➢ **Diagnosis:** Food elimination trial for 8 weeks (e.g. Pork, lamb and potato, sweet potato OR Hills Z/D®)
  - ✓ Atopy:
    - ➢ Hypersensitivity to environmental allergens (dust mites > pollen, mould)
    - ➢ Starts 6 months to 3 years
    - ➢ Moderate to severe pruritus also +/- rhinitis, cough, dyspnoea (asthma)
    - ➢ **Diagnosis:** History, clinical signs, response to corticosteroids and elimination of other allergies (food and flea allergy dermatitis)
  - ✓ Mosquito bite hypersensitivity:
    - ➢ Papules, crusting, swellings on nose, pinnae and periauricular regions
    - ➢ **Diagnosis:** History (mosquitos), clinical signs, response to corticosteroids
- **Parasites:**
  - ✓ Notoedric mange (*Notoedres cati*):
    - ➢ Crusted papules on head and neck → front and back toes, perineum → excoriations, alopecia and crusts
    - ➢ **Diagnosis:** Skin scrapings (mites/eggs)
  - ✓ Otodectic mange (*Otodectes cynotis*):
    - ➢ Otitis externa, dry waxy secretions (amber to black)
    - ➢ Crusted papules in ear canal
    - ➢ **Diagnosis:** Mites visualised with naked eye or magnification in ear canal or in wax, skin scrapings
  - ✓ Demodecosis (*Demodex gatoi/cati*):
    - ➢ Localised skin lesions on face (around ears, eyes and chin) and neck
    - ➢ Generalised skin lesions on trunk and limbs, can indicate systemic illness
    - ➢ Alopecia, erythema, scaling and crusts and pruritus
    - ➢ **Diagnosis:** Skin scrapings (mites/eggs), hair plucks

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- ✓ Cheyletiellosis (*Cheyletiella blakei*):
  - ➢ Crusted papules on back
  - ➢ **Diagnosis:** Skin scrapings (mites/eggs), hair pluck, flea comb and sticky tape impressions

- **Psychogenic alopecia:**
  - ✓ Pruritus despite corticosteroid therapy

- **Infectious:**
  - ✓ Superficial feline pyoderma:
    - ➢ Localised alopecia, +/- erythema, papules, pustules, erosions, ulcers and crusts
    - ➢ Primary rare in cats, usually secondary to allergies, mites, systemic disease (FHV) or immunosuppressive therapy
    - ➢ **Diagnosis:** Impression smears and sticky tape impressions
  - ✓ Dermatophytosis (Ringworm): *Microsporum canis*
    - ➢ Highly contagious, affects face, head, neck and limbs +/- trunk
    - ➢ **Lesions:** Localised or diffuse, alopecia, erythema, scaling, small crusted papules (miliary dermatitis)
    - ➢ **Diagnosis:** Fungal culture, skin scrape and hair pluck, woods lamp (50%)
  - ✓ Mycobacterial: *Mycobacteria fortuitum, chelonei, smegmatis*
    - ➢ Non-healing ulcerated nodules with fistulas tracts
    - ➢ Abdominal and inguinal regions usually
    - ➢ **Diagnosis:** Biopsy: Histopathology and culture and sensitivity (mycobacterial)

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Diarrhoea and Haematochezia

- **This chapter covers:**
  - ✓ General features and differentials of small and large intestinal diarrhoea
  - ✓ Differentials for haematochezia and protein losing enteropathy
  - ✓ Basic diagnostic pathways
  - ✓ Empirical management of diarrhoea patients
  - ✓ Treatment for specific conditions

- **History:**
  - ✓ Duration?
    - ➢ Acute or chronic
  - ✓ Any weight loss, vomiting, blood in diarrhoea, lethargy?
    - ➢ Determine the severity
  - ✓ Description of diarrhoea?
    - ➢ Determine if small or large intestinal, both
  - ✓ Diet history, vaccination, deworming, concurrent medication?
    - ➢ Determine the possible cause

Diarrhoea:	
Small intestinal:	**Large intestinal:**
Features: ▪ Increased faecal bulk/water ▪ No straining ▪ Projectile ▪ Melaena ▪ Not urgent	**Features:** ▪ Small amount of faeces ▪ More frequent ▪ Painful straining ▪ Mucous ▪ Fresh blood
Mixed bowel:	
▪ Increased faecal bulk/water ▪ Straining ▪ Fresh blood ▪ Mucous ▪ Not urgent	

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Small intestinal:

Acute:	Chronic:
▪ **Gastrointestinal disease:** ➢ Diet: • Indiscretion • Diet change ➢ Parasites: • Hookworm, roundworms, *Giardia* and *Coccidia* ➢ Viruses: • Parvovirus (common in dogs, rare in cats) • Coronavirus, rotavirus ➢ Bacterial: • Food poisoning • *Campylobacter, E. Coli, Salmonella, Clostridia* ➢ Toxicity: • Drugs (NSAID's) • Organophosphates ➢ Haemorrhagic gastroenteritis ➢ Structural obstructions: • Foreign body • Intussusceptions ▪ **Extra-gastrointestinal disease:** ➢ Pancreatic disease: • Pancreatitis • Exocrine pancreatic insufficiency ➢ Liver/kidney disease ➢ Hypoadrenocorticism ➢ Ischaemic: • Anything leading to shock or ischaemia of the GIT e.g. Heat stroke	▪ **Gastrointestinal disease:** ➢ Diet: • Intolerance • Hypersensitivity ➢ Infectious: • Parasites: Hookworm, roundworms, *Giardia* and *Coccidia* • Bacterial infection: Antibiotic responsive enteropathy (SIBO), *Campylobacter, Clostridia, Salmonella* • Fungal: *Histoplasmosis* and *Pythiosis* ➢ Infiltrative: • Inflammatory bowel disease • Lymphangiectasia • Neoplasia - lymphoma, adenocarcinoma ➢ Breed related enteropathy (German Shepherds and Basenjis) ➢ Structural obstructions: • Foreign body/intussusceptions ▪ **Extra-gastrointestinal disease:** ➢ Pancreatic disease: • Pancreatitis • Exocrine pancreatic insufficiency ➢ Liver/kidney disease ➢ Structural obstructions: • Foreign body • Intussusceptions • Volvulus ➢ Endocrine: • Hypoadrenocorticism • Hyperthyroidism (cats)

Large intestinal:

- Infectious:
  - ➢ Parasites: Whipworm, *Tritrichomonas, Cryptosporidia*
  - ➢ Bacterial: *Clostridia*
  - ➢ Fungal: *Pythiosis, Histoplasmosis*
- Diet:
  - ➢ Fibre-deficiency
  - ➢ Food intolerance
  - ➢ Indiscretion
- Infiltrative:
  - ➢ Inflammatory bowel disease
  - ➢ Lymphangiectasia
  - ➢ Neoplasia - lymphoma, adenocarcinoma)
- Structural obstructions:
  - ➢ Foreign body
  - ➢ Intussusceptions
- Strictures

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- **Causes of haematochezia:**
  - ✓ Inflammatory – inflammatory bowel disease, histiocytic ulcerative colitis (boxers)
  - ✓ Infectious:
    - ➢ Parasites: Whipworm, hookworm, *Giardia*
    - ➢ Bacterial: *Clostridia*
    - ➢ Viral: Parvovirus
    - ➢ Fungal: Histoplasmosis, Pythiosis
  - ✓ Neoplasia:
    - ➢ Lymphoma
    - ➢ Adenocarcinoma
  - ✓ Trauma and coagulopathy
  - ✓ Haemorrhagic gastroenteritis
  - ✓ Anal gland disorders

Diagnostics:

- **All cases of diarrhoea/haematochezia:**
  - ✓ General physical examination and rectal examination
  - ✓ PCV/TP
  - ✓ Coagulation testing if haematochezia
  - ✓ Faecal smears and faecal floatation:
    - ➢ Assess for parasitic causes
    - ➢ Stained: Normal bacterial population is mixed, uniform population is abnormal, large spore forming gram positive rods (clostridia – look like "safety pins")
    - ➢ Wet preparation: Assess for motile bacteria (shoot through the field)
  - ✓ *Giardia* ELISA test
  - ✓ Virus testing:
    - ➢ Parvovirus/coronavirus antigen test

- **Indications for further diagnostics:**
  - ✓ Hypoproteinaemia (DDx: protein losing enteropathy/nephropathy, liver disease)
  - ✓ Anaemia
  - ✓ Systemic signs of illness and abdominal pain
  - ✓ Reoccurring after symptomatic therapy
  - ✓ Older animal
  - ✓ Polyphagia, steatorrhea
  - ✓ Weight loss
  - ✓ Palpable abdominal or rectal abnormality

- **Other diagnostics for chronic diarrhoea/haematochezia:**
  - ✓ Biochemistry and haematology, urinalysis
  - ✓ UP:C to rule out extra-gastrointestinal causes of hypoproteinaemia
  - ✓ Total T4: Hyperthyroidism
  - ✓ Trypsin-like immunoreactivity: To assess for exocrine pancreatic insufficiency
  - ✓ FIV and FeLV
  - ✓ Imagery:
    - ➢ Radiography
    - ➢ Ultrasound +/- aspirate
  - ✓ Serum folate:
    - ➢ Decreased can be due to jejunal abnormalities leading to malabsorption of folate
    - ➢ Increase can be consistent with increased bacterial population e.g. Bacterial overgrowth

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- ✓ Serum cobalamin:
  - ➢ Decreased can be due to ileal abnormalities leading to malabsorption of cobalamin
  - ➢ Important in feline chronic gastrointestinal disease as supplementation can improve clinical outcome
- ✓ Endoscopy and mucosal biopsy
- ✓ Laparotomy and full thickness biopsy

- **Treatment according to clinical signs:**

Acute and not severe, systemically well:
▪ Symptomatic treatment: ➢ Diet change to novel or hydrolyzed diet ➢ Smaller meals of increased frequency ➢ +/- Fenbendazole 50mg/kg SID for 5 days ➢ +/- Antibiotics if large breed or suspecting antibiotic responsive enteropathy • Metronidazole 10mg/kg PO BID
Acute and severe, small intestinal:
▪ Hospitalise: Supportive therapy, IV fluid support and IV antibiotics ▪ Bowel rest (adults 24 hours, NOT in puppies) ▪ Further diagnostics
Chronic, large intestinal:
▪ Rectal examination: Palpate for any abnormalities such as mass lesions or narrowing ▪ Symptomatic treatment: ➢ Fibre supplementation psyllium 1-2 tablespoons per day or low residue diet ➢ Fenbendazole 50mg/kg SID for 3 days ▪ Failure to respond try hydrolysed or novel protein diet ▪ Endoscopy and biopsy
Chronic, small intestinal:
▪ Failure to respond to empirical trials: ➢ Diet change to novel or hydrolyzed diet ➢ Fenbendazole 50mg/kg SID for 3 days ➢ Antibiotics for 2-3 weeks if large breed or suspecting antibiotic responsive enteropathy (SIBO) • Metronidazole 10mg/kg BID OR • Tylosin 20mg/kg BID OR • Oxytetracycline 15mg/kg BID ▪ Blood profile: Assessment of systemic disease ▪ TLI: Assessment of EPI ▪ Imagery and endoscopy and biopsy

- **<u>Parasitic gastroenteritis:</u>**
- ***Giardia* (Zoonotic):**
  - ✓ Fenbendazole 50mg/kg PO SID for 3 days
  - ✓ Metronidazole 20mg/kg PO BID for 10 days (higher dose required – but beware neurological signs)
- **Intestinal worms:**
  - ✓ Roundworm, Hookworm, Whipworm:
    - ➢ Fenbendazole 50mg/kg PO SID for 3 days, off label in cats
  - ✓ Tapeworms:
    - ➢ Praziquantel 3-7mg/kg PO
    - ➢ 4 times label dose for *Spirometra*
- ***Coccidia spp*:**
  - ✓ 10 – 40µm in length depending on the species
  - ✓ Toltrazuril 20mg/kg PO SID for 2 days

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- **Cryptosporidium (Zoonotic) (5µm in length):**
  - ✓ Can be present in low numbers normally, and may be associated with other causes of diarrhoea e.g. parasites (worms and *Giardia)* and viruses but can contribute to the severity of the diarrhoea
  - ✓ Most commonly in young animals
  - ✓ Treat underlying disease process and manage symptomatically
- **Campylobacter (Zoonotic):**
  - ✓ Small, gram negative, curved rod, motile bacteria
  - ✓ Can be present in low numbers normally, and may be associated with other causes of diarrhoea e.g. parasites (worms and *Giardia)* and viruses but can contribute to the severity of the diarrhoea
  - ✓ Erythromycin 10mg/kg PO TID for 1 to 2 weeks
  - ✓ Tylosin 15mg/kg PO BID for 7 days

- **Haemorrhagic gastroenteritis:**
- **Pathophysiology:**
  - ✓ Thought to be due to either a hypersensitivity or Clostridia toxins
  - ✓ Inflammation leads to rapid loss of fluid into gastrointestinal tract leading to marked haemoconcentration and dehydration
  - ✓ Associated with acute vomiting and then diarrhoea with blood, symptoms of shock
  - ✓ Must rule out other causes of haemorrhagic vomiting and diarrhoea
- **Treatment:**
  - ✓ Aggressive IV fluid therapy, correction of perfusion and dehydration deficits and electrolyte abnormalities
  - ✓ Antibiotics: Ampicillin 22mg/kg TID or metronidazole 10mg/kg IV BID
  - ✓ Antiemetic: Metoclopramide CRI 1-2mg/kg/day IV and others
  - ✓ Gastric protectants: Ranitidine 2mg/kg BID, sucralfate 0.5-1gm PO TID, proton pump inhibitors
  - ✓ Anaemia: Blood transfusion, see **"Transfusion therapy"**
  - ✓ +/- Colloid therapy: If hypoproteinaemia develops either synthetic colloid or plasma transfusion

- **Viral diarrhoea:**
- See also **"Viral diseases and Vaccination"**
- **Features:**
  - ✓ Typically, in young unvaccinated puppies
  - ✓ Parvovirus is a very severe debilitating disease, coronavirus is usually less severe

- **Parvovirus:**
- **Pathophysiology:**
  - ✓ Parvovirus targets and destroys rapidly dividing cells such as intestinal lining and bone marrow
  - ✓ Typically, in young unvaccinated dogs, but can occur in previously vaccinated animals
  - ✓ False positives: Can occur up to 12 days post-vaccination especially if a modified live vaccine was used. Still manage as a positive if consistent clinical signs and history. Leukopenia can help provide supportive evidence.
  - ✓ Feline panleucopenia virus: Rare in cats, use canine parvovirus test to diagnose
  - ✓ 90% mortality rate without treatment, 80% survival rate with aggressive management
  - ✓ Resilient viruses, persist in the environment for up to 8 months, likely to be a major source of infection
  - ✓ Highly contagious viruses that are spread primarily by ingestion of affected animal's faecal material:
- **Diagnostics:**
  - ✓ Parvovirus antigen ELISA
  - ✓ PCV/TP, haematology, biochemistry
  - ✓ Others listed above (e.g. faecal analysis)

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- **Treatment:**
  - ✓ Isolation and barrier nursing
  - ✓ Supportive therapy: Keep warm and quiet
  - ✓ Fluid therapy: Aggressive IV fluid therapy, correction of perfusion and dehydration deficits and electrolyte abnormalities
  - ✓ Antiemetic: Metoclopramide CRI 1-2mg/kg/day IV, maropitant 1mg/kg SC SID for <5 days
  - ✓ Gastric protectants: Ranitidine 2mg/kg BID, sucralfate 0.5-1gm PO TID, proton pump inhibitors
  - ✓ IV antibiotics:
    - ➢ Broad spectrum bactericidal
    - ➢ If not leukopenia: Cephalothin 22mg/kg IV TID and metronidazole 10mg/kg IV BID
    - ➢ If leukopenia: Ticarcillin 50mg/kg IV QID
  - ✓ Transfusions:
    - ➢ Blood if anaemia develops
    - ➢ +/- Plasma for oncotic support if hypoalbuminaemia is present
  - ✓ Early enteral nutrition:
    - ➢ Important, start if anorexic >2 days
    - ➢ Micro-enteral feeding with electrolyte solutions via tube feeding (naso-oesophageal tubes) then progress to food
- **Monitoring:**
  - ✓ Temperature, pulse and respiration, hydration status QID, body weight, PCV/TP and electrolytes SID-BID
- **Prevention:**
  - ✓ Vaccination is very effective, vaccinate all animals
  - ✓ Isolation of infected animal as they can shed virus for up to 40 days after recovery
  - ✓ Beware the virus can remain in the environment for up to 8 months, do not allow unvaccinated animals access to that environment
  - ✓ Isolation of puppies away from other unvaccinated animals at least 2 weeks after final vaccination
  - ✓ Clean and disinfect the environment

- **<u>Protein losing enteropathy (PLE):</u>**
- **Pathophysiology:**
  - ✓ A cause of hypoproteinaemia, due to a loss of protein through the gastrointestinal tract
  - ✓ Always see a loss of albumin but usually also globulin
  - ✓ Diarrhoea with hypoalbuminaemia suggests PLE but hypoalbuminaemia without diarrhoea does not rule out PLE
  - ✓ May lose antithrombin III and predispose to thromboembolism
- **Caused:**
  - ✓ Generally, by chronic gastrointestinal disease such as inflammatory bowel disease, neoplasia (lymphoma and adenocarcinoma), infectious diseases (parasites, fungal infections), lymphanglectasia, gastric ulcerations, cardiac disease (RHS). May also be caused by acute gastrointestinal disease such as canine haemorrhagic gastroenteritis.
- **Clinical signs:**
  - ✓ Weight loss
  - ✓ +/- Diarrhoea, +/- vomiting
    - ➢ Lack of vomiting or diarrhoea does not rule out PLE
  - ✓ +/- Abdominal or pleural effusions, peripheral oedema
- **Diagnostics:**
  - ✓ Require full diagnostic work-up, including biopsies (via endoscopy or laparotomy) and histopathology

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- **Inflammatory bowel disease (IBD):**
  - ✓ Inflammation of the small and large intestine
  - ✓ Diagnosis is based on biopsy and histopathology
  - ✓ Types of inflammation:
    - ➢ Small intestine: Lymphocytic plasmacytic (most common form in both dogs and cats), but can be eosinophilic
    - ➢ Large intestine: Lymphocytic plasmacytic, eosinophilic, histiocytic ulcerative colitis (boxers and French bulldogs), fibre-responsive
  - ✓ Feline IBD is commonly associated with chronic pancreatitis or cholangiohepatitis

- **Lymphocytic plasmacytic inflammatory bowel disease:**
  - ✓ **Small intestinal:**
    - ➢ Diet change to novel or hydrolysed diet:
      - Important in cats
    - ➢ Immunosuppressive therapy:
      - Prednisolone at 1-2mg/kg PO BID until resolution, then 20% reduction every couple weeks
        - Dogs: Azathioprine or budesonide can be used for long term control or adjunctive therapy
        - Cats: Chlorambucil good for long term but monitor for immunosuppression
    - ➢ +/- Metronidazole 10mg/kg PO BID
    - ➢ Supplement:
      - Omega 3/6 fatty acids
      - Vitamin B12 injection at 250µg SC weekly or fortnightly
  - ✓ **Large intestinal:**
    - ➢ Diet change to novel or hydrolysed diet
    - ➢ If no response:
      - Trial fibre supplementation and sulfasalazine
      - Prednisolone at 1-2mg/kg PO BID until resolution, then 20% reduction every couple weeks

- **Eosinophilic inflammatory bowel disease:**
  - ✓ Must rule out hypersensitivity, hypereosinophilic syndrome and parasites
  - ✓ Immunosuppression therapy and novel or hydrolysed diet

- **Histiocytic ulcerative colitis:**
  - ✓ Usually in Boxers and French Bulldogs, associated with haematochezia
  - ✓ Responsive to enrofloxacin 5mg/kg/day PO 6-8 weeks

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Dysphagia and Oral Disease

- **This chapter covers:**
  - ✓ Dysphagia clinical features, differentials and general treatment
  - ✓ Basic information on common oral tumours

- **Dysphagia:**
- **Clinical signs:**
  - ✓ Inappetance, weight loss, halitosis, pawing at mouth, hypersalivation, facial swelling, oral haemorrhage, nasal discharge
- **Differentials:**
  - ✓ Dental disease:
    - ➢ Feline odontoclastic resorptive lesion (FORL), see following pages
    - ➢ Abscessation
  - ✓ Trauma
  - ✓ Foreign bodies
  - ✓ Neurological:
    - ➢ Cranial nerve dysfunction:
      - CN V: Trigeminal to pharynx
      - CN VII: Facial to oral voluntary muscles
      - CN XII: Hypoglossal to tongue
      - CN IX: Glossopharyngeal to pharynx
      - CN X: Vagus to pharynx and oesophagus
    - ➢ Myasthenia gravis (focal)
  - ✓ Temporomandibular joint disease
  - ✓ Fractures
  - ✓ Masticatory muscle myositis, see following pages
  - ✓ Abscessation:
    - ➢ Oral and retrobulbar
  - ✓ Neoplasia, see following pages
  - ✓ Oral ulceration, see following pages
  - ✓ Oesophageal disease
  - ✓ Salivary gland disease, see following pages
- **Diagnostics:**
  - ✓ History
  - ✓ General physical examination:
    - ➢ Able to open and close mouth properly
    - ➢ Signs of systemic disease
    - ➢ Palpation of the muscles of mastication and temporomandibular joint
    - ➢ Cranial nerve function
  - ✓ Oral examination (under general anaesthetic if required)
  - ✓ Imagery:
    - ➢ Radiology, scoping, CT, MRI
  - ✓ Biopsy and histopathology
  - ✓ FeLV, FIV, FHV, FCV (PCR on secretions and ELISA testing)

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- **Oral ulceration:**
- **Clinical signs:**
  - ✓ Clinical signs of dysphagia
- **Diagnostics:**
  - ✓ History and general physical examination
  - ✓ Haematology, biochemistry and urinalysis
  - ✓ Biopsy
  - ✓ Feline virus testing (PCR on secretions and ELISA testing)
- **Differentials:**
  - ✓ Caustic substance ingestion
  - ✓ Immune mediated diseases:
    - ➢ Systemic lupus erythematosus, vacuities, pemphigus, ulcerative gingivitis/stomatitis (Maltese)
  - ✓ Inflammatory:
    - ➢ Feline eosinophilic granuloma complex, see following pages
    - ➢ Feline gingivitis-stomatitis, see following pages
  - ✓ Infectious diseases:
    - ➢ FIV, FeLV
    - ➢ FHV, FCV: Clinical signs of upper respiratory tract (sneezing and nasal discharge), also +/- ocular discharge
    - ➢ Fungal (*Candida sp.*)
  - ✓ Palatine ulcers:
    - ➢ Ulcerative lesion on the roof of the mouth usually due to **over grooming**
    - ➢ Present for anaemia due to blood loss and erosion of palatine blood vessels
  - ✓ Neoplasia, see following pages
  - ✓ Systemic disease: E.g. renal failure

- **Masticatory muscle myositis:**
- **Pathogenesis:**
  - ✓ Immune attack on the type 2M fibres present in the temporalis and masseter muscles
  - ✓ Aggressive forms seen with Dobermans and Rottweilers
- **Clinical signs:**
  - ✓ Acute stage: Inflammation
    - ➢ Clinical signs of dysphagia, pain on opening of the mouth, swelling and pain of the masticatory muscles exophthalmos
  - ✓ Chronic stage: Fibrosis
    - ➢ Unable to open mouth, bilateral masticatory muscle atrophy
- **Diagnostics:**
  - ✓ Clinical presentation
  - ✓ Absence of temporomandibular joint disease
  - ✓ Difficulty in opening the mouth under general anaesthetic
  - ✓ Biopsy:
    - ➢ Histopathology
  - ✓ Positive serum 2M-antibodies
- **Treatment:**
  - ✓ Immunosuppression: Prednisolone 1-2mg/kg PO BID until resolution, then 20% reduction every couple weeks
  - ✓ Nutrition: Nasoesophageal/gastric or oesophageal tube

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Salivary gland disease:

Types:

- ✓ Sialocele aka "salivary mucocele":
  - ➢ Enlargement associated with a salivary gland due to accumulation of saliva within the surrounding tissue
  - ➢ Idiopathic (usually), trauma around that region
  - ➢ Ranula, under the tongue
- ✓ Sialoadenosis:
  - ➢ Bilateral non-inflammatory enlargement that is non-painful
- ✓ Sialoadenitis:
  - ➢ Bilateral inflammatory enlargement that is mildly painful
  - ➢ Typically, secondary to prolonged vomiting or regurgitation
- ✓ Salivary gland necrosis:
  - ➢ Bilateral inflammatory enlargement that is very painful
  - ➢ This condition is particularly associated with oesophageal disease such as *Spirocercosis*

- **Clinical signs:**
  - ✓ Clinical signs of dysphagia and uni/bilateral enlargement of salivary glands

Diagnostics:

- ✓ General physical examination, FNA, radiographs +/- iodine contrast

Feline eosinophilic granuloma complex:

Pathogenesis:

- ✓ Causes indolent ulcers on the lips or oral mucosa
- ✓ Unknown cause possible hypersensitivity

Diagnosis:

- ✓ Biopsy and histopathology
- ✓ +/- Intradermal skin testing, food elimination trial

- **Treatment:**
  - ✓ Prednisolone 2-4mg/kg/day PO BID until resolution, then 20% reduction every
  - ✓ Good ectoparasite control
  - ✓ Food elimination trial

Feline odontoclastic resorptive lesion (FORL):

Pathogenesis:

- ✓ Common in older cats
- ✓ Odontoclast cells become reactivated (cause unknown)
- ✓ Odontoclast attack the roots of the teeth, leading to cavities and gingival hyperplasia and pain

Diagnosis and treatment:

- ✓ Visual examination and probing under gums
- ✓ Dental radiographs
- ✓ Removal of all affected teeth

Feline chronic gingivitis-stomatitis:

- **Pathogenesis:**
  - ✓ Immune mediated, possible calicivirus infection
  - ✓ Typically, lymphocytic-plasmacytic inflammation
- **Clinical signs:**
  - ✓ Clinical signs of dysphagia
  - ✓ Ulcerations of the gingiva, buccal mucosa, tongue, pharynx
- **Diagnostics:**
  - ✓ Biopsy and histopathology

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✓ Virus testing: FIV, FeLV, feline calicivirus (PCR)

Treatment:

✓ Dental prophylaxis

✓ Medical management:

- ➢ Antibiotics:
  - Clindamycin 10mg/kg PO SID or
  - Metronidazole 10 mg/kg PO BID
- ➢ Anti-inflammatories:
  - Prednisolone 1mg/kg PO BID
- ➢ Pain relief:
  - Oral tramadol or buprenorphine

✓ Whole mouth extractions (+/- canines):

- ➢ Good success rates with mouth extraction but if calicivirus positive then may not do well:
  - Trial anti-viral agents if calicivirus positive
  - Trial cyclosporine at 5mg/kg PO SID as chronic immunosuppressive therapy

Oral tumours:

Dogs: Malignant melanoma > squamous cell carcinoma > fibrosarcoma

Cats: Squamous cell carcinoma >>> fibrosarcoma

Type:	Information:
Melanoma:	• **Features:** ➢ Highly malignant, most common malignant oral tumour (25%) ➢ Metastasis is common 80%, sub-mandibular lymph nodes and lung ➢ Arise from oral or gingival melanocytes ➢ Older middle dogs, rare in cats • **Treatment:** ➢ Surgical excision +/- intralesional chemotherapy +/- radiation therapy • **Prognosis:** 1-2 years if small and completely removed, but usually less
Squamous cell carcinoma:	• **Features:** ➢ Malignant, most common oral tumour in cats (75%), second most common malignant tumour in dogs (20%) ➢ If in the caudal mouth, then highly metastatic and high rate of reoccurrence ➢ Locally invasive into surrounding bone • **Treatment:** ➢ Nasal: Removal of nose and chemotherapy ➢ Maxillary/mandibular: Surgical removal not tolerated well in cats, dogs ok, and radiation • **Prognosis:** 60% for 1 year if in rostral mouth and good removal, if caudal mouth = poor due to metastatic spread
Fibrosarcoma:	• **Features:** ➢ Malignant, third most common malignant tumour in dogs (15%) ➢ Older large breed dogs ➢ If in younger animal may have a higher rate of metastatic spread ➢ Locally invasive, originating from gum or hard palate • **Treatment:** ➢ Surgical resection +/- radiation therapy • **Prognosis:** 50% for 1 year

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Epulis:	• **Features:** ➢ Most common benign oral tumour ➢ Older large breed dogs • **Types:** ➢ Acanthomatous: Arise from the periodontal ligament, locally invasive into bone and can cause dental disruption ➢ Fibromatous/ossifying: Arise from dental laminar epithelium, not very invasive • **Treatment:** ➢ Acanthomatous: Surgical removal with good margins if invades bone ➢ Fibromatous/ossifying: Surgical removal, not require bone margins • **Prognosis:** Excellent

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Ear Disease

- **This chapter covers:**
  - ✓ General information about ear disease
  - ✓ Common causes and diagnosis
  - ✓ Treatment options for different diseases

- **Otitis interna/media:**
  - ✓ Inflammation of the inner or middle ear
  - ✓ Can see signs of vestibular disease such as head tilt, nystagmus, ataxia, vomiting
  - ✓ Cranial nerve deficits e.g. Horner's, facial nerve paralysis
  - ✓ Causes:
    - ➢ Potentially spread from otitis externa to media to interna, haematogenous from the nasopharynx via the eustachian tube

- **Otitis Externa:**
  - ✓ Primary and predisposing causes:
    - ➢ Unilateral disease:
      - Foreign bodies (grass seeds)
      - Tumours/cysts and nasopharyngeal polyps (cats)
    - ➢ Bilateral disease:
      - Allergy: Atopy (most common cause – 1/3 only have ear disease) and food allergy
      - Endocrine: Hyperadrenocorticism, diabetes mellitus, hypothyroidism
      - Parasites: Mites
      - Other: Immune mediated (pemphigus)
    - ➢ Uni/bilateral disease:
      - Anatomical – stenosis of ear canals, neoplasia, hair
      - Otitis media
  - ✓ Secondary complications:
    - ➢ Infections: Bacteria and yeast
    - ➢ Canal stenosis due to chronic inflammation
    - ➢ Otitis media

- **Diagnostics:**
  - ✓ History:
    - ➢ Shaking head, head tilt, scratching back of ears, rubbing ears on furniture
  - ✓ Full dermatological and general physical examination – assess for predisposing causes or system disease:
    - ➢ Atopy: Itchy feet, ventrum, face
    - ➢ Endocrine disorders
  - ✓ Thorough otoscopic exam
  - ✓ Ear swab:
    - ➢ Cytology and staining
    - ➢ Culture and sensitivity - aerobic and anaerobic culture, results may not reflect sensitivity to topical agents
  - ✓ Skin scrape of pinnae:
    - ➢ Assess for mites (especially *demodex spp.*)
  - ✓ Other diagnostic tests: Low allergy diet, CBC, biochemistry (systemic disease)
  - ✓ Radiographs (otitis media):
    - ➢ Views: Open mouth, oblique, DV and lateral
    - ➢ Soft tissue/fluid opacity but can appear normal, not as sensitive as CT
  - ✓ CT

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Treatment:

Otitis Externa:

Treatment must involve elimination of underlying disease and predisposing factors

- ✓ **If ear drum is ruptured:**
- ✓ Avoid oil based or irritating substances (e.g. chlorhexidine)
- ✓ Avoid aminoglycoside antibiotic preparations (ototoxic)

Cleaning:

- ✓ Complete cleaning especially with severe cases or otitis media, under anaesthesia if un-cooperative
- ✓ Intact membrane: Dilute chlorhexidine (severe cases)
- ✓ Ruptured/unsure membrane: Warm saline
- ✓ Topical cleaner once or twice a week as prevention:
  - ➢ Cerumenolytics (↓ wax): Dioctyl sodium sulfosuccinate, carbamide peroxide
  - ➢ Antiseptics (↓ infectious organisms): Acetic acid, chlorhexidine
  - ➢ Astringents (↓ moisture): Isopropyl alcohol, boric acid, salicylic acid

- **Treatment:**
  - ✓ **Prevention:**
    - ➢ After treating the current ear disease – prevention of otitis externa:
      - Treatment of underlying disease e.g. Atopy
      - Once to twice weekly ear flushes
      - Can add 1mg of dexamethasone per 10ml of topical cleaner e.g. EpiOtic®
  - ✓ **Treatment duration:**
    - ➢ Acute at least 2 weeks
    - ➢ Chronic at least 1 week after resolution of clinical signs and negative cytology findings
    - ➢ Require recheck and cytology every 2 weeks
  - ✓ **Treatment of predisposing causes:**
    - ➢ Atopy: Severe atopy can cause thickening of the outer cartilage folds leading to narrowing and complete closure of the external auditory meatus:
      - MUST reduce the localised inflammation and the systemic allergic disease to effectively treat the otitis externa
      - **Localised inflammation:** Short term:
        - Methylprednisolone acetate - calculate the max dose of and inject into the cartilage folds (spread dose around both ears)
        - Dexamethasone 0.2mg/kg SC then follow with oral prednisolone on a reducing dose
      - **Systemic atopic disease:** Long term - see **"Dermatology"**
    - ➢ Other: foreign body, neoplasia, food allergy, diabetes mellitus, hyperadrenocorticism, hypothyroidism etc.
  - **Ruptured ear drums:**
    - ➢ Cleaning agents:
      - TrizEDTA®: Use as a cleaner, perform 1 hour before topical agents once a day
      - Acetic acid: E.g. MalAcetic otic® once a day
    - ➢ **Topical preparations safe for ruptured ear drums:**
      - Antibiotics: Ciprofloxacin, enrofloxacin, ofloxacin, ticarcillin, ceftazidime:
        - E.g. Enrofloxacin 1.5% and 5mg of dexamethasone
      - Anti-fungal: Clotrimazole, miconazole, nystatin
      - Anti-inflammatory: Dexamethasone

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Compounded ear formulations:

Drugs:	Formula:	Vehicle:
Enrofloxacin 20ml	• Add 6ml enrofloxacin injection 50mg/ml and 2ml 5% dexamethasone: • Added into 12ml vehicle • Final concentration - Enrofloxacin 15mg/ml and Dexamethasone 0.5mg/ml	• Saline or propylene glycol • Not Methopt tears → precipitates • Store refrigerated
Timentin 6% 22ml	• Add 6ml of water for injection to 3.1g of timentin • Divide into 3 syringes (2.5ml each) - freeze two syringes (lasts for 3 months) • Add one syringe into 17mls of saline and add 2.5ml of 5% dexamethasone • Final concentration of Ticarcillin 6% Dexamethasone 0.5mg/ml	• Saline or propylene glycol • Last 1 month with saline • Store refrigerated

Infection:	Cleaning:	Topical:	Systemic:
Yeast:	• Lactic and salicylic acid or acetic acid preparations: ➢ BID for 7 days ➢ Maintenance - twice a week	• Combination of corticosteroid / antifungal • Antifungals: ➢ Miconazole, clotrimazole • Administer: ➢ 0.3ml/ear BID for <10kg ➢ 0.5ml/ear BID for >30kg	• Not usually required • Dogs: Ketoconazole 10-20 mg/kg/day PO up to 6 weeks • Cats: Itraconazole 10mg/kg PO SID up to 6 weeks
Cocci bacteria:	Intact ear drum: • Lactic & salicylic acid: ➢ BID for 14 days Ruptured ear drum: • TrizEDTA®: ➢ Use as a cleaner - perform 1 hour before topical agents SID, safe for ruptured ear drums	• Combination corticosteroid / antiblotic • Intact ear drums: ➢ E.g. Polymyxin B • Ruptured ear drums: ➢ See above or safe drugs and compounded formulations - Enrofloxacin and dexamethasone SID • Administer: ➢ 0.3ml/ear BID for <10kg ➢ 0.5ml/ear BID for >30kg	• If severe or extends to pinna: • Cephalexin 22 mg/kg PO BID

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Infection:	Cleaning:	Topical:	Systemic:
Rod bacteria: • Recommend culture and sensitivity	Intact ear drum: • Lactic & salicylic acid: ➢ BID for 14 days Ruptured ear drum: • TrizEDTA®: ➢ Use as a cleaner - perform 1 hour before topical agents SID, safe for ruptured ear drums	• Combination corticosteroid / antibiotic • Intact ear drums: ➢ E.g. Polymyxin B • Either intact/ruptured ear drums: ➢ See above for safe drugs and compounded formulations - Enrofloxacin and dexamethasone SID • Administer: ➢ 0.3ml/ear BID for <10kg ➢ 0.5ml/ear BID for >30kg • Second line: ➢ Based on C&S: Note concentrations within ear canal are higher than serum	• Based on culture and sensitivity • Empirical: Amoxicillin clavulanic acid 25mg/kg PO BID
Mixed: Treat in order: ➢ 1st rods ➢ 2nd cocci ➢ 3rd yeast	Intact ear drum: • Acetic acid: ➢ SID for 7 days Ruptured ear drum: • Acetic acid: ➢ SID for 7 days • TrizEDTA®: ➢ Use as a cleaner - perform 1 hour before topical agents SID, safe for ruptured ear drums	• Treat as above, but in order of pathogenicity: ➢ 1st rods ➢ 2nd cocci ➢ 3rd yeast • Cocci and yeast infections can be covered with an antibiotic / antifungal preparation • Can alternate treatments – treat bacteria in AM and yeast in PM • Either intact/ruptured ear drums: ➢ See above for safe drugs and compounded formulations – Enrofloxacin and dexamethasone SID	• Treat as above, but in order of pathogenicity: ➢ 1st rods ➢ 2nd cocci ➢ 3rd yeast
arasitic: Mites • *Otodectes cyanotis*	• Treat all in contact • 3-4wks to cover life cycle	• Topical pyrethrins BID for 7 days then off for 7 days then again for 7 days • Off label products/uses: ➢ Ivermectin 300µg/kg PO/SC weekly for 3 weeks OR Frontline® 1-2 drops into ear fortnightly	• Spot on antiparasitic: ➢ Advocate ® / Revolution ® every 2 weeks for 6 weeks

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- **Otitis media/interna:**
- **Clinical signs:**
  - ✓ Intermittent head tilt, otorrhea, head shaking, pain on opening mouth, pain on touching ear
  - ✓ Vestibular disease (head tilt, nystagmus, ataxia, vomiting) and cranial nerve deficits e.g. Horner's, facial nerve palsy
- **Causes:**
  - ✓ **Extension of otitis externa: Present in 50% of chronic otitis externa**
  - ✓ Occasionally ascending infection via the eustachian tube
  - ✓ Iatrogenic causes
  - ✓ Nasopharyngeal polyps
  - ✓ Neoplasia of the middle ear
- **Diagnosis:**
  - ✓ Otoscope examination of ear drum – shape (bulging outward), colour (should be transparent)
  - ✓ Myringotomy:
    - ➢ Must clean ear canal completely
    - ➢ Aim for caudoventral quadrant using a 22-gauge spinal needle, aspirate middle ear contents
    - ➢ Send contents for cytology, culture and sensitivity
  - ✓ Survey radiographs: Increased opacity and lysis of the tympanic bullae, but poor sensitivity
  - ✓ CT and MRI: Best
- **Treatment:**
  - ✓ Topical preparations (water based – not ointments), based on cytology and culture and sensitivity
  - ✓ Systemic antibiotics for 6-8 weeks:
    - ➢ Amoxicillin clavulanic acid 25mg/kg PO BID – good first choice
    - ➢ Enrofloxacin 5mg/kg PO SID
  - ✓ Systemic antifungal drugs for 4-6 weeks:
    - ➢ Ketoconazole 10-20mg/kg/day PO
    - ➢ Itraconazole 5mg/kg PO SID for dogs, 10mg/kg PO SID for cats.

- **Surgical treatment options and indications:**
  - ✓ **Lateral wall resection of the vertical canal:**
    - ➢ When diseases of the external ear canal where the medial wall changes are reversible and the horizontal canal show no change
    - ➢ Tumours of the lateral vertical canal
    - ➢ To improve aeration of the horizontal canal and middle ear
    - ➢ To improve ease of medication
    - ➢ Reoccurrence of ear disease in 70% of patients
  - ✓ **Vertical canal ablation:**
    - ➢ Relapsing and non-responsive patients
    - ➢ Recurrent otitis externa/media or neoplasia
    - ➢ Only after elimination of underlying disease and predisposing causes and medical therapy based on culture and sensitivity for at least 6 weeks and elimination of otitis media
    - ➢ Extensive irreversible pathological changes: calcified/ulcerated/narrow canals, osteomyelitis of tympanic bullae
  - ✓ **Total ear canal ablation:**
    - ➢ Severe trauma of the ear
    - ➢ Horizontal canal or bulla neoplasia
    - ➢ Persistent otitis media OR unable to medicate
    - ➢ Irreversible hyperplasia of canal

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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 extracellular 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		

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Haemorrhagic:	Usually > 30 (>1.018)	

- Usually caused by trauma, neoplasia, coagulopathies
- True haemorrhagic i.e. not iatrogenic:
  - Should not see platelets or erythophagocytosis on smears and sample should not clot
- Time frame:
  - Assess history
  - Compare fluid PCV/TP to peripheral PCV/TP:
    - If PCV/TP is similar = recent bleed, if PCV is low and TP normal = chronic
    - If PCV is increasing or is higher than peripheral, then active bleeding
  - Presence of erythrophagocytosis = chronic

Other:	Variable	

- **Bile:**
  - Green-black tinged fluid
  - Presence of bile pigments
  - Abdominal fluid [bilirubin] > serum [bilirubin], and greater than two times higher
- **Urine:**
  - Abdominal fluid [creatinine] and [K+] > serum [creatinine] and [K+]
  - Perform radiographs, contrast excretory urogram, retrograde urethrography

- **Causes of effusions:**

	Pleural:	Abdominal:
Transudates:	• Reduced oncotic pressure e.g. hypoproteinaemia (albumin <15gm/L) ➢ PLN, PLE, liver disease • Excess IV fluids (cats)	• Reduced oncotic pressure e.g. hypoproteinaemia (albumin <15gm/L) ➢ PLN, PLE, liver disease
Modified transudate:	• Increased capillary hydrostatic pressure e.g. RHS CHF, LHS CHF (cats), pericardial disease • Diaphragmatic hernia • Neoplasia • Lymphatic obstruction e.g. neoplasia, diaphragmatic hernia, abscess • Increased permeability of vessels (blood and lymphatics) e.g. FIP	• Increased capillary hydrostatic pressure: e.g. Portal hypertension: pre/intra/post-hepatic. RHS CHF • Increased permeability of vessels (blood and lymphatics) e.g. FIP
Non-septic Exudate:	• Inflammation: FIP (can have high globulins), liver disease, lung torsion, hernia • Neoplasia	• Neoplasia • Inflammation: Bile (increased [bilirubin] compared to serum), FIP, pancreatitis, torsion, uroabdomen
Septic Exudate:	• Ruptured abscess, foreign body inhalation, penetrating thoracic injury, severe pulmonary infection (especially fungal), oesophageal perforation	• Ruptured abscess (splenic, hepatic, urinary, prostatic), foreign body perforation, penetrating injury, bowel rupture

See also Pleural Effusion in "Respiratory Disease"

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Electrolytes and Blood Gas

This chapter covers:

- ✓ Differentials for changes in electrolytes and blood gas parameters

Na+: Sodium

INCREASED:

- Loss of low-Na+ (hypotonic) fluid:
  - ➢ Diuresis: Drugs (frusemide, corticosteroids, mannitol), diabetes mellitus (osmotic), post obstructive (*low USG*)
  - ➢ GIT losses: Vomiting and diarrhoea (*high USG*)
  - ➢ Renal failure (acute or chronic) (*low USG*)
  - ➢ Burn injuries and third space losses (peritonitis and pancreatitis) (*high USG*)
- Loss of free water:
  - ➢ Diabetes insipidus (*low USG*)
  - ➢ Panting (heat stroke, pyrexia, exercise, seizures)
  - ➢ Reduced water intake
- Increased Na+ retention:
  - ➢ Primary hyperaldosteronism (Conn's syndrome)
  - ➢ Excessive Na+ from IV fluid or bicarbonate therapy, sodium phosphate enemas
- Ingestion of high Na+ foods/water

- **Clinical signs:** (due to shrinking of brain): Weakness, lethargy, ataxia, seizures, stupor coma
- **Mild:** <160
- **Severe** (Neurological CSx): >170

See **"Fluid therapy"** for how to correct

DECREASED:

- Hypoadrenocorticism (+/- ↑ *K+*) (rule out whipworm)
- Vomiting/diarrhoea
- Renal failure (*Azotaemia*)
- Over hydration – IV fluid diuresis
- Water retention:
  - ➢ Heart failure
  - ➢ Effusions (pleural and abdominal fluid)
- Artefact: Hyperlipidaemia, hyperproteinaemia, hyperglycaemia

- **Clinical signs:** (due to swelling of the brain): Lethargy, seizures, coma
- **Mild:** <140 (<150 cats)
- **Severe** (Neurological CSx): <110 – 120

- See **"Fluid therapy"** for how to correct

K+: Potassium

INCREASED:

- Reduced excretion:
  - ➢ Obstructive/rupture uropathy (FLUTD, urolith, stricture) *(azotaemia, anuria)*
  - ➢ Renal failure (anuric or oliguria) (*azotaemia*)
- Hypoadrenocorticism (↓ *Na+ can be normal*) (rule out whipworm and renal disease)
- Redistribution (ICF to ECF):
  - ➢ Crush injury (↑ *CK*)
  - ➢ Acidosis (↓ *pH*) – *commonly metabolic acidosis*
  - ➢ Thrombocytosis (↑ *CK*)
- Artefact:
  - ➢ Haemolysed sample
  - ➢ Very high WCC
  - ➢ Thrombocytosis

- **Clinical signs:** Weakness, collapse, flaccid paralysis, arrhythmia, bradyarrhythmias
- **Mild:** >5.5
- **Moderate:** >6.5
- **Severe:** >9

See **"Fluid therapy"** for how to correct

DECREASED:

- Persistent loss of high-K+ fluid
- Diarrhoea and vomiting
- Diuresis (*Frusemide, IV fluids low in K+*)
- Insulin therapy (shifts into cells)
- Renal failure (polyuric - ↑ excretion) – (*Azotaemia, ↓ USG*)
- Chronic anorexia
- Burmese polymyopathy (↑ *CK due to myopathy*)
- Metabolic alkalosis (*diuretics, vomiting, liver disease, bicarbonate therapy*)

- **Clinical signs:** Weakness, lethargy, anorexia, PU/PD, vomiting, neck ventroflexion (cats), crouched gait
- **Mild:** 3 – 3.5
- **Moderate:** 2.5 – 3
- **Severe:** <2.5

- See **"Fluid therapy"** for how to correct

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Cl-: Chloride

INCREASED:	DECREASED:
▪ Acidosis / hypernatraemia	▪ Alkalosis / hyponatraemia

HCO_3^-: Bicarbonate

INCREASED: **Metabolic alkalosis**	DECREASED: **Metabolic acidosis**
▪ Vomiting: Pyloric obstruction (loss of H+) ▪ ↑ Na+ or ↓ Cl- or ↓ K+ ▪ Hepatic insufficiency ▪ HCO_3- or bicarbonate precursor containing fluid therapy ▪ Diuretics: Furosemide ▪ Compensatory to hypercapnia ▪ See **"Fluid therapy"** for how to correct	▪ Diarrhoea ▪ Vomiting: Duodenal reflux ▪ Polyuric disorders ▪ Renal failure (loss of HCO_3-) ▪ Diabetes Mellitus - DKA ▪ Increased strong acids: Lactate/ketone bodies ▪ Compensatory to hypocapnia ▪ See **"Fluid therapy"** for how to correct

PCO_2 (art/venous):
▪ Measure of alveolar ventilation

INCREASED: **Respiratory acidosis**	DECREASED: **Respiratory alkalosis**
▪ Hypoventilation ▪ Stress, panting ▪ Rebreathing (↑ dead space) ▪ Neuromuscular disease ▪ Airway disease ▪ Compensatory to metabolic alkalosis	▪ Hyperventilation ▪ Hypotension ▪ Fever ▪ Sepsis ▪ Excitement/exercise ▪ Pain ▪ Pulmonary thromboembolism ▪ Compensatory to metabolic acidosis

PO_2 (art/venous):
▪ Measure of alveolar to arterial gas exchange:

INCREASED:	DECREASED:
	▪ Pneumonia ▪ Pulmonary oedema / fibrosis ▪ Sedation ▪ Muscle weakness: Diaphragm weakness – neurological or toxic (botulism, tetanus, tick paralysis / myasthenia gravis)

Anion Gap:
▪ Cations: (Na+, K+, Ca++, Mg++)
▪ Anions: (Cl-, HCO_3-, albumin*, organic acids*, SO_4*, HPO_4-*)
* anions that contribute to the anion gap

INCREASED:	DECREASED:
▪ Increase in anions ▪ Lactic acidosis ▪ Ketoacidosis ▪ Renal insufficiency: ↑ phosphate and sulphate ▪ Toxicities: Ethylene glycol, salicylate ▪ Rhabdomyolysis ▪ Hyperalbuminaemia	▪ Increase in cations (e.g. hypercalcaemia) ▪ Haemodilution ▪ Hypoalbuminaemia

pH [H+ ion]:

INCREASED:	DECREASED:
▪ **↑ pH = Alkalemia**	▪ **↓ pH = Acidemia**
▪ Respiratory or metabolic alkalosis	▪ Respiratory or metabolic acidosis

▪ See **"Fluid Therapy"** for treatment

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Endocrine Disease

- **This chapter covers:**
  - ✓ The common endocrine diseases seen in dogs and cats
  - ✓ General information, diagnostic and treatment options

- **<u>Common endocrine diseases:</u>**
  - ✓ Diabetes mellitus
  - ✓ Diabetes ketoacidosis
  - ✓ Hyperthyroidism
  - ✓ Hypothyroidism
  - ✓ Hyperadrenocorticism
  - ✓ Hypoadrenocorticism

<u>Diabetes mellitus:</u>

- **History:**
  - ✓ Obese animal with recent weight loss
  - ✓ Drinking more water and excessively hungry

- **Pathophysiology:**
  - ✓ ***Type I diabetes mellitus OR insulin dependent:***
    - ➢ No insulin secretion due to pancreatic beta cell destruction
    - ➢ Immune destruction of β-cells, islet cell hypoplasia, pancreatic destruction
    - ➢ **Dogs:** Almost always type 1 diabetes mellitus
  - ✓ ***Type II diabetes mellitus OR non-insulin dependent:***
    - ➢ Resistance to insulin due to diabetogenic hormones (e.g. steroids/hyperadrenocorticism, progesterone) or obesity (cats)
    - ➢ **Cats:** More like type 2 diabetes mellitus but if prolonged hyperglycaemia, glucose toxicity causes irreversible destruction of pancreatic cells ends up like type 1:
      - Obesity is a significant risk factor, stress hyperglycaemia complicates diagnosis and management
    - ➢ If diabetes persists after pregnancy, then more likely to be type 1
- **Risk and precipitating factors:**
  - ✓ Drugs: Corticosteroids, progestogens (should **spey intact females** as progesterone makes management of diabetes mellitus difficult – increases insulin resistance)
  - ✓ Obesity, pancreatitis, hyperadrenocorticism

- **Clinical signs:**
  - ✓ Polyuria, polydipsia, polyphagia, weight loss, lethargy, cataracts and hepatomegaly
  - ✓ Cats can develop diabetic neuropathy seen as hindlimb weakness

- **Diagnosis:**
  - ✓ Blood glucose (>14mmol/L or >250mg/dl = suggestive): See **"Biochemistry"** for other differentials:
    - ➢ Dogs: Measured on at least two occasions after a period of fasting (>8 hours)
    - ➢ Cats:
      - **Stress hyperglycaemia** rarely exceeds >16mmol/L or 290mg/dl
      - If concurrent glucosuria and consistently high blood glucose (remains >16mmol/L or >290mg/dl with repeated measurements) over an 8 hour period, the highly likely to be diabetes
      - Serum fructosamine can help differentiate between stress hyperglycaemia and diabetes
  - ✓ Urinalysis:
    - ➢ Glycosuria: Exceeds renal threshold (>12mmol/L or 210mg/dl)
      - Fanconi syndrome (glucosuria without hyperglycaemia)

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- Evidence of urinary tract infections
- Ketonuria

✓ Serum fructosamine:
- Mean blood glucose concentrations for the last 2 – 3 weeks
- Useful in the diagnosis in cats, rarely needed in dogs
- Not affected by stress hyperglycaemia
- >350µmol/L then indicates persistent hyperglycaemia

✓ Biochemistry:
- ↑ ALP and ALT (hepatic lipidosis)
- Hypercholesterolaemia and hyperlipidaemia, hypertriglyceridaemia
- +/- Ketonaemia

✓ Ultrasound:
- For animals presenting with concurrent signs of systemic illness - look for pancreatitis, cholangiohepatitis, hepatic lipidosis

Canine non-ketotic diabetes mellitus:

- **Treatment:**

✓ **Insulin therapy:**
- **Use intermediate acting or Lente insulin e.g. Caninsulin ®**
- Only if healthy and eating
- Offer food before administering insulin injection
- Dose depends on blood glucose:
  - Caninsulin®:
    - Peak effect within 4 hours, duration approximately 8 hours
    - If >20mmol/L or >360mg/gl start on 0.5U/kg
    - If <20mmol/L or <360mg/gl start on 0.25U/kg TWICE a day
  - Human intermediate acting insulin: 0.3-0.5U/kg TWICE a day
- Perform a blood glucose curve for the first 12 hours after commencing treatment (samples taken every 2 hours)
- If hypoglycaemia occurs <8mmol/L or if <16mmol/L when due for next dose, reduce insulin dose before sending home
- Next blood glucose curve 7 – 10 days
- DO NOT GIVE IF:
  - If not sure if injection went in or missed one, wait until next dose is due
  - If dog does not seem right

✓ **Diet:**
- Initially anything palatable and complete and balanced, later avoid soft moist foods as can cause severe post-prandial hyperglycaemia
- Aim for high fibre and complex carbohydrate diet (e.g. Hill's diets), can blunt post-prandial hyperglycaemia
- Feed half the daily intake twice day coinciding with insulin injections:
  - Thin dogs: Avoid further weight loss as starvation increases ketone production
  - Non-obese: Diet that pet will eat reliably to keep calorie intake constant
  - Obese: Reduce intake to 60% of requirement (of ideal body weight)
- If not eating do not give insulin injection, if only partially eating adjust dose according to how much was eaten, e.g. if only ate half its normal amount of food give half its normal dose of insulin

✓ **Exercise:**
- Only after stabilised
- Regular exercise (same time each day), start low intensity and short duration and gradually increase over couple weeks while monitoring for signs of hypoglycaemia (behaviour changes, weakness, seizures)

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- ➢ May need to reduce amount of insulin given
- ➢ Avoid exercise 6-8 hours after insulin injections (prone to hypoglycaemia)

- **Monitoring:**
  - ✓ Indications of adequate control:
    - ➢ Resolution of clinical signs can be used as a primary indicator of adequacy of glycaemic control:
      - Reduction in clinical signs: Polyuria and polydipsia, polyphagia, cessation of weight loss
    - ➢ 24 hour water intake <60ml/kg/day
    - ➢ Glucosuria but no ketones, if no glucosuria then could be hypoglycaemic
  - ✓ Monitoring at home:
    - ➢ Owners can use a dipstick to measure urine glucose and ketones
    - ➢ Blood glucose monitoring with a glucometer
    - ➢ Measurement of the amount of water drank per day
    - ➢ Monitoring of re-emergence of clinical signs such as polyuria and polydipsia, polyphagia and lethargy
  - ✓ Monitoring at clinic:
    - ➢ Evaluations performed fortnightly:
      - Stable control can take 1-2 months to establish
      - Any changes made after a glucose curve require a repeat curve performed 1 week later
    - ➢ Blood glucose curve (gold standard):
      - Collect initial pre-insulin sample, feed normal type and amount of food, administer insulin once eaten
      - Measurement of blood glucose concentrations every 2 hours for 12 hours
      - Aim of blood glucose curve is to identify:
        - o Pre-insulin blood glucose
        - o Lowest blood glucose reading = NADIR
        - o Time of when NADIR occurs = time of peak effect
        - o Duration of insulin action
      - Aim for:
        - o NADIR to occur between 4-8 hours post insulin and to be between 6-8mmol/L or 110-140mg/dl
  - ✓ **Indications for adjusting insulin dosage:**
    - ➢ NADIR < 5mmol/L or 90mg/dl, need dose reduction
    - ➢ NADIR > 8mmol/L or 140mg/gl, need dose increase
    - ➢ Blood glucose at time of next dosage <10mmol/L or <180mg/dl, need dose reduction
    - ➢ If pre-insulin >25mmol/L or 450mg/dl OR if poor response to insulin and current dose >1.5U/kg – MUST assess for causes of insulin resistance **see below**
    - ➢ Short duration of effect:
      - For example, blood glucose concentrations are only within 6-8mmol/L or 110-140mg/dl for short duration then rapid increase after, consider a change to longer acting insulin (or administer BID if not already)
    - ➢ Prolonged effect: Change to shorter acting insulin
    - ➢ **If having difficulty stabilising, then refer to product manual for trouble-shooting tips**
  - ✓ Fructosamine:
    - ➢ Performed every 4 months
    - ➢ Good glycaemic control = ~400µmol/L
    - ➢ Poor control = >500µmol/L
  - ✓ Indications for investigation:
    - ➢ Persistent polyuria, polydipsia, polyphagia, weight loss, ketonuria, post-insulin weakness, altered behaviour, seizures
- **Insulin resistance:**
  - ✓ Suspect if >1.5U/kg and pre-insulin >25mmol/L or 450mg/dl
  - ✓ If see hyperglycaemia MUST rule out somoygi effect from recent hypoglycaemia

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- ✓ Reasons for insulin resistance/antagonism:
  - ➢ Corticosteroids administration, hyperadrenocorticism, pregnancy, obesity, systemic illness/concurrent disease, insulin anti-bodies (rare)
- ✓ After resolving the cause of the insulin resistance MUST carefully monitor blood concentrations carefully as will need to reduce insulin dose

- **Complications:**
  - ✓ Cataracts, usually due to poor control, occurs within 2 years (~50%)
  - ✓ Seizure/coma (insulin overdose)
  - ✓ Anaemia and haemoglobinaemia
  - ✓ Severe hypophosphataemia
  - ✓ Urinary tract infections: Perform a urinalysis and sediment exam (bacteria, neutrophils etc.)
- **Female non-desexed dogs with diabetes mellitus:**
  - ✓ Progesterone (when in season/pregnant) causes increased insulin resistance, develop symptoms of diabetes when in season
  - ✓ Control diabetes first then desex before next season, achieving control can be very difficult
  - ✓ Predisposed to urinary tract infections, perform urine cultures regularly
  - ✓ Diabetes may resolve once desexed

Feline non-ketotic diabetes mellitus:

- **Insulin therapy:**
  - ✓ Treatment using long acting Lantus: Glargine = Synthetic insulin – very long-acting (very low pH)
  - ✓ Conservative insulin therapy:
    - ➢ If blood glucose >20mmol/L or >360mg/gl → 0.5U/kg of 'ideal body weight' BID
    - ➢ If blood glucose <20mmol/L or <360mg/gl → 0.25U/kg of 'ideal body weight' BID
  - ✓ Post-insulin monitoring:
    - ➢ Perform 12 hour glucose curves for first 3 days sampling every 4 hours:
      - 0 hours (before morning insulin) then every 4 hours
    - ➢ DO NOT increase dose for the first week:
      - Little decrease in [blood glucose] in first 3 days but don't increase dose
    - ➢ Decrease dose and treat if biochemical or clinical hypoglycaemia occurs
    - ➢ Recheck at 1, 2, 3 and 4 weeks after the cat is sent home, and then as required
- **Diet and exercise:**
  - ✓ Obesity and inactivity are significant risk factors for diabetes
  - ✓ Palatable and nutritionally balanced - **high protein, low carbohydrate** (e.g. Hills m/d, fresh meat)
  - ✓ Don't need to co-ordinate food and insulin → Cats don't have significant postprandial hyperglycaemia
  - ✓ Feed ad lib until weight loss stops then restrict if obese and start to lose weight:
    - ➢ Reduce intake to 70% of requirement for animal's ideal body weight
    - ➢ Reduce by only 1-2% of body weight per week, monitor weight once a fortnight
    - ➢ If too rapid, can cause hepatic lipidosis and liver failure
  - ✓ Regular exercise (may need to reduce amount of insulin given)
- **Monitoring:**
  - ✓ Water intake (better than urine glucose):
    - ➢ Dry food ~60ml/kg/day, wet food ~10ml/kg/day if well controlled, otherwise still hyperglycaemic
  - ✓ Urine glucose: Well controlled cats should almost always be 0 or 1+ for urine glucose:
    - ➢ 2+ or greater, may require a dose increase
    - ➢ Glargine, long action therefore minimal periods when blood glucose >16mmol/L or 290mg/dl in cats treated for >2-3 weeks
  - ✓ Fructosamine: Maintain <400µmol/L
  - ✓ Clinical signs:
    - ➢ Poor control: Polyuria, polydipsia, polyphagia and loss of body weight
    - ➢ Episodic weakness/ataxia:

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- Could be hypoglycaemia caused by insulin overdose
- Beware as hypoglycaemia can be fatal

➢ Somogyi effect:

- Rebound hyperglycaemia due adrenalin release during hypoglycaemia
- Especially with higher doses of non-glargine insulin

- **Adjusting insulin dosage:**

Blood Glucose Parameter:	Insulin Dose:
Pre-insulin >20mmol/L or 360mg/dl	Increase by 0.5U
Pre-insulin 15-20mmol/L or 270-360mg/dl &/or Nadir 7-9mmol/L or 130-160mg/dl	Same
Pre-insulin 12-14mmol/L or 215-250mg/dl	Reduce by 0.5U
Pre-insulin <12mmol/L or 215mg/dl	Withhold and check for remission
Nadir 5-7mmol/L or 90-120mg/dl	Reduce by 0.5U
Nadir <5mmol/L 90mg/dl	Reduce by 1U
Clinical hypoglycaemia	Reduce by 50%

- **Diabetic remission:**
  - ✓ >75% remission rate for Glargine + low-carbohydrate diet (c.f. 30% remission rate with other insulins)
  - ✓ Need 1-3 months of good glycaemic control (< 5-9mmol/L or 90-120mg/dl)
  - ✓ β cells recover from glucose toxicity and produce own insulin → more likely if steroids/progestogens in previous 3 months
  - ✓ Is cat in remission??:
    - ➢ After minimum 2 weeks insulin with blood glucose consistently <10mmol/L or 180mg/dl
    - ➢ If pre-insulin BG <10mmol/L or <180mg/dl, withhold insulin and perform 12 hour glucose curve
    - ➢ If at next dosing time:
      - Blood glucose >12mmol/L or >220mg/gl then give 1U BID insulin
      - Blood glucose <12mmol/L or <220mg/gl, stop insulin and discharge with a follow-up visit in 1 week
  - ✓ Is cat in coming out of remission??:
    - ➢ Polyuria, polydipsia, poor body condition (below ideal level)
    - ➢ Urine ketones, is always an abnormal finding

Diabetes ketoacidosis (DKA):

- **Pathophysiology:**
  - ✓ Lack of insulin (either absolute lack or relative due to increased resistance) leads to increased lipolysis releasing free fatty acids from adipose tissue. These are converted to ketones. Excessive production of ketones results in a metabolic acidosis or ketoacidosis
  - ✓ **Typically, this is precipitated by an underlying disease that increases the production of stress hormones which increase insulin resistance**

- **Clinical signs:**
  - ✓ ***Diabetes mellitus:*** Polyuria, polydipsia, polyphagia, weight loss, lethargy
  - ✓ ***Diabetic ketoacidosis (DKA):*** Lethargy, vomiting, anorexia, severity depends on degree of metabolic acidosis and nature of concurrent disease

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- **Diagnosis:**
  - ✓ **Must investigate for precipitating factors**
  - ✓ Diagnosis = hyperglycaemia, glucosuria, ketonuria, and metabolic acidosis
    - ➢ *Diabetes Mellitus:* Blood glucose (persistent fasting >14mmol/L or >250mg/dl) and glucosuria
  - ✓ Urinalysis and dipstick:
    - ➢ Must perform urinalysis and culture and sensitivity to investigate concurrent disease
    - ➢ Urine specific gravity – if low with azotaemia then concurrent intrinsic renal failure
    - ➢ Ketonuria = diabetic ketosis:
      - Detects only acetoacétic acid, does not detect β-hydroxybutyrate or acetone BUT rarely does DKA develop without production of acetoacetic acid
  - ✓ Blood gas:
    - ➢ Metabolic acidosis, pH <7.1 is life threatening
  - ✓ Haematology:
    - ➢ Increased PCV due to dehydration
    - ➢ Stress or inflammatory leukogram
  - ✓ Biochemistry:
    - ➢ Hyperglycaemia
    - ➢ Hyponatraemia and hypokalaemia – can be due to osmotic loss into the urine and osmotic haemodilution
    - ➢ +/- Azotaemia
    - ➢ Other changes depending on underlying disease:
      - E.g. Liver disease, pancreatitis, hyperadrenocorticism
  - ✓ Ultrasound:
    - ➢ Commonly see pancreatitis, cholangiohepatitis
  - ✓ +/- Radiographs
- **Prognosis:**
  - ✓ Mortality rate of dogs and cats with DKA is 30 - 40%
  - ✓ Increased mortality rates with dogs with underlying disease

- **Treatment:**
- **Goals:**
  - ✓ Insulin to suppress breakdown of production of ketones
  - ✓ Correct fluid and electrolyte imbalances
  - ✓ Correct metabolic acidosis
  - ✓ Identify predisposing factors
  - ✓ Slow correction of hyperglycaemia

- **Treatment protocol for healthy DKA:**
- Bright alert and responsive, eating and drinking
  - ✓ Correct underlying fluid deficits and electrolytes derangements
  - ✓ Commence insulin therapy:
    - ➢ Can start with either short acting regular crystalline insulin (e.g. Actrapid ®) at 0.2 U/kg SC TID OR intermediate acting insulin at 0.5 U/kg SC BID
    - ➢ Give food with insulin and free access to water
    - ➢ Monitor blood glucose levels closely every 2 hours
    - ➢ Monitor of any signs of illness
  - ✓ Identification and treatment of underlying cause
  - ✓ When ketoacidosis has resolved can start intermediate acting insulin with less intensive monitoring
  - ✓ Ketonuria may persist for several days despite treatment

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- **Treatment protocol for sick DKA:**
- Depressed, anorexic and dehydrated with severe fluid and electrolyte derangements and marked acidosis
  - ✓ **Fluid therapy:**
    - ➢ Correction of perfusion deficits and hypokalaemia:
      - Selection of IV fluids should be based on sodium balance see **"Fluid Therapy"** for treatment of hyponatraemia which is commonly seen with severe DKA's
    - ➢ Begin correction of dehydration deficits whilst factoring in maintenance requirements and ongoing losses:
      - Restore half of fluid deficits over the following 6 hours, then the rest over 24-36 hours
    - ➢ Potassium:
      - Levels will decrease as therapy continues, hypokalaemia is common complication
      - Insulin therapy move potassium back into cells
      - Hyperglycaemia and acidosis will mask hypokalaemia as potassium shifts from intracellular space to extracellular space. This is then lost into the urine, leads to a whole body potassium deletion.
    - ➢ Phosphate:
      - Hypophosphataemia = serum PO4 <0.5mmol/L
      - Haemolytic anaemia, weakness, ataxia, seizures
      - Supplement with potassium phosphate at 0.01-0.03mmol/kg/hr in Ca+ free fluids recheck in 6 hours or give half K+ supplementation as KCL and half as KPO4 (potassium phosphate)
  - ✓ **Insulin therapy:** Use regular crystalline insulin (e.g. Actrapid ®)
    - ➢ **Aim for gradual reduction of blood glucose levels:**
      - **Do not** decrease blood glucose levels by >3-4mmol/L/hr or >70mg/dl/hr and serum osmolality more than 0.5 to 1osmol/hr as can cause osmotic cerebral oedema
    - ➢ Intermittent low-dose intramuscular injection:
      - Loading dose of regular insulin at 0.2 U/kg IM, followed by 0.1 U/kg IM every hour until blood glucose is <15mmol/L
      - When blood glucose is <14mmol/L or <250mg/dl start a 2.5% - 5% dextrose drip and give regular insulin 0.1 to 0.4 units/kg IM every 4 - 6 hours or SC every 6 - 8 hours until the patient is stable enough to begin intermittent acting insulin
    - ➢ Continuous low-dose intravenous infusion:
      - **More gradual reduction of blood glucose**
      - Regular insulin at 0.05 U/kg/hr (cat) to 0.1 U/kg/hr (dog) continuously in a separate IV line. Use an infusion pump to insure accuracy
      - **OR** add 25U to 500mls of 0.9% saline and run at 1ml/kg/hr (cat) or 2ml/kg/hr (dog), must run through 50mls of solution to coat infusion set to allow correct administration
      - When blood glucose is <14mmol/L or <250mg/dl change IV fluids to 0.9% NaCl + 5% dextrose (remove 100ml out of 1L of saline and add 100ml of 50% glucose) and add K+ if required
  - ✓ **Dextrose drips:**
    - ➢ Provision of carbohydrates to prevent hypoglycaemia
    - ➢ Do not stop insulin therapy (unless hypoglycaemic), increase and decrease concentration of dextrose infusion to prevent hyperglycaemia and hypoglycaemia
  - ✓ **Transition to intermediate acting insulin therapy:**
    - ➢ When blood glucose is 6-12mmol/L or 110-210mg/dl, animal is drinking and eating, remaining hydrated with minimal ketones (in urine and serum), begin treating as an uncomplicated case
  - ✓ **Bicarbonate therapy:**
    - ➢ Controversial
    - ➢ Indications: patient has pH < 7.2 or total C02 (HCO_3) is < 12mmol/L
    - ➢ Restore deficits slowly (over 36 - 48 hours)
      - Amount: mEq of HCO_3 = BWt(kg) x 0.3 x (base deficit OR the amount of HCO_3 you want to correct), give 1/3 IV and the rest into IV fluids and give slowly over 12 hours **OR**

        1mmol/kg or 1ml/kg of 8.4% solution slowly over 20mins
      - Monitor ionised calcium levels (may drop with correction of acidosis)

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- Adverse effects of acidosis: Vasodilation, hypotension, CNS and respiratory depression, arrhythmias
- Why therapy is controversial: Both fluid and insulin therapy will help to resolve acidosis. One mmol of HCO3 is generated from each mmol of ketoacid metabolized
- Adverse effects of HCO3: May worsen hypokalaemia, cause paradoxical cerebral acidosis (with associated CNS dysfunction) and delay decrease in blood lactate and ketone levels

- **Monitoring:**
  - ✓ Insulin therapy will usually resolve hyperglycaemia over 6 - 8 hours and ketosis over 10 - 48 hours (although ketone synthesis is immediately inhibited)
  - ✓ Ketouria may get worse before it gets better as β-hydroxybutyrate is initially converted to acetoacetate
  - ✓ Evaluate blood glucose levels every hour and electrolyte/acid-base status every 4 - 6 hours. Adjust therapy accordingly

- **Complications of therapy:**
  - ✓ **Hypoglycaemia:**
    - Treatment:
      - Give 5ml/kg dextrose 5% IV or 0.5–1ml/kg dextrose 50% IV diluted with saline
      - Continue with glucose supplementation in IV fluid as indicated above
  - ✓ **Hypokalaemia:**
    - Due to insulin and resolution of acidosis driving potassium into cells
    - Treatment:
      - See **"Fluid Therapy"** and add 20mmol/L of potassium into IV fluids as a maintenance adjust accordingly
  - ✓ **Hypophosphataemia:**
    - Due to insulin and resolution of acidosis driving phosphorus into cells
    - Treatment:
      - See **"Fluid Therapy"**
  - ✓ **Renal failure:**
    - Due to a combination of pre-renal and primary renal disease:
      - Pre-renal disease due to hypovolemia and hypotension
    - Monitor urine output by placing a urinary catheter:
      - Aim for >1 - 2 ml/kg/hr after correction of perfusion and hydration deficits and after obtaining normotension
    - Treatment:
      - Correct underlying fluid deficits, if normotensive but no urine output then treat as per acute kidney injury. See **"Renal Disease"**
  - ✓ **Cerebral oedema:**
    - Fast reductions in blood glucose can cause cerebral oedema → brain makes sorbitol when the serum is hyperglycaemic to maintain osmolality and retain water → when hyperglycaemia is resolved → water moves into the brain cells due to the osmotic draw of the sorbitol → swelling and cerebral oedema
    - Prevent by slow reduction in BG <3-4mmol/L/hr or <70mg/dl/hr and slow reduction serum osmolality <0.5 to 1osmol/hr:

Serum osmolality (mOsm/kg) = 2 x (Na mmol/L) + (glucose (mg/dl) / 18) + (BUN (mg/dl) / 2.8)

    - Clinical signs:
      - Neurological signs, altered mentation, seizures
    - Treatment:
      - Mannitol 0.5-1gm/kg slow IV QID
      - Slow down rate of BG reduction

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Hyperthyroidism:

Signalment:

- ✓ Hyperthyroidism in dogs is very rare, but common in cats

Feline hyperthyroidism:

Pathophysiology:

- ✓ Elevated circulating levels of thyroxine (T4) and triiodothyronine (T3)
- ✓ Most common cause is functional adenomatous hyperplasia which causes gland enlargement (goitre), can be bilateral (80%) or unilateral (20%)
- ✓ Mainly older cats (average 9-12 years)
- ✓ Affect multiple organs systems:
  - ➢ Renal effects: Hyperthyroidism can lead to CRF due to increased GRF and proteinuria, they can have either overt CRF or seem normal ("masked") only to have it become overt once the hyperthyroidism is treated
  - ➢ Cardiovascular effects: Can lead to the development of hypertrophic cardiomyopathy, systemic hypertension, see **"Cardiovascular Disease"**

Clinical signs:

- ✓ Weight loss with normal to increased appetite, vomiting +/- diarrhoea or increased faecal volume, polyuria and polydipsia, anxiety, restlessness and excitability poor unkempt hair coat
- ✓ Tachycardia (> 240bpm), systolic murmurs, gallop rhythms and arrhythmias. Secondary hypertrophic cardiomyopathy may lead to congestive heart failure
- ✓ Palpable thyroid mass (goitre) anywhere between larynx and thoracic inlet, more commonly bilateral but can be unilateral

Diagnosis:

- ✓ Biochemistry: Elevated ALP and ALT without primary hepatic disease, occasionally azotaemia, hyperglycaemia
- ✓ Urinalysis: Variable USG and proteinuria
- ✓ Serum total T4:
  - ➢ Elevated levels are diagnostic if >50nmol/L but in young cats >70nmol/L can be normal
  - ➢ If not elevated and clinical signs suggestive of hyperthyroidism repeat test at a later date or do a free T4 if elevated, then diagnostic

Treatment:

- ✓ **Must assess renal function** before and after treatment as the hyperthyroidism may be masking CRF – if CRF is evident prior to treatment, then treatment of the hyperthyroidism may not be warranted
- ✓ Blocking thyroid hormone synthesis:
  - ➢ Oral carbimazole:
    - 5 mg/cat PO BID if <100nmol/L, TID if >100nmol/L
    - Carbimazole is metabolised into methimazole
    - Side effects:
      - Short term: in 20% of patients but only short term, vomiting, anorexia, and lethargy
      - Long term: Skin rashes and pruritus, blood dyscrasias (reduced platelets and granulocytes)
  - ➢ Transdermal methimazole:
    - 0.05-0.1ml on skin BID-SID (5mg methimazole/0.1ml)
    - Applied to the inside of the ear, takes longer to reduce thyroid levels
- ✓ Surgery:
  - ➢ Thyroidectomy
  - ➢ If underlying chronic renal failure is unmasked by a trial on medical therapy this may not be indicated
  - ➢ Potential complication is hypoparathyroidism 2-3 days post-operatively
  - ➢ Treated with IV calcium gluconate then long term with oral calcium and vitamin D

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- ✓ Radioactive iodine therapy
  - ➢ Considered gold standard
  - ➢ Relatively low risk, can provide lifelong euthyroidism in 80-90% of patients
  - ➢ If underlying chronic renal failure is unmasked by a trial on medical therapy this may not be indicated

- **Monitoring:**
  - ✓ Poor owner compliance is the main cause of failure
  - ✓ Ideally repeat serum total T4, haematology and biochemistry every 2 weeks for 12 weeks but otherwise at least every 4 weeks:
    - ➢ Serum total T4:
      - Aim for mid normal serum total T4
      - If massive drop from >100nmol/L to normal, then trial then reduce frequency
      - If no decrease in serum total T4, then increase dose by 5 mg SID
    - ➢ Renal enzymes and urinalysis:
      - If mild azotaemia then can monitor and treat, see **"Renal Disease"**
      - If overt azotaemia, then may need to stop hyperthyroid therapy and monitor
    - ➢ Blood dyscrasias: Reduced platelets and granulocytes
  - ✓ Once stable then repeat every 3-6 months

Hypothyroidism:

- **Signalment:**
  - ✓ Hypothyroidism is rare is cats, but common in dogs

- **Canine hypothyroidism:**
- **Pathophysiology:**
  - ✓ **Congenital:** Congenital hypothyroidism is rare
  - ✓ **Acquired hypothyroidism:** Common and usually in dogs between 4-10 years old (giant breeds can be younger):
    - ➢ Primary acquired hypothyroidism:
      - Most common acquired form, 95% of cases
      - Autoimmune destruction of thyroid gland, leading to lymphocytic thyroiditis resulting in impaired production and secretion of thyroid hormones
    - ➢ Secondary acquired hypothyroidism
      - Rare, only 5% cases
      - Deficiency of thyroid stimulating hormone (TSH) leading to thyroid follicular atrophy
      - Caused by extrathyroid gland illness – neoplasia, systemic illness, drug therapy and "sick euthyroid syndrome"
    - ➢ Tertiary hypothyroidism: Very rare, due to reduced thyrotropin-releasing hormone (TRH)

  - ✓ **Sick euthyroid syndrome:**
    - ➢ Normal thyroid gland, but transient suppression of thyroid gland activity due to systemic illnesses or drug administration (phenobarbitone, NSAIDs, corticosteroids)
    - ➢ Results in low resting serum total T4, free T4 will also reduce if illness is severe
    - ➢ MUST treat the underlying illness then perform screening tests when animal has recovered

- **Clinical signs:**
  - ✓ Hypothyroidism affects many body organs as it is required for normal cellular metabolic functions
  - ✓ Clinical signs are gradual, subtle and vague:
    - ➢ Lethargy
    - ➢ Weakness and muscle wasting, weight gain without an increase in appetite, heat-seeking, corneal lipidosis

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- Dull, brittle hair coat with alopecia that is bilateral symmetrical, over pressure points and tail, hyperpigmentation, myxoedema "tragic" facial expression
- Neurological signs: Weakness, ataxia, vestibular signs and facial paralysis

- **Diagnosis:**
  - ✓ Commonly over-diagnosed, and require multiple tests and may need to delay testing if concurrent illness is present due to "sick euthyroid syndrome"
  - ✓ Treatment trial without diagnostics is NOT appropriate
  - ✓ Drugs can lower T4 levels: Corticosteroids, phenobarbitone, trimethoprim sulfa
  - ✓ Haematology and biochemistry:
    - Non-specific changes, non-regenerative anaemia (normocytic and normochromic), hypercholesterolaemia
  - ✓ **Endocrine testing:**
    - Overall low total T4, a low free T4 by equilibrium dialysis with high TSH = 98% specific for hypothyroidism
    - Low total T4 and low free T4 by equilibrium dialysis with normal TSH can be due to non-thyroidal illness or drugs
  - ✓ **Serum total T4:**
    - Low normal levels are suggestive of hypothyroidism but can be affected by "sick euthyroid syndrome" and drugs
    - Normal levels rule out hypothyroidism in 90% of cases
  - ✓ **Free T4:**
    - More specific than total T4
    - If low <10nmol/L are consistent with hypothyroidism, strong indicator of hypothyroidism but can be reduced with concurrent illness, drugs and hyperadrenocorticism
    - Best if measured by modified equilibrium dialysis (MED)
  - ✓ **TSH (thyroid stimulating hormone):**
    - High TSH confirms hypothyroidism with a low total T4 or free T4 and with concurrent clinical signs
    - Can be normal in 30% of dogs with hypothyroidism

- **Treatment:**
  - ✓ Lifelong therapy with synthetic T4 hormone
  - ✓ Synthetic L-thyroxine:
    - 0.02mg/kg PO BID - generic products are not recommended
    - If concurrent diabetes, hypoadrenocorticism then monitor closely as thyroid supplementation can lead to hyperglycaemia and ketoacidosis, and also hypoadrenal crisis

- **Monitoring:**
  - ✓ Re-evaluate after 6-8 weeks
  - ✓ Response to therapy is the main indicator of treatment success:
    - Should see improvements in clinical signs over six weeks – increased activity, weight loss, hair regrowth may take longer
  - ✓ Assess serum total T4, 6 hours after administration of the hormone, should be within normal range if not increase dose
  - ✓ If there has been no response to therapy with normal post-pill serum total T4 levels after 4-5 months, then re-evaluate diagnosis
- Overdose of thyroid medication:
  - ✓ Develop clinical signs of hyperthyroidism, stop medication, clinical signs abate over a few days, restart supplementation at reduced dose

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Hyperadrenocorticism:

- **Signalment:**
  - ✓ Rare in the cat, common in dogs

- **Canine hyperadrenocorticism:**
- **Pathophysiology:**
  - ✓ Elevated cortisol levels
  - ✓ **Pituitary-dependent hyperadrenocorticism (PDH): 85%**
    - ➢ Excessive production of ACTH from a tumour in the pituitary gland, leads to bilateral adrenal gland cortex hyperplasia
  - ✓ **Functional adrenal tumour or adrenal dependant hyperadrenocorticism (ADH): 15%**
    - ➢ Secretion of excess quantities of cortisol, due to adrenal gland adenomas or adenocarcinomas, results in atrophy of uninvolved gland
    - ➢ Adenomas are benign, adenocarcinomas are malignant (poor prognosis)
  - ✓ **Iatrogenic:**
    - ➢ Long term use or long acting corticosteroids

- **Clinical signs:**
  - ✓ Polyuria, polydipsia, polyphagia, pot-bellied (due to hepatomegaly and muscle wasting)
  - ✓ Panting (muscle wasting), acute dyspnoea and hypoxia caused by a pulmonary thromboembolism
  - ✓ Lethargy and weakness due to muscle wasting, can cause cruciate ligament ruptures
  - ✓ Skin and hair coat changes:
    - ➢ Alopecia bilateral and symmetrical, hyperpigmentation
    - ➢ Thin skin, able to see blood vessels more easy and calcinosis cutis
    - ➢ Predisposes to recurrent and chronic bacterial infections (skin/urinary tract)
  - ✓ Neurological signs from pituitary tumours causing compression or increased intracranial pressure

- **Diagnosis:**
  - ✓ Haematology, biochemistry and urinalysis:
    - ➢ Stress leukogram (neutrophilia, monocytosis, lymphopenia and eosinopenia)
    - ➢ Elevated ALP (can be very high, with no concurrent hyperbilirubinemia) and ALT (usually mild), hypercholesterolaemia, hyperlipidaemia, hyperglycaemia:
      - Steroid isoenzyme can also be increased by diabetes mellitus, hepatic disease, pancreatitis, congestive heart failure and malignancies
    - ➢ Dilute urine, +/- bacteria (culture urine regardless), +/- proteinuria, increased risk of calcium uroliths
  - ✓ Diabetes mellitus and hyperadrenocorticism:
    - ➢ Difficult to diagnose hyperadrenocorticism in a dog with diabetes
    - ➢ Adrenal screening tests are affected by non-adrenal illness, test only after stabilising diabetes

- **Screening tests** (to positively diagnose hyperadrenocorticism):
  - ✓ **Low-Dose Dexamethasone Suppression Test (LDDST):**
    - ➢ Testing for hyperadrenocorticism should be delayed if the dog is ill as LDDST is affected by non-adrenal illness (false positives), or use the ACTH stimulation test
    - ➢ Best screening test, very sensitive (85-95%) but less specific (50-70%)
    - ➢ Can differentiate between PDH and ADH in some cases
    - ➢ Protocol:
      - Collect blood for a resting cortisol level (8 and 10am)
      - Administering dexamethasone 0.01 mg/kg IV
      - Collect 4 and 8 hours post injection
    - ➢ Interpretation:
      - Normally the pituitary-adrenal axis is suppressed by exogenous dexamethasone

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- Cortisol levels >30nmol/L at 8 hours post dexamethasone = hyperadrenocorticism
- Cortisol levels <50% or <40nmol/L at 4 hours post dexamethasone = PDH
  - Seen in 20% of cases, if no suppression at 4 hours then still could be PDH

✓ **ACTH stimulation response test:**

➢ Less sensitive (80%) but highest specificity (80-90%)

➢ USED if concurrent illness (not as affected by non-adrenal illness) and also to monitor the response to treatment

➢ Protocol:

- Synthetic ACTH (tetracosactrin, Synacthen ®)
- Collect blood for a resting cortisol level
- Inject 5ug/kg of ACTH IM or IV, or 1 vial for large dog or ½ a vial for a small dog
- Collect a second blood sample 1-hour later

➢ Interpretation:

- Normal dogs: 2-3-fold increase in cortisol compared to resting levels
- 1 hour cortisol level:
  - >550nmol/L = hyperadrenocorticism (ideally >600nmol/L)
  - 400-600 "Grey zone" = false positive results may occur
- Reduced response:
  - Adrenocortical atrophy = hypoadrenocorticism

✓ **Urinary Cortisol: Creatinine Ratio:**

➢ Normal urinary cortisol:creatinine ratio = NOT hyperadrenocorticism

Discrimination tests

✓ To distinguish the various causes of the disease

✓ **Ultrasonography:**

➢ If bilateral enlargement – likely PDH but 20% of PDH can have no enlargement

➢ If unilateral enlargement and atrophy of the other gland – likely ADH

✓ **High-Dose Dexamethasone Suppression Test:**

➢ Same protocol as LDDST except administer 0.1mg/kg:

- Suppressed cortisol concentration at 1 hour = PDH
- Failure to suppress cortisol concentration at 1 hour = ADH

Treatment:

Pituitary dependent hyperadrenocorticism:

✓ **Mitotane:**

➢ Potent adrenocorticolytic action

➢ Beware patients with pre-existing diabetes as may need to reduce insulin dosage

✓ Induction phase:

➢ Mitotane at 50mg/kg daily (into two doses) given with food

➢ Dispense prednisolone if accidently overdose occurs

➢ Start maintenance dose when indicators of "end-point of therapy" therapy have occurred:

- Water consumption <60ml/kg/day OR
- Reduction in appetite occurs OR
- Vomiting, diarrhoea or listlessness

➢ Perform an ACTH stimulation test:

- If both pre and post-ACTH cortisol levels are < 30nmol/l then = "end-point of therapy" → start maintenance phase

✓ Maintenance phase:

➢ <10 days to reach "end-point of therapy" start maintenance dosage of 25 mg/kg weekly

➢ >10 days to reach "end-point of therapy" start maintenance dosage of 50 mg/kg weekly

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- Divide weekly dose into two to four treatments per week
- Clinical signs may take up to 6 months to abate

✓ Relapses during maintenance phase:
- Restart induction phase - mitotane 50mg/kg/day (divided)
- Then increase the weekly maintenance dose by 25-50% to prevent future relapse when "end-points of therapy" have occurred

✓ Hypoadrenocorticism during maintenance phase:
- Clinical signs of overdose/iatrogenic hypoadrenocorticism: Anorexia, vomiting, lethargy, weakness, ataxia
- Perform ACTH stimulation test
- Treat with short-term prednisolone 0.25 - 0.5 mg/kg SID, then reduce the weekly maintenance dose by 25-50%

✓ Monitoring:
- Water intake
- Re-examination and undergo ACTH stimulation tests every 3 months

✓ **Trilostane:**
- Inhibits 3β-hydroxysteroid dehydrogenase activity → reducing adrenal steroid synthesis
- MUST not be handled by pregnant owners
- Not used in dogs with hepatic and renal impairment
- Small risk of adrenal crisis or adrenal lysis syndrome
- Clinical signs may take up to 6 months to abate

✓ Recommended dosage regimes:

Weight:	Dose:
< 5kg	15mg PO BID
5-20kg	30mg PO BID
20-40kg	60mg in AM, 30mg in PM
40kg	60mg PO BID
Larger the dog (>25kg)	Use the least amount to reduce clinical signs

✓ **Monitoring:**
- Water intake
- Repeat ACTH stimulation tests performed 4-6 hours after dosing
- 2 weeks, 1 month, 3 months and then every 3 months after starting trilostane

	Cortisol level:	Degree of control:
Baseline cortisol:	25 – 75nmol/L	Normal baseline
Baseline and 1hr cortisol:	<15nmol/L	Excessive control
1hr cortisol:	25-80nmol/L	Tight control
1hr cortisol:	80-125nmol/L	Acceptable control

- <u>Adrenal dependent hyperadrenocorticism:</u>

✓ Surgery: Unilateral adrenalectomy is the treatment of choice

✓ Medical:
- As above with PDH
- May be more difficult to stabilise

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Hypoadrenocorticism:

- **Signalment:**
  - ✓ Uncommon in the dog and rare in the cat

- **Canine hypoadrenocorticism:**
- **Pathophysiology:**
  - ✓ **Primary hypoadrenocorticism** (Addison's disease):
    - ➢ Destruction of the adrenal cortex most commonly immune mediated
    - ➢ Leads to mineralocorticoid and glucocorticoid deficiencies
  - ✓ **Isolated hypocortisolism** (Atypical Hypoadrenocorticism):
    - ➢ Hypocortisolaemia without mineralocorticoid deficiency:
      - Isolated destruction of the zona fasciculate
      - **Secondary:** Reduced ACTH secretion due to pituitary gland pathology
      - **Iatrogenic:** Post corticosteroid therapy or treatment of hyperadrenocorticism

- **Clinical signs:**
  - ✓ Acute presentation = "Addisonian crisis"
    - ➢ Vomiting and diarrhoea, lethargy and weakness, anorexia and PU/PD
  - ✓ Chronic intermittent gastrointestinal signs

- **Diagnosis:**
  - ✓ Biochemistry, haematology, electrolytes and urinalysis:
    - ➢ No stress leukogram (No eosinopenia, lymphopenia and neutrophilia) and mild anaemia (possibly masked by dehydration)
    - ➢ +/- Azotaemia (pre-renal), hypocholesterolaemia, hypoalbuminaemia
    - ➢ +/- Hypoglycaemia, +/- hypercalcaemia
  - ✓ Electrolyte abnormalities: Due to aldosterone deficiency
    - ➢ Hyponatraemia (Na+ <140mmol/L)
    - ➢ Hyperkalaemia (K+ >5.6mmol/L)
    - ➢ Sodium:potassium ratio (Na+:K+):
      - Ratio less than 25:1 is considered suggestive
      - Ratio less than 15:1 virtually diagnostic
      - Differentials for low Na+:K+ ratio include whipworm, acute kidney injury, effusions, gastrointestinal inflammation
    - ➢ NOTE:
      - Electrolyte abnormalities are due to mineralocorticoid deficiency, in "isolated hypoadrenocorticism" the electrolytes are normal or may get hyponatraemia
      - Dehydration: May mask the hyponatraemia and hypochloraemia
      - Blood results may appear like renal failure
    - ➢ USG: <1.030 due to medullary washout from Na+ loss
  - ✓ ACTH stimulation test:
    - ➢ Definitive diagnostic test
    - ➢ Hypoadrenocorticism = No response in cortisol concentration to stimulation = pre and post-ACTH cortisol measurements < 5nmol/L
    - ➢ Normal cortisol response to ACTH testing = Extremely unlikely to be hypoadrenocorticism
    - ➢ Does not distinguish between primary and secondary

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- **Treatment:**
- **Emergency therapy of acute adrenal crisis:**
  - ✓ Correct perfusion and dehydration deficits
  - ✓ Correct electrolyte and acid-base imbalances
  - ✓ Supply mineralocorticoids and glucocorticoids
- Protocol:
  - ✓ Collect blood for baseline diagnostics
  - ✓ Haematology, biochemistry, electrolytes and resting cortisol levels
  - ✓ IV catheter and select/create IV fluid with Na+ concentration within 10mmol of the patient's Na+
    - ➢ Severe hyponatraemia: See **"Fluid Therapy"**, aim for gradual increase less than 1mmol/hr if less than 130mmol/L
  - ✓ Correct perfusion deficits with a low sodium fluid
  - ✓ Correct dehydration deficits, half of fluid deficits over the following 6 hours, then the rest over next 24 hours
  - ✓ Perform an ACTH stimulation test and avoid glucocorticoid therapy until completion
  - ✓ Steroid supplementation:
    - ➢ Dexamethasone phosphate 0.5mg/kg IV, then reduce to 0.1mg/kg IV BID
    - ➢ Hydrocortisone sodium succinate CRI 0.5mg/kg/hr
    - ➢ Commence oral therapy once eating and no vomiting (see below)
  - ✓ Mineralocorticoid supplement → to correct electrolyte derangement:
    - ➢ Single injection of desoxycortisone pivalate 2.2mg/kg IM/SC q25days OR Fludrocortisone (Florinef ®) 0.01mg/kg/day PO BID
    - ➢ Not required in less common cases of glucocorticoid-dependent (secondary) hypoadrenocorticism
  - ✓ Severe hyperkalaemia: See **"Fluid Therapy"**
  - ✓ Metabolic acidosis: See **"Fluid Therapy"**
  - ✓ Once stabilised and eating, transition to maintenance therapy, **see below**
- **Monitoring:**
  - ✓ Reassessment of vitals, CRT and pulse pressure and hydration status every 4 hours
  - ✓ ECG: Monitored every two hours until hyperkalaemia changes subside
  - ✓ Electrolytes: Every four to eight hours
  - ✓ Others: PCV/TP, CVP, urine output, and renal function, blood gases

- **Maintenance therapy:**
- Mineralocorticoids: Lifelong therapy may be required
  - ✓ Fludrocortisone [Florinef ®]
    - ➢ Given orally at 0.01mg/kg/day BID
    - ➢ Dosage adjusted by measuring serum electrolytes
    - ➢ Re-evaluated every week until stabilised, then every 3 to 4 months
    - ➢ May need to increase dose over the first 12 to 18 months, until fully stable
  - ✓ Desoxycortisone pivalate (DOCP):
    - ➢ Pure mineralocorticoid, long-acting IM injection every 25 days
    - ➢ Starting dose of 2.2mg/kg every 25 days
    - ➢ Electrolytes are re-evaluated every week until stabilised, then every 3 to 4 months
    - ➢ Can try to extend dosing interval with DOCP:
      - If normal electrolytes increase dose interval by 1 to 2 days each time up to 30 day dosing if stable
      - If levels are abnormal, reduce the interval
- Glucocorticoids:
  - ✓ May need in conjunction with mineralocorticoid supplementation
  - ✓ Prednisolone: Prednisolone 0.1-0.3mg/kg PO BID, in times of stress increase dose to 1-2mg/kg PO
  - ✓ Cortisone acetate: 0.5mg/kg PO SID to BID

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Fluid Therapy

- **This chapter covers:**
  - ✓ Assessment of hydration
  - ✓ Basic principles of fluid therapy – types of fluids, indications and rates of administration
  - ✓ How to correct electrolyte imbalances

- **Assessment of hydration status:**
  - ✓ Assessment is based primarily on clinical examination findings, can be supported with laboratory findings

Status:	Clinical signs:
Mild (5%):	▪ Skin and mucous membranes are tacky
Moderate (8-10%):	▪ Capillary refill time >2 seconds ▪ Skin lost elasticity and retains an abnormal position (tenting) ▪ Oral mucous membranes are dry ▪ Eyes sunken into sockets - dehydration of peri-orbital tissue
Severe (12-15%): Concurrent perfusion deficits with this degree of dehydration	▪ Capillary refill >5 seconds ▪ Markedly shrunken eyeballs ▪ Involuntary muscle twitching ▪ Cold extremities and hypothermia ▪ Circulatory failure and death occur

- **Typical electrolyte abnormalities:**

Disease process:	Electrolyte abnormalities:
Dehydration	↑ Na+, Cl-
Diabetes ketoacidosis	↓ K+, Na+ and HCO_3-
Diarrhoea	↓ K+, Na+, Cl- and HCO_3-
Hypoadrenocorticism	↓ Na+, Cl- and ↑ K+
Kidney failure (acute)	↑ K+, Na+, Cl-
Urethral obstruction	↑ K+
Vomiting	↓ K+, Na+, Cl-

- **Types of fluid:**
  - ✓ **Tonicity:**
    - ➢ Isotonic: Solute levels similar to plasma
      - E.g. NaCl 0.9%, Hartmanns, PlasmaLyte 148
    - ➢ Hypotonic: Solute levels lower than plasma
      - E.g. Glucose 5% (isotonic in the bottle) and 2.5%, NaCl 0.45%
    - ➢ Hypertonic: Solute levels greater than plasma
      - E.g. NaCl 7.5%, 10% glucose, Mannitol
  - ✓ **Types:**
    - ➢ **Crystalloids:**
      - Isotonic crystalloids: E.g. NaCl 0.9%, Hartmanns, Plasmalyte 148
        - o Solutions containing mineral salts and water soluble molecules that can pass through into the intracellular space, does not increase oncotic pressure
      - Hypertonic crystalloids: E.g. 7.2 – 23% saline
        - o Used to increase intravascular volume rapidly via water shift into the intravascular space from the extravascular space down an osmotic gradient produced by increasing intravascular sodium concentration

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- Short acting intravascular volume expansion, via water shift into the intravascular space down an osmotic gradient
- 5ml/kg of 7% hypertonic saline produces a similar haemodynamic effect similar to 60-90ml/kg of replacement crystalloid, but lasts only 30 minutes
- Resulting hypernatraemia limits the amount that can be safely administered
- Contraindications of hypertonic saline: Dehydrated patients, hyperosmolar patients
- Potential adverse effects: Rapid respiratory rate, hypotension (vagally or osmolality mediated), bradycardia, hypernatraemia

➢ **Colloids:**

- E.g. dextran, starches, gelatins
- Fluids containing large molecules that remain in the intravascular space and helps retain water in the intravascular space
- Smaller volumes are required to correct perfusion deficits and restore normovolemia
- Large molecules persist for longer; small molecules have stronger effect initially
- Crystalloids are still required to correct dehydration deficits but when correcting perfusion deficits reduce crystalloids volumes by 40%
- Use for the maintenance of colloid osmotic pressure and the prevention and management of odema formation in hypoproteinemic patients has recently come under question
- Possible complications: Anaphylactic type reactions, prolonged coagulation times and increased risk of bleeding, initiation or exacerbation of acute kidney injury

✓ **Final pH in the body:**

➢ Alkalinising fluids: For acidotic processes

- Fluids that contain acetate, gluconate, lactate which is transformed into bicarbonate
- E.g. Hartmanns, PlasmaLyte 148

➢ Acidifying fluids: For alkalotic processes

- Fluids that do not contain an alkalising agent and that has high chloride, it also has an acidifying effect by dilution
- E.g. NaCl 0.9%

Type:	Osm:	pH:	Na+:	Cl-:	K+:	Ca+:	Mg+	HCO_3^-
Plasma:	**300**	**7.35**	**147**	**115**	**4**	**2**	**-**	**21**
Hartmanns / Lactated Ringers:	272	6.5	131	110	5	2	-	Lactate 28
NaCl 0.9%:	310	5.5	154	154	-	-	-	-
PlasmaLyte 148:	295	6.3	140	96	5	-	1.5	Gluconate 23 Acetate 27
Glucose 5%:	280 (in bottle)	4.5	-	-	-	-	-	-

▪ **Aims of therapy:** See below for rates

✓ **Perfusion:**

➢ Replace fluid deficits in the intravascular compartment. Fluid choice can include colloids as they remain in the intravascular space longer and also hypertonic saline but must also include crystalloids

✓ **Rehydration:**

➢ Replace fluid deficit in the interstitial compartment.

➢ Composition of the fluid should resemble that of the extracellar fluid space e.g. replacement fluids such as PlasmaLyte 148 or Hartmanns/Lactated Ringers

✓ **Maintenance:**

➢ Used to replace ongoing fluid lost from normal daily losses

➢ The solution should be lower in Na+ and higher in K+ (add 10-20mmol/L of K+)

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- Anorexic puppy or kitten then also add 2.5% dextrose (50ml/L of 50% dextrose)

1). Correction of perfusion:

- ✓ Perfusion:
  - Fluid deficit in the intravascular space, aim to replace deficits rapidly while reassessing for **"end-point resuscitation variables"**
- ✓ Options:
  - Crystalloid fluid resuscitation:
    - Give 10ml/kg crystalloid boluses IV, repeated until reach **"end-point resuscitation variables"**
  - Combination fluid resuscitation:
    - Give 5ml/kg boluses of a colloid IV with 5-10ml/kg crystalloid boluses
    - Can give hypertonic saline maximum 3-5ml/kg IV but not in hyponatraemic or dehydrated patients, in combination with 5-10ml/kg crystalloid boluses
    - Colloids and hypertonic saline reduce the volume required to correct hypovolaemia
    - Repeat boluses while reassessing **"end-point resuscitation variable"**
- ✓ **IF** administered more than a half a blood volume (45ml/kg for dogs and 30ml/kg for cats) and not responding:
  - Assess for cardiogenic shock: Assess for jugular pulses and cardiac disease
  - Assess for obstructive shock: GDV, pericardial effusion, tension pneumothorax
  - Assess for distributive shock: Assess central venous pressure or trial vasopressor therapy
  - Assess for ongoing losses: External and internal haemorrhage (consider abdominal counter pressure)
  - **If still in shock and needs more fluids, consider:**
    - TP if <45g/L or low oncotic pressure, consider colloids or whole blood
    - PCV if <30% or anaemic/haemorrhagic patients, consider pRBC or whole blood and monitor PCV and lactic acid levels
    - If clinically dehydrated, elevated PCV then continue with crystalloids

- **End-point resuscitation variables:**
  - ✓ Resuscitation end-points are physical and laboratory parameters that are used as an indication of adequate blood flow to vital organs
  - ✓ Indicates when to stop resuscitation fluid therapy and continue with rehydration and maintenance therapy
  - ✓ Downstream parameters:
    - Physical examination parameters are not as sensitive for ongoing perfusion deficits as downstream parameters
    - Downstream parameters do not contribute to perfusion but rather depend on having adequate perfusion
    - Downstream parameters can provide early recognition of occult shock and be a source of feedback for resuscitation efforts and thus provide a guide for continued therapy

Variables:	Value:
Mentation:	Alert
Heart rate (beats per minute):	Dog: 90–140 Cat: 180
Mucous membrane colour:	Pink
Capillary refill time (seconds):	1–2
Rectal temperature:	>37.5°C
Mean arterial Pressure (mm Hg):	70–80
Systolic blood pressure (mm Hg):	100

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Urine output (mL/kg/hr): **(not to be used in isolation as a marker of hypovolaemia)**	>1 >2 (if on fluids)
SpO2 (%):	>95
***SvO2 Mixed venous O_2 saturation (%):** ***PcvO2 (mmHg):**	>70 >35mmHg
***Lactate (mmol/L):**	<2
***Base deficit (mEq/L):**	> -4

*** = downstream parameters**

Amount = HYDRATION DEFICIT + MAINTENANCE REQUIREMENTS + ONGOING LOSSES

2). **Correction of dehydration:**

- ✓ **Rehydration:** Correction of fluid deficit in the interstitial space
- ✓ Dehydration deficits is based primarily on physical examination parameters, see **"Assessment of hydration status"**
- ✓ Use replacement crystalloid:
  - ➢ Correct 25 to 50% of the fluid deficit over 4 – 6 hours, then the remainder over the next 18-24 hours

HYDRATION DEFICIT (L) = % dehydration X BWt (kg) / 100

3). **Providing for maintenance needs:**

- ✓ **Maintenance:**
  - ➢ ml/kg/day = 80 x body weight $^{0.75}$ (cats) or 132 x body weight $^{0.75}$ (dogs)
  - ➢ **OR** 30 x body weight (kg) + 70
  - ➢ Add on top of the rehydration rates

4). **Covering for ongoing losses:**

- ✓ **Ongoing losses:**
  - ➢ Losses from vomiting and diarrhoea
  - ➢ Can estimate or weigh (1gm of liquid = 1ml of fluid)
  - ➢ Add on top of the rehydration and maintenance rates

- **Complications of fluid therapy:**
  - ✓ Tonicity:
    - ➢ Rapid administration of hypotonic fluids can cause haemolysis
    - ➢ Rapid administration of hypertonic fluids can cause crenation
  - ✓ Electrolyte imbalances:
    - ➢ Commonly hypokalaemia or hypernatraemia with use of replacement fluids for maintenance
    - ➢ Potential for exacerbation of acute kidney injury with saline 0.9% due to excess chloride
  - ✓ Acid-base derangements:
    - ➢ Acidification (e.g. hyperchloraemic metabolic acidosis) or alkalinisation
  - ✓ Volume overload:
    - ➢ Excessive fluid administration that exceeds ability to remove it
    - ➢ Tissue oedema and delayed wound healing e.g. intestinal anastomosis sites and prolong recovery times
    - ➢ Clinical signs:
      - Pulmonary oedema: Seen first especially in small dogs and cats

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- Increased respiratory rate, crackles, soft and moist cough, serous nasal discharge
- Subcutaneous oedema: Seen around paws and ventral areas

✓ Haemodilution:
- ↓ **Oncotic pressure:** Consider use of colloids if TP <45g/L
- ↓ **PCV:** Consider administering whole blood or pRBC and monitor PCV
- Dilution of coagulation factors

Treatment:

- **Hypoglycaemia:**
  - ✓ Give dextrose 0.5–1ml/kg of 50% IV diluted with saline 1:3 (to reduce vasculitis) given over 5 minutes
  - ✓ Offer food if able to i.e. Patient is bright and there are no contraindications
  - ✓ Dextrose CRI at 2.5 to 5% if require longer term glucose supplementation
    - 2.5% = 50ml of 50% dextrose in 950ml
    - 5% = 100ml of 50% dextrose in 900ml
  - ✓ Insulin overdose:
    - If due to insulin overdose must continue glucose supplementation with 2.5% glucose solution (or higher), until insulin wears off (i.e. blood glucose starts to increase) then start insulin again at 50-75% of dose, **see Diabetes Mellitus in "Endocrine Disease"**
    - Severe insulin overdose requires higher rates of glucose supplementation and occasional boluses of dextrose
    - Must monitor electrolytes especially potassium as this will shift intracellularly resulting in hypokalaemia, this can occasionally require aggressive potassium supplementation greater than the recommended 0.5mEq/kg/hr
    - Hypophosphatemia can occur as it is shifted intracellularly by insulin
    - Glucagon CRI: Persistent and severe hypoglycaemia e.g. Severe insulin overdose or insulinoma, consider starting glucagon CRI – administer a 50ng/kg bolus followed by a CRI of 5-40ng/kg/min

- **Hyperglycaemia:**
  - ✓ Stress hyperglycaemia:
    - Cats but also dogs can have significant hyperglycaemia due to stress
    - It can exceed the proximal tubule reabsorption leading to glucosuria
  - ✓ Differentiating between stress and diabetes:
    - Assessment of the patient and history: Is there a history of PU/PD, weight loss, excessive appetite
    - Glucose curve: The glucose will drop as the patient's stress resolves
    - Fructosamine: Can give an indication of the patient's prior glycaemic status
  - ✓ Glucose free fluids
  - ✓ Monitor blood glucose every 2-4 hours

- **Hypernatraemia:**
  - ✓ **In moderate to severe hypernatraemia aim to REDUCE sodium by less than 0.5mEq/L per hour** – otherwise can cause cerebral oedema:
    - If neurological signs occur treat with mannitol 0.5-1g/kg IV over 20 minutes OR 7% saline at 3-5ml/kg over 20 minutes
  - ✓ Access to water should not be permitted if severe

  - ✓ **Types of hypernatraemia:**
    - Hypervolaemic hypernatraemia:
      - Hypernatremia results from increased sodium intake or reabsorption
      - Most likely sodium gain from IV fluids or food, also from acute kidney injury, hyperaldosteronism (Conn's syndrome)
      - Distended jugular veins, high CVP, pulmonary oedema
    - Normovolaemic hypernatraemia:

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- Hypernatremia with euvolemia is a decrease in total body water with near-normal total body Na (pure water deficit)
- Free water loss with no signs of dehydration, usually results in very high sodium
- Diabetes insipidus, hypodipsia

➢ Hypovolaemic hypernatraemia:

- Hypernatremia associated with hypovolaemia occurs with Na+ loss accompanied by a relatively greater loss of water from the body
- Hypotonic or free water renal or gastrointestinal loss
- Signs of dehydration and hypovolaemia i.e. Dry and or pale mucus membranes, prolonged capillary time, skin tenting, weak pulse pressure and low blood pressure

✓ **Hypovolaemic hypernatraemia** = free water loss, dehydration:

➢ Correct perfusion deficits with crystalloid solution supplemented with sodium to within 5 - 10mEq/L of the serum

➢ See formula below to work out how much hypertonic saline to add

➢ If the hypernatraemia has developed over a 24 hour period, replace free water deficits slowly over a 2 – 3 day period, correct slower than 0.5mEq/L per hour

➢ Subsequent improvement in renal blood flow and function should correct the hypernatraemia by increased renal excretion

➢ Monitor electrolytes every 4 hours

➢ **Supplementing IV fluid with sodium:**

- Add additional NaCl from hypertonic saline (e.g. NaCl 7%) to increase Na+ content of the IV fluid
- Use the formula below, to find out how much hypertonic saline is required:

$$\text{Volume (mls) to be added} = \frac{([\text{Current IV fluid Na+}] - [\text{Desired IV fluid Na+}]) \times 1000}{([\text{Desired IV fluid Na+}] - [\text{Hypertonic saline Na+}])}$$

NOTE: This is the volume that needs to be added to the IV fluid bag

Example: Patient has a Na+ concentration of 180mEq/L and you want to increase the Na+ in a bag to 175mEq/L using 7% hypertonic saline as your Na+ source

Desired IV fluid sodium concentration is 175 mEq/L

Current IV fluid Na+ concentration is 150mEq/L (0.9% NaCl)

Supplemental IV fluid Na+ concentration 1155mEq/L (7% saline)

$$\text{Volume (mls) to be added} = \frac{(150 - 175) \times 1000}{(175 - 1155)} = 25.5\text{ml of 7\% saline}$$

- If no improvement in hypernatraemia after trying to correct hypovolaemia, then consider free water supplementation with 5% dextrose as per "Normovolaemic hypernatraemia"

✓ **Normovolaemic hypernatraemia** due to free water deficit:

➢ Oral fluid replacement is also ideal but some of these animals will be unable to drink

➢ Calculate free water deficit and replace slowly over the number of hours it takes to decrease Na+ by 0.5mEq/L/hr:

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Free water deficit = (([Current Na+] / [Desired Na+]) – 1) x (0.6 x body weight)

Number of hours to decrease Na+ (by 0.5mEq/L/hr) = ([Current Na+] – [Desired Na+]) / 0.5

How many ml/hr of 5% dextrose = (ml of free water required / number of hours)

OR

3.7ml/kg/hr of 5% dextrose to decreases sodium by 0.5mmol/hr (*Haskins*)

- Cover maintenance and any ongoing losses (e.g. Diarrhoea or vomiting) with another isotonic crystalloid
- Monitor electrolytes every 4 hours
- If neurological signs occur treat with mannitol 0.5-1g/kg IV over 20 minutes OR hypertonic 7% saline at 3-5ml/kg over 20 minutes
- Avoid and monitor for over hydration – can measure central venous pressure as a guide

✓ **Hypervolaemic hypernatraemia** due to excess Na+:

- Rate of correction depends of rate of increase. If it was a rapid increase due to salt ingestion or hypertonic saline administration, can correct rapidly. If chronic then correct slowly by 0.5mEq/L/hr
- Correct free water deficit with 5% dextrose as per normovolaemic hypernatraemia
- A loop diuretic such as furosemide can be trialled 2mg/kg IV to reduce Na+ reabsorption
- If neurological signs occur treat with mannitol 0.5-1g/kg IV over 20 minutes OR hypertonic 7% saline at 3-5ml/kg over 20 minutes

- **Hyponatraemia:**

✓ **Rate of INCREASE should not exceed 0.5mEq/L per hour** – otherwise can cause cerebral dehydration as the plasma is hypertonic to the brain and cause central pontine myelinolysis:

- **Especially important until reach >130mEq/L**
- Monitoring electrolytes every 4 hours

✓ **Hypovolaemic hyponatraemia** due to hypotonic losses or hypoadrenocorticism:

✓ Reduced circulating volume from fluid losses results in release of ADH, this results in increased free water reabsorption and also increased thirst, therefore reducing plasma sodium

✓ Hyponatraemia in hypoadrenocorticism is due to reduced aldosterone section, this results in reduced sodium reabsorption

✓ Clinical signs a consistent with dehydration and hypovolaemia - weakness, rapid and weak pulses, prolonged capillary refill times, cold extremities and hypotension

✓ Volume resuscitation and rehydration with a fluid where the sodium concentration is within 5mEq/L of the patient's plasma sodium level.

- Can use a mix of 5% dextrose in water with Hartmanns to get the required sodium concentration in the IV fluid, use the formula below

✓ Maintenance requirements can be administered with an IV fluid of a high sodium such as 0.9% saline if sodium concentration does not increase

$$\textbf{Volume (mls) to be added} = \frac{([\text{Current IV fluid Na+}] - [\text{Desired IV fluid Na+}]) \times 1000}{([\text{Desired IV fluid Na+}] - [\text{Supplemental IV fluid Na+}])}$$

Example: Patient has a Na+ concentration of 110mEq/L and you want to decrease the Na+ in a bag to 110mEq/L using 5% dextrose and water as your free water source

Desired IV fluid sodium concentration is 110mEq/L

Current IV fluid Na+ concentration is 135mEq/L (Hartmanns)

Supplemental IV fluid Na+ concentration 0mEq/L (5% dextrose and water)

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$$\text{Volume (mls) to be added} = \frac{(135 - 110) \times 1000}{(110 - 0)} = 227\text{ml of D5W}$$

- ✓ **Hypervolaemic hyponatraemia** due to retention of free water
- ✓ Clinical signs reflect underlying disease: Oedema, ascites, pleural effusion
- ✓ Water restriction only for inappropriate ADH secretion or primary polydipsia (both rare) – consult medical text
- ✓ **Congestive heart failure** – normal body sodium levels but excess free water
  - ➢ Reduced cardiac output is interpreted as reduced circulating volume. Renin-angiotensin activation leads to release of ADH and aldosterone, resulting in sodium and free water reabsorption and increased thirst. Ultimately leading to an excess of free water.
- ✓ **Advanced liver (cirrhosis) or renal failure (nephrotic syndrome)**
  - ➢ Both can result in hypoalbuminaemia leading to shift of fluid in the interstitial space and third spacing, the resultant reduce effective circulating volume leads to activation of ADH leading to free water accumulation in an attempt to restore circulating volume.

- ✓ **Treatment of hypervolaemic hyponatraemia:**
  - ➢ Loop diuretics inhibit Na+ and Cl- reabsorption at the thick ascending loop of Henle
    - Give frusemide with the saline to promote free water excretion and prevent ECF volume expansion
    - The rate of change of serum sodium concentration must be monitored every 2 to 3 hours and the infusion adjusted as needed

- **Hyperkalaemia:**
  - ✓ Ensure patency of the urinary outflow tract
  - ✓ Choice of fluid
    - ➢ Traditionally potassium free fluid but any balanced isotonic fluid can be used
    - ➢ Increasing intravascular volume will dilute serum potassium, alkalinisation will result in an exchange for K+ going into the cell with H+ coming out of the cell also increase renal excretion
  - ✓ Mild to moderate elevations: 5.5 – 6.5
    - ➢ IV crystalloids: Correct volume and dehydration deficits
  - ✓ Moderate to severe elevations: >6.5 OR when cardiotoxic effects, metabolic acidosis
    - ➢ IV crystalloids: Correct volume deficits
    - ➢ Calcium gluconate 10% 0.5-1.5 mL/kg slow IV over 10 minutes:
      - Acts within minute to protects against cardiotoxic effects by altering the threshold potential. This enables the previously hypo-polarized cells to depolarize again.
      - Must instigate other treatments as only protects for 20 minutes and does not alter serum K+
    - ➢ Insulin and dextrose OR dextrose alone:
      - Insulin (regular short acting) 0.25-0.5U/kg IV **ALWAYS** with .....
      - 2g of dextrose per unit of insulin = 4ml of 50% dextrose OR 40ml of 5% dextrose for each unit of insulin **followed** with CRI of 2.5% glucose at maintenance rates until insulin wears off, 6-8 hours
      - Insulin drives K+ back into cells but must be followed with dextrose CRI to prevent hypoglycaemia from the insulin therapy
      - Dextrose by itself will stimulate endogenous insulin production
      - Monitor blood glucose and potassium levels every 2 hours
    - ➢ Bicarbonate therapy:
      - For severe hyperkalaemia or metabolic acidosis (HCO_3<10mmol/L)
      - 1mmol/kg or 1ml/kg of 8.4% solution over 30 minutes is generally safe. Adverse effects of bicarbonate therapy are related to speed of administration so be cautious.
      - Acts within 15mins by inducing a metabolic alkalosis
      - Metabolic alkalosis results in H+ ions being drawn out of the cell in exchange for potassium

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- Bicarbonate may negate the cardioprotective effects of intravenous calcium by altering the pH resulting in increased binding of calcium to albumin

- **Hypokalaemia:**
  - **Potassium chloride:** 1mmol = 1mEq of K+ (and 1mEq of Cl-) = 75mg
  - Run the fluids at a desired rate based on hydration etc. BUT adjust K+ so not to administer above the max infusion rate
  - Administer slowly, aim for < 0.5mEq/kg/hr IV
  - Beware of high fluid rates if required to correct perfusion, as it can exacerbate hypokalaemia, ensure to adjust K+ supplementation based on frequent electrolyte reassessment
  - Should add 10-20mmol of K+ per litre for maintenance fluid therapy

Serum K^+ (mEq/L)	Add K (mmol/L in 1L fluid bag)	Total mEq K+ in 1L fluid after addition of KCl-	**Maximum infusion rate of IV fluids (0.5mEq/kg/hr)**
<2.0	80	80	6 mL/kg/hr
2.1-2.5	60	60	8 mL/kg/hr
2.6-3.0	40	40	12 mL/kg/hr
3.1-3.5	30	30	18 mL/kg/hr
3.6-5.0	20	20	25 mL/kg/hr

✓ **Mild:** 3 – 3.5
- Best by restoration of normal feeding
- Oral supplementation at 1-3mEq/kg/day PO divided BID-TID:
  - Potassium gluconate (best) 1gm = 4.3mEq of K+
  - Potassium chloride 1gm = 13.4mEq of K+
- Once levels have normalised then maintain on 1mEq/kg/day divided BID OR adjust dose as necessary based on electrolyte levels

✓ **Moderate:** 2.5 - 3
- Oral supplementation combined with IV therapy

✓ **Severe:** < 2.5
- IV therapy

Hypocalcaemia:

✓ Acute therapy:
- If concurrent seizures/tetany: Use IV calcium therapy in conjunction with diazepam IV
- Calcium gluconate 10% 0.5-1ml/kg given slow IV over 15 minutes, slowly to effect monitoring heart rate and ECG
- Can continue as a CRI e.g. 0.25ml/kg of Calcium gluconate 10% maintenance infusion

✓ Long term therapy (i.e. treatment of primary hypoparathyroidism):
- Vitamin D: Increases intestinal calcium absorption
- Calcitriol (1,25 Dihydrocholecalciferol = active form of vitamin D) at 0.005 – 0.015µg/kg/day with a calcium supplement (e.g. Calcium carbonate)
- Monitor calcium concentrations monthly until stabilized – avoid hypo/hypercalcaemia
- Also monitor phosphate and albumin concentrations

Hypercalcaemia:

✓ Treatment of underlying disease

✓ Acute therapy:
- Fluid therapy (0.9% saline), correction of volume and dehydration deficits to enhance urinary excretion

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- +/- Diuretics: E.g. Furosemide 1-2mg/kg IV, SC, PO BID
  - Must ensure fully hydrated before commencing
  - Match urine output with fluid input and if possible and can tolerate it have water available
- +/- Corticosteroids:
  - Only if a definitive diagnosis has been made and they are indicated
- If life threatening:
  - Chelating agents e.g. Sodium or potassium phosphate (0.02mmol) at 0.25-0.5mmol/kg IV over 4 hours
  - Sodium bicarbonate 1mEq/kg or 1ml/kg of 8.4% solution slowly over 20 minutes:
    - Decreases both ionized and total calcium
    - Must monitor acid-base balance
    - Best if concurrent metabolic acidosis

- **Hyperphosphataemia:**
  - ✓ Treatment of underlying disease
  - ✓ Acute therapy:
    - Fluid therapy 0.9% saline
    - Can give insulin to promote cellular uptake same protocol as for hyperkalaemia
    - Correction of hypocalcaemia (if present)

- **Hypophosphataemia:**
  - ✓ Treatment should be considered if <0.65nmol/L, initiated if <0.30nmol/L
  - ✓ Sodium or potassium phosphate (0.02mmol) at 0.01-0.05mmol/kg/hr until phosphate levels normalise
  - ✓ Do not supplement if there is a concurrent hypercalcaemia or if anuric or oliguria
  - ✓ Amount:
    - Phosphate supplementation = 0.01-0.05 mmol/kg/hr
    - Add into IV fluids
    - Alternatively, determine the amount of potassium supplementation needed, give half the amount as KCl and the other half as KPO4 (potassium phosphate)
  - ✓ Treatment:
    - Administer in Ca+ free fluids and avoid if patient is hypercalcaemic
    - Discontinue when serum phosphorus = 0.9 mmol/L
    - Monitor phosphate levels every 6 hours

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Haematology

- **This chapter covers:**
  - ✓ Basic changes seen on a haematology panel and changes seen on blood smear
  - ✓ Interpretation of changes in PCV/TP and in the serum colour

White blood cells:

Stress Leukogram: ↑ Neutrophils (no left-shift) and macrophages, ↓ lymphocytes and eosinophils
Pancytopenia: ↓ WBC, ↓ RBC and ↓ platelets

Cell Type:	Increased:	Decreased:
Neutrophils:	• **Neutrophilia:** **With right shift:** ➢ Fear, excitement or exercise ➢ Corticosteroids (stress leukogram) **With left shift:** ➢ Infection ➢ Immune mediated disease ➢ Neoplasia (esp. tumour necrosis) ➢ Inflammation ➢ Bone marrow neoplasia - leukaemia	• **Neutropenia:** ➢ Decreased production – disease affecting precursors in bone marrow e.g. myeloproliferative disease, parvovirus, FIV, FeLV, drugs, toxicities ➢ Pancytopenia **With degenerative left shift:** ➢ Overwhelming demand or acute inflammation/infection
Lymphocytes:	• **Lymphocytosis:** ➢ Physiologic: e.g. young animals, excitement ➢ Prolonged immune stimulation ➢ Protozoal e.g. *Ehrlichiosis* ➢ Addison's disease ➢ Leukaemia	• **Lymphopenia:** ➢ Stress response ➢ Corticosteroids ➢ Immunosuppressive drugs ➢ Viruses ➢ Endotoxemia and severe bacterial infection ➢ Loss of lymph ➢ Pancytopenia
Eosinophils:	• **Eosinophilia:** ➢ Parasites ➢ Allergies and allergic dermatitis ➢ Asthma ➢ Neoplasia ➢ Fungi	• **Eosinopenia:** ➢ Corticosteroids (stress response) ➢ Catecholamine's ➢ Acute infection
Monocytes	• **Monocytosis:** ➢ Stress leukogram ➢ Chronic inflammation – infection, malignancy, necrosis, pyogranulomatous inflammation ➢ Intracellular bacteria e.g. *Mycobacteria, Brucella* ➢ Foreign body reactions ➢ Immune-mediated disease ➢ During recovery from neutropenia	

- **Neutrophils:**
  - ✓ **Neutrophilia:**
    - ➢ Inflammation uncommon to go above 40 x 10^9/L
    - ➢ Neoplasia/paraneoplastic: Consider if counts >70 x10^9/L for dogs & >50 x10^9/L for cats
  - ✓ **Right shift:**
    - ➢ Increased numbers of mature "segmented" neutrophil. Usually due to increased corticosteroids (stress, hyperadrenocorticism, drugs)
  - ✓ **Left shift:**
    - ➢ Increased numbers of immature neutrophils "bands" the peripheral blood. Occurs in inflammatory conditions
    - ➢ Band neutrophils: 'U' shaped nucleus with no segments
      - **Regenerative left shift:**
        - o Neutrophilia due to increased immature neutrophils but segmented "mature" neutrophils are still predominant
      - **Degenerative left shift:**
        - o More immature neutrophils than segmented "mature" ones
        - o Can have normal numbers or neutropenia
        - o Unable to meet demand, due to overwhelming infection/inflammation
  - ✓ **Toxic change:**
    - ➢ Number of morphological changes in neutrophils indicating shortened production times with incomplete maturation in the bone marrow due to intense stimulation
    - ➢ 3 common changes seen on blood smear:
      - Reduced granulation resulting in increased cytoplasmic basophilia
      - Cytoplasmic vacuolation
      - Dohle bodies: Light blue-grey, oval inclusions in the periphery of the cytoplasm
  - ✓ **Hypersegmentation:**
    - ➢ 6 or more nuclear lobes
    - ➢ Caused by vitamin B12 and folate deficiencies, prolonged corticosteroids administration

Red blood cells:

- **Terminology:**
  - ✓ **Anisocytosis:**
    - ➢ Variation in cells size, seen with increased reticulocytes and spherocytes
  - ✓ **Poikilocytes:**
    - ➢ Abnormally shaped red blood cells
  - ✓ **Polychromasia:**
    - ➢ Increased variation in red blood cell colour, seen with increased reticulocytes
  - ✓ **Hypochromasia:**
    - ➢ Decreased red blood cell colour. Usually seen as increased central pallor with reduced haemoglobin concentration
  - ✓ **Howell-Jolly body:**
    - ➢ Nuclear remnant that has remained within the red blood cell, usually basophilic. Increased frequency with regenerative anaemias due to increased red blood cell release
  - ✓ **Heinz bodies:**
    - ➢ Clumps of haemoglobin due to oxidative damage, seen as pale blue structures protruding from the red blood cell. Cats can have upto 10% normally.
  - ✓ **Eccentrocytes:**
    - ➢ Haemoglobin has moved to one side, leaving a hollow area on the other side. Due to oxidative injury or following Heinz body
  - ✓ **Nucleated:**
    - ➢ Commonly seen in young kittens <12 weeks of age, conditions of increased RBC production (eg. IMHA), myeloproliferative conditions involving splenic metastases or splenic disease

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- ✓ **Spherocytes:**
  - ➢ Small dark red blood cells that lack central pallor, have normal MCV but reduced surface area
  - ➢ Most frequently seen in immune-mediated haemolytic anaemia, but also post-transfusion and Heinz body anaemias
- ✓ **Schistocytes:**
  - ➢ Irregular RBC fragments due to damage/shearing from intravascular fibrin strands or turbulent flow through microvasculature
  - ➢ Most frequently seen in DIC, hemanglosarcoma, glomerulonephritis, CHF etc.

Appearance:

- ✓ **Dog:** Bi-concave, uniform in size

Parameter:	Increased:	Decreased:
MCV (mean corpuscular volume): Mean size of RBC	**Macrocytosis:** • Regenerative response • FeLV • Myeloproliferative disease • Old samples >24 hours • Autoagglutination	**Microcytosis:** • Iron deficiency • Liver disease and PSS • Anaemia of chronic disease
RDW (red blood cell distribution width): Degree of variation in size of RBC	• ↑ Numbers of reticulocytes • Anaemia with large variation in cells sizes	
MCHC (mean corpuscular Hb concentration): Mean concentration of Hb in RBCs	**Hyperchromasia:** • Intravascular haemolysis • Heinz body formation • Agglutination • Lipaemia (artefact)	**Hypochromasia:** • Large numbers of immature RBC (regenerative anaemia) • Iron deficiency • Old samples >24 hours

- ✓ **Cat:** Mild anisocytosis, little central pallor, Howell-jolly bodies (<1%)

Anaemia:

- ✓ See also **'Anaemia and Pale Mucous Membranes'** for diagnostic pathway and causes of anaemia

Degree of anaemia:

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

Types of anaemias:

- ✓ Pre-regenerative: Bone marrow takes 3-4 days to respond (refer to history)
- ✓ Regenerative anaemia: When increased number of reticulocytes in peripheral circulation
- ✓ Non regenerative: No increase in reticulocytes in peripheral circulation

Regenerative anaemia:

- ✓ When increased number of reticulocytes in peripheral circulation
- ✓ Cats: Two types of reticulocytes:
  - ➢ Aggregate type: Only count this type when assessing response to anaemia
  - ➢ Punctate type: Increased numbers if regenerative response been for up to 3-4 weeks

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- **Reticulocyte:**
  - ✓ 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)

Degree:	Dogs:	Cats:
Mild:	1.5 – 4%	0.5 - 2%
Moderate:	5 – 20%	3 – 4 %
Marked:	>20%	> 4%

Packed cell volume and total solids:

- **Appearance of PCV tube:**
  - ✓ Large buffy coat = ↑ WBC, bone marrow neoplasia
  - ✓ Serum:
    - ➢ Lipaemic (white): Pancreatitis, post-prandial lipaemia, hyperadrenocorticism, diabetes mellitus, hypothyroidism
    - ➢ Haemolysed (red): Poor collection, intravascular haemolysis
    - ➢ Icteric (yellow): Pre-hepatic, hepatic, post-hepatic

- **Alterations in packed cell volume and total solids:**
  - ✓ HCT is equivalent to PCV but expressed as a percentage
  - ✓ Hb is the amount of haemoglobin in the blood (free and inside the cells), in haemolysis the PCV and HCT will be reduced but the haemoglobin will be normal

PCV:	TS:	Interpretation:
↑	**Normal**	Polycythaemia (rare), splenic contraction, dehydration with hypoproteinaemia
↑	↑	Dehydration, fluid shift
↑	↓	Severe dehydration with loss of protein Haemorrhagic gastroenteritis, haemorrhage
Normal	**Normal**	Normal hydration Acute haemorrhage
Normal *or* ↑	↓	Splenic contraction after blood loss Protein loss (GIT, renal) or ↓ protein production (liver)
Normal	↑	Anaemia with dehydration Normal hydration with hyperproteinaemia/hyperglobulinaemia
↓	**Normal**	Chronic RBC destruction or ↓ production, if haemolysed or icteric serum → haemolytic anaemia Anaemia of chronic disease Bone marrow disorders
↓	↓	Aggressive fluid therapy, blood dilution (>3hr post-haemorrhage)
↓	↑	Lymphoproliferative disease, anaemia of chronic disease

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Platelets:

- Usually if platelets are clumped on a blood smear then there are adequate numbers for coagulation
- If platelet numbers are $>30x10^9$/L, then should be enough for coagulation
- Platelet clumping will artificially reduce platelet counts on haematology panel

Increased:	Decreased:
▪ **Thrombocytosis:** ➢ Inflammation ➢ Neoplasia ➢ Acute haemorrhage (rebound) ➢ Chronic bleeding and iron deficiency ➢ Stress ➢ Drug induced	▪ **Thrombocytopenia:** ➢ Immune mediated destruction ➢ Splenomegaly/hypersplenism (sequestration) ➢ Disseminated intravascular coagulation (consumption) ➢ Aplastic anaemia (pancytopenia) ➢ Acute blood loss ➢ Bone marrow disease

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Hepatobiliary Disease

- **This chapter covers:**
- Common diseases affecting the hepatobiliary system:
  - ✓ **Jaundice**
  - ✓ **Acute hepatopathy / hepatitis:**
  - ✓ **Canine chronic hepatitis:**
  - ✓ **Feline hepatic lipidosis:**
  - ✓ **Feline inflammatory liver disease:**
  - ✓ **Hepatic encephalopathy**
- For more information on changes seen in hepatic and biliary parameters in a biochemistry panel, See **"Biochemistry"**

- **<u>Jaundice:</u>**
- **Pathophysiology:**
  - ✓ Divided into three types:
  - ✓ Pre-hepatic:
    - ➢ ↑ Production of haemoglobin due to haemolysis
  - ✓ Hepatic:
    - ➢ ↓ Uptake and conjugation due to hepatic failure
    - ➢ ↓ Hepatic excretion
  - ✓ Post-hepatic:
    - ➢ ↓ Removal due to biliary obstruction
- **Causes:**
  - ✓ Pre-hepatic:
    - ➢ Immune mediated haemolytic anaemia
    - ➢ Toxic: Snake, onions/garlic, paracetamol
    - ➢ Bacterial: *Mycoplasma haemofelis*
    - ➢ See **"Anaemia and Pale mucous membranes"** for differentials for haemolysis
  - ✓ Hepatic:
    - ➢ Hepatitis: Severe acute
      - Toxic: Plants, mycotoxin
      - Inflammatory: Hepatitis, cholanglohepatitis, neoplasia
      - Infectious: Bacterial (leptospirosis), parasitic (migrating larvae, toxoplasma)
  - ✓ Post-hepatic:
    - ➢ Biliary tract obstruction: Biliary stones or mucocele
    - ➢ Duodenal foreign body: Blocking duodenal papillae
    - ➢ Pancreatitis
- **Clinical signs:**
  - ✓ Icteric mucous membranes/sclera
  - ✓ Clinical signs associated with underlying cause
- **Diagnostics:**
  - ✓ Pre-hepatic:
    - ➢ PCV/TP, icteric serum
    - ➢ Spherocytes
    - ➢ Autoagglutination
  - ✓ Hepatic:
    - ➢ ↑ Bilirubin, ↑ ALP, GGT, +/- ↑ ALT, AST
    - ➢ Ultrasound: +/- Liver pathology
  - ✓ Post-hepatic:
    - ➢ ↑ Bilirubin, ↑ ALP, GGT
    - ➢ Ultrasound: Biliary tract congestion, biliary stones, mucocele, pancreatitis

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- **Treatment:**
  - ✓ As per the underlying condition
  - ✓ See below for hepatic disease
    - ➢ Consider antibiotics for post-hepatic obstruction as biliary stasis is a high risk of infection

- **Acute hepatopathy/hepatitis:**
- **Clinical signs:**
  - ✓ Acute onset, anorexia, vomiting, cranial abdominal pain, jaundice, bleeding, hepatic encephalopathy, seizures
- **Diagnostics:**
  - ✓ Biochemistry:
    - ➢ Elevated liver enzymes especially ALT and AST, ALP, bilirubin, bile acids, hypoproteinaemia, hypoglycaemia
  - ✓ Haematology:
    - ➢ Anaemia, thrombocytopenia
  - ✓ Ultrasound: May not see any specific changes
  - ✓ Biopsy: May show non-specific changes
- **Causes:**
  - ✓ Infectious (bacterial, viral, protozoal, parasitic, algae), toxicity (paracetamol, cycad ingestion), neoplastic, pancreatitis, IBD etc.
- **Treatment:**
  - ✓ Supportive therapy:
    - ➢ IV fluids and electrolytes
    - ➢ Antiemetic: Metoclopramide 0.5mg/kg TID, maropitant 1mg/kg SC SID for <5 days
    - ➢ Gastric protectants: Proton pump inhibitors, H_2 antagonist (famotidine), sucralfate
  - ✓ Antibiotics:
    - ➢ Depending on cause
    - ➢ Metronidazole 7.5mg/kg BID and ampicillin 22mg/kg TID
  - ✓ Coagulopathy:
    - ➢ Plasma for coagulopathy, see **"Coagulopathy"**
    - ➢ Vitamin K1 injection, single dose 5mg/kg SC, +/- continue with 2.5mg/kg PO BID
  - ✓ Adjunctive therapy:
    - ➢ Anti-oxidants:
      - Vitamin E 400 IU PO SID and Vitamin C
      - S-Adenosy-L-methionine (SAMe): Potent antioxidant 20mg/kg PO SID
    - ➢ Choleretic:
      - Ursodeoxycholic acid (Actigall ®): 10-15mg/kg PO SID, only if no biliary tract obstruction
    - ➢ Paracetamol toxicity:
      - N-Acetylcysteine: 140mg/kg IV initially (diluted in saline), then 70mg/kg IV or PO QID for 5 doses
  - ✓ Neurological signs:
    - ➢ See **"Seizures Disorders"** - avoid benzodiazepines, use propofol to stop seizures and begin phenobarbitone or levetiracetam
    - ➢ Treatment **"Hepatic encephalopathy"** see following pages

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- **Canine chronic hepatitis:**
- **Pathophysiology:**
  - ✓ Syndrome characterised by hepatic degeneration and necrosis leading to fibrosis, causes can include:
    - ➢ Idiopathic: Most common
    - ➢ Recurrent pancreatitis/IBD, biliary tract disease (obstruction, inflammation)
    - ➢ Toxicity/drug: Chronic exposure
    - ➢ Inflammation (immune-mediated, fungal, bacterial, viral (adenovirus))
    - ➢ Breed specific copper accumulation disorders (Bedlington terriers, West Highland Terriers, Dalmatians, Dobermans)
- **Clinical signs:**
  - ✓ Anorexia, lethargy, vomiting, weight loss, jaundice, PU/PD, ascites (portal hypertension or hypoalbuminaemia)
  - ✓ Behaviour changes: Disorientation, head pressing, ataxia, pacing, seizures, circling
- **Diagnostics:**
  - ✓ Haematology and biochemistry:
    - ➢ Variable changes on haematology
    - ➢ Usually elevated ALT, bilirubin and paired serum bile acids
    - ➢ But signs of liver dysfunction: Low albumin, urea, glucose, coagulopathy
  - ✓ +/- Coagulopathy
  - ✓ Ultrasound
  - ✓ Biopsy: Definitive diagnosis, histopathology and culture and sensitivity
- **Treatment:**
  - ✓ Treatment for specific disease according to diagnosis based on biopsy
  - ✓ Supportive therapy:
    - ➢ IV fluids and electrolytes
    - ➢ Antiemetic: Metoclopramide 0.5mg/kg TID, maropitant 1mg/kg SC SID for <5 days
    - ➢ Gastric protectants: Proton pump inhibitors, H_2 antagonist (famotidine), sucralfate
    - ➢ Coagulopathies: Plasma and Vitamin K, see **"Coagulopathy"**
    - ➢ Neurological signs see **"Hepatic encephalopathy"**
  - ✓ Adjunctive: Based on definitive diagnosis
    - ➢ Diet as for **"Hepatic encephalopathy"** - protein restriction to minimise hepatic workload or protein intolerance
    - ➢ Immunosuppressive therapy:
      - Prednisolone 0.5mg/kg PO 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
    - ➢ Anti-fibrotic:
      - Colchicine 0.03mg/kg PO SID
    - ➢ Copper chelation:
      - D-penicillamine 10-15mg/kg PO BID
      - Zinc acetate 5mg/kg PO BID (max dose 100mg BID)
    - ➢ Anti-oxidants:
      - Vitamin E 400 IU PO SID
      - S-Adenosy-L-methionine (SAMe): Potent anti-oxidant 20mg/kg PO SID
      - Milk thistle
    - ➢ Choleretic:
      - Ursodeoxycholic acid (Actigall ®): 10-15mg/kg PO SID
      - Only if no biliary tract obstruction
    - ➢ Ascites:
      - Spirolactone +/- furosemide
      - Abdominocentesis

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- **Feline hepatic lipidosis:**
- **Pathophysiology:**
  - ✓ Increased hepatocellular accumulation of lipids and cholestasis leading to hepatic failure
  - ✓ Typically seen in middle age obese cats that have experienced a period of prolonged anorexia resulting in rapid weight loss:
    - ➢ Most commonly secondary to pancreatitis, inflammatory bowel disease, cholangiohepatitis and diabetes mellitus
    - ➢ Other causes include hepatotoxins, other systemic illness, surgery, severe prolonged stress
- **Clinical signs:**
  - ✓ Weight loss and anorexia, lethargy, vomiting, jaundice, hypersalivation, hepatomegaly
- **Diagnostics:**
  - ✓ Haematology:
    - ➢ +/- Non-regenerative anaemia
  - ✓ Biochemistry:
    - ➢ Significant increases in ALP, GGT and bilirubin, also increased ALT and AST
    - ➢ ALP increase is usually significantly higher than ALT
  - ✓ Cytology: Hepatocytes with cytoplasmic vacuolisation
  - ✓ Biopsy: Definitive diagnosis
  - ✓ Ultrasound: Diffusely hyperechoic liver +/- hepatomegaly
- **Treatment:**
  - ✓ Supportive therapy:
    - ➢ IV fluids and electrolytes
    - ➢ Antiemitics: Metoclopramide 0.5mg/kg TID, maropitant 1mg/kg SC SID for <5 days
    - ➢ Gastric protectants: Proton pump inhibitors, H_2 antagonist (famotidine), sucralfate
  - ✓ Antibiotics:
    - ➢ +/- Amoxicillin or second generation cephalosporin +/- low dose metronidazole
    - ➢ Change according to culture and sensitivity
  - ✓ Nutrition:
    - ➢ **Very important** via nasoesophageal/gastric or oesophageal tubes
    - ➢ Based on 60kcal/kg/day of ideal body weight (= total caloric intake), using a high protein complete and balanced diet
    - ➢ Start at 25% and increase by 25% per day over 4 days
    - ➢ Monitor electrolytes for refeeding syndrome: Hypophosphataemia, hypokalaemia, hypomagnesaemia
  - ✓ Adjunctive:
    - ➢ Vitamin K1 2.5mg/kg SC BID for 2 days then once weekly
    - ➢ Vitamin B12 250µg SC weekly
    - ➢ S-Adenosy-L-methionine (SAMe): Potent anti-oxidant 20mg/kg PO SID

- **Feline inflammatory liver disease:**
- **Pathophysiology:**
  - ✓ Cholangitis is inflammation of the biliary tract and surrounding liver, common syndrome in cats, rare in dogs.
  - ✓ **Suppurative form:**
    - ➢ Acute neutrophilic cholangitis:
      - Neutrophilic inflammation of the portal triads and bile ductules
      - Ascending bacterial infection or biliary system pathology
      - Acute onset, severe pyrexia illness, usually younger males
    - ➢ Chronic neutrophilic cholangitis:
      - Possible progression of acute neutrophilic form or possible immune mediated disease
      - Often concurrent pancreatitis and IBD

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- Mixed inflammation of the portal areas
- Chronic mild to moderate illness (weeks) occasionally pyrexic, usually middle aged males

✓ **Non suppurative form:**

➢ Lymphocytic portal hepatitis: Lymphocytic and plasmacytic

- Nonspecific lymphocytic infiltration of the portal regions without concurrent cholangitis
- Common causing chronic mild illness (weeks), rarely pyrexic, older aged cats

➢ Lymphocytic cholangitis:

- Lymphocytic infiltration of the portal area with biliary hyperplasia and fibrosis
- Less common, possible immune mediated pathogenesis

▪ **Clinical signs:**

✓ Vomiting, diarrhoea, anorexia, weight loss, jaundice, hepatomegaly, rarely ascites

▪ **Diagnostics:**

✓ Haematology:

➢ +/- Inflammatory leukogram, anaemia, thrombocytopenia

✓ Biochemistry:

➢ Increased bilirubin, bile acids and ALT

➢ ALP increase seen more with chronic forms, GGT is more sensitive in cats

➢ +/- Hypoproteinaemia, hypoglycaemia

➢ +/- Elevated feline pancreatic lipase immunoreactivity

✓ Ultrasound: May not see any specific changes

✓ Biopsy: Definitive diagnosis, histopathology and culture and sensitivity

▪ **Treatment:**

✓ General:

➢ Suppurative form: Long term antibiotics at least 5 weeks

➢ Non suppuratives form: Long course of corticosteroids +/- antibiotics

✓ Supportive therapy:

➢ IV fluids and electrolytes

➢ Antiemitics: Metoclopramide 0.5mg/kg TID, maropitant 1mg/kg SC SID for <5 days

➢ Gastric protectants: Proton pump inhibitors, H_2 antagonist (famotidine), sucralfate

✓ Nutrition: Important via nasooesophageal/gastric or oesophageal tubes and feed as per **"Feline hepatic lipidosis"**

✓ Antibiotics:

➢ Amoxicillin 22mg/kg TID or cephalothin 22mg/kg TID +/- metronidazole 7.5mg/kg BID

➢ Change according to culture and sensitivity

✓ Anti-inflammatories:

➢ If non-responsive to antibiotics, indicated with chronic neutrophilic cholangitis and lymphocytic cholangitis

➢ Prednisolone 1mg/kg PO BID, then tapered over 6 weeks to lowest EOD dose

➢ Chlorambucil: If cannot tolerate prednisolone can trial chlorambucil

✓ Adjunctive:

➢ Anti-oxidants:

- Vitamin E 400 IU PO SID and Vitamin C
- S-Adenosy-L-methionine (SAMe): Potent anti-oxidant 20mg/kg PO SID

➢ Choleretic:

- Ursodeoxycholic acid (Actigall ®) 10-15mg/kg PO SID
- Only if no biliary tract obstruction

➢ Vitamin K 2.5mg/kg SC BID for 2 days then once weekly

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- **Hepatic encephalopathy:**
- **Pathophysiology:**
  - ✓ Can occur when >70% of hepatic function is lost
  - ✓ Secondary to any hepatic disease process, most commonly portosystemic shunts
  - ✓ Younger animals most commonly shunts, older animals most commonly neoplasia
- **Clinical signs:**
  - ✓ Anorexia, vomiting, PU/PD, ascites
  - ✓ Behavioural changes: Disorientation, head pressing, ataxia, pacing, seizures, circling
- **Causes:**
  - ✓ Acute or chronic hepatic failure, portosystemic shunts (congenital and acquired)
- **Diagnostics:**
  - ✓ Haematology, biochemistry and urinalysis:
    - ➢ Microcytic anaemia +/- target cells, acanthocytes, poikilocytes
    - ➢ Variable changes in liver enzymes (ALT, AST, GGT, ALP) and bilirubin
      - Signs of liver dysfunction: Low albumin, urea, glucose, coagulopathy
    - ➢ Paired serum bile acids (usually elevated)
    - ➢ Blood ammonia levels may be elevated
    - ➢ Urinalysis: Low USG and ammonia urate crystals
  - ✓ Imaging:
    - ➢ Ultrasound
    - ➢ Scintigraphy, portography
- **Treatment of acute hepatic encephalopathy:**
  - ✓ IV fluids and electrolytes
  - ✓ Reduce ammonia and urease producing bacteria:
    - ➢ Nil per os 12-24 hours
    - ➢ Enema to remove toxins and reduce ammonia producing bacteria:
      - Warm water to remove contents
      - Retention enema (5-10ml/kg) e.g. 3 parts lactulose to 7 parts water every 6 hours or until colon is empty in crisis states
    - ➢ +/- Gastric lavage if recent ingestion of hepatoxic agent
    - ➢ IV antibiotics: Metronidazole 7.5mg/kg IV BID or amoxicillin 20mg/kg IV TID
  - ✓ Seizures:
    - ➢ Treatment for seizures see **"Seizures Disorders"** BUT avoid benzodiazepines, use propofol and begin phenobarbitone
    - ➢ Cerebral oedema: Mannitol 1g/kg IV over 20 minutes
  - ✓ Gastric protectants: Proton pump inhibitors, H_2 antagonist (famotidine), sucralfate
  - ✓ Coagulopathies
    - ➢ Plasma see **"Coagulopathy"**
    - ➢ Vitamin K1 2.5mg/kg SC BID for 2 days then once weekly.
- **Treatment of chronic hepatic encephalopathy:**
  - ✓ Treatment for underlying disease
  - ✓ Reduce ammonia production
    - ➢ Lactulose 0.5-2ml/kg PO BID-TID OR psyllium (1-3 tsp./day)
  - ✓ Antibiotics:
    - ➢ Used intermittently during relapse, metronidazole 7.5mg/kg PO BID
  - ✓ Diet:
    - ➢ Low protein but high biological value, high carbohydrate to reduce ammonia production e.g. Hills L/D®
    - ➢ Small meals frequently
    - ➢ Moderate fibre content to reduce ammonia production and constipation
    - ➢ Vitamin B and C

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Nasal and Nasopharyngeal Disease

- **This chapter covers:**
  - ✓ Differentials for common clinical signs associated with nasal and nasopharyngeal disease
  - ✓ Including:
    - ➢ **Rhinitis**
    - ➢ **Sneezing**
    - ➢ **Nasal discharge**
    - ➢ **Reverse sneezing**
    - ➢ **Epistaxis**
  - ✓ Lists differentials, methods of diagnosis and treatment principles

- **Nasal discharge and sneezing:**
  - ✓ Differentials:
    - ➢ Sneezing:
      - Acute onset: Foreign body, allergic, infectious – viral, trauma
      - Chronic: Infectious (viral, bacterial (secondary), fungal, parasitic), neoplastic, foreign body
      - Reverse Sneezing: Foreign body, nasal mites, allergic, vomited contents
    - ➢ Nasal discharge:
      - Serous: Viral, allergic, foreign body
      - Purulent: Chronic foreign body, secondary bacterial infection, fungal infection, lower respiratory tract disease, neoplasia, dental disease
      - Sanguineous (epistaxis): Neoplasia, fungal, trauma, foreign body, systemic coagulopathy, hypertension, dental disease
    - ➢ +/- Systemic illness, +/- facial deformity

- **Differentials and treatment:**

Lower respiratory tract disease: ▪ E.g. Pneumonia ▪ Typically see bilateral mucopurulent nasal discharge and signs of lower respiratory tract involvement	
▪ **Diagnosis:** ➢ See **"Respiratory Disease"** ➢ Radiographs of chest ➢ +/- Culture of nasal discharge (but best if lower airway sample collected)	▪ **Treatment:** ➢ Treatment of lower airway disease
Dental disease: ▪ Tooth root abscesses, oral nasal fistulas	
▪ **Diagnosis:** ➢ Oral examination ➢ Radiographs	▪ **Treatment:** ➢ Dental treatment e.g. removal of teeth ➢ Antibiotics ➢ Surgical correction of fistulas
Foreign body: ▪ Acute onset, usually unilateral serous nasal discharge, sneezing and reverse sneezing	
▪ **Diagnosis:** ➢ Nasal flush ➢ Scoping ➢ +/- Radiographs (4-5 day lag phase in radiographic changes)	▪ **Treatment:** ➢ Removal of foreign body ➢ +/- Antibiotics

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Fungal disease:

Aspergillosis:

- Typically, canine disease, especially young dogs with long nose usually *Aspergillus fumigatus*
- Purulent to sanguineous discharge, pain on palpation, ulceration of nasal planum and signs of systemic illness
- Can have neurological signs

Cryptococcus:

- Typically, feline disease, and usually *Cryptococcus neoformans*
- Can have neurological signs with *Cryptococcus var gatti*
- LCAAT is used for monitoring titre levels as an indication of response to therapy

Diagnosis:

- Serology – fungal antibody titres
- Scoping (fungal plaques) and biopsy, cytological demonstration of organisms
- Culture

- **Treatment:**
  - *Aspergillosis:*
    - Topical (best) and systemic antifungals combined with local debridement
  - *Cryptococcus:*
    - Systemic antifungals (amphotericin B if showing neurological signs) combined with surgical debridement

leoplasia:

Commonly adenocarcinoma, squamous cell carcinoma and lymphoma (cats)

- Systemically well, unilateral to bilateral discharge, advanced cases see facial deformity

Diagnosis:

- Radiographs and CT
- Scoping and biopsy

- **Treatment:**
  - Radiation and chemotherapy

Lymphocytic plasmacytic rhinitis:

- Diagnosis of exclusion, based on repeat biopsy findings of lymphocytic plasmacytic inflammation 4 weeks apart

Diagnosis:

- Repeat biopsy

- **Treatment:**
  - Corticosteroids at immunosuppressive doses, once under control can try inhaled

iral infections:

See **"Viral disease and Vaccination"** for a table of comparison

- Typically, FHV and FCV usually with concurrent ocular signs
- Can be concurrently infected with both and cause similar clinical signs, and contribute towards chronic infection:
  - FHV can also have severe conjunctivitis and corneal ulceration
  - FCV can have mild ocular signs, oral ulcers and gingivitis, lameness (self-limiting)
- Chronic viral disease can lead to mucosal destruction and secondary bacterial infection leading to a syndrome called "chronic snufflers":
  - Unilateral/bilateral mucopurulent nasal discharge
  - +/- Chronic sneezing

- See **"Viral disease and Vaccination"** for more information

Diagnosis:

- PCR on conjunctival swab

Chronic snufflers:

- Rule out other causes of mucopurulent nasal discharge
- Radiographs
- Scoping
- +/- Culture of nasal discharge

- See **"Viral disease and Vaccination"** for more information
- **Treatment:**
  - Supportive therapy: Nebulisation and humidification, lysine 500mg BID
  - Antibiotics: Doxycycline to prevent secondary bacterial infection
- **Chronic snufflers:**
  - 8 weeks of doxycycline or clindamycin (good bone penetration)
  - +/- Short courses for relapse
  - +/- Saline nebulisation
  - Anti-viral medication

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Bacterial infections:

- *Mycoplasma, Bordetella Bronchiseptica, Chlamydophila felis* (cats) can cause primary respiratory tract infections
- *Mycoplasma* and *Chlamydophila felis* can cause conjunctivitis

• **Diagnosis:** ➢ Culture of nasal swab (not recommended as almost always secondary to a predisposing factor/cause e.g. Viral infection) ➢ Conjunctival swab and PCR	• **Treatment:** ➢ Treatment of the underlying disease ➢ Supportive therapy: Nebulisation and humidification, nutritional support (smell is important for cat's appetite) ➢ Antibiotics: Doxycycline for up to 3 weeks

Parasitic infections:

- Nasal mites (e.g. *Pneumonyssoides caninum*), varies between geographic regions, typically more common in kennels

• **Diagnosis:** ➢ Scoping ➢ Cytology	• **Treatment:** ➢ Ivermectin 300µg/kg PO once a week (care with collies and it is off label use)

Nasal polyps:

➢ Condition of cats

• **Diagnosis:** ➢ Scoping of nasal cavity and pharynx ➢ Radiographs	• **Treatment:** ➢ Surgical removal

- **Reverse sneezing:**
  - ✓ Increased inspiratory effort through the nose, typically caused by caudal nasal or nasopharyngeal disease
- **Differentials:**
  - ✓ Foreign body
  - ✓ Nasal mites (e.g. *Pneumonyssus caninum*), **see above**
  - ✓ Lymphocytic plasmacytic rhinitis, **see above**
  - ✓ Nasal polyps (cats)
  - ✓ Fungal
  - ✓ Post vomiting
  - ✓ Also other nasal diseases, **see differential list for sneezing above**
- **Diagnostics:**
  - ✓ History:
    - ➢ Any vomiting (vomitus lodged in nasopharynx), eating grass
  - ✓ Visualisation of the nasopharyngeal region and nasal cavity:
    - ➢ Ideally with scope, otherwise spey hook and mirror
  - ✓ Flushing of the nasal cavity with saline:
    - ➢ Intubate and place swabs in the back of the pharynx to absorb fluid and collect debris
  - ✓ Imagery:
    - ➢ Radiographs or CT but can take up to a week before see changes and typically non-specific
    - ➢ Scoping
  - ✓ Biopsy:
    - ➢ Best via scoping but can also be done blind (do not pass the medial cantus of the eye)

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Epistaxis:

- ✓ Bleeding from the nasal cavity
- ✓ Caused by local and systemic disease processes

Differentials:

- ✓ Neoplasia, see previous pages
- ✓ Fungal, see previous pages
- ✓ Trauma
- ✓ Foreign body
- ✓ Systemic coagulopathy, see "Coagulopathy"
- ✓ Hypertension, see "Cardiovascular Disease"
  - ➢ Dental disease

Diagnostics:

- ✓ History: Access to rodenticides, trauma
- ✓ General physical exam
- ✓ Assessment for coagulopathy, see "Coagulopathy"
- ✓ Blood pressure measurements, see "Cardiovascular Disease"
- ✓ Haematology and biochemistry: Assess for systemic disease
- ✓ Imagery:
  - ➢ Radiographs or CT but can take up to a week before see changes but typically non-specific
- ✓ Visualisation via scoping:
  - ➢ Foreign body, fungal (visualisation, cytology and culture/PCR)
- ✓ Biopsy
  - ➢ Best via scoping but can also be done blind (do not pass the medial cantus of the eye)

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Neurological Disease

- **This chapter covers:**
  - ✓ Steps of a basic neurological examination
  - ✓ Differentials for diseases affecting the different areas of the nervous system
  - ✓ **Also covers:**
    - ➢ **Vestibular disease**
    - ➢ **Horner's syndrome**

- **Neurological examination:**
- **Order of tests:**
  - ✓ 1) Mental status: Alteration can indicate intracranial disease
  - ✓ 2) Gait and Posture: Alteration can indicate intracranial disease
  - ✓ 3) Cranial Nerves: Alteration indicate intracranial disease
  - ✓ 4) Proprioception
  - ✓ 5) Spinal reflexes: Patella, withdrawal/flexion, cutaneous trunci and pain
  - ✓ 6) Back pain

- **1) Mental status:**

Sites of damage:	Features:
Cerebrum:	▪ Altered mentation and behaviour ▪ Circling (usually to side of lesion) ▪ Blindness
Brain stem:	▪ Inability to arouse ▪ Stupor or coma

- **2) Gait and posture:**
  - ✓ **Ataxia:**

Type of ataxia:	Features:
Ascending proprioceptive pathways: (sensory)	▪ Limb crossing ▪ Proprioceptive deficits ▪ Weakness ▪ No head tremors
Cerebellum:	▪ Head tremors ▪ Intention tremors ▪ Hypermetria (exaggerated movements) ▪ Dysmetria ▪ Wide base stance ▪ No weakness ▪ Normal proprioception
Vestibular:	▪ Head tilt (different from head turn) ▪ Circling towards lesion ▪ Nystagmus ▪ No weakness ▪ Normal proprioception

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3) **Cranial nerve examination:**

✓ **Symmetry of head and face:**

Disorder:	Cranial nerves:
• Temporal muscle atrophy	• CN V (motor)
• Failure to close mouth	• CN V (motor)
• Abnormal pupil position	• CN III (ventrolateral strabismus), IV, VI (abduction strabismus)
• Abnormal pupil size	• Dilated (CN II and III, brain stem lesion) • Constricted (↓ Sympathetic input – diffuse brain lesions)
• Nystagmus	• CN VIII
• Head tilt	• CN VIII
• Drooping: ➢ Ears ➢ Commissures of lips • Eyelids: ➢ Inability to close	• CN VII
Exams:	**Cranial nerves:**
• Pupillary response test	• CN II, CN III (constriction)
• Ability to blink	• CN V (sensory), CN VII (motor)
• Ability to feel sensation to face	• CN V (sensation) ➢ Ophthalmic branch (medial canthus) ➢ Maxillary branch (lateral canthus) ➢ Mandibular branch (base of ear)
• Jaw tone	• CN V (motor)
• Vestibular nystagmus: ➢ Lift head – eyes should stay in middle of orbit	• CN VIII
• Eating and drinking, gag reflex	• CN IX, X
• Action of tongue / tongue position (centre of mouth)	• CN XII (motor)
Tests that require normal brain functionality – i.e. Tests central involvement	
• Response to noise	• CN VIII
• Physiological nystagmus: ➢ Lift head – affected eye/side will drop	• Vestibular system (where the head is in space) → medial longitudinal fasciculus (MLF) → cerebellum → motor to the eyes → CN III (ventrolateral strabismus), IV, VI (abduction strabismus)
• Menace	• CN II (visual), VII (blink) • Whole visual pathway and cerebellum and cortex

- 4) **Proprioception and hopping:**
  - ✓ Knuckling: Support weight and knuckle
  - ✓ Placing reactions (useful in cats): Bring to table should lift leg up when gets to edge of table, can blind fold
  - ✓ Hopping: Hold one limb off ground and push body to other side

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- **5) Spinal reflexes:**
  - ✓ Used to help localise site of lesion

Test:	Location:	How:
Patella reflex	• L4 – L6 and femoral nerve	• Patella tendon tap
Withdrawal and flexion	• Brachial and sacral nerve plexus • Hindlimb: ➢ Medial toe: L4-L6 and femoral nerve ➢ Lateral toe: L6-S2 and sciatic nerve • Forelimb (middle toe): ➢ Dorsal aspect: C6-T2 and radial nerve ➢ Ventral aspect: C6-T2 and median nerve	• Pinch and pull interdigital skin
Perineal test	• S1 – S3	• Touch/pinch either side of anus
Cutaneous trunci	• T3 – L3 (sensory) and C8 – T1 (motor) • Lack of twitch means lesion is 2 vertebrae cranial to that location • Can be unilateral or bilateral	• Pinch the skin either side of spine until skin flicks • Start at wing of ilium and work cranially
Pain sensation	• Tests pain pathways	• Pinch skin over toes, more pressure required the deeper the lesion • MUST acknowledge (look, bite, yelp)

Segments:	Dysfunctions:
Cervical C1-C5	• UMN to all limbs • Urinary incontinence
Cervicothoracic C6-T2 Brachial plexus	• LMN to forelimb and UMN to hindlimbs • Horner's syndrome • Root signature
Thoracolumbar T3-L3	• UMN to hindlimbs • Urinary incontinence (UMN bladder) • ***Schiff Sherrington posture*** – rigid extension of forelimb and flaccid paralysis of hindlimbs
Lumbosacral L4-S3 Lumbosacral plexus	• LMN loss to hind limbs • Loss of perineal reflexes/sensation • Urinary and faecal incontinence (LMN bladder) • Root signature
Sacral S1-S3	• Normal all limbs • Loss of sciatic function • Loss of perineal reflexes/sensation

Type:	UMN:	LMN:
Paresis/paralysis	Spastic	Flaccid
Spinal Reflexes	Normal to ↑	↓ or absent
Muscle tone	Normal to ↑	↓
Muscle atrophy	Disuse (mild)	Neurogenic (severe)
Gait	Long stride Stiff ataxic	Short stride Lame like gait

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- 6) Neck and back pain:
  - ✓ Palpation with ball of fingers on the sides of each vertebra, range of neck movement with food
  - ✓ For more information, see **"Spinal Pain"** below and **"Spinal Disease"** in **"Skeletal Disease"**

********** CLINICAL SIGNS and DIFFERENTIALS **********

Localising neurological dysfunction:

- **Intracranial:**

Parts:	Clinical Signs and tests:
Cerebrum: Intelligence and goal directed behaviour	• **Clinical signs:** ➢ Seizures ➢ Blindness, absent menace (contralaterally) ➢ Abnormal behaviour ➢ Circling ➢ Proprioceptive deficits • **Tests:** Behaviour (history), menace, nasal septum stimulation, proprioceptive positioning
Brainstem: Alertness and CN functions (only III - XII)	• **Clinical signs:** ➢ CN deficits: Only CN III - XII ➢ Mental depression: Dullness/stupor ➢ Irregular respiration ➢ UMN to all limbs • **Tests:** CN tests, reflexes
Cerebellum: Co-ordinates and fine tunes movement	• **Clinical signs:** ➢ Tremors ➢ Cerebellar ataxia: Dys/hypermetria, wide base stance and gait ➢ Absence of menace: Ipsilateral with unilateral disease ➢ Vestibular signs ➢ Decerebellate rigidity ➢ Paradoxical vestibular syndrome (uncommon) ➢ Normal proprioceptive position • **Tests:** Menace and rule out others

- **Differentials for neurological dysfunction:**
  - ✓ Should be considered for **any neurological clinical signs seen:**

DIFFERENTIALS FOR NEUROLOGICAL DYSFUNCTION	
• **Extracranial:** ➢ Liver disease ➢ Renal disease ➢ Electrolyte and glucose derangements ➢ Toxins ➢ Endocrine disease ➢ Dietary – Thiamine deficiency (fish diets in cats and cooked meat diets in dogs)	• **Intracranial:** ➢ Congenital abnormalities • Hydrocephalous • Storage diseases ➢ Inflammatory CNS diseases (**see next page**): • Non-infectious inflammatory disorders • Infectious inflammatory disorders ➢ Trauma ➢ Neoplasia ➢ Coagulopathies/vascular disorders

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NON-INFECTIOUS INFLAMMATORY DISORDERS			
Type:	**Features:**	**Diagnosis:**	**Treatment:**
• **Granulomatous meningoencephalitis:** • Small breeds, 2-6 year old • Cause unknown, mononuclear accumulations around vessels in the parenchyma and meninges of brain and spinal cord	• **Focal** (50%) • Acts like a focal space occupying lesion' • CSx vary according to where the lesion is • **Disseminated** (50%) • CSx seizures, ataxia, neck pain, stupor	• CSF cytology: Primarily mononuclear, but up to 20% neutrophilic • Lesions on MRI	• Immunosuppressive doses of corticosteroids • Other therapies: cyclosporine, cytarabine, cytosine arabinoside • **Prognosis:** Poor, diffuse form carries an even worse prognosis
• **Steroid responsive meningitis arteritis:** • Young <1 years old large breed dogs • Perivascular suppurative process of unknown cause	• **Acute onset of neck pain** • Lethargy • Inappetance • Reluctance to move • Fever is common	• CSF cytology: >90% neutrophils, but no degenerative neutrophils or bacteria • Peripheral neutrophilia	• Corticosteroid therapy: Tapering over 6 months minimum • Return to normal within 24-48 hours • **Prognosis:** Usually good with complete resolution • Grown out by 18months
• **Necrotizing meningoencephalitis:** • Breed specific: Pug, Maltese, Yorkshire terriers • <1 year to young adult • Mononuclear pleocytosis deep in the white matter of the cerebrum and thalamus	• Neurological CSx: Seizures, ataxia	• 90% lymphocytes in CSF • Lesions on MRI	• Supportive therapy • **Prognosis:** Poor

INFECTIOUS INFLAMMATORY DISORDERS			
Type:	**Features:**	**Diagnosis:**	**Treatment:**
• **Bacterial:** • Not common – usually spread from adjacent structures	• Clinically extremely sick • Neurological signs • Neck pain • Pyrexia	• CSF cytology: Neutrophils (degenerative mainly) • +/- Inciting organism • CSF culture: <70% sensitive	• IV antibiotics and supportive therapy • Poor prognosis
• **Viral:** • FIV, FIP, distemper, rabies	• See **"Viral Disease and Vaccination"**	• See **"Viral Disease and Vaccination"**	• See **"Viral Disease and Vaccination"**

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INFECTIOUS INFLAMMATORY DISORDERS - Continued			
Type:	**Features:**	**Diagnosis:**	**Treatment:**
▪ **Fungal:** ***Cryptococcus*:** ▪ Most common ▪ Common in cats ***Aspergillosis*:** Young <5 years, large breed dogs **Other:** Blastomycoses, Histoplasmosis	▪ Often with upper respiratory tract disease ▪ Usually from inhalation	▪ CSF PCR and culture ▪ Demonstration of organism in CSF ▪ Cryptococcus: LCAT cryptococcal latex agglutination test ▪ Aspergillosis antibody levels	▪ Antifungals: Long term therapy up to years – until pair titres are zero ▪ Prognosis is poor for dogs
▪ **Protozoal:** **Toxoplasma** (dogs and cats) ▪ Usually asymptomatic: Reactivation in older dogs causes disease **Neospora:** (dogs) ▪ < 6 months of age ▪ Older dogs See **"Parasitic Disease"**	▪ **Toxoplasma:** ▪ Can cause any neurological clinical signs ▪ +/- Muscle pain due to myositis ▪ Can also cause systemic disease depending of site of infection ▪ **Neospora:** ▪ <u>≤6 months old</u> ▪ Ascending hindlimb weakness (LMN paresis) ▪ +/- Rigid hyperextension ▪ Hyperaesthesia ▪ <u>Older dogs:</u> ▪ Neurological CSx: Tremors, seizures, paresis/paralysis occasionally systemic illness ▪ See **"Parasitic Disease"**	▪ CSF PCR ▪ Serology: ➢ Increasing IgG titres (>4x) ➢ Elevated IgM with CSx ▪ Check with local pathology lab for protocol ▪ CSF cytology: Mixed or eosinophilic pleocytosis ▪ Haematology: Eosinophilia or monocytosis ▪ ↑ CK ▪ Definitive diagnosis is difficult as need to demonstrate the organism or its DNA ▪ See **"Parasitic Disease"**	▪ Prednisolone 1-2mg/kg PO BID ▪ Clindamycin 15mg/kg PO BID ▪ Doxycycline 5mg/kg PO BID ▪ May require lifelong therapy ▪ See **"Parasitic Disease"**
▪ **Parasitic:** ***Anglostrongylus cantonensis*:** ▪ Rat lungworm	▪ Eosinophilic meningitis ▪ Larval migration into spinal cord via ingesting snails ▪ See **"Parasitic Disease"**	▪ CSF cytology: Eosinophilic pleocytosis +/- larvae ▪ Serum and CSF antibody PCR ▪ See **"Parasitic Disease"**	▪ Prednisolone 1-2mg/kg PO BID ▪ Fenbendazole (mass die off can incite severe anaphylaxis) and acutely worsen clinical signs ▪ See **"Parasitic Disease"**
▪ **Other:** ▪ Rickettsial ▪ Ehrlichia	▪ Regionally specific	▪ Serology	

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Spinal Pain:

- Differentials for spinal pain: See **"Skeletal Disease"**
  - ✓ Inflammatory CNS disorders **(see previous pages)**
  - ✓ Discospondylitis
  - ✓ Vertebral malformation
  - ✓ Polyarthritis or osteoarthritis of joint facets
  - ✓ Intervertebral disc disease
  - ✓ Caudal cervical spondylomyelopathy (Wobbler syndrome)
  - ✓ Cauda equine syndrome (Lumbosacral stenosis)
  - ✓ Neoplasia
  - ✓ Vascular: Fibrocartilaginous emboli, infarct
  - ✓ Trauma: Fracture/luxation
  - ✓ Parasitic migration: e.g. *Angiostrongylus cantonensis* causing eosinophilic meningitis

- **Spinal shock:**
  - ✓ A syndrome of neurological dysfunction after spinal injury due to depression of spinal reflexes
  - ✓ Often transient and usually abates after 24 hours
  - ✓ Repeat examinations are important

- **Root signature:**
  - ✓ Neurogenic pain in a limb due to compression of the nerve roots to that limb
  - ✓ Rule out musculoskeletal disease and if proprioceptive deficits are present then likely root signature
  - ✓ Forelimb root signature indicates C4-C7 lesion

- **Depth of damage and prognosis:**

Loss of:	Depth of damage:	Prognosis:
Proprioception	Superficial	Good
Voluntary motor	Superficial	Fair
Above and **cutaneous pain**	Middle	Moderate
Above and **deep pain**	Deep	Poor

- **Localising the site of damage:**

Test:	Location:	How:
Patella reflex	• L4 – L6 and femoral nerve	• Patella tendon tap
Withdrawal and flexion	• Brachial and sacral nerve plexus • Hindlimb: ➢ Medial toe: L4-L6 and femoral nerve ➢ Lateral toe: L6-S2 and sciatic nerve • Forelimb (middle toe): ➢ Dorsal aspect: C6-T2 and radial nerve ➢ Ventral aspect: C6-T2 and median nerve	• Pinch and pull interdigital skin
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Segments:	Functions:
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Lumbosacral L4-S3 Lumbosacral plexus	▪ LMN loss to hindlimbs ▪ Loss of perineal reflexes/sensation ▪ Urinary and faecal incontinence (LMN bladder) ▪ Root signature
Sacral S1-S3	▪ Normal all limbs ▪ Loss of sciatic function ▪ Loss of perineal reflexes/sensation

Paresis and Paralysis:

- **Weakness or reduced motor function**

Clinical Signs:	
Central nervous system:	**Peripheral nervous system and Myopathies:**
▪ Brain: ➢ Ataxia and conscious proprioceptive deficits in all limbs ➢ +/- Cranial nerve deficits and other neurological clinical signs ▪ Spinal lesions: ➢ Must be between C1 – T2 to cause tetraparesis ➢ C1 – C5: • UMN tetraparesis to all limbs ➢ C6 – T2: • LMN paresis to forelimbs • UMN paresis to hindlimbs	▪ Peripheral nervous system: ➢ LMN tetraparesis – flaccid paresis and paralysis ▪ Muscular (myopathies): ➢ Paresis with normal reflexes and no proprioceptive deficits

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Differentials:	
Central nervous system:	**Peripheral nervous system and Myopathies:**
Same as differentials for "Neurological Dysfunction" and "Spinal Pain" • **Differentials of "Neurological Dysfunction"** ➢ Metabolic disease: • Hepatic and renal disease • Thiamine deficiency • Diabetic neuropathy (cats) • Hypothyroidism • Hyperadrenocorticism • Hypokalaemia • Hypoglycaemia • Hypercalcaemia ➢ Toxins ➢ Congenital abnormalities • Hydrocephalus • Storage diseases ➢ Inflammatory CNS diseases (**see previous pages**): • Infectious • Non-infectious ➢ Trauma ➢ Neoplasia ➢ Coagulopathies/Vascular disorders • **Differentials of "Spinal pain"** ➢ Inflammatory CNS diseases (**see previous pages**): • Infectious/non-infectious ➢ Discospondylitis ➢ Vertebral malformation ➢ Polyarthritis or osteoarthritis of joint facets ➢ Intervertebral disc disease ➢ Caudal cervical spondylomyelopathy (Wobbler syndrome) ➢ Cauda equine syndrome (Lumbosacral stenosis) ➢ Neoplasia ➢ Trauma: Fracture ➢ Polyradiculoneuritis (idiopathic immune mediated) inflammation of spinal nerve roots ➢ Parasitic migration e.g. *Angiostrongylus cantonensis* causing eosinophilic meningitis	• **Other differentials:** • **Traumatic** • **OTHER causes of lameness:** ➢ Osteoarthritis ➢ Polyarthritis: Commonly hocks/carpus pain • **Neoplastic** ➢ Spinal column neoplasia ➢ Thymomas – antibodies to thymus cells resembles ACH receptors • **Myopathies** ➢ Systemic lupus erythematous • **Metabolic disease** • **Toxins:** ➢ Tick paralysis: Hindlimb and respiratory weakness, vomiting ➢ Snake bite: +/- Coagulopathy, haematuria ➢ Organophosphates ➢ Carbamates ➢ Lead ➢ Tetanus: • Hyperaesthetic (tactile, visual, auditory) • Localised signs include trismus, facial muscle spasm generalised signs include stiff gait ➢ Botulism: • Initially stiff gait then a progressive tetraparesis leading to paralysis • Unable to close mouth and eyes • Megaoesophagus and reduced gag • **Idiopathic polyradiculoneuritis:** ➢ Idiopathic immune mediated inflammation of the spinal nerve roots ➢ Usually large breed dogs, sudden onset in a previously healthy animal ➢ See ascending LMN tetraparesis, paralysis, +/- spinal hyperaesthesia ➢ +/- Change in bark, respiratory paralysis (severe) ➢ Usually no megaoesophagus or gag deficits ➢ Systemically well can usually wag tail and move neck normally, continence is usually maintained • **Degenerative myelopathy:** ➢ Mostly in large dogs especially males • *Initial signs:* ➢ Slowly progressive ataxia and weakness of pelvic limbs ➢ Generally symmetrical ➢ Loss of proprioception to hindquarters ➢ Toe scuffing ➢ Hypermetria and hyper-reflexia (UMN)

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	▪ *Chronic cases:* ➢ Hindquarter paralysis ➢ Faecal and urinary incontinence ➢ Foreleg and brain stem involvement ➢ No neurogenic back pain ▪ **Myasthenia gravis:** ▪ *Focal form:* ➢ Megaoesophagus: Regurgitation ➢ +/- Laryngeal/facial dysfunction: Gagging ▪ *Generalised form:* ➢ Episodic weakness (after exercise) ➢ +/- Clinical signs of focal form ▪ *Acute fulminant:* ➢ Acute rapidly progressive weakness ➢ With signs of focal forms

Diagnosis:	
Central nervous system:	**Peripheral nervous system and Myopathies:**
▪ All should have full general physical and neurological examination ▪ Haematology, biochemistry and urinalysis ▪ Radiographs of spine and chest ▪ Myelogram ▪ +/- Serology and PCR on CSF and serum ➢ *Toxoplasma, Cryptococcus, Neospora,* Distemper virus ▪ CSF: ➢ Cytology and culture ▪ MRI	▪ All should have full general physical and neurological examination ▪ Ticks (seasonal): Complete body clip ▪ Metabolic weakness: ➢ Haematology, biochemistry and urinalysis ➢ T4 and ACTH stimulation ▪ High CK (>5 x ↑) = myopathy → EMG and muscle biopsy ▪ Normal CK: EMG, muscle/nerve biopsy, CSF ▪ Radiographs: Osteoarthritis ▪ Joint taps and culture: Polyarthritis ▪ **Specific:** ▪ Snake bite: ➢ ACT, APTT/PTT, venom detection kit ▪ Myasthenia Gravis: ➢ Tensilon response test (anticholinesterase), Anti-ACh antibody titres ▪ Polyradiculoneuritis: ➢ EMG, nerve biopsy

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Vestibular Disease:

Class:	***Central vestibular disease:*** Brainstem, cerebellum	***Peripheral vestibular disease:*** Inner ear and distal CN VIII
Common clinical signs for both central and peripheral:	▪ Heat tilt, falling and leaning, circling (tight circles) → to side of lesion ▪ **Note: Head turn** indicates cerebral disease not vestibular – see above **"Differentials for neurological dysfunction"** ▪ Ataxia: Wide based stance and falling to side of lesion ▪ Nystagmus: ➢ Physiological: Normal nystagmus – fast phase in direction of head movement ➢ Spontaneous: Involuntary – fast phase away from side of lesion ➢ Positional: Nystagmus that changes direction/intensity when head is placed in an unusual position ▪ Strabismus: Ventral or ventrolateral on ipsilateral side ▪ Vomiting (nausea)	
Differentiating clinical signs:	▪ Nystagmus (<66 beats/min): ➢ Horizontal, rotary, vertical ➢ "Positional" nystagmus: May change when change head position ➢ Fast phase varies with head movement and can be toward or away from head tilt ▪ Altered mentation: Depressed, stupor, obtunded ▪ +/- Ipsilateral postural and proprioception deficits ▪ +/- Multiple cranial nerve deficits ▪ Hemiparesis (if unilateral lesion) ▪ Tetraparesis (if multifocal)	▪ Nystagmus (>66 beats/min): ➢ Horizontal or rotary ➢ Nystagmus not change when change head position ➢ Fast phase in the direction opposite the head tilt ▪ NORMAL postural and proprioception reactions ▪ +/- Concurrent Horner's
Causes:	▪ Traumatic ▪ Haemorrhage/vascular infarct ▪ Infectious/inflammatory disorders: GME (dogs), FIP, FeLV, FIV ▪ Neoplasia ▪ Thiamine deficiency (cats)	▪ Feline idiopathic vestibular syndrome (young to middle aged) ▪ Geriatric vestibular disease (older) ▪ Otitis media/interna ▪ Middle ear tumours/nasopharyngeal polyps ▪ Trauma ▪ Congenital vestibular syndrome (purebred) ▪ Drugs (Aminoglycoside ototoxicity, ear cleaners) ▪ Hypothyroidism
Diagnosis:	▪ +/- Biochemistry, haematology and electrolytes ▪ Imaging (CT/MRI) ▪ CSF (cytology/culture) ▪ Serology from CSF or serum (Toxoplasma, Cryptococcus, Neospora)	▪ +/- Biochemistry, haematology and electrolytes ▪ Neurological examination ▪ Otoscopic examination ▪ Pharyngeal exam ▪ Imaging (radiographs/CT) ▪ Myringotomy (cytology/culture)
Treatment of **idiopathic Disease:**		▪ Sedatives – diazepam (severe disorientation and ataxia) ▪ Antiemetic (of anti-motion sickness drugs): e.g. dimenhydrinate, chlorpromazine, maropitant ▪ Antibiotics: If suspect otitis media
Prognosis for **idiopathic Disease:**		▪ Nausea and nystagmus should improve dramatically in 3 days ▪ Significant improvement in 7-10 days ▪ Back to normal in 2-3 weeks

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- **Horner's syndrome:**
- Disruption of the sympathetic innervation to the eye and peri-orbital region
  - ✓ Disruption in one of three regions:
    - ➢ First order: Hypothalamus to cervical spinal cord
    - ➢ Second order: Cervical spinal cord to the sympathetic chain then to the cranial cervical ganglion (near the tympanic bullae)
    - ➢ Third order: Cranial cervical ganglion through the tympanic bullae to the orbit
  - ✓ Caused by:
    - ➢ Idiopathic, most common especially in golden retrievers
    - ➢ Brainstem disease
    - ➢ Inflammation of the trigeminal nerve
    - ➢ Otitis interna/media
    - ➢ Trauma to neck, skull, orbit
    - ➢ Retrobulbar neoplasia/infection
- Clinical signs:
  - ✓ Almost always unilateral
  - ✓ See miosis (constriction), enophthalmos, ptosis, protrusion of 3rd eyelid
- Diagnosis:
  - ✓ History of trauma/infection
  - ✓ General physical examination
  - ✓ Neurological examination
  - ✓ Lesion localisation:
    - ➢ Administer 10% phenylephrine onto eye and time how long it takes for >75% mydriasis:
    - ➢ Third order: <20 minutes
    - ➢ Second order: 20-45 minutes
    - ➢ First order: >45 minutes
  - ✓ Radiographs:
    - ➢ Neck, skull, tympanic bullae
- Treatment:
  - ✓ Treat the underlying cause
  - ✓ Idiopathic: No treatment, can come back to normal in days to months

Cognitive dysfunction syndrome (CDS):

- ✓ Cognition: Aspects of mentation that relate to perception, awareness, learning, memory
- ✓ Age-related cognitive behaviour impairment in dogs that is not related to sensory or motor impairment or a medical condition referred to as **"old dog syndrome"** or **"doggy Alzheimer's"**

Clinical signs: One or more of four signs of behavioural dysfunction in absence of any physical cause:

- ✓ Disorientation
- ✓ Interacts less and decreased activity
- ✓ Sleep pattern disturbed
- ✓ Housetraining lost

Diagnosis of CDS only after other medical conditions that can cause behavioural changes have been ruled out:

- ✓ Thorough behavioural and medical history
- ✓ A complete physical exam, including neurological exam
- ✓ Appropriate laboratory tests and imaging to rule out other medical conditions

Treatment:

- ✓ Propentophylline (Vivitonin), Gabapentin, Dietary management (Hills B/D diet)

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- **Urinary bladder dysfunction:**
  - ✓ Treatment: See **"Urinary Tract Disease - Urinary Incontinence"**
  - ✓ **Lower motor neuron bladder:**
    - ➢ Urine dribbling, large flaccid bladder that is easily expressed
  - ✓ **Upper motor neuron bladder:**
    - ➢ Large firm bladder that is difficult to express

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Ophthalmology

- **This chapter covers:**
  - ✓ Common terminology
  - ✓ Basic ocular examination and principles
  - ✓ Differentials and treatment for commonly seen lesions and diseases

Diagnostics and physical examination:

1). Oral examination:

- ✓ Suggests retrobulbar or orbital disease - pain on opening mouth or swelling behind molars

2). Ocular discharge:

- ✓ Type, unilateral/bilateral, duration (acute vs chronic)

3). Retroillumination:

- ✓ Darkened room, stand at arm's length in front of the dog at eye level or looking slightly up, bright light next to your face pointing at dog's eye
- ✓ Assessment of the transparency of the ocular structures by comparing fundic reflexion between eyes
- ✓ Assessment of the pupil symmetry, size and shape:
  - ➢ Small pupil size: Uveitis (usually)
  - ➢ Large pupil size: Glaucoma (usually

4). Neurological tests:

- ✓ Vision tests: If fail all then indicates central blindness
  - ➢ Menace reflex: Tests sensory structures and the visual pathways (CN II) and ability to blink (CN VII)
  - ➢ Tracking of objects/light: Tests sensory structures and the visual pathways (CN II) motor to eye muscles (CN III, IV, VI)
  - ➢ Ability to navigate the room: Like menace reflex, requires intact visual pathways
- ✓ Palpebral and corneal reflexes: Sensation from eye and associated structures (CN V) and ability to blink (CN VII)
- ✓ Third eyelid protrusion: Loss of CN VI (motor)
- ✓ Pupillary light reflexes (PLR):
  - ➢ Tests sensory structures in the eye and sensory pathway (CN II) to the mid-brain and CN III nucleus and parasympathetic output to the iris
  - ➢ Direct and consensual PLR
  - ➢ Can be normal in cortically blind animals, not a sign of visual ability
- ✓ Cranial nerves and function:

Cranial Nerve:	Function:
CN II	Sensory to CNS
CN III	Dorsal, medial and ventral rectus muscles Parasympathetic to iris (constriction)
CN IV	Dorsal oblique muscle
CN V	Sensation from the eye (cornea, conjunctiva, lacrimal gland) and skin around eye
CN VI	Lateral rectus and retractor bulbi muscles
CN VII	Ability to blink

5). Globe size:

- ✓ Look from top down to assess symmetry

6). Retropulsion:

- ✓ Don't do if ulcer
- ✓ Push in all directions to identify if orbital disease is present

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7). Assessment of conjunctiva and sclera:

✓ **Conjunctival blood vessels → indicates WHERE the lesion is**

SUPERFICIAL DISEASE: (Conjunctivitis and keratitis)	DEEP DISEASE: (Intraocular disease)
▪ **Superficial blood vessels:** ➢ Fine diameter ➢ Branch ➢ Mobile (use a cotton tip) ➢ Blanch rapidly: Couple seconds (2-5% Phenylephrine diluted 1:10) ➢ Form loops at limbus	▪ **Deep blood vessels:** ➢ Wide diameter ➢ Branch rarely ➢ Immobile (don't move) ➢ Blanch slowly: 40 seconds (2-5% Phenylephrine diluted 1:10) ➢ Stop before limbus

8). Schirmer tear test:

✓ Assessment of aqueous tear production:

➢ Not reliable in cats

➢ Normal 15-20mm/minute

➢ Keratoconjunctivitis sicca: <10mm/minute

9). Examination of the eyelids:

10). Assessment of the iris and lens:

11). Assessment of the anterior chamber:

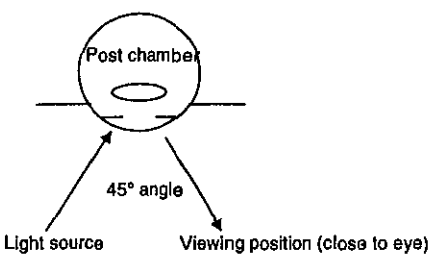
✓ Examination of the eye from the side

✓ Presence of aqueous flare:

➢ Aqueous flare caused by fibrin, plasma proteins and cells in the anterior chamber usually caused by uveitis

➢ How:

- In a darkened room point a bright pinpoint light at the eye and look at the eye very closely at a 45° angle direction to the incoming light source
- If aqueous flare will be seen as a "beam of brightness" between two "slips of grey"



12). Assessment of posterior chamber:

✓ Direct and Indirect ophthalmoscopy: To observe the retina, optic nerve

13). Fluorescein stain:

✓ Assessment of corneal damage, patency of lacrimal duct

14). Assessment of intraocular pressure:

✓ Tonometry:

➢ Normal = 10-25 mmHg

➢ Uveitis: Low pressure

➢ Glaucoma: High pressure

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- **Drugs used during examination:**
  - ✓ Mydriatic agents: Cause dilation
    - ➢ Tropicamide: More rapid onset (20 minutes) and shorter duration (<12 hour)
    - ➢ Atropine: Blocks cholinergic responses of iris sphincter muscles, may persist for up to weeks, use once diagnosis has been made
  - ✓ Local anaesthetic drops:
  - ✓ Phenylephrine:
    - ➢ Used to diagnose Horner's syndrome
    - ➢ See "Neurological Disease"
  - ✓ Anti-inflammatory drugs:
    - ➢ Prednisolone 1% most potent topically
    - ➢ Dexamethasone 0.1% potent and penetrates into eye best

- **Terminology:**
  - ✓ Miosis: Pupillary constriction
  - ✓ Mydriasis: Pupillary dilation
  - ✓ Epiphora: Excessive tear production
  - ✓ Blepharospasm: Narrowed palpebral fissure due to pain
  - ✓ Photophobia: Aversion to light
  - ✓ Enophthalmos: Sunken globe
  - ✓ Exophthalmos: Bulging globe
  - ✓ Buphthalmos: Pathologically enlarged globe
  - ✓ Distichiasis: Hairs growing along the eyelid margin, originating from the ducts of the meibomian glands
  - ✓ Trichiasis: Contact of normal hair with the eye
  - ✓ Ectopic cilia: Hair growing on the conjunctival side of the eyelid, usually the upper eye lid

Specific Conditions:

- **Blindness:**
- Caused by:
  - ✓ Disease affecting structures of the eye:
    - ➢ Cornea, anterior and posterior chambers, iris, lens, retina and optic disc
  - ✓ Disease affecting neurological pathways and CNS:
    - ➢ Cranial nerves and CNS
- Blind and normal PLR: = Central blindness
  - ✓ Bilaterally normal PLR: Brain trauma, seizures, tumours, inflammatory CNS diseases, toxicity, vascular/ischemic accidents, inflammatory CNS disease
  - ✓ Unilateral normal PLR: Contralateral cerebral lesion - neoplasia, vascular/ischemic accidents, toxoplasma, inflammatory CNS disease
- Blind with abnormal PLR:
  - ✓ Unilateral abnormal PLR: Disease associated within the affected eye or damage to its optic nerve, **see Fundus in following pages**
  - ✓ Bilateral abnormal PLR: Disease associated with both eyes or both optic nerves or chiasm, e.g. optic neuritis, infections (Cryptococcus, toxoplasma), inflammatory CNS disease, neoplasia, **see Fundus in following pages**

- **Strabismus:**
  - ✓ Deviation in one or both eyes away from normal
  - ✓ DDx: Extraocular muscle (cranial nerve denervation (see above), atrophy, scarring), congenital, orbital disease

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- **Nystagmus:**
  - ✓ Uncontrolled oscillatory movement of the eye
  - ✓ Can be normal in Siamese cats
  - ✓ Disease affecting the vestibular system either peripheral or central disease:
    - ➢ See Vestibular disease in **"Neurological Disease"**

- **Enophthalmos:**
- Displacement of the globe caudally, +/- secondary third eyelid protrusion, and drooping of upper eyelid
- Reduced orbital fat and muscle
- Dehydration:
  - ✓ CSx: Systemic signs of dehydration
  - ✓ Tx: Fluid replacement therapy and treat underlying cause
- Horner's syndrome:
  - ✓ Loss of sympathetic innervation – most commonly idiopathic especially in Golden Retrievers
  - ✓ See Horner's syndrome in **"Neurological Disease"**
  - ✓ CSx: Miosis, third eyelid protrusion, upper lid droop "ptosis"
  - ✓ DDx: Orbital disease (e.g. tumour, abscess), cervical pathology (trauma, disc disease, tumour), middle ear (infections, trauma, tumour, polyp)
  - ✓ Dx: GPEx – head, neck, chest and ear examination, radiographs and advanced imagery
  - ✓ Tx: Treat underlying disease otherwise can spontaneously resolve after couple months
- Ocular pain
- Globe collapse/rupture

- **Exophthalmos:**
  - ✓ Displacement of the globe cranially
  - ✓ **MUST** protect cornea – lubrication to prevent exposure keratitis
  - ✓ **Treat** corneal ulceration if present
- Orbital tumours:
  - ✓ CSx: Usually non-painful, unilateral/bilateral
  - ✓ Dx: Imagery (ultrasound, CR/MRI), fine needle aspirates/biopsy
  - ✓ Tx: Surgical removal of orbital contents, +/- radiation and chemotherapy
- Orbital haemorrhage:
  - ✓ CSx: +/- Scleral or intraocular haemorrhage
  - ✓ Dx: History of trauma, coagulation testing
  - ✓ Tx: Anti-inflammatory pain relief, corneal protection
- Retrobulbar abscesses:
  - ✓ CSx: Pain of palpation of eye, pain on opening mouth, anorexia
  - ✓ Dx: Ultrasound of the orbit, FNA behind last molar or carnassial tooth, dental radiographs
  - ✓ Tx: Establishing drainage, antibiotics and removal of underlying cause (generally tooth root infection)
- Salivary gland abnormalities: e.g. Zygomatic mucoceles
  - ✓ CSx: Unilateral
  - ✓ Dx: Aspiration (straw coloured +/- blood tinged mucoid material)
  - ✓ Tx: Surgical removal
- Masticatory myositis:
  - ✓ CSx: Pain/inability to opening mouth, pain or atrophy of temporalis and masticatory muscles, anorexia
  - ✓ Dx: Clinical signs, biopsy and histopathology, serum 2M-antibodies
  - ✓ Tx: See Masticatory muscle myositis in **"Dysphagia"**
- Prolapse or Proptosis:
  - ✓ CSx: Generally, eye is trapped in front of the eyelids
  - ✓ Tx: Replacement of globe or enucleation
  - ✓ Positive prognostic indicators:

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- Miotic pupil
- Positive consensual light reflex and menace response
- Short duration of proptosis
- Minimal extraocular and soft tissue damage

✓ Negative prognostic indicators:
- Haemorrhage into eye
- Extraocular muscle avulsion

- **Eyelid Disorders:**
- Abnormal extra hair: (Distichiasis)
- Abnormal contact of normal hair with eye: (Trichiasis)
  - ✓ Can be secondary to entropion
  - ✓ CSx: Epiphora, blepharospasm, corneal ulceration
  - ✓ Tx: Electrical or thermal (cold) destruction of hair follicle, treatment of entropion
- Entropion: (Inward rolling of eyelid)
  - ✓ CSx: Epiphora, blepharospasm, corneal ulceration
  - ✓ Tx: Surgical correction if not secondary to pain (spastic entropion) and >10-12 months old (may grow into excess skin) can temporarily evert until then with tacking sutures or temporary tarsorrhaphy
- Eyelid gland inflammation (meibomian adenitis) or stye (hordeolum):
  - ✓ Tx of Stye: Lance and topical antibiotic anti-inflammatories
  - ✓ Tx of Meibomian adenitis: Topical anti-biotic anti-inflammatories +/- systemic antibiotics
- Eyelid gland neoplasia:
  - ✓ Dogs: Meibomian gland adenoma – most common especially in middle to old aged, behave benign
  - ✓ Cats: Squamous cell carcinoma – locally and invasive and can metastasize, due to UV exposure especially on non-pigmented area
  - ✓ Tx: Surgical resection
- Third eyelid protrusion:
  - ✓ See all causes of enophthalmos – e.g. ocular pain (see epiphora and blepharospasm), Horner's syndrome (see "enophthalmos"), drugs
  - ✓ Bilateral third eyelid protrusion in cats = "Haws Syndrome" idiopathic self-limiting condition that resolves over 5-6 weeks
- Third eyelid gland prolapsed aka "Cherry Eye":
  - ✓ Not: Important for production of aqueous component of tears (approximately 1/3 of total tear production), chronic cherry eye can lead to keratoconjunctivitis sicca
  - ✓ Tx: Surgical replacement best, eye lubrication until surgery to reduce further inflammation
- Third eyelid neoplasia:
  - ✓ CSx: Swellings or masses on the third eye lid
  - ✓ Tx: Surgery, chemotherapy (lymphoma in cats)

- **Conjunctiva:**
- Conjunctivitis:
  - ✓ CSx: Discharge (serous to mucopurulent), hyperaemia of conjunctival blood vessels, blepharospasm, chemosis

SUPERFICIAL DISEASE **Conjunctivitis and keratitis:**	DEEP DISEASE **Intraocular disease:**
• **Superficial blood vessels:** ➢ Fine diameter ➢ Branch ➢ Mobile (use a cotton tip) ➢ Blanch rapidly: Couple seconds (2-5% Phenylephrine diluted 1:10) ➢ Form loops at limbus	• **Deep blood vessels:** ➢ Wide diameter ➢ Branch rarely ➢ Immobile (don't move) ➢ Blanch slowly: 40 secs (2-5% Phenylephrine diluted 1:10) ➢ Stop before limbus

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- ✓ DDx: Bacterial (*Chlamydophila*), allergic, viral (FHV, FCV, distemper), foreign body, KCS, trauma, secondary to other ocular disease (corneal ulceration, uveitis, neoplasia), eosinophilic keratoconjunctivitis (raised pink/white lesions):
  - ➢ Cats: More likely infectious in nature FHV (1st), *Chlamydophila* (2nd), FCV and mycoplasma
  - ➢ Dogs: More likely non-infectious e.g. Dry eye, eye lash irritation, allergic
- ✓ Dx: Schirmer tear test (all dogs), fluorescein stain, PCV (FHV, FCV, *Chlamydophila*), rarely biopsy and culture
- ✓ Tx: Treat underlying disease
  - ➢ Allergic: Once ruled out ulceration and foreign body, treat with antibiotic/anti-inflammatory eye drops
  - ➢ FHV: Usually self-limiting, but anti-viral agents (e.g. Famciclovir oral 40mg/kg TID) if severe, corneal ulceration or relapsing, topical antibiotics, lysine supplementation 500mg BID (reduces viral replication)
  - ➢ *Chlamydial*: Topical antibacterial ointments TID (chloramphenicol), systemic doxycycline 5mg/kg PO BID

- **Lacrimal system:**
- Reduced aqueous tear production aka "Dry eye" or "Keratoconjunctivitis Sicca":
  - ✓ CSx: Mucopurulent discharge, conjunctivitis, corneal ulceration and pigmentation
  - ✓ DDx: Drug induced (trimethoprim-sulfa, sulfasalazine), immune mediated (most common), others e.g. surgery
  - ✓ Dx: Schirmer tear test <10mm/minute (hard to interpret when ulcer is present), also fluorescein stain or ulceration
  - ✓ Tx: Cyclosporine topical ointment for 4 weeks then retest, artificial tears (e.g. Viscotears®), surgery (not recommended)
- Reduced drainage:
  - ✓ CSx: Epiphora (usually without conjunctivitis)
  - ✓ DDx: Dacryocystitis (inflammation of the lacrimal sac/duct), lacrimal puncta disorders (small or absent), nasolacrimal duct obstructions
  - ✓ Dx: Lack of fluorescein stain drainage from ipsilateral nostril, expression of discharge from lacrimal punta
  - ✓ Tx: Nasolacrimal cannulation

- **Cornea:**
- Superficial/simple ulcers:
  - ✓ CSx: Epiphora, blepharospasm, conjunctivitis
  - ✓ DDx: Trauma, foreign body, KCS, eyelid/lash disorders, viral (FHV), bacterial (*Chlamydophila*), allergic:
    - ➢ Cats: FHV is most common, relapses due to stress, treat aggressively (no steroids)
  - ✓ Dx: Schirmer tear test, assessment of eyelids, conjunctiva, fluorescein staining
  - ✓ Tx: Treat predisposing cause e.g. FHV and *Chlamydophila* see above in "conjunctivitis" and in **"Nasal and nasopharyngeal disease"**
    - ➢ Topical antibiotic ointment, +/- systemic doxycycline 5mg/kg PO BID
    - ➢ If miosis then mydriatic therapy (atropine drops until mydriasis), e-collar and analgesia
  - ✓ Should resolve in 7 days
- Deep ulcers:
  - ✓ Dx: Thick rim of fluorescein stain uptake
  - ✓ Tx: Aggressive treatment, topical potent antibiotics q2hrs e.g. ocufloxacin and systemic doxycycline 5mg/kg PO BID, mydriatic therapy (single application), serum drops q2hrs, systemic pain relief, if very deep +/- surgical/corneal conjunctival grafts
- Descemetoceles:
  - ✓ Dx: Deep rim of positive stain uptake and a central area of no stain uptake (descemet's membrane)
  - ✓ Tx: Require surgical repair with a corneal/conjunctival grafts with topical and systemic antibiotics

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- Indolent ulcer:
  - ✓ Layer of non-adherent cornea, common in boxers
  - ✓ Tx: Corneal debridement (topical local anaesthetic), grid keratotomy and topical antibiotics
- Melting ulcer:
  - ✓ Liquefaction of stroma by proteases produced by bacterial (*Pseudomonas spp.*) and fungal infections or inflammatory cells (sterile process)
  - ✓ Tx: Serum drop q2hrs (anti-proteases), topical antibiotics e.g. ocufloxacin and systemic antibiotics e.g. doxycycline 5mg/kg PO BID and antifungals based on C&S results
- Sequestrum:
  - ✓ Focal brown/black plaque often raised +/- associated with ulceration
  - ✓ Syndrome of cats, secondary to chronic corneal irritation/ulceration typically FHV
  - ✓ Tx: Topical antibiotics q4hrs e.g. ocufloxacin, artificial tears, anti-viral medication (if FHV), +/- keratectomy with conjunctival graft (treatment of choice)
- Pannus:
  - ✓ Chronic immune mediated keratitis, chronic inflammation leads to cellular infiltration and vascularisations, later leading to granulation, common in German Shepard's and greyhounds
  - ✓ Tx: Lifelong, topical corticosteroids or cyclosporine
- Corneal infiltrations:
  - ✓ Lipid: Do not cause ulcers
    - ➢ DDx: Idiopathic, hyperadrenocorticism, hypoadrenocorticism, hypothyroidism and diabetes
  - ✓ Calcific: Appear more crystalline, can cause breakout and cause ulcers
    - ➢ DDx: Hyperadrenocorticism, hypoadrenocorticism, hypothyroidism and hypercalcaemia
- Pigmentation:
  - ✓ Secondary to chronic corneal irritation

- **Lens pathology:**
- Nuclear sclerosis:
  - ✓ Non-pathological progressive central opacity (blue-grey) due to compression of lens cells, looks like a "foggy windscreen", common in dogs >8 years old
- Cataracts:
  - ✓ Irreversible lens opacity due to changes in lens fibres/proteins, looks like "shattered windscreen"
  - ✓ DDx: Congenital, hereditary (bilateral and progressive), diabetic (osmotic draw), secondary to uveitis (usually unilateral), retinal pathology
  - ✓ Tx: Surgical removal (many options, not all are candidates depends on pathogenesis), +/- lens implantation
- Lens luxation:
  - ✓ Displacement of lens (anterior, posterior)
  - ✓ DDx: Primary luxation (present unilateral but is bilateral condition) or secondary due to trauma, glaucoma, uveitis, tumours, cataracts
  - ✓ Tx: Surgical removal as can lead to glaucoma, medical management to trap the lens posteriorly where it causes less problems than anteriorly but does not prevent glaucoma

- **Uvea:**
- Uveitis:
  - ✓ Inflammation of the uvea
  - ✓ CSx: Photophobia, epiphora, blepharospasm, miosis, deep scleral injection, corneal oedema, aqueous flare, hyphema, hypopyon, synechiae, reduced intraocular pressure, others
  - ✓ Chronic changes: Iris pigmentation, cataract, globe collapse/shrinkage, glaucoma, synechiae
  - ✓ DDx: Trauma, infectious (bacterial, fungal, viral (FIP, FeLV, FIV), parasitic (toxoplasma)), corneal and lens pathology, tumour, immune mediated, hypertension
  - ✓ Tx: Topical anti-inflammatory e.g. dexamethasone/prednisolone acetate 1% QID, mydriatics topical atropine QID until mydriasis, topical antibiotics q4hrs e.g. ocufloxacin, pain relief e.g. tramadol or systemic anti-inflammatories (NSAIDs or corticosteroids), treatment of underlying cause

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- Hyphema:
  - ✓ Blood in the anterior chamber
  - ✓ Causes: Traumatic, coagulopathy (rodenticide, IMTP), neoplastic, systemic hypertension, uveitis
  - ✓ Tx: Topical anti-inflammatory e.g. dexamethasone/prednisolone acetate 1% QID, mydriatics e.g. topical atropine QID until mydriasis, systemic pain relief, treatment of underlying cause
  - ✓ Should improve over several days, but prognosis depends on the amount of blood present
- Glaucoma:
  - ✓ Elevated intraocular pressure >25mmHg with clinical signs
  - ✓ Disrupts retinal and optic nerve functionality
  - ✓ CSx: Globe enlargement, pain, corneal oedema, blepharospasm, episcleral injection, mydriatic non-responsive pupil, lens subluxation, vision loss
  - ✓ DDx: Primary due to inherited defects in the iridocorneal filtration angle or secondary to uveitis, lens subluxation, tumour
  - ✓ Tx:
    - ➢ Topical therapy, recheck IOP in 2 hours:
      - Latanoprost 0.005% (Xalantan) – increases fluid drainage
      - Dorzolamide 2% (Trusopt) – reduces aqueous humour
      - Timolol – reduces intraocular pressure, can be combinted with Dorzolamide (Cosopt)
    - ➢ If no reduction, then can try mannitol 1-2g/kg slow IV
    - ➢ Long term surgery e.g. shunt implant, cyclocryotherapy or laser
    - ➢ Eye enucleation
    - ➢ Treatment of the underlying cause if secondary

- **Fundus:**
- Inherited retinopathy:
  - ✓ Progressive retinal atrophy
  - ✓ CSx: Loss of vision (progressive – starts with night blindness), pupils are mydriatic and reduced responsiveness
  - ✓ Dx: Tapetal hyperreflectivity, attenuated retinal blood vessels, optic disc pallor, electroretinography
  - ✓ Tx: No treatment
- Sudden acquired retinal degeneration syndrome (SARDS):
  - ✓ Retinal degeneration of unidentified cause
  - ✓ CSx: Loss of vision (acute), middle aged dogs showing signs of polyuria/polydipsia, weight gain, lethargy
  - ✓ Tx: No treatment
- Taurine deficiency retinal degeneration:
  - ✓ Associated with taurine deficient diets fed to cats, e.g. dog food
- Retinal detachment:
  - ✓ DDx: Hypertension (most common in cats), coagulopathy, infections, trauma, uveitis, cataracts, lens luxations
  - ✓ CSx: Loss of vision (acute), pupils are mydriatic and non-responsive
  - ✓ Dx: Visualisation of retinal detachment, ultrasound
  - ✓ Tx: Treat underlying cause
- Chorioretinitis: - Retinal inflammation
  - ✓ DDx: Primary inflammation due to infectious agent or secondary to systemic disease
  - ✓ Dx: Dull regions on the retina (reduced reflectivity), focal spots (granulation tissue), inflammation around retinal blood vessels appearing blurred, retinal detachment, haemorrhage

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Pancreatic Disease

- **This chapter covers:**
  - ✓ Features of pancreatitis and exocrine pancreatic insufficiency
  - ✓ Diagnosis and treatment principles

Pancreatitis:

- **Pathophysiology:**
  - ✓ Inactive pancreatic enzymes are activated prematurely within the pancreas leading to autodigestion. Exhaustion of anti-inflammatory mediators combined with hypotension can result in shock and multiple organ failure.
  - ✓ Risk factors:
    - ➢ Ingestion of high fat foods
    - ➢ Bile reflex and pancreatic duct blockage
    - ➢ Pancreatic trauma (+/- abdominal surgery), ischaemia
    - ➢ Drugs: Corticosteroids, potassium bromide and phenobarbitone combination therapy?
    - ➢ Part of feline inflammatory bowel disease and cholangiohepatitis
  - ✓ Feline pancreatitis:
    - ➢ Usually low grade chronic process, associated with inflammatory bowel disease, cholangiohepatitis, diabetes mellitus
    - ➢ Clinical signs are non-specific i.e. lethargy and anorexia
    - ➢ Difficult disease process to definitively diagnose
    - ➢ Biochemistry profile likely to reflect concurrent hepatic involvement
    - ➢ Ultrasound does not commonly show typical changes
    - ➢ Treatment is supportive and should be directed at the inflammatory bowel disease and/or cholangiohepatitis rather than the pancreatitis, early enteral nutrition (tube feeding) is important

Clinical signs:

- ✓ Dogs: Anorexia, vomiting, abdominal pain, pyrexia
- ✓ Cats: Lethargy, anorexia, hypothermia most frequently, less commonly vomiting and abdominal pain

Differential diagnosis:

- ✓ Acute gastroenteritis, gastric ulceration, inflammatory bowel disease, obstruction, peritonitis, liver failure, renal failure, neoplasia, hypoadrenocorticism and others

- **Diagnostics:**
  - ✓ Technically difficult, should be based on combination of supportive clinical signs and diagnostics
  - ✓ See **"Species differences"** above
  - ✓ Biochemistry:
    - ➢ Amylase: Poor specificity, should be > 3-4 times normal
    - ➢ Lipase: More specific than amylase, serum levels should be increased by 3-4 times normal level
    - ➢ Hyperlipidaemia
    - ➢ +/- Other secondary changes: Azotaemia, hepatopathy
    - ➢ Cats:
      - Typically signs of liver disease (↑ liver enzymes and bilirubin)
      - Less commonly increases in amylase and lipase
  - ✓ Pancreatic lipase immunoreactivity (cPLI and fPLI):
    - ➢ Conflicting evidence and anecdotal reports regarding sensitivity and specificity
    - ➢ False positive occur with non-pancreatic disease e.g. gastroenteritis, inflammatory bowel disease, neoplasia, renal disease, diabetes mellitus
    - ➢ May be used to track the progression/resolution of the disease if it confirmed by other methods
    - ➢ Should not be used as the sole diagnostic test for the diagnosis of pancreatitis
  - ✓ Imagery:
    - ➢ Ultrasonography: non-invasive gold standard
      - Cats commonly don't see any changes as compared to dogs

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- Radiography: Not very sensitive

- **Treatment:**
  - ✓ Antibiotics:
    - Dogs: Not proven to be beneficial, only if signs of bacterial translocation, peritonitis
    - Cats: Antibiotics can be considered due to concurrent cholangiohepatitis
  - ✓ Analgesia:
    - Opioid pain relief e.g. methadone QID or fentanyl patch or CRI, see **"Anaesthesia and Analgesia"**
  - ✓ Fluids and electrolytes:
    - Most important, correction of perfusion and hydration deficits and electrolyte derangements
    - See **"Fluid Therapy"**
  - ✓ Antiemetic: Metoclopramide CRI, maropitant 1mg/kg SC SID for <5 days
  - ✓ Gastroprotectants: Proton pump inhibitors, H_2 antagonist
  - ✓ NPO if persistent vomiting
  - ✓ Nutrition:
    - Micro-nutrition: If >2 days without food, begin micro-enteral nutrition 0.2ml/kg/hr slow infusion of electrolyte solution via nasoesophageal tube helps to maintain health of enterocytes
    - Begin nutrition with small frequent highly digestible low fat food +/- enzyme supplementation
  - ✓ Monitor biochemistry (especially albumin) and electrolytes for imbalances/improvements
- **Long term management:**
  - ✓ Low fat highly digestible food +/- pancreatic enzymes (powdered) at 1tsp each meal per 10kg BWt

Exocrine Pancreatic Insufficiency (EPI):

- **Pathophysiology:**
  - ✓ Loss of exocrine pancreatic function, clinical signs when >90% of functionality lost
  - ✓ Pancreatic acinar atrophy is the most common cause in younger dogs, especially German Shepherds and Rough Coated Collies, otherwise secondary to chronic pancreatitis in older dogs
  - ✓ Not common in cats, affected cats commonly have concurrent disease such as inflammatory bowel disease, hyperthyroidism and diabetes mellitus
  - ✓ Reduced digestive enzymes leads to maldigestion
  - ✓ Reduce anti-bacterial secretions leads to small intestinal bacterial overgrowth, this can lead to vitamin B12 malabsorption and other chronic nutrient deficiencies
- **Clinical signs:**
  - ✓ Weight loss despite polyphagia
  - ✓ +/- Vomiting and diarrhoea
  - ✓ Large volumes of greasy grey faeces
- **Diagnostics:**
  - ✓ Trypsin-like immunoreactivity (TLI):
    - Performed when fasted for 12 hours
    - Low levels are diagnostic
  - ✓ Serum cobalamin and folate levels:
    - Should be performed in all cases of suspected EPI
- **Treatment:**
  - ✓ Diet modification:
    - Highly digestible, high quality protein and nutritionally balanced
    - Supplementation with fat soluble vitamins (A, K, E, D) and B12 (cobalamin)
  - ✓ Pancreatic enzymes (powdered) at 1tsp each meal per 10kg BWt
  - ✓ Metronidazole 10mg/kg PO BID for 2 - 3 weeks for small intestinal bacterial overgrowth

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Paralysis Tick

- **This chapter covers:**
  - ✓ Features of tick paralysis by *Ixodes Holocyclus*
  - ✓ Treatment principles and prevention options

- **Features:**
  - ✓ Lag phase: Ticks generally attach for 1 – 2 days before clinical signs
  - ✓ Pathophysiology: Toxin inhibits presynaptic release of acetylcholine at the neuromuscular junction
- **Clinical signs:**
  - ✓ Dysphonia, retching/gagging, drooling
  - ✓ Ascending paresis/paralysis, dilated pupils
  - ✓ Dyspnoea, cyanosis
- **Differential Diagnosis:**
  - ✓ Trauma, spinal cord disease, myasthenia gravis, myopathy, snake envenomation, see **Paresis and Paralysis** in **"Neurological Disease"**

- Stages (1A - 4D):

Gait score:	Respiratory score:
1. Able to **walk** but weak/ataxic	A. **Normal** breathing
2. Able to **stand** but unable to walk	B. **Increased** respiratory rate
3. Unable to stand but can **sit** up	C. **Laboured** breathing
4. Unable to right without aid	D. **Severe** dyspnoea and cyanosis

- **Treatment:**
- Remove ticks: Immediately
- Administer tick anti-toxin:
  - ✓ If any clinical signs are present, then treat immediately
  - ✓ Best administered pre-emptively as the patient will get worse before getting better
  - ✓ Dose: 0.5 - 1ml/kg, up to 2ml/kg in cats slow IV
  - ✓ Premedication:
    - ➢ Feline: Prednisolone sodium succinate 2mg/kg slow IV &/or adrenaline 0.02mg/kg IM 10mins later (but just prior to antitoxin administration)
  - ✓ Beware of acute side effects of cardiovascular, shock, collapse:
    - ➢ If bradycardia and hypotension (poor mucous membrane colour), stop anti-toxin administration and atropine 0.04mg/kg IV
    - ➢ If anaphylactic: Collapse, vomiting, hypotension, stop anti-toxin administer oxygen, IV fluid boluses, adrenalin 0.02mg/kg IV and prednisolone sodium succinate 10mg/kg slow IV
- Symptomatic and supportive care:
  - ✓ General care and monitoring:
    - ➢ Bladder expression, change bedding, eye lubrication, maintenance of hydration (maintenance IV fluids), repeat searches q4 hours
  - ✓ Stress:
    - ➢ Sedation: Combinations of acepromazine 0.01-0.03mg/kg IV, butorphanol 0.1-0.3mg/kg IV/SC
    - ➢ Sedation is important as it improves breathing dynamics and oxygen demand by reducing stress
  - ✓ Gastrointestinal dysfunction:
    - ➢ Nil per os until normal gag reflex
    - ➢ Pharyngeal suctioning
    - ➢ Antiemetic: Metoclopramide 0.5mg/kg IV then CRI in IV fluids, maropitant 1mg/kg SC SID for <5 days
    - ➢ Gastric protectants: Ranitidine 2mg/kg IV BID, esomeprazole 1mg/kg IV SID

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- ✓ Respiratory complications:
  - ➢ Sedation: Combinations of acepromazine 0.01-0.03mg/kg IV, butorphanol 0.1-0.3mg/kg IV/SC
  - ➢ Oxygen therapy: Nasal line 50-100ml/kg/min per line
  - ➢ +/- Ventilation if respiratory effort is unsustainable, hypoxaemic (SPO2 <90%, or PaO2: <60mmHg) despite oxygen therapy or hypercapnic (PCO2: >60mmHg)
  - ➢ Antibiotics if aspiration has occurred
  - ➢ Atropine 0.1ml SC BID to reduce salivation, not with pneumonia
- ✓ Myocardial dysfunction and left-sided congestive heart failure:
  - ➢ Rarely clinical
  - ➢ Diuretics, oxygen

- Tick search and clip:
  - ✓ Very important repeat searches q4 hours
- Tick preventive bath

- Tick control advice:
  - ✓ Daily searching, most important
  - ✓ Clip long haired dogs
  - ✓ Avoid tick habitats, long grass, forest areas
  - ✓ Pharmacological control measures: Should not be relied upon as the only control measure
    - ➢ Tick collars:
      - Amitraz/flumethrin: 2 months **(dogs only)**
      - Deltamethrin: 3 months **(dogs only)**
    - ➢ Frontline spray®: 2 week protection
    - ➢ Frontline PLUS® spot-on: 2 week protection, dogs and cats (off-label use in cats)
    - ➢ Advantix® imidacloprid and permethrin spot-on: 2 week protection **(dogs only)**
    - ➢ Permethrin shampoo/rinses: 1 week protection **(dogs only)**
    - ➢ Natural pyrethrins shampoos: Safe on cats, 3 day protection
    - ➢ Proban® tablets (Cythioate - organophosphate): Given every 48 hours **(dogs only)**
    - ➢ NexGard® tablets (Afoxolaner): Monthly **(dogs only)**
    - ➢ Bravecto® tablets (Fluralaner): 3 monthly tablets **(dogs only)**

- **Discharge:**
  - ✓ Only if can walk, urinate, swallow properly and no retching
  - ✓ Reduced activity for 4 weeks, small risk of sudden cardiac arrest

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Parasitic Disease

This chapter covers:

- ✓ Anti-parasitic medications
- ✓ Parasites of different body systems – features, clinical signs, diagnosis, treatment

Minimum ages for parasite control preparations:

	Minimum ages:	
Product:	**Puppies:**	**Kittens:**
Advantix	8 weeks	-
Advantage	Weaning	Weaning
Advocate	7 weeks	9 weeks
Bravecto	6 months (>1.8kg)	-
Capstar	4 weeks	4 weeks
Dimmitrol	2 weeks	-
Frontline	8 weeks	8 weeks
Frontline spray	2 days	2 days
Killtix	12 weeks	-
Milbemax	2 weeks	-
Nexguard	8 weeks (>2kg)	-
Panoramis	8 weeks	-
Profender	8 weeks	-
Proheart	4 weeks	-
Revolution	6 weeks	6 weeks
Sentinel	2 weeks	-

Prevention of gastrointestinal worms:

- **Check product information as to what they cover/don't cover and use an adjunct as necessary**

Signalment:	**Timing:**
Puppies:	▪ Start at 2 weeks, then every 2 weeks until 12 weeks, then as adults
▪ **Kittens:**	▪ Start at 2 weeks, then every 3 weeks until 12 weeks, then as adults
Adults:	▪ If never been dewormed before or not recently, two doses of a broad spectrum dewormer 2 weeks apart ▪ Monthly spot-on or tablet dewormers ▪ Three monthly broad spectrum dewormers
▪ **Pregnant:** Products safe during pregnancy: ➢ Panacur (fenbendazole) is safe for both cats and dogs Other: Heartguard, Interceptor, Revolution, Frontline Plus	▪ **Bitches:** ➢ Mating ➢ During pregnancy to reduce placental transmission ➢ Third week post-partum to reduce mammary transmission ➢ At weaning kill ones from pups ▪ **Queens:** ➢ Late pregnancy to reduce mammary transmission ➢ Third week post-partum to reduce mammary transmission

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Skin parasites:

Fleas:

- *Ctenocephalides canis* (dog)
- *Ctenocephalides felis* (cat and dog)

Features:	**Clinical signs:**	**Diagnosis:**	**Treatment:**
• Contagious • *Ctenocephalides felis:* Intermediate host of *Dipylidium caninum* **Distribution:** • Head, neck, dorsum, tail, legs	• +/- Anaemia • Flea allergy dermatitis • Dogs: ➢ Pruritus + papules ➢ Alopecia and lichenification ➢ Hyperpigmentation ➢ Secondary pyoderma • Cats: ➢ Miliary dermatitis ➢ Alopecia ➢ Excoriations	• Visualisation • Flea dirt	• Integrated flea control • Adulticidal and insect growth regulator • All pets • Environmental control

- **Flea control:**

Features:	
• Flea eggs are resistant to desiccation and can remain in the environment for months • Life cycle of the flea is temperature dependant, varies from weeks to months: ➢ I.e. faster in warmer months and longer in cooler months • The population of fleas is 95% in the environment, only 5% is on the pet • MUST continue flea control all year around	
Integrated flea control:	
• Flea control is most successful when using all the following steps in combination	
• Flea control on the pet:	• Combination of an effective adulticide and an insect growth regulator • The adulticide will kill the fleas on the pet before eggs can be laid • Insect growth regulator will prevent any eggs that are laid from hatching
• Flea control in the environment:	• Vacuuming: ➢ Facilitates eggs removal from the environment ➢ Must vacuum under furniture, in all the corners and cracks ➢ Vacuuming twice will increase success as it will stimulate the pupae to hatch enabling them to be vacuumed • Washing of pets bedding: ➢ Hot water and dried out in direct sunlight • Spraying of flea habitats in the environment with chemicals: ➢ Dark shady areas of bare dirt (e.g. under a raised house) ➢ Limited efficacy, best to prevent access • Prevention of access to flea habitats: ➢ Fence/corner off

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Lice:
- *Trichodectes canis* (dogs)
- *Linognathus setosus* (cats)

• **Features:** • Contagious • **Distribution:** • Head and dorsum	• **Clinical signs:** • Pruritus • Scaliness	• **Diagnosis:** • Sticky tape impressions	• **Treatment:** • Topical preparation fortnightly for 6 weeks • Treat all animals

Demodex mites:
- *Demodex canis* (dog)
- *Demodex cati* (cat)

• **Features:** • Non-contagious • Localised: ➢ <5 focal lesions • Generalised: ➢ >5 lesions or an entire body area affected ➢ Rarely cats • Juvenile onset (up to 15 months) • Adult onset: ➢ Immunosuppressed • **Distribution:** • *Demodex cati:* ➢ Head and ears	• **Clinical signs:** • Localised: ➢ Small scales non-pruritic regions of alopecia • Generalised: ➢ Diffuse erythematous scaling dermatitis ➢ +/- Secondary pyoderma • Pododemodicosis: ➢ Hyperpigmented and thickened • *Demodex cati:* ➢ Head lesions ➢ Otitis externa	• **Diagnosis:** • Skin scrape and hair pluck • +/- Histopathology for difficult areas e.g. toes	• **Treatment:** • Juvenile / localised: ➢ Spontaneously regress ➢ Topical spot on • Generalised / juvenile or adult: ➢ Amitraz baths, weekly for 6 weeks ➢ Ivermectin 300-600µg/kg PO SID ➢ Beware collies/shelties (start low and titrate) or test for gene mutation • Treat until two negative skin scrapes • Treat concurrent pyoderma See **"Dermatology"** for treatment

Sarcoptid mites:
- *Sarcoptes scabiei* (dogs)
- *Notoedres cati* (cats)

• **Features:** • Contagious • **Distribution:** • *Sarcoptes scabiei:* ➢ Pinnae, elbows ➢ Hocks, vent abdomen • *Notoedres cati (cats):* ➢ Pinnae, face, eyelids, neck ➢ Feet and perineum	• **Clinical signs:** • *Sarcoptes scabiei:* ➢ Red papules, pruritus, alopecia • *Notoedres cati:* ➢ Pruritus and self-trauma ➢ Hyperkeratosis ➢ Miliary dermatitis	• **Diagnosis:** • Deep skin scrape • Response to treatment	• **Treatment:** • *Sarcoptes scabiei:* ➢ Amitraz baths, weekly for 6 weeks ➢ Ivermectin 300µg/kg PO/SC fortnightly • *Notoedres cati:* ➢ Amitraz baths, ½ dose, weekly for 6 weeks ➢ Ivermectin at 200µg/kg once then 200µg/kg PO twice per week for 4 weeks ➢ Treat all animals

Ear mites:

- *Otodectes cynotis* (dogs/cats)

• **Features:** • Contagious • **Distribution:** • Ears and ear canal	• **Features:** • Otitis externa with thick brown crusty discharge	• **Diagnosis:** • Otoscopic exam • Cytology	• **Treatment:** • Insecticidal ear drops • Ivermectin 300µg/kg PO/SC fortnightly

Fur mites:

- *Cheyletiella spp*
- *Lynxacarus radovsky* (cat)

• **Features:** • Contagious	• **Clinical signs:** • Scaly dandruff +/- pruritus	• **Diagnosis:** • Otoscopic exam	• **Treatment:** • Ivermectin 300µg/kg PO/SC fortnightly

Ticks:

- *Ixodes holocyclus*, See **Paralysis Tick** for more information on **Tick Paralysis**
- *Rhipicephalus sanguineus* (brown dog tick), See **Anaemia and Pale Mucous Membranes** for more information on **Babesiosis**

• **Features:** • *Ixodes holocyclus:* ➢ Paralysis tick ➢ East Coast Australia • *Rhipicephalus sanguineus:* ➢ Vector for *Babesia canis* ➢ Worldwide	• **Clinical signs:** • *Ixodes holocyclus:* ➢ Hindlimb paresis ➢ Leads to complete body and respiratory muscle paralysis ➢ Retching and vomiting	• **Diagnosis:** • Visualisation • Clinical signs	• **Treatment:** • Daily tick searches • Topical spot-on fortnightly • Baths (every 3 days) • Monthly to tri-monthly tablets

- *Amblyomma americanum* (Lone Star Tick)
- See **Coagulopathy** for more information on **Ehrlichiosis**

• **Features:** • North America • Vector for: ➢ *Francisella tularensis* (potentially zoonotic) ➢ *Borriela lonestari* ➢ *Erhlichia canis (Erlichiosis)*	• **Clinical signs:** • *Tularemia:* • Cats: ➢ Fever ➢ Lymphadenomegaly, splenomegaly and hepatomegaly ➢ Oral ulcers ➢ Icterus ➢ Panleukopenia • Dogs: ➢ Relatively resistant • *Canine monocytotropic ehrlichiosis:* ➢ Anorexia, weight loss ➢ Bleeding - Petechiae, ecchymosis, epistaxis ➢ Lymphadenomegaly and splenomegaly ➢ +/- Ocular, neurological and cardiovascular signs	• **Diagnosis:** • *Tularemia:* ➢ Serology titres ➢ ELISA ➢ Tissue/exudate C&S is definitive • *Canine monocytotrophic ehrlichiosis:* ➢ Blood smear (look for morulae) ➢ Haematology (thrombocytopenia) ➢ Serology - ELISA ➢ PCR	• **Treatment:** • *Tularaemia:* ➢ 1st: Aminoglycosides ➢ Other Antibiotic classes reportedly less effective. • *Canine monocytotrophic ehrlichiosis:* ➢ Doxycycline (2 to 4 weeks course) ➢ Chloramphenicol if refractory ➢ Imidocarb diproprionate 5mg/kg IM repeated in 2 weeks ➢ Severe cases: Low dose corticosteroids

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- *Dermacentor variabilis*
- See **Coagulopathy** for more information on **Rocky Mountain Spotted Fever**

Features:	**Clinical signs:**	**Diagnosis:**	**Treatment:**
• North America • Vector for: ➢ *Rickettsia rickettsii (Rocky Spotted Mountain Fever)*	• Fever • Oedema/hyperaemia of extremities • Skin lesions • Musculoskeletal inflammation • Bleeding – Petechiae, ecchymosis • Neurological signs	• Haematology (thrombocytopenia) • Serology - ELISA • PCR	• 1st: Doxycycline for at least 7 days • 2nd: Chloramphenicol or Fluoroquinolones

- *Ixodes scapularis*
- See **Coagulopathy** for more information on **Erhlichiosis**

Features:	**Clinical signs:**	**Diagnosis:**	**Treatment:**
• North America • Vector for: ➢ *Borrelia burgdorferi (Lyme disease)* ➢ *Erhlichia canis (Erlichiosis)*	• *Lyme borreliosis:* • Dogs: ➢ Polyarthritis ➢ Fever ➢ Renal disease ➢ Meningitis • Cats: ➢ More resistant, polyarthritis warrants investigation	• *Lyme borreliosis:* ➢ Difficult ➢ Known tick exposure ➢ Clinical signs ➢ Laboratory findings ➢ Response to antibiotic therapy ➢ Serology: False positive common ➢ Immunoblotting ➢ PCR	• *Lyme borreliosis:* ➢ 1st: Doxycycline ➢ 2nd: Amoxicillin, Azithromycin, Ceftriaxone (meningitis). minimum 30 days ➢ Relapse common ➢ Anti-inflammatory for polyarthritis

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Nematodes:

Heartworm:

- *Dirofilaria immitis*

- **Features:**
- 6 months from bite to worms in pulmonary arteries
- Microfilaria do not develop with dog/cat
- *Dog:*
  - RHS congestive heart failure
  - Pulmonary thromboembolism
- *Cats:*
  - Occult infections common
  - Low numbers or single sex infection
  - Interstitial pneumonitis
  - Difficult to diagnose and treat
- **Transmission:**
- Larvae spread by mosquitos
- **Distribution:**
- Pulmonary artery and right atrium and ventricle

- **Clinical signs:**
- Dogs:
  - Weakness, lethargy
  - Heart murmur
  - Pulmonary oedema
  - Ascites
  - Dyspnoea and coughing
  - Haemoptysis
- Cats:
  - Tachypnoea, coughing, haemoptysis
  - Acute death due to hypersensitivity

- **Diagnosis:**
- HW antigen test:
  - Specific, detects females
  - Reduce sensitivity if low numbers or no adults
- HW microfilaria testing:
  - Blood test for microfilaria
  - False negatives if same sex, single worm or immature worms not producing larvae
- Blood smear:
  - Microfilariae on blood smear
  - Not very sensitive
- Radiography:
  - RHS congestive heart failure
  - Enlarged pulmonary arteries

- **Interpretation:**
  - Positive HW antigen test = heartworm disease
    - If with positive microfilaria = adult and microfilaria
    - If with negative microfilaria = adults only
  - Negative HW antigen test = No heartworm disease:
    - If with positive microfilaria = possible transfusion/transplacental infection

- **Treatment:**
- *Prevention:*
  - MUST know if free of heartworm before starting prevention – perform HW antigen test
  - Monthly spot-ons or tablets – kills larvae (2 month reach back)
  - Long acting injection – yearly
  - Daily tablets: Must be given daily (narrow window of effect), if adult HW are present, will cause anaphylaxis
- *Treatment:*
  - Risk of pulmonary thromboembolism
  - Read medicine text for protocol
  - Treatment in cats can be contraindicated
- Corticosteroids (before and after):
  - Prednisolone 0.5mg/kg PO SID
- Adulticidal:
  - Melarsomine (two doses one month apart)
  - Pulmonary thromboembolism highest risk about 1 week post dose
  - Complete rest
- Microfilaricidial:
  - 1 month after adulticidal
  - Milbemycin oxime
- Treatment success:
  - Repeat HW antigen test 6 months post adulticide, should be negative, make sure that there are no larval forms have been missed
  - Repeat microfilaria test 1 month post microfilaricide, should be negative

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Nematodes:

Hookworm:
- *Ancylostoma spp*
- *Uncinaria stenocephala*

Features:	Clinical signs:	Diagnosis:	Treatment:
• Can cause: ➢ Cutaneous larval migrans ➢ Eosinophilic enteritis • **Distribution:** • Small intestinal • Can migrate through body e.g. Lungs • **Transmission:** • Ingestion of larvae or paratenic host • Transmammary / placental / dermal	• Diarrhoea +/- blood • Anaemia • Dermal lesions • Respiratory signs	• Faecal flotation • *Ancylostoma spp*  • *Uncinaria stenocephala* 	• Fenbendazole 50mg/kg PO SID for 3 days • Macrocyclic lactones (ivermectins) • Pyrantel

Roundworm:
- *Toxocara canis* (dogs)
- *Toxocara cati* (cats)
- *Toxascaris leonina* (dogs/cats)

Features:	Clinical signs:	Diagnosis:	Treatment:
• **Distribution:** • Small intestinal • Can cause: ➢ Ocular larval migrans ➢ Visceral larval mirgans • **Transmission:** • Ingestion of egg, larvae or paratenic host • Transmammary / placental • *Toxocara cati:* no transplacental • *Toxascaris leonine:* no transmammary / placental	• Diarrhoea +/- vomiting • Pot belly • Ocular disease • Hepatitis • Pneumonia	• Faecal flotation • Faecal ELISA • *Toxocara canis* (dogs)  • *Toxocara cati* (dogs)  • *Toxascaris leonine* (dogs/cats) 	• Fenbendazole 50mg/kg PO SID for 3 days

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Whipworm:

- *Trichuris vulpis*

• **Features:** • **Distribution:** • Caecum and colon • Can cause: ➢ Organ larval migrans • **Transmission:** • Ingestion of egg, larvae	• **Clinical signs:** • Haemorrhagic diarrhoea • Anorexia • Weight loss	• **Diagnosis:** • Faecal flotation repeated (3 times on 3 separate days) 	• **Treatment:** • Fenbendazole 50mg/kg PO SID for 3 days

Threadworm:

- *Strongyloides stercoralis* (dogs/cats)

• **Features:** • **Distribution:** • Small intestine • **Transmission:** • Ingestion of larvae • Transdermal • Transmammary	• **Clinical signs:** • Diarrhoea • Bronchopneumonia	• **Diagnosis:** • Faecal flotation (Baermann)	• **Treatment:** ➢ Ivermectin 300µg/kg PO/SC repeated

Cestodes:

Tapeworms:

- *Taenia spp.*
- *Echinococcus granulosus*
- *Dipylidium caninum*

• **Features:** • **Distribution:** • Small intestine • **Transmission:** • Ingestion of egg by intermediate host Ingestion of hydatid cysts by definitive host	• **Clinical signs:** • No clinical signs in definitive hosts	• **Diagnosis:** • Faecal flotation • Proglottis in faeces • *Dipylidium caninum* 	• **Treatment:** • Praziquantel • *Dipylidium caninum:* ➢ Flea control

Zipperworm:

- *Spirometra erinacei* (cats)

• **Features:** • **Distribution:** • Small intestine • **Transmission:** • Ingestion of copepods or spargana – eating and drinking	• **Clinical signs:** • No clinical signs	• **Diagnosis:** • Faecal flotation • Proglottis in faeces	• **Treatment:** • Praziquantel at 4 x standard dose

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Protozoa:

Giardia:

- *Giardia duodenalis* (dogs/cats)

Features:	Clinical signs:	Diagnosis:	Treatment:
Distribution: • Small intestinal **Transmission:** • Ingestion of oocysts	• Diarrhoea • Vomiting • +/- Blood	• Faecal analysis – identification of organisms • Faecal ELISA	• Fenbendazole 50mg/kg PO SID for 3 days • Repeat ELISA 10 days post treatment

Coccidia:

- *Isospora canis* and *felis*
- *Cryptosporidium spp.*

Features:	Clinical signs:	Diagnosis:	Treatment:
• *Isospora canis* & *felis:* ➢ Large oocysts 30-50µm ➢ Problem in catteries ➢ +/- Pulmonary migration • *Cryptosporidium spp.:* ➢ Small 5µm ➢ Can increase in numbers secondary to other diseases and immunosuppression **Distribution:** • Small intestinal **Transmission:** • Ingestion of oocysts	• *Isospora canis* & *felis:* ➢ Vomiting, diarrhoea, +/- blood • *Cryptosporidium spp.:* ➢ Asymptomatic ➢ Watery diarrhoea	• *Isospora canis* & *felis:* ➢ Faecal flotation (oocysts) • *Cryptosporidium spp.:* ➢ May need to send away • *Isospora canis (oocysts)*  • *Isospora felis(oocysts)* 	• *Isospora canis* & *felis:* ➢ Trimethoprim-sulpha 20-30mg/kg/day PO ➢ Or Tylosin 10mg/kg PO BID for 2 weeks ➢ Or Toltrazuril 20mg/kg PO SID for 2 days • General hygiene and reduce over crowding • *Cryptosporidium spp.:* ➢ No definitive ➢ treatment

Respiratory parasites:

Nematodes:

Lungworm:

- *Anglostrongylus vasorum*
- *Aelurostrongylus abstrusus* (Feline lungworm)

Features:	Clinical signs:	Diagnosis:	Treatment:
Distribution: • *Anglostrongylus vasorum:* ➢ Bifurcation of trachea • *Aelurostrongylus abstrusus:* ➢ Pulmonary parenchyma **Transmission:** • Ingestion of snail/slug	• *Anglostrongylus vasorum:* ➢ Asymptomatic • *Aelurostrongylus abstrusus:* ➢ Usually asymptomatic ➢ Cough, sneezing ➢ Crackles and wheezes	• *Anglostrongylus vasorum:* ➢ Transtracheal wash ➢ Endoscopy • *Aelurostrongylus abstrusus:* ➢ Faecal flotation (Baermann) ➢ Larvae shed in faeces	• *Anglostrongylus vasorum:* ➢ Ivermectin 300µg/kg PO/SC fortnightly ➢ Fenbendazole 50mg/kg PO SID for 3 days • *Aelurostrongylus abstrusus:* ➢ Ivermectin 300µg/kg PO/SC for 5 days ➢ Fenbendazole 50mg/kg PO SID for 3 days

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Neurological system parasites:

Nematodes:			
Rat lungworm: • *Angiostrongylus cantonensis*			
• **Features:** • Can cause eosinophilic meningitis • **Distribution:** • Small intestinal • Can migrates through body • **Transmission:** • Ingestion of snails/slugs	• **Clinical signs:** • Diarrhoea • Ascites • Neurological signs	• **Diagnosis:** • Faecal flotation	• **Treatment:** • No anthelmintic • Corticosteroids
PROTOZOAN: • *Toxoplasma gondii* **(see below)** • *Neospora caninum* **(see below)**			

- **Toxoplasma:**
- **Features:**
  - ✓ Intracellular protozoan parasite
  - ✓ Cats are definitive host but can infect dogs as well
  - ✓ Infection is via:
    - ➢ Ingestion of cysts from infected animals (most important)
    - ➢ Ingestion of oocysts from faecal contaminated soil/food
    - ➢ Transplacental (public health concerns)
  - ✓ Rapid replication in the gastrointestinal tract leads to dissemination of the parasite around the body into any body tissue where it replicates intracellularly
  - ✓ Ultimately destruction of the cells occurs if this is severe enough then clinical signs become apparent
  - ✓ Typically, immune response limits this and no clinical signs are seen
  - ✓ Therefore, clinical signs are rarely seen and they depend on which organ is affected
  - ✓ Immunosuppressive diseases can exacerbate the infection e.g. FIV, FeLV, FIP
- **Clinical signs:**
  - ✓ Rarely seen, but usually in cats
  - ✓ Vary according to organ systemic infected
  - ✓ Can vary with different age groups:
    - ➢ Young cats:
      - See pulmonary and hepatic disease
    - ➢ Older cats:
      - Due to activation of tissue cysts due to immunosuppression
      - See neurological or ocular signs
  - ✓ Nonspecific signs include:
    - ➢ Anorexia, lethargy, pyrexia
  - ✓ Ocular signs:
    - ➢ Most common clinical sign seen
    - ➢ Uveitis and blindness
  - ✓ Respiratory signs:
    - ➢ Dyspnoea due to pneumonia
    - ➢ Target organ in cats

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- ✓ Neurological signs:
  - ➢ Ataxia, seizures, circling, paresis and paralysis due to encephalomyelitis
- ✓ Muscular:
  - ➢ Stiff gait, pain and weakness due to myositis
- ✓ Alimentary:
  - ➢ Vomiting, diarrhoea, jaundice +/- ascites due to hepatitis / cholangiohepatitis / pancreatitis
- ✓ Reproductive:
  - ➢ Still born or fading kittens

- **Diagnostics:**
  - ✓ Combination of clinical signs and serology +/- PCR
  - ✓ Serology:
    - ➢ Assessment of IgM and IgG antibody titres
    - ➢ IgM:
      - Provides most information
      - Elevation of IgM titres along with clinical signs provides strong supportive evidence
      - Clinically IgM titres follow the clinical course of the disease i.e. increase with active infection and reduces with resolution
    - ➢ IgG:
      - Delay in increased means that positive result only indicates infection has occurred could be prior or active
  - ✓ PCR:
    - ➢ Only identifies the organism
    - ➢ Does not indicate active infection
- **Treatment:**
  - ✓ Does not kill tissue cysts
  - ✓ 50% survival rate
  - ✓ Combination of the following medications:
    - ➢ Azithromycin / clarithromycin
    - ➢ Clindamycin 15-20mg/kg PO BID (drug of choice – crosses blood brain barrier)
- **Prevention:**
  - ✓ No raw meats, reduce hunting (mice, birds, insects)
  - ✓ Frequent cleaning of the litter tray
  - ✓ Wash hands regularly, wear gloves when working in the garden
  - ✓ Cover sandboxes
  - ✓ Wash vegetables, cutting boards, cook meat

- **<u>Neospora:</u>**
- **Features:**
  - ✓ Protozoan *Neospora caninum*
  - ✓ Infection is via:
    - ➢ Ingestion of cysts from infected animals (most important)
    - ➢ Ingestion of oocysts from faecal contaminated soil/food
    - ➢ Transplacental
  - ✓ Usually affect dogs especially puppies
  - ✓ Causes an encephalomyelitis but also polyradiculoneuritis and fibrosing polymyositis
- **Clinical signs:**
  - ✓ Multifocal but neuromuscular signs are seen most frequently
  - ✓ See a progressive lower motor neuron paresis and ataxia that starts in the hindlimbs and ascends cranially, leads to paralysis
  - ✓ Eventually leads to hindlimb extensor rigidity, and muscle atrophy and contracture

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- ✓ Various other signs depending on organ system affected - arrhythmias and respiratory signs such as dyspnoea and cough due to pneumonia and alimentary signs

- **Diagnostics:**
  - ✓ Based on combination of clinical signs and diagnostic findings
  - ✓ Biochemistry: Elevated CK and AST
  - ✓ CSF: Mononuclear and neutrophilic pleocytosis
  - ✓ Serology: ELISA and immunofluorescent antibody testing
- **Treatment:**
  - ✓ Poor prognosis especially if severe neurological signs
  - ✓ Clindamycin 15-20mg/kg PO BID (drug of choice)

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Prostatic Disease

- **This chapter covers:**
  - ✓ Common disease affecting the prostate
  - ✓ Different features, clinical signs and diagnostic principles
  - ✓ General treatment principles

- **Pathophysiology**
  - ✓ **Benign prostatic hyperplasia:**
    - ➢ Most common prostatic disorder
    - ➢ Enlargement of the prostate gland under the influence of sex hormones
    - ➢ Typically, in middle aged to older entire males
  - ✓ **Prostatic/Paraprostatic cysts:**
    - ➢ Cystic structures within the prostate gland or adjacent to the prostate (paraprostatic)
    - ➢ Prostatic cysts are typically secondary to benign hyperplasia, but can also be secondary to infections and neoplasia
  - ✓ **Prostatic neoplasia:**
    - ➢ Least common prostatic pathology
    - ➢ Most commonly adenocarcinoma, can get signs of hypercalcaemia
    - ➢ +/- Systemic illness if leads to peritonitis
    - ➢ Mass lesions or pain on caudal abdominal palpation
    - ➢ Poor prognosis
  - ✓ **Prostatitis and prostatic abscessation:**
    - ➢ Inflammation of the prostate gland usually due to infections of the urinary tract
    - ➢ May be secondary to hyperplasia or urinary tract disorders and can lead to abscessation
    - ➢ Typically signs of systemic illness, can get septic peritonitis if it ruptures
    - ➢ **Acute prostatitis:**
      - Dysuria, stranguria, haematuria
      - Pyrexia and signs of systemic illness lethargy
      - +/- Pain on prostate palpation, +/- caudal abdominal pain
      - Enlarged prostate can be asymmetrical
      - +/- Blood or purulent discharge from penis independent of urination
      - +/- Haematuria, bacteriuria, pyuria
    - ➢ **Chronic prostatitis:**
      - Reoccurring pyrexia and UTI
      - Usually not systemically ill
      - +/- Pain on prostate palpation, can be symmetrically enlarged

- **Clinical signs:**
  - ✓ Stranguria, haematuria, urinary incontinence, blood dripping from penis (independent of urination)
  - ✓ Tenesmus, dyschezia, haematochezia
  - ✓ Abdominal pain or caudal abdominal swelling
  - ✓ Pyrexia, inappetance
  - ✓ +/- Perineal hernia secondary to straining
  - ✓ Altered hindlimb gait

- **Differentials:**
  - ✓ Other causes of tenesmus and dyschezia, see **"Rectal and Perianal Disease"**
  - ✓ Other causes of urinary tract disease, see **"Urinary Tract Disease"**

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- **Diagnostics:**
  - ✓ General physical examination:
    - ➢ Abdominal palpation: Lumbosacral and caudal abdominal pain
    - ➢ Rectal examination: Prostatic palpation, enlarged and painful
  - ✓ Urinalysis:
    - ➢ May not be helpful in diagnosis, urinalysis and culture may be negative, despite prostatic disease
  - ✓ Fine needle aspirates: Cytology and culture, leaked fluid can cause peritonitis
  - ✓ Prostatic wash: Cytology and culture
  - ✓ Imagery:
    - ➢ Radiology:
      - Prostatic enlargement, see **"Radiology"**
    - ➢ Ultrasonography:
      - Prostatic disease: Prostatomegaly, cysts, neoplasia, peri-prostatic fluid and inflammation
      - Sublumbar lymph node enlargement
      - Biopsy specimens
  - ✓ Haematology and biochemistry

- **Treatment:**
  - ✓ **Benign prostatic hyperplasia:**
    - ➢ Castration is essential for non-breeding dogs, reduction in prostate size over 4 weeks
    - ➢ Anti-androgens e.g. Delmadinone acetate (Tardak®) can take up to 5 days for an effect
    - ➢ Enema for constipation, faecal softeners
  - ✓ **Prostatitis and prostatic abscessation:**
    - ➢ Antibiotics:
      - Trimethoprim-sulpha 15mg/kg BID and/or enrofloxacin 5mg/kg PO/IV SID
      - Ideally 4 weeks and then stopped based on negative prostatic fluid culture
      - Change based on culture and sensitivity of prostatic fluid
      - Chronic prostatitis:
        - o Antibiotic choice should be based on culture and sensitivity
        - o Long course at least 8 weeks, then repeat culture
    - ➢ Prostatic involution:
      - Castration
      - Anti-androgens e.g. Delmadinone acetate (Tardak®) can take up to 5 days for an effect
    - ➢ If peritonitis or very large abscess:
      - Surgery
      - IV antibiotics and supportive therapy
  - ✓ **Prostatic/Paraprostatic cysts:**
    - ➢ Fine needle drainage but usually reoccurs
    - ➢ Prostatic cyst:
      - Castration may lead to reduction in size of prostatic cysts
      - Surgery if not responding or very large cyst
    - ➢ Paraprostatic cyst:
      - Surgical excision
  - ✓ **Prostatic neoplasia:**
    - ➢ Surgical removal
    - ➢ +/- Chemotherapy

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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 vertical 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

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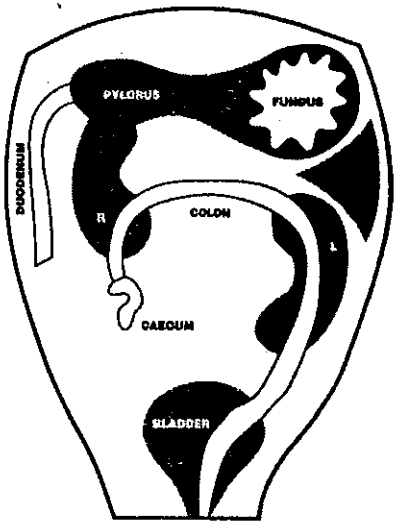
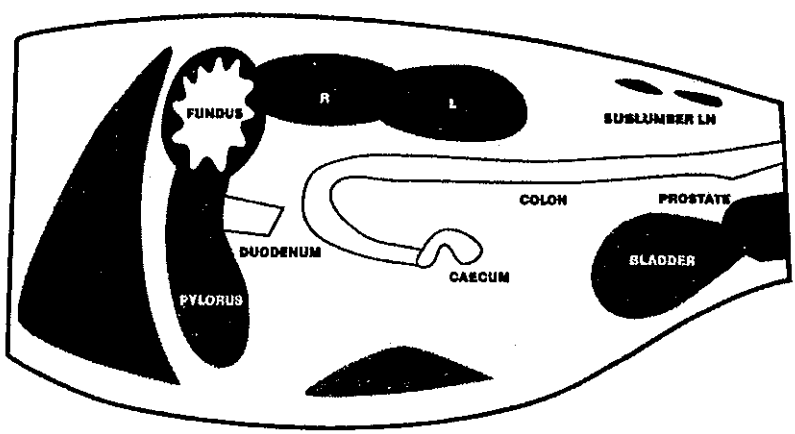


- Abdominal cavity – VD view



- Abdominal cavity – Lateral view

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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

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- **Colon:**
  - ✓ May require contrast studies or endoscopy to diagnose
- Pathology:
  - ✓ Abnormal position:
    - ➢ Ventral: Enlargement of sublumbar lymph node or retroperitoneal space pathology
    - ➢ Dorsal: Enlargement of uterus, prostate, bladder and abdominal masses
  - ✓ Localised dilation:
    - ➢ Foreign body, neoplasia, intussusception, strictures, diverticuli
  - ✓ Generalised dilation:
    - ➢ Constipation
    - ➢ Megacolon: Idiopathic, recurrent constipation, neurological (spinal disease), pelvic narrowing

- **Liver:**
- Assessment of size:
  - ✓ Dependant on serosal detail
  - ✓ Angle of the gastric axis on lateral view: Gastric axis should run parallel to the costal margins
  - ✓ Can protrude about 2 x with of L2 vertebral bodies past the costal margins on lateral view
  - ✓ Displacement of body organs: RHS kidney will be displaced caudally when enlarged
  - ✓ Rounding of liver edges – can indicate hepatomegaly
- Pathology:
  - ✓ Hepatomegaly:
    - ➢ Neoplasia, venous congestion, infiltrative diseases, cysts/abscesses
  - ✓ Microhepatica:
    - ➢ End stage liver disease (cirrhosis), diaphragmatic hernia/trauma, porto-systemic shunts

- **Spleen:**
- Assessment of size:
  - ✓ Varies between patients and can be affected by sedation
  - ✓ Look for displacement of surrounding organs
- Position of the spleen can vary but usually:
  - ✓ VD/DV view: Head of spleen is next to the fundus of the stomach, tail of the spleen can vary from the RHS of the abdomen to along the LHS
  - ✓ Lateral view: Head of spleen is next to the fundus of the stomach dorsally to the kidney (may not see), tail of the spleen anywhere along the ventral abdomen
- Pathology:
  - ✓ Splenomegaly:
    - ➢ Neoplasia, sedation (acepromazine), venous congestion, infiltrative diseases, torsion, inflammatory disease (IMHA)

- **Kidneys:**
- Assessment of size:
  - ✓ Size: 2.5-3 times the length of L2 on VD
  - ✓ Enlargement can cause displacement of body organs
  - ✓ Retroperitoneal space is separate from peritoneal cavity can lose serosal detail separate to peritoneal cavity
  - ✓ Left is lower and may not see the cranial pole of the right as it is within the liver
  - ✓ If unsure about patency of urinary tract perform an excretory urogram

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- Pathology:
  - ✓ Renomegaly:
    - ➢ Neoplasia (unilateral or bilateral), amyloidosis (bilateral), inflammation (uni/bilateral), hydronephrosis (urethral obstruction – unilateral or bilateral), cystic lesions (unilateral or bilateral) or abscess (unilateral)
  - ✓ Small kidneys:
    - ➢ Inflammation (renal disease, pyelonephritis, glomerulonepritits can be unilateral or bilateral), congenital

- **Uterus:**
  - ✓ Features:
    - ➢ Not normally seen
  - ✓ Enlargement:
    - ➢ Displacement of adjacent organs – bladder, colon and small intestines
    - ➢ Tubular soft tissue opacity
- Pathology/Pregnancy:
  - ✓ Enlargement:
    - ➢ Pregnancy: Not see skeletal structures until >40 days
    - ➢ Other: Pyometron, hydrometron, mucometron, cystic endometrial hyperplasia, neoplasia

- **Prostate:**
  - ✓ Positioned at the neck of the bladder, near the cranial border of the pelvis
  - ✓ Assessment of size:
    - ➢ Lateral view:
      - Normal size <75% height between pubis and ventral sacrum or <1.5 x length of L2
      - Displacement into the caudal abdomen (bladder displaced cranially as well), colon displaced dorsally
- Pathology:
  - ✓ Prostatomegaly:
    - ➢ Benign prostatic hyperplasia, prostatitis, neoplasia, cystic structures (can look like the bladder)

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Systematic review of thoracic radiographs:

- **Views:**
  - ✓ Lateral views allow visualisation of the **non-dependent** lung field
- **Differentiation:**
  - ✓ Does not always apply to cats
  - ✓ **Right lateral:** Left and right crura of the diaphragm lie parallel, heart is more upright and oval
  - ✓ **Left lateral:** Left and right crura intersect at the level of the caudal vena cava, heart is more rounded

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
    - o 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:
    - o Unstructured
    - o Structured
    - o Miliary unstructured – small multiple lesions but not fully discrete
  - Bronchial pattern
  - Vascular pattern

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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, fluid overload, 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), coagulopathy
  - ✓ Neoplasia
  - ✓ Atelectasis: Anaesthesia and bronchiectasis

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- **Interstitial pattern:**
- Features:
  - ✓ Unstructured:
    - ➢ Thickened interstitium due to fluid or cellular infiltrates, seen as "diffuse haziness"
    - ➢ Can still vascular patterns unlike alveolar pattern
    - ➢ Differentials:
      - Neoplasia, early oedema, pneumonia, pulmonary fibrosis (normal in older dogs)
      - Expiratory radiograph
  - ✓ Structured:
    - ➢ Miliary: Multiple small opacities
      - Differentials:
        - o Physiological mineralisation, end on blood vessels, neoplasia (metastatic)
    - ➢ Nodular: Circumscribed increased opacities >4mm in diameter
      - Differentials:
        - o Neoplasia (metastatic), granuloma (fungal, foreign body), abscess/cysts

- **Cardiac silhouette:**

View:	Dog:	Cat:
Lat:	▪ Width: 3 intercostal spaces (2.5-3.5) ▪ Height: 2/3 depth of thorax	▪ Width: 2 intercostal spaces (cranial 5th to caudal 7th ribs) ▪ Height: 70% of thorax
DV:	▪ Width: 2/3 width of thorax ▪ Length: Between 3rd and 8th ribs (5 intercostal spaces)	▪ Width: Half width of thorax

- **Assessment of cardiac size:**
- **Vertebral heart size:**
  - ✓ Height: Ventral border of the bronchus to the distal aspect of the apex
  - ✓ Width: Width of the heart at its widest point
  - ✓ Add the width and height and start at the cranial aspect of the 4th thoracic vertebra and count the number of vertebrae that it covers:
    - ➢ All breeds: <10.7
    - ➢ Boxers: <13
    - ➢ Labrador: <12
    - ➢ Cavalier king Charles: <11.5
    - ➢ Cats: <8

* see Cardiology References

- **Chamber enlargement:**
- **Note:** Inverted "D" shape in feline patients is not specific of any condition
  - ✓ RHS cardiac enlargement:
    - ➢ Increased sternal contact (LAT), inverted "D" shape (VD)
  - ✓ LHS chamber enlargement:
    - ➢ Taller cardiac silhouette (LAT)
    - ➢ Dorsally displaced trachea (LAT)
  - ✓ Enlarged LHS atrium:
    - ➢ Bulge on the dorsocaudal border (LAT)

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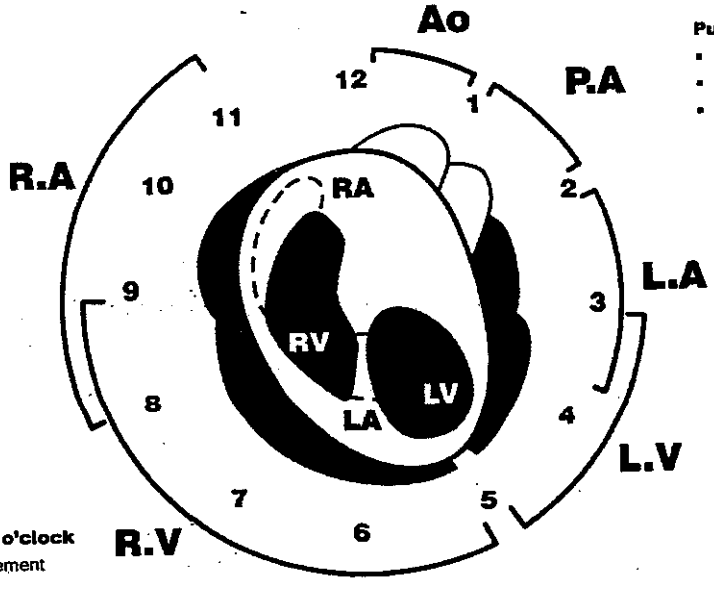
Global enlargement
- Dilated cardiomyopathy
- Pericardial effusion:
  - Congestive heart failure
  - Right atrial tumour
  - Heart base tumour
  - Benign pericarditis
  - Cracked left atrium

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**

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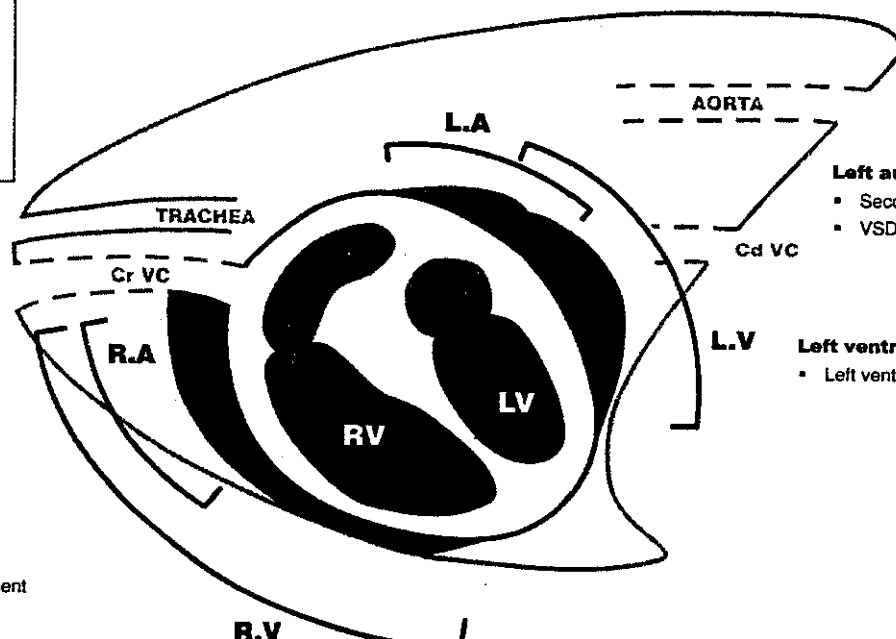
Global enlargement:
- Dilated cardiomyopathy
- Pericardial effusion:
  - Congestive heart failure
  - Right atrial tumour
  - Heart base tumour
  - Benign pericarditis
  - Cracked left atrium



Left auricle:
- Secondary to mitral endocardiosis
- VSD

Right atrium:
- Heartworm
- Pulmonic stenosis
- Right atrial tumour

Left ventricle:
- Left ventricular enlargement

Right ventricle:
- Right ventricular enlargement

- **Lateral view of the thoracic cavity**

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- **Typical changes associated with heart disease:**
  - ✓ Cardiac silhouette or chamber enlargement, **see above:**
    - ➢ LHS enlargement common with valvular disease
    - ➢ Variable chamber enlargement with dilated cardiomyopathy
    - ➢ Globular heart with loss of chamber outline with pericardial effusion
  - ✓ Elevation of the trachea and compression of the main stem bronchi
  - ✓ +/- Congestion of pulmonary veins and interstitial/alveolar pattern with LHS congestive heart failure
  - ✓ +/- Hepatomegaly and ascites with RHS congestive heart failure

- **Other thoracic organs:**
- **Trachea:**
- Narrowing:
  - ✓ Generalised:
    - ➢ Trachea hypoplasia: Congenital, narrow along it entire length
    - ➢ Tracheal collapse: Can be localised or generalised
  - ✓ Localised:
    - ➢ Strictures: ET tube, tracheal surgery
    - ➢ Mass lesions: Neoplasia, granulomas (parasitic)
  - ✓ Thickening of the dorsal tracheal membrane: Tracheal collapse, inflammation, coagulopathies
- Mineralisation:
  - ✓ Older dog, hyperadrenocorticism

- **Oesophagus:**
- Signs of oesophageal pathology:
  - ✓ Gas or fluid filled lumen, foreign body
  - ✓ Trachea displacement: Ventral and to the right
  - ✓ Tracheal band sign: Outline of the trachea due to negative contrast behind trachea
  - ✓ Signs associated with oesophageal pathology:
    - ➢ Alveolar or interstitial lung patterns: Secondary to aspiration
    - ➢ Pneumomediastinum/thorax and pleural effusion: Perforation of the oesophagus
- Pathology:
  - ✓ Generalised dilation:
    - ➢ Megaoesophagus: Secondary to many disease processes, see **"Vomiting"** for list of differentials
  - ✓ Localised dilation:
    - ➢ Internal pathology:
      - Foreign body, strictures, intussusception (gastro-oesophageal), intraoesophageal masses, oesophageal fistulas
    - ➢ External pathology: Compression and obstruction due to extraoesophageal pathology
      - Vascular ring abnormalities, intrathoracic masses

- **Pleural space:**
- Pneumothorax:
  - ✓ Features:
    - ➢ Retraction of lungs and pulmonary structures from the chest wall with negative opacity
    - ➢ Cardiac silhouette elevation (LAT)
  - ✓ Differentials:
    - ➢ Trauma (open or closed pneumothorax)
    - ➢ Pnuemomediastium
    - ➢ Tracheal or pulmonary bullae rupture

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- Pleural effusion:
  - ✓ Features:
    - ➢ Retraction of lungs from chest wall with fluid opacity
    - ➢ Leafing of lung lobes with interlobar fissures
  - ✓ Differentials:
    - ➢ Inflammatory (neoplasia, infections), chyle, haemorrhagic, transudate

- **Diaphragm:**
- Diaphragmatic hernias:
  - ✓ Features:
    - ➢ Inability to completely outline diaphragm
    - ➢ Presence of abdominal organs within thoracic cavity
    - ➢ Displacement of thoracic and abdominal organs
  - ✓ Diagnosis:
    - ➢ Radiographs +/- contrast studies:
      - Inject water soluble contrast into the peritoneal cavity and repeat radiographs in 20 minutes
      - Other contrast studies e.g. oesophageal
    - ➢ Ultrasound
- Diaphragm paralysis:
  - ✓ Features:
    - ➢ Inability to inflate lungs, can be unilateral
    - ➢ Flattened diaphragm

- **Mediastinum:**
- Pneumomediastinum:
  - ✓ Features:
    - ➢ Enhanced visualisation of mediastinal organs (trachea, oesophagus, aorta and cranial vena cava)
    - ➢ Can lead to pneumothorax (but not reverse) and subcutaneous emphysema
  - ✓ Differentials:
    - ➢ Trauma: Neck, tracheal rupture, thoracic
    - ➢ Oesophageal perforation
    - ➢ Pneumoretroperitoneum
- Enlargement or mass lesions:
  - ✓ Features and differentials:
    - ➢ Cranial mediastinum:
      - Dorsal or lateral displacement of the trachea
      - Increased width when > 2 x width of thoracic vertebral body
      - Neoplasia (thymic or lymphomas), pulmonary inflammation, abscessation, haemorrhage
    - ➢ Sternal:
      - Soft tissue enlargement on the dorsal aspect of the second sternabrae
      - Thymic neoplasia, peritoneal pathology causing enlargement (sternal LN drains the abdomen)
    - ➢ Tracheobronchial:
      - Soft tissues enlargement over the heart base at bifurcation of trachea into the main stem bronchi
      - Lymphadenopathy, oesophageal pathology, pulmonary inflammatory

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Skeletal system:

- **Vertebral column:**
- Beware of parallax: Artificial narrowing of disc spaces due to angle of x-ray beam

- **Intervertebral disc space pathology:**
  - ✓ Features:
    - ➢ Narrowed intervertebral disc space, could indicate prolapsed disc
    - ➢ Mineralisation of disc may incidental
  - ✓ See **"Skeletal Disease"** for more information on the following:
    - ➢ Cervical disc disease
    - ➢ Caudal cervical spondylomyelopathy (= Wobbler syndrome)
      - Narrowing or wedging of the IVD space, vertebral tipping C4-C7
      - Ventral spondylosis is present likely to be clinically relevant if present with clinical signs (but require further diagnostics)
    - ➢ Thoracolumbar disc disease
    - ➢ Cauda equine syndrome

- **Spondylosis:**
  - ✓ Features:
    - ➢ Bony projections extending between adjacent vertebrae due to osteophyte formation
    - ➢ Ventrally or laterally along the vertebral column
    - ➢ Normal in middle aged to older animals
    - ➢ Could be clinically significant in the caudal cervical vertebrae and lumbosacral region, may indicate instability, could be an indicator or wobbler and cauda equine syndromes

- **Discospondylitis:**
  - ✓ Features:
    - ➢ Narrowing of the intervertebral disc space, but may appear wider initially
    - ➢ Loss of outline of the adjacent vertebral end plates, due to a mix of lysis of the end places and sclerosis of surrounding bone
  - ✓ See **"Skeletal Disease"** for more information:

- **Specific axial conditions:**
- **Osteoarthritis:**
  - ✓ Features:
    - ➢ Increased radiopacity of the joint due to inflammatory alterations to the synovial fluid
    - ➢ Variation in the width of the joint space (wider/thinner)
    - ➢ Osteophyte formation on perichondral enthesophyte formation of non-weight-bearing surfaces
    - ➢ Alterations in subchondral structure and opacity
    - ➢ Abnormal mineralisation of articular soft tissues

- **Hip dysplasia and aseptic necrosis of the femoral head:**
  - ✓ See **"Skeletal Disease"** for more information:
  - ✓ Features:
    - ➢ Lack of head congruence
    - ➢ Lack of parallelism between head and acetabula and squaring of the femoral head
    - ➢ Thickening of the neck of the femur
    - ➢ Secondary arthritic changes

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- **Osteochondrosis dissecans (OCD):**
  - ✓ Common locations and features:
    - ➢ Shoulder: Flattening or lucent deficit of the caudal aspect of the humeral head (lateral view)
    - ➢ Elbow: Radiolucent defect in the medial humeral condyle
    - ➢ Stifle: Flattening or lucent defect in the lateral femoral condyle on the cranial caudal view
    - ➢ Tarsus: Cartilage flap or lucent defect in the medial ridge of the trochlear of the tarsal bone

- **Elbow dysplasia:**
  - ✓ See **"Skeletal Disease"** for more information:
- Pathophysiology:
  - ✓ **Osteochondrosis dissecans of the medial humeral condyle:**
    - ➢ Flattening or lucent deficit of the caudal aspect of the humeral head (lateral view)
    - ➢ Radiolucent defect on the medial humeral condyle
    - ➢ Secondary osteoarthritic change
  - ✓ **Ununited anconeal process (UAP):**
    - ➢ Separation between anconeus and ulna +/- osteoarthritic changes
  - ✓ **Fragmented medial coronoid process (FCP):**
    - ➢ Diagnosis of exclusion, no UAP or OCD
    - ➢ +/- Secondary osteoarthritic changes (associated with the medial coronoid process and anconeal process), +/- sclerosis of the trochlear notch, +/- abnormal contour or lack of visualization of the medial coronoid process
- Radiographs views:
  - ✓ Both elbows
  - ✓ Three standard views:
    - ➢ Relaxed mediolateral view with an inside angle of approximately 120°
    - ➢ Flexed mediolateral view with an inside angle of 45°
    - ➢ Craniocaudal view
  - ✓ Other views:
    - ➢ Craniocaudal view with 15° pronation (highlights medial coronoid process)
    - ➢ 90° flexed lateral, best for assessing elbow incongruity

- **<u>Miscellaneous skeletal conditions:</u>**
  - ✓ **Periosteal proliferation:** Healing fracture
  - ✓ Osteomyelitis – **see below**
  - ✓ Neoplasia
  - ✓ Panosteitis:
    - ➢ Young large breed dogs, progressive increased radiodensity of the medulla of long bones
  - ✓ Hypertrophic osteodystrophy:
    - ➢ Immune medicated vasculitis that affects the metaphysis of the radius and ulna of young large breed puppies
  - ✓ Hypertrophic osteopathy:
    - ➢ Middle aged, usually secondary to intrathoracic/intraabdominal disease, affects the diaphysis of long bones typically begins in the phalanges and extends cranially
  - ✓ Nutritional secondary hyperparathyroidism:
    - ➢ Deficiency of calcium and vitamin D
  - ✓ Osteophytes

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- **Lytic bone lesions:**
  - ✓ Healing fractures
  - ✓ Osteomyelitis:
    - ➢ Infectious process leading to causes a mixed area of lysis and sclerosis, sometimes with callus formation
    - ➢ Fungal: German Shepherds commonly due to aspergillosis
    - ➢ Secondary to surgery or open fractures
  - ✓ Neoplastic bone disease:
    - ➢ Either primary (most common) or secondary seen as focal lesions of lysis and usually periosteal proliferation
    - ➢ Osteosarcoma: Towards to stifle, away from the elbow

- **Classification of fractures:**
  - ✓ According to type of fracture:
    - ➢ Pathological/non-pathological:
      - Pathological fractures are associated with regions of bone weakness – i.e. Lysis secondary to neoplasia or nutrition
  - ✓ According to stability stable/unstable
  - ✓ According to accompanying wound:
    - ➢ Closed fracture: If there is associated skin trauma even if not full thickness then manage as an open fracture
    - ➢ Open fracture
  - ✓ According to the extent of bone damage:
    - ➢ Complete fractures, incomplete
  - ✓ According to the direction of the fracture:
    - ➢ Transverse, oblique, spiral, comminuted
  - ✓ According to the location of the fracture:
    - ➢ Diaphyseal: Proximal, midshaft and distal thirds
    - ➢ Metaphyseal
    - ➢ Epiphyseal
    - ➢ Physeal fracturé: Possible premature growth place closure and bone growth deformity
    - ➢ Salter–Harris classification of physeal fractures
  - ✓ According to fracture displacement:
    - ➢ Displaced and non-displaced

- **Salter–Harris classification of physeal fractures:**
  - ✓ Type 1: Occurs along physis
  - ✓ Type 2: Involves the physis and a small corner of metaphysis attached
  - ✓ Type 3: Involves the physis then through epiphysis and into the joint
  - ✓ Type 4: Involves the physis with both an epiphysis and metaphyseal fragment
  - ✓ Type 5: No displacement but with growth plate is crushed (seen as narrowing)

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Contrast radiography:

- **Gastrointestinal contrast study:**
- Preparation:
  - ✓ No food for 24 hours, water is ok
  - ✓ +/- Enema 2-4 hours before
  - ✓ Avoid sedative drugs or drugs that affect motility
- Contrast medium:
  - ✓ Barium sulphate suspension at 6-12 ml/kg for physiological distension
  - ✓ Or iodine based preparations if concerned about perforation or may need to go to surgery
- Timing of radiographs:
  - ✓ Lateral and VD projections:

Dogs:	Cats:
• Immediate – stomach • 20 minutes – stomach and duodenum • 35 minutes – stomach, duodenum & jejunum • 4 hours – stomach and small intestine • 5 hours – small intestine and colon • 24 hours – colon, abnormal residual	• Immediate – stomach • 15 minutes – stomach and duodenum • 35 minutes – all small intestine • 1 hours – small intestine and colon
• Gastric outflow obstruction: >60 minutes • Small intestinal obstruction: >5 hours	• Gastric outflow obstruction: >60 minutes • Small intestinal obstruction: >3 hours

- **Excretory urogram:**
- Ideal preparation:
  - ✓ No enable complete visualisation of the urinary tract
  - ✓ +/- No food for 24 hours, water is ok
  - ✓ +/- Enema 4 hours before
- Indications:
  - ✓ Determine the patency of the urinary tract – kidneys, ureters, bladder, urethra
  - ✓ Evaluation of renal pathology
- How:
  - ✓ Intravenous injection of water soluble iodine contrast medium at a dose of 850mg of iodine per kg:
    - ➢ Administer IV as a bolus
  - ✓ Radiograph:
    - ➢ Ensure factor settings are optimal before starting, radiographs are taken in VD
    - ➢ Immediately, 2 minutes, 5 minutes, 10 minutes (take a LAT at this stage as well), 15 minutes

- **Cystography:**
- Ideal preparation:
  - ✓ No enable complete visualisation of the urinary tract
  - ✓ +/- No food for 24 hours, water is ok
  - ✓ +/- Enema 2-4 hours before
- Indications:
  - ✓ Assessment of bladder pathology – neoplasia, cystoliths
- How:
  - ✓ Place a urinary catheter and empty the bladder
  - ✓ Types:
    - ➢ Positive contrast:
      - Administer 3-5ml of water soluble iodine contrast medium
    - ➢ Double contrast:

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- Infuse 1ml/5kg up to 5mls for large dogs of water soluble iodine contrast medium
- Move the patient around or manipulate the bladder to coat the bladder wall
- Infuse 5-10ml/kg of room air or until bladder feels firm

✓ Radiograph:
  - ➢ Immediately, combination of VD, LAT and oblique views

- **Retrograde urethrography (males) / Vaginourethrography (females):**
- Ideal preparation:
  - ✓ +/- No food for 24 hours, water is ok
  - ✓ +/- Enema 2-4 hours before
- Indications:
  - ✓ Assessment for urethral pathology – neoplasia, calculi, structures
- How:
  - ✓ Catheterise penis/vulva distal to the site in question
  - ✓ Hold the prepuce/vulva closed to create a seal rather than inflate the bulb
  - ✓ Infuse 1ml/kg of saline diluted water-soluble iodine contrast material:
    - ➢ Aim for a final concentration of 200mg of iodine per ml
  - ✓ Radiograph:
    - ➢ Immediately combination of VD, LAT and oblique views

Radiology: Evaluating Radiograph Exposure

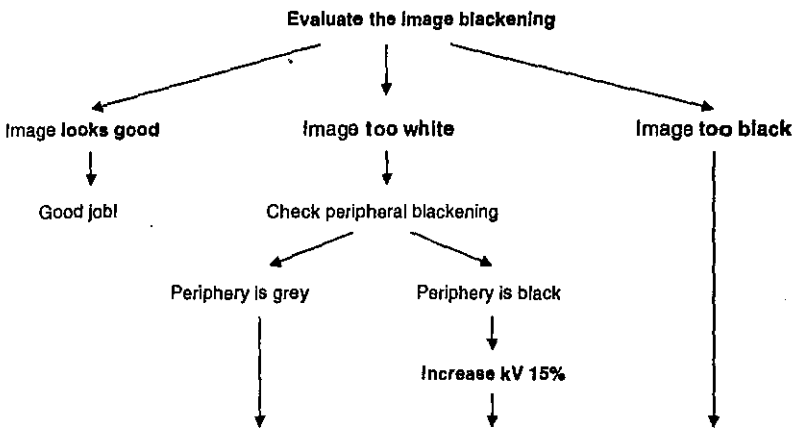

Table 1: Film is too light

- ***Common causes***
  - Insufficient technique: **Double the mAs**
  - Used wrong technique chart
  - Measured incorrectly
  - X-ray tube height is too high
  - X-ray tube not aligned with grid
- ***Less common causes: Processor problems***
  - Developer exhausted
  - Developer diluted
  - Inadequate developer replenishment
  - Developer temperature too low
  - Processor timer malfunction
- ***Rare causes***
  - X-ray machine miscalibration
  - X-ray tube failure
  - X-ray machine timer malfunction

Table 2: Film is too black

- ***Common causes:***
  - Excessive technique:
    - **↓ mAs by 50%**
    - **Decrease kVP by 15%**
  - Double exposure time
  - X-ray tube height is too low
- ***Less common causes: Processor problems***
  - Developer too strong
  - Developer temperature too high
  - Processor timer malfunction
- ***Less common causes: Darkroom problems***
  - Light fog
  - Safety light malfunction
  - X-ray machine time malfunction
  - X-ray machine miscalibration

Factor:	Adjustment:
▪ Emaciated animals	▪ ½ mAs
▪ Juveniles	▪ ½ mAs
▪ Pleural effusion	▪ ↑ by 50 to 100% mAs
▪ Ascites	▪ ↑ by 50 to 100% mAs
▪ Obesity	▪ Double mAs
▪ Plaster casts	▪ Double mAs

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Rectal and Perineal Disease

- **This chapter covers:**
  - ✓ Common disease affecting rectum and perineal area
  - ✓ Clinical signs, common differentials and diagnostic principles
  - ✓ General treatment principles

- **Clinical signs:**
  - ✓ Straining to defecate - tenesmus
  - ✓ Difficulty defecating - dyschezia
  - ✓ Licking and rubbing perineal region
  - ✓ Perineal swellings

- **Differentials:**
  - ✓ Anal gland disorders:
    - ➢ Infection (sacculitis)
    - ➢ Abscessation
    - ➢ Neoplasia (adenocarcinoma)
  - ✓ Constipation: See **"Constipation and Tenesmus"**
  - ✓ Pelvic canal narrowing:
    - ➢ Fracture or neoplasia
  - ✓ Prostatic disease: See **"Prostatic Disease"**
    - ➢ Prostatomegaly, prostatitis
  - ✓ Perineal hernia
  - ✓ Rectal strictures and polyps
  - ✓ Perianal gland adenomas

- **Diagnostics:**
  - ✓ General physical examination
  - ✓ Abdominal palpation:
    - ➢ Constipation, lumbosacral swelling
  - ✓ Rectal examination:
    - ➢ Rectum swellings on walls, polyps, strictures, hernias
    - ➢ Prostatic palpation
    - ➢ Expression of anal gland
  - ✓ Imagery:
    - ➢ Radiology:
      - Pelvic anatomy and mass lesions
      - Prostatic disease
      - Constipation
    - ➢ Ultrasonography:
      - Prostatic disease
      - Sublumbar lymph nodes (adenocarcinoma)
    - ➢ Endoscopy

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Perineal diseases:

- **Anal gland impaction, sacculitis, rupture:**
- **Pathophysiology:**
  - ✓ Exact cause is unknown, likely multifactorial
  - ✓ Dogs, especially small breeds
  - ✓ Chronic and recurrent impaction may predispose to impaction and abscessation
- **Clinical signs:**
  - ✓ Scooting, straining, licking the perineal region
  - ✓ Perineal swelling and inflammation
  - ✓ +/- Discharging sinus
- **Diagnosis:**
  - ✓ Physical and rectal exam and anal gland expression
  - ✓ Expression of contents:
    - ➢ Purulent or blood discharge indicates inflammation and infection
  - ✓ Swollen and painful anal gland +/- discharging sinus
- **Treatment:**
  - ✓ If contents don't indicate infection:
    - ➢ Expression only: May require frequent expression to reduce the risk of abscessation
  - ✓ If contents indicate infection but no discharging sinus:
    - ➢ Expression, flushing and infusion of antibiotic/anti-inflammatory preparation into gland
    - ➢ +/- Systemic antibiotics
    - ➢ May require frequent expression to reduce the risk of abscessation
  - ✓ Obvious swelling that is unable to be expressed:
    - ➢ Fine needle aspirate, lancing and flushing under anaesthesia
    - ➢ Flushing of the duct to ensure patency
    - ➢ E-collar
    - ➢ Systemic antibiotics and non-steroidal anti-inflammatories
  - ✓ Chronic reoccurrence:
    - ➢ +/- Surgical resection

- **Anal gland adenocarcinoma:**
- **Pathophysiology:**
  - ✓ Highly malignant neoplasia arising from anal gland
  - ✓ Spreads early to sublumbar lymph nodes, can have extensive metastatic spread at time of diagnosis
  - ✓ Typically, older female dogs
  - ✓ Guarded prognosis
- **Clinical signs:**
  - ✓ Anal sac neoplasia can be small and non-clinical
  - ✓ +/- Tenesmus due to compression of the colon or urinary tract
  - ✓ Clinical signs of hypercalcaemia due to paraneoplastic syndrome:
    - ➢ Weakness, lethargy, PU/PD, vomiting, +/- diarrhoea, +/- constipation
  - ✓ +/- Palpable mass in dorsocaudal abdomen due to sublumbar lymph node metastasis
- **Diagnosis:**
  - ✓ Palpation of an unexpressible anal sac mass
  - ✓ Fine needle aspirate and cytology, +/- excisional biopsy and histopathology
  - ✓ Assessment of metastatic spread:
    - ➢ Ultrasound of abdomen: Sublumbar lymph nodes, spleen, liver
    - ➢ Radiographs: Chest
  - ✓ Biochemistry: Assess for hypercalcaemia and renal disease

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- **Treatment:**
  - ✓ Palliative chemotherapy +/- surgical removal (but reoccurrence is likely)
  - ✓ Treatment of hypercalcaemia, as can cause acute kidney injury

- **Perineal hernias:**
- **Pathophysiology:**
  - ✓ Develops when the muscles of the pelvis weaken and the rectum loses support and deviates to the side, bladder may prolapse
  - ✓ Typically, older entire male dogs
  - ✓ Possibly secondary to straining from prostatic enlargement or chronic colitis
- **Clinical signs:**
  - ✓ Swelling of the perineal region (unilateral or bilateral)
  - ✓ Dyschezia or constipation
  - ✓ Stranguria if bladder has prolapsed
- **Diagnosis:**
  - ✓ Rectal examination: Palpable deficit in the supporting structure of the lateral rectal wall
  - ✓ Radiology +/- contrast if concerned about bladder prolapsed
- **Treatment:**
  - ✓ Surgical correction

- **Perianal fistulas:**
- **Pathophysiology:**
  - ✓ Multiple chronic ulcerative lesions around the anus, can also have draining sinuses
  - ✓ German Shepherds are predisposed
  - ✓ Unknown cause, possibly immune mediated
  - ✓ Long term control is difficult
- **Clinical signs:**
  - ✓ Perianal ulcerative lesions and draining sinuses
  - ✓ Tenesmus, dyschezia, constipation
- **Diagnosis:**
  - ✓ General physical and visualisation of the lesions
  - ✓ Biopsy and histopathology and culture and sensitivity
- **Treatment:**
  - ✓ Nursing: Regular clipping and cleaning of the area
  - ✓ Medical management:
    - ➢ Immunosuppression:
      - Cyclosporine 5 mg/kg BID, when combined with ketoconazole 5mg/kg BID can lower dose of cyclosporine 0.5mg/kg BID
      - Tacrolimus (0.1%) topical application, not as successful as oral cyclosporine
    - ➢ Antibiotics:
      - Depends on culture and sensitivity
      - Initially broad spectrum: Amoxicillin clavulanic acid 25mg/kg PO BID and metronidazole 10mg/kg PO BID
  - ✓ Dietary management:
    - ➢ Faecal softeners
    - ➢ +/- Hypoallergenic diet – worth a try
  - ✓ Surgery

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- **Perianal gland tumours:**
- **Pathophysiology:**
  - ✓ Typically, adenomas/adenocarcinomas arise from modified sebaceous glands
  - ✓ Adenomas:
    - ➢ Usually older, entire males
  - ✓ Adenocarcinomas:
    - ➢ Typically, older females
    - ➢ Metastasis to sublumbar lymph nodes
- **Clinical signs:**
  - ✓ Adenomas:
    - ➢ Firm circumscribed nodules around the anus
    - ➢ Perianal pruritus and inflammation
  - ✓ Adenocarcinomas:
    - ➢ Invasive thickening and ulceration around the anus
    - ➢ Appear like perianal fistulae
    - ➢ Clinical signs of hypercalcaemia due to paraneoplastic syndrome:
      - Weakness, lethargy, PU/PD, vomiting, +/- diarrhoea, +/- constipation
- **Diagnosis:**
  - ✓ Biopsy: Cytology and histopathology
  - ✓ Adenocarcinomas:
    - ➢ Ultrasound of abdomen: Assess for metastatic spread
    - ➢ Radiographs: Chest to assess for metastatic spread
    - ➢ Biochemistry: Assess for hypercalcaemia and renal disease
- **Treatment:**
  - ✓ Adenomas: Surgical excision combined with castration
  - ✓ Adenocarcinomas: Surgical excision combined with chemotherapy

Rectal diseases:

- **Rectal and anal prolapse:**
- **Features:**
  - ✓ Commonly secondary to repeated tenesmus (see differentials in previous pages) and recurrent diarrhoea
  - ✓ Most commonly seen in dogs with gastrointestinal parasites
- **Clinical signs:**
  - ✓ Tenesmus and dyschezia
  - ✓ Haematochezia
  - ✓ Everted anus or prolapsed rectal mucosa seen as a red tube mass
- **Diagnosis:**
  - ✓ Findings on physical and rectal exam
  - ✓ Faecal analysis: Flotation, smears, *Giardia* ELIZA
  - ✓ Radiography:
    - ➢ Assess for causes of straining: e.g. Prostatic disease
    - ➢ Concurrent constipation
- **Treatment:**
  - ✓ Treatment of the underlying cause e.g. parasites, prostate disease, diarrhoea
  - ✓ Reduction of the prolapsed tissue:
    - ➢ Can use mannitol and glucose solutions to reduce swellings
    - ➢ Reduce the prolapsed gently with the use of lubricants
    - ➢ Placement of the purse-string suture that is wide enough to pass soft faeces, keep in place for up to a week

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- Administration of lactulose 1ml/4kg TID to soften and aid in the passage of faeces, for up to a month

✓ Surgical removal if the tissue is non-viable:
- Last resort

- **Other rectal diseases:**

Rectal polyps:			
Features: ▪ Usually benign growths in the rectum ▪ Vary in size and shape	**Clinical signs:** ▪ Tenesmus ▪ Dyschezia ▪ Haematochezia	**Diagnosis:** ▪ Physical and rectal exam ▪ Speculum examination of the rectum ▪ Endoscope ▪ Radiography +/- contrast ▪ +/- Biopsy and cytology and histopathology	**Treatment:** ▪ Surgical resection
Rectal adenocarcinomas:			
Features: ▪ Narrowing of the rectal lumen	**Clinical signs:** ▪ As above	**Diagnosis:** ▪ As above	**Treatment:** ▪ Surgical resection ▪ Piroxicam ▪ Faecal softeners
Rectal stricture:			
Features: ▪ Narrowing of the rectal lumen ▪ Secondary to inflammation, neoplastic, fungal disease	**Clinical signs:** ▪ As above	**Diagnosis:** ▪ As above	**Treatment:** ▪ Faecal softeners ▪ Treatment for underlying disease ▪ Balloon dilation ▪ +/- Surgical resection

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Renal Disease - Acute and Chronic

- **This chapter covers:**
  - ✓ Principles of renal disease
  - ✓ Features of acute and chronic renal failure
  - ✓ Causes, features, clinical signs and diagnostic principles
  - ✓ General treatment and management principles

- **Renal disease:** Lesions in the kidneys
- **Stages of renal dysfunction:**
  - ✓ **Renal insufficiency:**
    - ➢ >70% loss of functional mass = Loss of concentrating ability leading to dilute urine
  - ✓ **Renal failure:**
    - ➢ >80% loss of functional mass = Loss of ability to excrete wastes leading to azotaemia and dilute urine in a normally hydrated animal
- **Azotaemia:** Elevated concentrations of BUN and creatinine
  - ✓ Can be pre-renal, renal (acute or chronic), post renal in origin
- **Uraemia:** Combination of the presence of azotaemia and clinical signs of illness
  - ✓ Clinical signs: Lethargy, inappetance, weight loss, vomiting, diarrhoea, stomatitis

- **Azotaemia:**

Pre-renal: No direct parenchymal damage – unless prolonged	
Causes: • Due to reduced renal perfusion (moderate to severe elevations): ➢ Hypovolemia – shock, dehydration, haemorrhage ➢ Cardiogenic – reduced perfusion • Due to increased protein catabolism (mild elevations): ➢ Necrosis, starvation, infection, fever	**Laboratory changes:** • ↑ BUN and creatinine • Oliguria • +/- ↑ PCV and TP • USG > 1.030 in Dogs or >1.035 in Cats • +/- ↑ Amylase • Rapid correction of azotaemia following fluid therapy
Renal: Direct parenchymal damage Acute or chronic (See table below for differentiation)	
Causes: • Tubular disease: ➢ Toxins: Common • NSAID's, ACE inhibitors, *Lilium spp.*, antifungals, gentamicin, ethylene glycol, grapes, pigmenturia, hypercalcaemia (>3.7mmol/L) ➢ Renal ischemia: Common • Hypovolemia, hypotension, heart disease • Interstitial disease: ➢ Pyelonephritis (bacterial e.g. *Leptospirosis*) • Glomerular disease (see ↑ proteinuria): ➢ Immune complex deposition due to infections, immune mediated disease, severe inflammation	**Laboratory changes:** • +/- ↑ BUN and creatinine • Polyuria/Oliguria • +/- Metabolic acidosis (↓ HCO_3 production and ↓ H+ excretion) • ↑ K+ (due to acidosis and ↓ excretion) • ↑ Phos • +/- ↑ Ca+ • USG 1.008 – 1.014 **but can be** < 1.030 in Dogs or <1.035 in Cats • Granular casts = hall mark of acute injury • Proteinuria, calcium oxalate crystals

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Post-renal: Failure to remove urine from the body	
Causes: ▪ Rupture or obstruction or ureters, bladder, urethra ▪ +/- Large painful bladder ▪ +/- Inability to urinate ▪ +/- Abdominal pain ▪ +/- Uroabdomen	**Laboratory changes:** ▪ ↑ BUN and creatinine ▪ +/- ↑ K+, ↓ Na+ ▪ Oliguria, anuria ▪ USG varies ▪ Uroabdomen: [creatinine] and [K+] of abdominal fluid > [creatinine] and [K+] of serum

- **Normal water intake:**
  - ✓ **NOTE:** These may increase depending on activity levels and temperature

	Dogs:	Cats:
Intake - Daily water ingested	<60ml/kg	<50ml/kg (dry food) <10ml/kg (wet food)

- **Acute versus chronic renal failure:**

	Acute kidney injury:	Chronic renal failure:
Clinical signs:	▪ Acute onset anorexia and lethargy, +/- vomiting ▪ +/- Painful kidneys	▪ Chronic anorexia, lethargy, vomiting ▪ +/- Small irregular kidneys
Water intake:	▪ Reduced	▪ Polydipsia
Body condition and weight:	▪ Good body condition ▪ No/minimal weight loss (apart from dehydration losses)	▪ Poor body condition ▪ Chronic weight loss
Urine production:	▪ Oliguria	▪ Polyuria (with secondary polydipsia)
Haematology:	▪ Non-anaemic to increased PCV	▪ +/- Anaemia: Non-regenerative
Biochemistry:	▪ Azotaemia (previously normal) ▪ +/- Hyperkalaemia ▪ Metabolic acidosis (moderate to severe) ▪ Hyperphosphataemia ▪ Hypercalcaemia	▪ Azotaemia (previously high) ▪ +/- Hypokalaemia ▪ Metabolic acidosis (mild) ▪ Hyperphosphataemia
Urinalysis:	▪ USG variable: ➢ Pre-renal: >1.030 ➢ Renal: 1.008 - <1.030 ▪ +/- Inflammation ▪ +/- Pathogens ▪ +/- Casts: Granular casts are a hallmark of acute injury ▪ +/- Calcium oxalate crystals with ethylene glycol	▪ USG: 1.008 - <1.030 ▪ Proteinuria without inflammatory changes

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Acute kidney injury:

- **Features:**
  - ✓ Sudden onset failure of the kidneys – leading to inability to filter the blood and excrete wastes
  - ✓ If the patient has a concurrent disease process that impairs urine concentrating ability (e.g. Hyperadrenocorticism or diabetes insipidus) then they are at risk of acute kidney injury
  - ✓ Acute kidney injury may be reversible
- **Chronic versus acute kidney injury:**
  - ✓ Acute kidney injury should not have non-regenerative anaemia, history of loss of body condition and polyuria/polydipsia
  - ✓ Acute on chronic: Chronic renal disease that has developed signs of uraemia due to conditions that promote dehydration, once resolution of fluid deficits and azotaemia, manage as per chronic renal failure
- **Phases:**
  - ✓ **Oliguric/anuric:** Reduced urine production
  - ✓ **Polyuric:** Excessive urine production due to inability to concentrate – usually during the recovery phase
- **Clinical signs:**
  - ✓ Lethargy, inappetance, vomiting and diarrhoea +/- blood
- **Causes:**
  - ✓ **See table on previous page**
  - ✓ Haemodynamic leading to reduced perfusion (ie. primary pre-renal leading to renal): e.g. hypovolemia, hypotension, heart disease
    - ➢ Compounded by pre-existing polyuric disorder (e.g. Hyperadrenocorticism or diabetes insipidus)
  - ✓ Drugs and toxins: NSAID's, ACE inhibitors, *Lilium spp.*, antifungals, ethylene glycol, grapes, gentamicin
  - ✓ Haemoglobinuria and myoglobinuria
  - ✓ Infections: e.g. *Leptospirosis*, ascending or haematogenous bacterial infections
  - ✓ Hypercalcaemia: Severe >3.7mmol/L
- **Treatment:**
  - ✓ **Treatment of underlying cause:**
    - ➢ Decontamination if toxic
    - ➢ Antibiotics if pyelonephritis
    - ➢ Resolution of obstruction to urine outflow or urinary tract rupture
  - ✓ **Oliguric phase:**
    - ➢ Place urinary catheter
    - ➢ Correct fluid perfusion rapidly, then hydration deficits, acid-base and electrolyte abnormalities:
      - Aim to be normovolemic and normotension within the first hour
      - Restore to normal hydration within 6 hours, if clinically hydrated aim to correct at least 5% dehydration as less than this is clinically undetectable
      - Then continue at 1.5 to 3 x maintenance to promote ongoing diuresis and urine output >1ml/kg/hr
      - Monitor closely for volume overload
    - ➢ Correction of hyperkalaemia: see **"Fluid Therapy"**
    - ➢ Correction of acid-base imbalances: Usually metabolic acidosis, alkalizing fluids +/- bicarbonate therapy see **"Fluid Therapy"**
    - ➢ Monitoring:
      - Blood pressure and urine output
      - Aim for minimum urine production of 1-2ml/kg/hr after correcting perfusion, obtaining normotension and correcting some of the hydration defects
        - o Anuria: <0.25ml/kg/hr, poor prognosis
        - o Absolute oliguria: <0.5-1ml/kg/hr
        - o Polyuria: >2ml/kg/hr – better prognosis

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- Promote diuresis:
  - If inadequate urine output and achieved normovolaemia, normotension, normohydration, trial a 10ml/kg IV crystalloid fluid challenge, if no improvement in urine output then consider osmotic or diuretic therapy
  - Mannitol 0.25-1g/kg over 10 mins, if diuresis then continue as a CRI 1mg/kg/min, total daily dose <2g/kg/day. Do not repeat if no diuresis in 1 hour
    - Do not administer if dehydrated or overhydrated
  - Furosemide:
    - Cat: 1-2mg/kg IV, dog: 1-4mg/kg IV
    - Should see results in 30 minutes, if not then repeat or increase dose
    - Can continue as a CRI 0.2-1mg/kg/hr after diuresis is obtained
- Maintain polyuria until regains normal renal functionality
- Manage systemic signs of uraemia/azotaemia:
  - Gastroprotectants:
    - Sucralfate 0.5-1gm PO TID
    - H2 antagonists: Ranitidine 2mg/kg SC/IV SID, extend dosage interval renally excreted
    - Proton pump inhibitors: Omeprazole 1mg/kg IV/PO SID
  - Vomiting: NPO until subsides
    - Metoclopramide 0.2-0.5mg/kg SC/IV/IM TID
    - Maropitant 1mg/kg SC SID for <5 days

✓ **Polyuric phase:**

- IV fluids to maintain normovolaemia: Monitor urine output and matching in's and out's is the most effective way
- Correction of electrolyte and acid-base imbalance
- Ensure provision of adequate calorie intake (↓ dietary protein, ↓ phosphorous and Na+) – specially formulated renal diet is best but anything is better than nothing, may need to place a feeding tube
- Treatment of underlying disease

Chronic renal failure:

- **Pathophysiology:**
  - ✓ Presence of azotaemia and minimally concentrated urine (<1.030) due to a primary renal pathology that has been long standing. Azotaemia indicates a loss of at least 80% functional capacity
- **Chronic verves acute kidney injury:**
  - ✓ Acute kidney injury should not have non-regenerative anaemia, history of loss of body condition and polyuria/polydipsia
  - ✓ Can have acute exacerbation or chronic renal disease "acute -on-chronic" where pre-renal/renal causes exacerbate pre-existing chronic renal disease
- **History:**
  - ✓ History of polyuria and polydipsia, loss of body condition, vomiting and diarrhoea
- **Clinical signs:**
  - ✓ *Compensated:* Progressive loss of body condition, polyuria and polydipsia
  - ✓ *Decompensated:* Lethargy, inappetance, anaemia of chronic disease, vomiting and diarrhoea (+/- blood), stomatitis, neurological signs
- **Causes:**
  - ✓ Glomerulonephritis
  - ✓ Secondary to acute renal failure
  - ✓ Infectious disease: Bacterial, viral (e.g. FIP in cats), fungal, rickettsial
  - ✓ Metabolic renal disease: Hypercalcaemia, amyloidosis, drugs and toxins (grapes, NSAID's, anti-freeze)
  - ✓ Tubular disease
  - ✓ Neoplasia
  - ✓ Polycystic kidneys and renal dysplasia
  - ✓ Hypotensive or hypoperfused states: Circulatory or vascular disease leading to prolonged reduce renal perfusion

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- **Staging degree of chronic renal failure:**
- Combination of fasted plasma creatinine concentrations, urine specific gravity and degree of proteinuria
- International Renal Interest Society, 2006 Staging of Chronic Kidney Disease. Web Site: www.iris-kidney.com

Stage and severity:	Creatinine:	
	µmol/L	mg/dl
Stage 1:		
Dog:	<125	<1.4
Cat:	<140	<1.6
No clinical signs but minimally concentrated urine, proteinuria		
Mild: Stage 2		
Dog:	125-179	1.4-2
Cat	140-249	1.6-2.8
Moderate: Stage 3		
Dog:	180-439	2.1-5
Cat:	250-439	2.9-5
Severe: Stage 3		
Dog:	>440	>5
Cat:	>400	>5

- **Treatment:**
  - ✓ **Goals:**
    - ➢ Resolve clinical signs of uraemia
    - ➢ Elimination of risk factors e.g. urinary tract infections
    - ➢ Correction of fluid and electrolyte deficits
    - ➢ Maintenance of electrolytes, vitamins and minerals
    - ➢ Maintenance of body condition
    - ➢ Reduce the progression of renal failure
  - ✓ **Diet:**
    - ➢ Supplying sufficient calories to stop protein catabolism
    - ➢ Typically, high fat, low protein but high biological value to minimise excess urea production
    - ➢ Phosphate restriction to reduce the risk of secondary renal hyperparathyroidism, it is associated with progression of renal disease
    - ➢ Sodium restriction to reduce hypertension
    - ➢ Supplementation of omega-3 PUFAs (NOT omega-6) has been shown to have renoprotective anti-inflammatory properties
    - ➢ If hypoalbuminaemia, weight loss and loss of condition then increase protein content of diet
  - ✓ **Secondary renal hyperparathyroidism:**
    - ➢ Hyperphosphataemia due to reduced excretion leads to secondary renal hyperparathyroidism. Elevated parathyroid hormone leads to an increase in calcium
    - ➢ Hypercalcaemia results in renal osteodystrophy, metabolic calcification, dystrophic calcification
    - ➢ Hypercalcaemia is a major factor in the progression of the renal failure due to calcification
    - ➢ Dietary restriction of phosphate:
      - Typically, sufficient in renal diets but may need further supplementation if phosphate levels remain persistently elevated

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- Phosphorous binding therapy:
  - Aluminium hydroxide 100mg/kg/day or calcium carbonate at 30-70 mg/kg/day as needed
  - Ipakitin: Phosphate chelator, only if normocalcaemic and not on calcitriol therapy or calcium supplementation
- Calcitriol therapy (Vitamin D):
  - Aim to reduce parathyroid hormone levels due to renal hyperparathyroidism
  - Begin therapy after resolution of hypercalcaemia and hyperphosphataemia.
  - Dose at 1.5-3µg/kg/day
  - Monitoring is essential:
    - Calcium, phosphate, parathyroid hormone levels (PTH), biochemistry at 2 weeks, 1 month then every 2 months
    - If PTH is persistently high after 1 month of therapy, increase calcitriol dose

✓ **Management of hypertension:**
- Slow down progression of renal disease
- Most common cause of feline hypertension
- Monitor response to anti-hypertensive treatment every week for one month then every 3 months
- Dietary sodium reduction
- Amlodipine:
  - Calcium channel blocker, drug of choice
  - Dose for dogs is 0.1mg/kg/day and dose for cats is 0.7 - 1.2 mg/cat/day
  - Average reduction approximately 40-50mmHg
  - MUST monitor electrolytes as can get hypokalaemia
- ACE inhibitors:
  - E.g. Benazepril 0.5-1.0mg/kg PO SID
  - Only mild reduction in blood pressure approximately 5-15mmHg
  - Can combine with calcium channel blocker treatment (amlodipine)

✓ **Hypokalaemia:**
- Potassium loss is common in cats, can cause myopathy and weakness
- Typically, renal diets have been supplemented with potassium but further supplementation is sometimes required
- Supplemented with potassium gluconate 3-5mEq/day PO SID-BID or potassium citrate

✓ **Metabolic acidosis:**
- Renal diets are usually supplemented with enough bicarbonate or bicarbonate precursors to maintain acid-base balance
- +/- Supplement with sodium bicarbonate at 10mg/kg PO BID or potassium citrate at 50mg/kg PO BID

✓ **Gastrointestinal ulceration and vomiting:**
- Gastric protectants:
  - H2 antagonist e.g. ranitidine 2mg/kg BID-TID
  - Proton pump inhibitors e.g. omeprazole 1mg/kg SID
  - Sucralfate 0.5-1gm/kg PO TID
- Antiemetic:
  - Metoclopramide 0.2-0.5mg/kg TID
  - Maropitant 1mg/kg SID

✓ **Proteinuria:**
- Treatment is recommended where UPC is >0.5
- ACE inhibitor: e.g. Benazepril 0.5-1.0mg/kg PO SID, not if dehydrated or azotaemic
- Omega-3-fatty acid supplementation
- Urinary loss of anti-thrombin III can increase risk of thromboembolism:
  - Aspirin 0.5mg/kg PO SID a platelet inhibitor, can reduce the risk of thromboembolism

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- ✓ **Anaemia of chronic disease:**
  - ➢ Non-regenerative, characteristically a normochromic, normocytic anaemia
  - ➢ +/- Vitamin B12 (cobalamin), worth a try
  - ➢ Blood transfusion but only temporary solution, usually no clinical signs of anaemia due to chronicity

- **Monitoring:**
  - ✓ Body weight
  - ✓ Blood pressure and proteinuria
  - ✓ PCV/TP
  - ✓ Urea and creatinine
  - ✓ Potassium and phosphorus levels

Pyelonephritis:

- **Pathophysiology:**
  - ✓ Inflammation of the renal pelvis and parenchyma due to bacterial infection
  - ✓ Most commonly due to ascending infection rather than haematogenous spread
  - ✓ Usually requires an immunosuppressive disease process or predisposing factors e.g. Ectopic ureters, Cushing's or steroid administration, dilute urine, urine retention, diabetes mellitus, renal failure, uroliths, urinary tract neoplasia
- **Clinical signs:**
  - ✓ Presentation varies
  - ✓ +/- Signs of lower urinary tract inflammation such as dysuria, stranguria, haematuria
  - ✓ Acute:
    - ➢ Systemically ill, lumbar or caudal abdominal pain (kidney pain), pyrexia, vomiting and inappetance
  - ✓ Chronic:
    - ➢ History of polyuria and polydipsia, weight loss, lethargy
- **Diagnostics:**
  - ✓ Normal diagnostics do not rule out pyelonephritis
  - ✓ Biochemistry:
    - ➢ +/- Azotaemia, +/- hyperphosphataemia due to renal failure, metabolic acidosis
  - ✓ Haematology:
    - ➢ +/- Leucocytosis but often normal
  - ✓ Urinalysis:
    - ➢ Absence of pathology on urinalysis does not rule out pyelonephritis
    - ➢ Minimally concentrated or isothenuric urine with bacteriuria, pyuria, haematuria, proteinuria, granular casts
    - ➢ Send sample for culture and sensitivity
  - ✓ Ultrasound:
    - ➢ Hyperechoic renal cortex, increased kidney size, poor corticomedullary distinction, dilated renal pelvis
- **Treatment:**
  - ✓ IV fluids:
    - ➢ Correction of fluid deficits, electrolytes
    - ➢ Resolution of azotaemia
  - ✓ Antibiotics:
    - ➢ IV broad spectrum antibiotics followed with oral antibiotics for up to 8 weeks
    - ➢ Perform a culture and sensitivity after first week to assess response the management, then 1 and 4 weeks after antibiotics to determine resolution
  - ✓ Management of the underlying or predisposing disease

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Reproductive Organ Disease

- **This chapter covers:**
  - ✓ General information on pregnancy duration, modes of detection
  - ✓ Outlines stages of parturition
  - ✓ Pathophysiology of uterine inertia and dystocia and the indications for intervention
  - ✓ Information on pregnancy hypocalcaemia (eclampsia), pyometron, mastitis, post-partum metritis

	Dog:	Cat:
Onset of oestrus:	▪ Small breed dogs and cats: 5-10 months ▪ Large breeds 14-24 months old	▪ 5 – 9 months
Pro-oestrus:	▪ Behavioural: ➢ Attracted to males but will not stand ▪ Physical: ➢ Vulval swelling and vaginal bleeding ▪ Average 9 days (2-25 days)	▪ Behavioural: ➢ Increased affectionate behaviour, vocalising, rolling, tail raising ➢ Males are attracted to them ▪ Physical: ➢ No external physical changes ▪ Average 2 days (2-3 days)
Oestrus:	▪ Behavioural: ➢ Attracted to males and will stand ▪ Physical: ➢ Vulval swollen and thickened ➢ Reduced bleeding – blood to straw coloured ▪ Spontaneous ovulators: ➢ Average 9 days (3 – 20)	▪ Behavioural: ➢ Increased vocalising, rolling, restlessness, tail and back raising ➢ Increased male seeking behaviours (trying to go outside) ➢ Will stand for males ▪ Physical: ➢ No external physical changes ▪ Induced ovulators: ➢ Average 4 days (if mated) ➢ Average 10 days (if not mated)
Gestation period:	▪ 63 days from ovulation ▪ 57 days from day 1 of dioestrous	▪ 65 days +/- 4 days
Interoestrus interval: If not bred	▪ 5 – 10 months	▪ 1 week to 5 months ▪ Cat can bread as early as a week after parturition

- **Detecting pregnancy:**

Method:	Day from ovulation:
▪ Ultrasound	18 - 24
▪ Palpation	21 - 32
▪ Radiographs	>40
▪ Auscultation	>55

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- **Stages of parturition:**
- Stage 1:
  - ✓ Onset of uterine contractions and dilation of the cervix
  - ✓ Changes in behaviour such as restlessness, panting, shivering, hiding
  - ✓ Canine: Approximately 6 to 12 hours
  - ✓ Feline: 6 to 24 hours
- Stage 2:
  - ✓ Release of amniotic fluid, abdominal contractions, delivery of puppies/kittens
  - ✓ Canine: Approximately 3 – 6 hours but up to12 hours
  - ✓ Feline: Usually within 6 hours but can take up to 24 hours
- Stage 3:
  - ✓ Passage of all of the placenta, usually <15 minutes after each puppy/kitten

- **<u>Dystocia:</u>**
- **Pathogenesis:**
  - ✓ Functional dystocia or uterine inertia:
    - ➢ Primary:
      - Lack of uterine contractions at the end of gestation, >24 hours after drop in rectal temperature (<37.2°C)
      - Large litter: Stretching the uterine muscles
      - One or few foetuses
    - ➢ Secondary:
      - Lack of uterine contractions due to difficult/protracted parturition
      - Large litters, females of poor body condition
  - ✓ Obstructive dystocia:
    - ➢ Inability to deliver foetus due to either foetal oversize or maternal pelvic canal narrowing
- **Diagnosis:**
  - ✓ Complete history: Length of gestation, previous dystocia, progression of parturition
  - ✓ Full general physical examination
  - ✓ Palpation of the birth canal
    - ➢ Ferguson reflex via palpation of the dorsal vaginal vault, it is neuroendocrine reflex that induces contraction. If absent, then medical management is unlikely to be successful
  - ✓ Bloods: PCV and TP, blood glucose, calcium (ionised best), urea, +/- progesterone levels (<2.0ng/ml)
  - ✓ Radiographs:
    - ➢ Foetal number, size, position and pelvis conformation
    - ➢ Foetal death >12 hours if gas present, bending of spine and overlap of bones
  - ✓ Ultrasound:
    - ➢ Assess foetal viability
    - ➢ Foetal heart rate:
      - Canine: Normal = 180 - 220bpm, if <160bpm require immediate intervention
      - Feline: Normal = >220bpm, if <180bpm require immediate intervention

- **Intervention is required when:**
  - ✓ Evidence of foetal distress i.e. Depressed heart rates
  - ✓ Sudden lethargy in bitch/queen
  - ✓ If had dystocia previously OR if radiographs indicate obstructive dystocia
  - ✓ >2 hours since start of stage 2 without delivery of neonate (>2 hours decreases rates of survival)
  - ✓ >1 hours between delivery of neonates with active labour

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- **Surgical intervention i.e. caesarean section is required when:**
  - ✓ Previous history of dystocia or breeds prone to dystocia
  - ✓ Dystocia of any reason with >5 pups remaining
  - ✓ Any evidence of obstructive dystocia: Foetal oversize/malposition, birth canal obstruction
  - ✓ Unresponsive uterine inertia
  - ✓ Systemically ill bitch/queen
  - ✓ 20 minutes of intense labour without delivering a neonate
  - ✓ 10 minutes of intense labour with a neonate present in the birth canal
  - ✓ Fresh blood from vagina for more than 10 minutes (possible uterine rupture)
  - ✓ Purulent discharge from vulva
  - ✓ >30 minutes after green discharge noted without neonate produced

- **Medical management:**
  - ✓ Only if bitch/queen is healthy (normal blood glucose and calcium), no obstructive dystocia/foetal abnormalities, cervix is dilated, no foetal stress (evident as low heart rates) and non-protracted parturition:
    - ➢ Physical activity
    - ➢ Stimulation of the dorsal vaginal vault:
      - Ferguson reflex via palpation of the dorsal vaginal vault, it is neuroendocrine reflex that induces contraction. If absent, then medical management is unlikely to be successful.
    - ➢ Oxytocin 0.25 – 3 IU SC/IM can repeat every 30-60 minutes:
      - If no puppy after 2 injections, then caesarean section
      - Increases frequency of contractions
    - ➢ Calcium and/or glucose (if blood concentrations indicate):
      - Calcium gluconate 10% 0.2ml/kg slow IV (whilst auscultating chest – stop if bradycardia, dysrhythmia):
        - o Increases strength of contractions
      - Glucose 50% 0.5–1ml/kg IV diluted with saline slow
    - ➢ Oxygen therapy:
      - Advised to reduce the risk of foetal hypoxia

- **Partially delivered foetus:**
  - ✓ Attempts to manually remove the foetus lodged in the birth canal can be attempted only if the bitch/queen is in good health, parturition is not protracted, no foetal oversize
  - ✓ Surgery is indicated if not successful within 20 minutes
  - ✓ Lubricant, fingers best (no instruments unless dead), pull on body not limbs, pull gently ventrally

- **Caesarean section:**
  - ✓ Consult a surgery textbook for more information:
    - ➢ Avoid pre-operative sedation and pain relief – give after removal of the pups but before wake up
  - ✓ Puppy care:
    - ➢ Oxygen and warmth, vigorous rubbing, suction of oral and respiratory secretions
    - ➢ Respiratory support: Intubation or masked positive pressure ventilation, 26g needle into nasal philtrum (acupressure point)
    - ➢ Cardiovascular stimuli: 1 drop of 1:10,000 adrenaline under tongue, cardiac compressions
    - ➢ Reversal of opioids: Naloxone 1 drop under tongue

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- **Puerperal tetany (eclampsia):**
- **Pathogenesis:**
  - ✓ Most often in small breeds of dog and less commonly in the larger breeds and the cat
  - ✓ Sometimes before parturition but usually during lactation
  - ✓ Rapid onset and clinical course:
    - ➢ Early signs: Restlessness, panting and nervousness, 8-12 hours later can progress to ataxia, trembling, muscular tetany and seizures
    - ➢ Hyperthermia (>40°C): Due to increased muscular activity
- **Diagnostics:**
  - ✓ History and signalment
  - ✓ Serum calcium or ionised calcium (best)
- **Treatment:**
  - ✓ Acute therapy:
    - ➢ Slow IV calcium gluconate 10% 0.5-1ml/kg diluted administer slow IV
    - ➢ Cessation of tetanic spasms and improvement in other signs occurring within 15 minutes:
    - ➢ Monitor heart rate with ECG and avoid ventricular fibrillation and cardiac arrest
  - ✓ Maintenance therapy: Oral Calcium supplementation until all milk production ceases
  - ✓ Suckling puppies: Weaned if mature enough otherwise OR fed milk substitute/fostered
- **Prevention:**
  - ✓ Feed a good quality, balanced diet (e.g. Puppy or kitten food) with a Ca+/P+ ratio of 1:1 or less that provides the required (but not excessive) amount of calcium during gestation

- **Pyometron:**
- **Pathophysiology:**
  - ✓ Accumulation of purulent exudates within the uterus, usually due to infection of the uterus secondary to cystic endometrial hyperplasia
  - ✓ Occurs during dioestrus when progesterone stimulates endometrial growth and glandular secretion after being primed by oestrogen
  - ✓ Usually have concurrent urinary tract infections
  - ✓ Bacterial infection is usually due an ascending E. coli, streptococcus, staphylococcus
  - ✓ Typically occurs in middle aged, after repeated heats without pregnancy
  - ✓ Occurs typically within 3 months of last oestrus
  - ✓ Can be an open or closed pyometron
  - ✓ Non-infectious mucometra can also occur (but usually does not have left-shift neutrophilia)
- **Clinical signs:**
  - ✓ Signs of systemic illness: Lethargy, inappetance, pyrexia, vomiting, +/- PU/PD
  - ✓ +/- Vaginal discharge (depends if cervix is open or closed), purulent +/- blood
- **Diagnostics:**
  - ✓ History and timing since last oestrus
  - ✓ Not pregnant
  - ✓ Haematology:
    - ➢ Increased number of band neutrophils (>20% is highly specific indicator)
  - ✓ Biochemistry:
    - ➢ +/- Azotaemia, hyperproteinaemia and hyperglobulinaemia
  - ✓ Imagery:
    - ➢ Radiographs: Enlarged uterus (not usually see)
    - ➢ Ultrasound: Fluid filled uterus, lack of pregnancy
- **Treatment:**
  - ✓ Considerations:
    - ➢ Does the owner want to breed?
    - ➢ Is it an open or closed pyometron?
  - ✓ Stabilise systemically:

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- IV fluids: Correction of perfusion, hydration and electrolyte abnormalities
- IV antibiotics: Cephalothin 22mg/kg IV TID, enrofloxacin 5mg/kg IV SID

✓ Surgical:
- Ovariohysterectomy, treatment of choice as it is a progressive disease

✓ Medical:
- Not recommended due to risks of septicaemia and death
- Anecdotal reports of uterine catheter placement and repeated uterine lavage with sterile saline have increased success rates of medical management
- With open cervix a suggested protocol:
  - Antiprogesterone: Aglepristone10mg/kg SC on day 1, 2 and 7
    - Can help dilate closed cervix if so usually within 48 hours

and

  - Antibiotics: Amoxicillin-clavulanic acid 20mg/kg PO BID and enrofloxacin 5mg/kg PO SID

and

  - Cloprostenol 1µg/kg SC day 3 and 8
    - Synthetic prostaglandin analogue causes luteolysis (reduce progesterone concentration) and cause contraction of the myometrium

- **Mastitis:**
- **Pathophysiology:**
  - ✓ Bacteria most commonly cultured are E. Coli, Staphylococci, β-haemolytic streptococci
  - ✓ Usually affects bitches during the post-partum period, less commonly affecting queens
  - ✓ Caused by trauma, infection up teat canal, haematological spread
  - ✓ Can be localised affecting only one gland or diffuse affecting more than one
  - ✓ Severe mastitis can be life threatening, leading to sepsis and septic shock and gland necrosis
  - ✓ Rule out mammary adenocarcinoma
- **Clinical signs:**
  - ✓ Painful warm and swollen / firm mammary glands
  - ✓ Normal to purulent or blood tinged purulent discharge
  - ✓ Systemic signs include: Pyrexia, lethargy and anorexia
  - ✓ +/- Abscessed and necrotic gland tissue
- **Diagnostics:**
  - ✓ Sample of milk:
    - Culture and sensitivity
    - Cytology: Presence of extracellular and intracellular bacteria
    - pH:
      - For acidic milk use weak base antibiotics: Clindamycin, erythromycin
      - For alkaline milk use weak acids: Cephalothin, amoxicillin, amoxicillin clavulanic acid
      - Either: Enrofloxacin (but avoid if nursing)
  - ✓ Haematology: Inflammatory leukogram with left-shift, dehydration
  - ✓ Biochemistry: Secondary organ dysfunction e.g. azotaemia
- **Treatment:**
  - ✓ Stop puppies and kittens suckling if:
    - Severe inflammation, abscessation
    - Manual teat expression every 4 hours if they are removed
  - ✓ Pain relief
  - ✓ Supportive:
    - Alternating warm and cold compressors in between expressions
  - ✓ IV fluids:
    - Correct hydration and perfusion deficits and electrolytes

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- ✓ Antibiotics:
  - ➢ Empirical therapy:
    - Based on mild pH (see above)
    - Cephalothin, amoxicillin, trimethoprim sulfa, clindamycin, erythromycin, enrofloxacin (only if not nursing
  - ➢ Based on culture and sensitivity
  - ➢ If still nursing, avoid tetracyclines or fluoroquinolones
- ✓ Severe abscessated or gangrenous mastitis:
  - ➢ Surgical debridement
- ✓ Chronic mastitis:
  - ➢ Require antibiotics that are highly lipophilic and based on culture and sensitivity as antibiotic penetration will be reduced with the presence of active inflammation

- **Postpartum metritis:**
- **Pathophysiology:**
  - ✓ Usually develops in the immediate postpartum period i.e. within a week.
  - ✓ Generally due to ascending infection - most commonly cultured are *E. Coli, Staphylococcus, Streptococci, Klebsiella*
  - ✓ Pathogenesis is incompletely understood, normally bacterial contamination is eliminated during uterine involution
  - ✓ Increased risk if dystocia or retained foetal membranes or tissues
  - ✓ Can become systemically ill due to septicaemia or endotoxemia
- **Clinical signs:**
  - ✓ Systemic signs: Pyrexia, lethargy and inappetance
  - ✓ Polyuria and polydipsia
  - ✓ Vaginal discharge is different to lochia – purulent, odourus, blood tinged
  - ✓ +/- Abdominal pain and palpable uterine enlargement
- **Diagnostics:**
  - ✓ Culture and sensitivity of a direct uterine sample is best or a sample as close to the cervix as possible
  - ✓ Cytology: Degenerate neutrophils with extracellular and intracellular bacteria
  - ✓ Haematology: Inflammatory leukogram with left-shift, dehydration
  - ✓ Biochemistry: Secondary organ dysfunction e.g. azotaemia
  - ✓ Urinalysis: Cystocentesis best, +/- isothenuria, bacteriuria
  - ✓ Imagery:
    - ➢ Radiography: Assess for retained foetuses
    - ➢ Ultrasound: Echogenic material may indicate foetal remnants
- **Treatment:**
  - ✓ Pain relief
  - ✓ IV fluids:
    - ➢ Correct hydration and perfusion deficits and electrolytes abnormalities
  - ✓ Antibiotics:
    - ➢ Bactericidal antibiotics: Amoxicillin-clavulanic acid 25mg/kg SC/PO, cephalothin 22mg/kg IV TID, enrofloxacin 5mg/kg IV SID (avoid if nursing)
  - ✓ Supportive care for the neonates
  - ✓ Medical management:
    - ➢ Usually considered if want to future breed and also to avoid interrupting neonate nursing
    - ➢ Uterine contraction:
      - Prostaglandin F2α 10-40ug/kg TID for 3 - 5 days or until uterine evacuation, start low dose and increase over a couple days
      - Causes luteolysis (reduce progesterone concentration) and cause contraction of the myometrium

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- Uterine catheter placement and repeated uterine lavage with sterile saline have increased success rates of medical management

✓ Surgical management:
- Ovariohysterectomy: If systemically ill, not responding to medical management and not intending future breeding

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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

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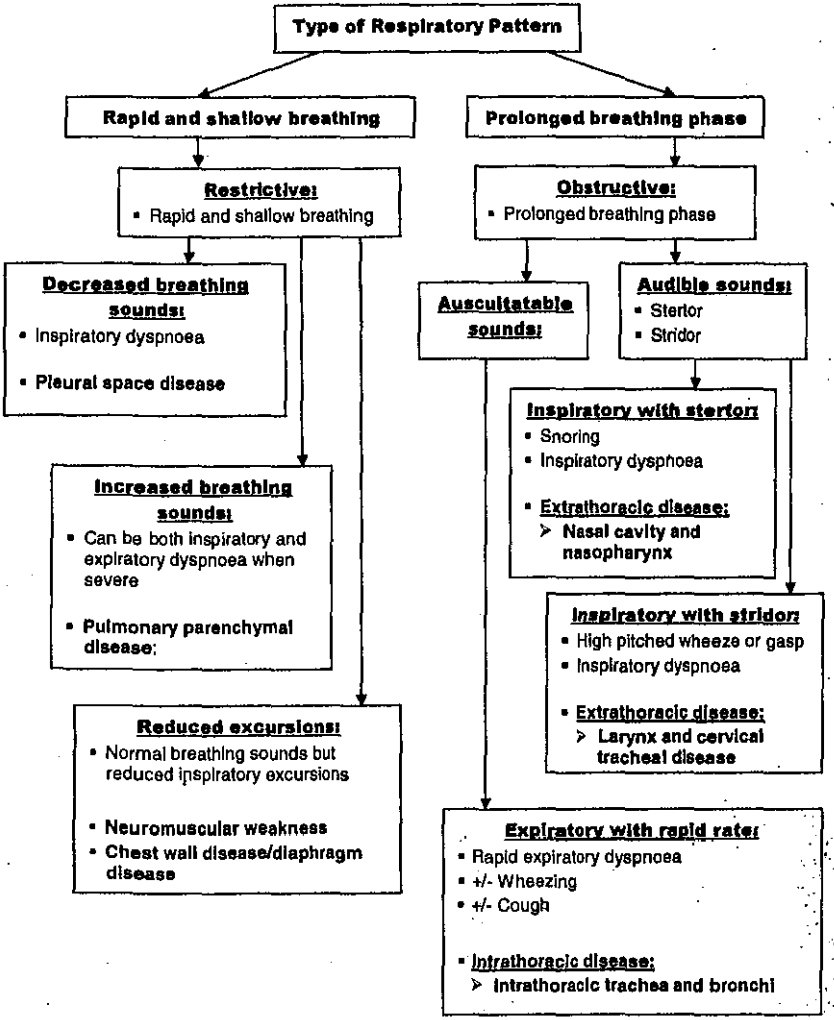

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Restrictive pattern and diseases:

- Due to **restriction of lung expansion:**
  - **Inspiratory dyspnoea usually rapid and shallow**

Disease:	Differentials:	Diagnosis:
Pleural space disease: • Inspiratory tachypnoea • Reduced breath sounds • Reduced sounds dorsally: ➢ Pneumothorax • Reduced sounds ventrally: ➢ Pleural effusion	• Pleural effusion • Pneumothorax • Pleural mass • Diaphragmatic hernia	• Radiographs • Thoracentesis (cytology and culture)
Pulmonary parenchymal disease: • Inspiratory tachypnoea • +/- Expiratory if severe • Increased breath sounds	• Pulmonary oedema • Pneumonia ➢ Aspiration ➢ Viral ➢ Bacterial ➢ Fungal • Pulmonary fibrosis • Neoplasia • Inflammatory: ➢ Allergic ➢ Pulmonary infiltration with eosinophils (PIE) • Parasitic: ➢ Lungworm, heartworm	• Radiographs • BAL (cytology, culture and sensitivity) • Haematology and biochemistry
Neuromuscular weakness: • Normal breathing sounds • Reduced inspiratory excursions	• Tick paralysis • Snake envenomation • Botulism • Neuromuscular disease - polyradiculoneuritis	• Snake venom detection kit • Search for paralysis tick
Chest wall disease or diaphragm disease:	• Trauma • Neoplasia • Diaphragmatic hernia	• Radiographs +/- contrast

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Obstructive pattern and diseases:

- **Due to airway obstruction:**

- **Extrathoracic:**
  - ✓ Inspiratory dyspnoea and mild tachypnoea (<45 breaths per minute)
  - ✓ **Stridor:**
    - ➢ High pitched wheezy/gasping sound during inspiration
    - ➢ Typically originates from larynx
  - ✓ **Stertor:**
    - ➢ Snoring
    - ➢ Typically originating from nasal, nasopharyngeal region

- **Intrathoracic:**
  - ✓ Expiratory dyspnoea and tachypnoea with wheeze
  - ✓ Prolonged expiration and increased expiratory effort

Disease:	Differentials:	Diagnosis:
Nasal cavity and nasopharynx: • Inspiratory obstructive dyspnoea with stertor See **"Nasal and Nasopharyngeal Disease"**	• Brachycephalic airway syndrome • Foreign body • Fungal infection • Inflammatory • Neoplasia • Rhinitis • Nasopharyngeal polyps • Stenosis	• Examination of the nasal cavity • Radiographs • Scoping +/- biopsy • Nasal and nasopharynx flush • CT
Larynx and cervical tracheal disease: • Inspiratory obstructive dyspnoea with stridor • +/- Coughing **See below** for information on tracheal collapse and laryngeal paralysis	• Laryngeal disease: ➢ Stenosis ➢ Inflammation ➢ Paralysis ➢ Foreign body ➢ Neoplasia • Tracheal disease: ➢ Collapse (goose honk cough) ➢ Neoplasia ➢ Foreign body ➢ Hypoplasia ➢ Compression (extraluminal) ➢ Parasitic disease: *Filaroides spp, Capillaria spp.*	• Examination of laryngeal function under light anaesthesia • Radiographs • Scoping +/- biopsy • Fluoroscopy
Intrathoracic trachea and bronchi: • Prolonged obstructive expiration and increased expiratory effort • +/- Coughing **See below** for information on tracheal collapse, canine chronic bronchitis and feline bronchial disease	• Tracheal disease: ➢ Collapse (goose honk cough) ➢ Neoplasia ➢ Foreign body ➢ Hypoplasia ➢ Compression (extraluminal) ➢ Parasitic disease: *Filaroides spp, Capillaria spp.* • Bronchial disease: ➢ Canine chronic bronchitis ➢ Feline bronchial disease ➢ Parasitic disease: Lungworm and heartworm ➢ Infectious bronchitis	• Radiographs • Scoping +/- biopsy • Transtracheal wash or bronchoalveolar lavage

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Selected respiratory diseases:

- **See also "Nasal and Nasopharyngeal Disease"** for:
  - ✓ **Sneezing**
  - ✓ **Reverse sneezing**
  - ✓ **Nasal discharge**
  - ✓ **Epistaxis**

- **Acute respiratory distress:**
  - ✓ 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

- **Haemoptysis:**
- **Features:**
  - ✓ Coughing up blood
- **Causes:**
  - ✓ Coagulopathy
  - ✓ Pulmonary contusions
  - ✓ Congestive heart failure
  - ✓ Heartworm disease
  - ✓ Neoplasia
  - ✓ Pneumonia
  - ✓ Thromboembolism
  - ✓ Hypertension
- **Diagnostics:**
  - ✓ Radiographs:
    - ➢ 3 views: Inspiratory views
    - ➢ Tracheal narrowing due to swelling of the dorsal tracheal membrane can be seen with coagulopathy
  - ✓ See **"Coagulopathy"** to assess for coagulation disorder
    - ➢ ACT, APTT, PT, platelet counts
  - ✓ CBC and biochemistry
  - ✓ Heartworm testing, see **"Parasitic Disease"**
  - ✓ Bronchoalveolar lavage:
    - ➢ After ruling out coagulation disorder
    - ➢ Cytology, culture and sensitivity, PCR
  - ✓ Echocardiogram: If suspect cardiac disease
- **Treatment:**
  - ✓ Treatment of underlying disease
  - ✓ See **"Coagulopathy"** for coagulopathic diseases

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- **Brachycephalic airway syndrome:**
- **Pathophysiology:**
  - ✓ Syndrome of upper airway obstruction caused by a number of airway changes can these include:
    - ➢ Stenosis of the nares, soft palate elongation, eversion of the laryngeal saccules, laryngeal collapses, and hypoplastic trachea (English Bulldogs)
  - ✓ Narrowing of the airways leads to a self-perpetuating cycle of increased airway turbulence leading to further inflammation and then airway narrowing
- **Clinical signs:**
  - ✓ Obstructive inspiratory dyspnoea with stertor
  - ✓ Respiratory distress with open mouth breathing
  - ✓ Reduce exercise intolerance
- **Diagnostics:**
  - ✓ Signalment and clinical signs
  - ✓ Visualisation of the pharynx and larynx under light anaesthesia
  - ✓ +/- Radiographs of neck and thorax
- **Treatment:**
  - ✓ Acute stabilisation:
    - ➢ SPO_2 and oxygen therapy
    - ➢ Sedation:
      - Butorphanol 0.1-0.3mg/kg IM/IV or methadone 0.3mg SC/IM – anxiolytic and antitussive effects
      - Acepromazine 0.01-0.03mg/kg IV/IM if certain that respiratory distress is not due to cardiac disease
    - ➢ +/- Intubation
  - ✓ Medical management:
    - ➢ Anti-inflammatories:
      - Initially dexamethasone 0.2mg/kg IV then long term prednisolone 0.5mg/kg PO BID for 2 weeks then taper, then inhaled anti-inflammatories
    - ➢ Strict rest in a cool environment
  - ✓ Surgical management:
    - ➢ No treatment for hypoplastic trachea
    - ➢ Emergency tracheostomy
    - ➢ Surgical correction of upper airway abnormalities:
      - Widening of nares, trimming of elongated soft palate and removal or laryngeal saccules
    - ➢ Surgery is best performed when symptoms are mild

- **Infectious tracheobronchitis aka Kennel cough:**
- **Pathophysiology:**
  - ✓ Commonly caused by *Bordetella bronchiseptica* and/or parainfluenza virus infection
  - ✓ Extremely infectious, direct contact with respiratory secretions and fomites are the main method of spread
  - ✓ Typically, history of taken to kennels, vet clinic, dog park
  - ✓ Cats can get *Bordetella* infection from dogs
  - ✓ Incubation can be up to a week
  - ✓ Clinical signs are exacerbated by panting, exercise and excitement
- **Clinical signs:**
  - ✓ Acute onset paroxysmal harsh hacking cough, often followed by retching and gagging, often productive (white froth)
  - ✓ Cough can be stimulated by pinching the trachea
  - ✓ Typically, systemically well
  - ✓ Can lead to systemic illness if *Bordetella pneumonia* occurs

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- **Diagnostics:**
  - ✓ Combination of history and clinical signs
  - ✓ If systemically ill, older, heart disease then radiographs +/- bronchoalveolar lavage
- **Treatment:**
  - ✓ Can take about 2 weeks to resolve unless *Bordetella* bronchopneumonia occurs
  - ✓ Isolation of affect animals
  - ✓ Infected animal shed the parainfluenza virus for up to two weeks, and the *Bordetella* bacteria for up to a month
  - ✓ Antibiotics:
    - ➢ Not proven to alter out come
    - ➢ Doxycycline 5mg/kg PO BID or amoxicillin clavulanic acid 20mg/kg PO BID for 4 weeks
    - ➢ Antitussives e.g. Codeine 0.5mg/kg PO TID or tramadol 2-4mg/kg PO BID-TID
      - Avoid cough suppressants if ill and suspect bronchopneumonia

- **<u>Tracheal collapse:</u>**
- **Pathophysiology:**
  - ✓ Disease of small breed middle aged to older dogs
  - ✓ Can affect different areas of the trachea, extrathoracic, thoracic inlet, intrathoracic regions and also the bronchi:
    - ➢ Extrathoracic regions = inspiratory obstructive dyspnoea
    - ➢ Intrathoracic region and bronchi = expiratory obstructive dyspnoea
  - ✓ Caused by a weakening of the tracheal cartilages, suspected to be caused by a reduction in the glycosaminoglycan content of the cartilage rings, leading to a loss of support of the dorsal tracheal membrane which then collapses into the lumen
  - ✓ Can be exacerbated by secondary factors: Allergic airway disease, respiratory tract infection, obesity, heart disease +/- pulmonary oedema and laryngeal paralysis
  - ✓ Can lead to secondary cor pulmonale due to pulmonary hypertension
- **Clinical signs:**
  - ✓ "Goose honk" cough
  - ✓ +/- Dyspnoea and collapse
- **Diagnostics:**
  - ✓ Fluoroscopy:
    - ➢ Best as can visualise collapse
    - ➢ Can find that site of collapse on radiographs is different to actual collapse site seen on fluoroscopy
  - ✓ Radiographs:
    - ➢ 3 views both inspiratory and expiratory views
    - ➢ Rule out coagulopathy as it can also cause the appearance of a collapsed dorsal tracheal membrane
  - ✓ Endoscopy:
    - ➢ Visualisation of collapse
    - ➢ +/- Bronchoalveolar lavage if concurrent pulmonary disease
  - ✓ Biochemistry:
    - ➢ +/- Concurrent hepatopathy that is similar to hyperadrenocorticism possibly due to chronic hypoxia
- **Treatment**
  - ✓ Acute stabilisation:
    - ➢ SPO_2 and oxygen therapy
    - ➢ Sedation:
      - Butorphanol 0.1-0.3mg/kg IM/IV or methadone 0.3mg SC/IM, anxiolytic and antitussive effects
      - Acepromazine 0.01-0.03mg/kg IV/IM
    - ➢ Anti-inflammatories:
      - Initially dexamethasone 0.2mg/kg IV, then long term prednisolone 0.5mg/kg PO BID for 2 weeks then taper

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- ✓ Long term:
  - ➢ Management of exacerbating factors:
    - Treatment of concurrent disease, e.g. heart disease, pneumonia
    - Weight loss
    - Reduce allergen exposure
  - ➢ Medical management:
    - Antitussives: Codeine oral liquid or tramadol 2-4mg/kg PO BID
    - +/- Bronchodilators: Worth a try but not a bronchoconstrictive disease
    - Anti-inflammatory inhalers: Fluticasone puffer
  - ➢ Surgery:
    - Last resort

- **Laryngeal paralysis:**
- **Pathophysiology:**
  - ✓ Paralysis of the muscles controlling the arytenoids cartilages, leading to failure of abduction during inspiration, can be unilateral or bilateral
  - ✓ Typically seen in middle aged to large breed dogs, rare in cats
  - ✓ Can have chronic problem that is exacerbated by e.g. stress and heat, leading to acute presentation of dyspnoea
- **Causes:**
  - ✓ Idiopathic: Most common cause
  - ✓ Congenital, trauma or lesion to cervical region, pathology in the cranial thorax, polyneuropathy, myopathy, tick paralysis
- **Clinical signs:**
  - ✓ Inspiratory obstructive dyspnoea
  - ✓ Can be subclinical then present in acute respiratory distress
  - ✓ +/- Concurrent neurological deficits
- **Diagnostics:**
  - ✓ Visualisation of the pharynx and larynx under light anaesthesia
  - ✓ Can administer doxapram 0.3-0.5ml IV to stimulate a couple deep breaths to exaggerated arytenoids cartilage movement
  - ✓ +/- Radiographs of neck and thorax
- **Treatment:**
  - ✓ Acute stabilisation:
    - ➢ SPO_2 and oxygen therapy
    - ➢ Sedation:
      - Butorphanol 0.10.3mg/kg IM/IV or methadone 0.3mg SC/IM – anxiolytic and antitussive effects
      - Acepromazine 0.01-0.03mg/kg IV/IM
    - ➢ +/- Intubation, active cooling if required
    - ➢ Anti-inflammatories:
      - Initially dexamethasone 0.2mg/kg IV
  - ✓ Medical management:
    - ➢ Treatment of concurrent aspiration pneumonia or other respiratory disease
    - ➢ Not likely to be successful in all cases, can be worth a try but be prepared for acute decompensation
    - ➢ Anti-inflammatories:
      - Initially dexamethasone 0.2mg/kg IV then long term prednisolone 0.5mg/kg PO BID for 2 weeks then taper
    - ➢ Strict rest in a cool environment +/- sedatives
  - ✓ Surgical management:
    - ➢ Treatment of choice, consult surgery textbook

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- **Canine chronic bronchitis:**
- **Pathophysiology:**
  - ✓ Chronic inflammation of the conducting airways (bronchi and bronchioles)
  - ✓ Typically, small breeds
  - ✓ Self-perpetuating disease process where inflammation leads to fibrosis and mucus which leads to further airway inflammation
  - ✓ No identifiable cause, possibly due to airborne allergens (e.g. smoke)
  - ✓ +/- Development of atelectasis secondary to airway mucus obstruction, chronic obstructive pulmonary disease
- **Clinical signs:**
  - ✓ Chronic hacking cough (>2 months)
  - ✓ Obstructive expiratory dyspnoea
  - ✓ +/- Sinus arrhythmia
- **Diagnostics:**
  - ✓ Clinical signs, rule out other disease processes
  - ✓ Radiographs:
    - ➢ 3 views: +/- Bronchial/interstitial pattern
  - ✓ Bronchoscopy:
    - ➢ Excess mucus, hyperaemic mucous membranes
  - ✓ Bronchoalveolar lavage:
    - ➢ Increased mucus
    - ➢ Cytology, culture and sensitivity, PCR for *Mycoplasma*
- **Treatment:**
  - ✓ Anti-inflammatories:
    - ➢ Initially dexamethasone 0.2mg/kg IV then long term prednisolone 0.5mg/kg PO BID for 2 weeks then taper, then trial inhaler
  - ✓ Antibiotics:
    - ➢ Doxycycline 5mg/kg PO BID for 4 weeks, for possible *Mycoplasma*
  - ✓ Bronchodilators: Worth a try
  - ✓ Adjunctive therapy:
    - ➢ Reduce airborne allergens
    - ➢ Coupage
    - ➢ Nebulisation

- **Feline bronchial disease:**
- **Pathophysiology:**
  - ✓ Inflammation of the conducting airways (bronchi and bronchioles)
  - ✓ Syndrome that encompasses:
    - ➢ Chronic bronchitis:
      - Clinically seen as a chronic cough
    - ➢ Asthma:
      - Caused by acute reversible narrowing of the airways due to bronchoconstriction, clinically seen as acute onset dyspnoea, triggered usually by airborne allergens
      - Diagnosis is based on clinical presentation, response to bronchodilators, an inflammatory bronchoalveolar lavage (typically eosinophilic) and evidence of hyperplasia of the mucus glands and smooth muscle
  - ✓ Not all cats affected by bronchial disease will have asthma, and some cats will present in acute distress due to asthma without a history of coughing
  - ✓ +/- Development of atelectasis secondary to airway mucus obstruction, see in right middle lung lobe
  - ✓ Must rule out heart disease and other diseases affecting the pulmonary system

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- **Clinical signs:**
  - ✓ Chronic cough more than 2 months
  - ✓ Mild to severe respiratory distress usually obstructive expiratory dyspnoea
  - ✓ Wheezing
- **Diagnostics:**
  - ✓ Radiographs:
    - ➢ 3 views: +/- Bronchial pattern and hyperinflation of lung fields, but can be normal
  - ✓ Bronchoalveolar lavage:
    - ➢ Usually eosinophilic or neutrophilic
    - ➢ Cytology, culture and sensitivity, PCR or *Mycoplasma* culture
- **Treatment:**
  - ✓ Acute stabilisation:
    - ➢ SPO_2 and oxygen therapy
    - ➢ Sedation:
      - Butorphanol 0.1-0.3mg/kg IM/IV or methadone 0.3mg SC/IM, anxiolytic and antitussive
      - Acepromazine 0.01-0.03mg/kg IM/IV if certain that respiratory distress is not due to cardiac disease
    - ➢ Bronchodilator:
      - Terbutaline 0.01mg/kg IV/IM
      - If reduction in rate and effort, supportive of the diagnosis of asthma
      - If heart rate increases >200bpm then drug is working
    - ➢ Anti-inflammatories:
      - Initially dexamethasone 0.2mg/kg IV
  - ✓ Long term treatment:
    - ➢ Principles:
      - Management includes:
        - o Anti-inflammatories and bronchodilators typically inhalation preparations
        - o Reduction in airborne allergens is very important
      - Anti-parasitic:
        - o Rule out parasitism as a cause of eosinophilic bronchoalveolar lavage by treating prophylactically with fenbendazole 50mg/kg PO SID for 5 days
      - Antibiotics:
        - o Culture for *Mycoplasma* before starting, doxycycline 5mg/kg PO SID for 4 weeks
    - ➢ Mild to moderate cases:
      - Anti-inflammatory:
        - o Fluticasone 250µg BID
        - o Moderate cases start with prednisolone 1mg/kg PO BID for 7 days tapering
      - Bronchodilators:
        - o Salbutamol 100-200ug as needed
    - ➢ Severe cases: Frequent cough and dyspnoea
      - Acute stabilisation as above
      - Anti-inflammatory:
        - o Start with prednisolone 1mg/kg PO BID for 7 days tapering, may require intermittent or constant oral therapy for example every other day
      - Bronchodilators:
        - o Salbutamol 100-200ug can give every 30 minutes to stabilize then QID
        - o Oral terbutaline or theophylline

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- **Pneumonia:**
- **Pathophysiology:**
  - ✓ Inflammation of the lung parenchyma
  - ✓ Causes include:
    - ➢ Bacterial
    - ➢ Protozoal: E.g. *Pneumocystic carinii*
    - ➢ Fungal: Cryptococcal, aspergillosis
    - ➢ Parasites
    - ➢ Chemical
  - ✓ Primary bacterial pneumonia is rare, more commonly secondary to:
    - ➢ Aspiration from either loss of consciousness, seizures, oesophageal and laryngeal disease, general anaesthetic, bottle feeding
    - ➢ Immunosuppression due to systemic disease, endocrinopathies, drugs, viral infection
- **Clinical signs:**
  - ✓ Sometimes no clinical signs for days after inciting event
  - ✓ Soft cough common in dogs but not frequent in cats
  - ✓ Dyspnoea starts with inspiratory restrictive pattern that can progress to both an inspiratory and expiratory restrictive pattern when severe
  - ✓ Increased respiratory sounds: Crackles and wheezes
  - ✓ +/- Nasal discharge typically mucopurulent
  - ✓ +/- Signs of systemic illness: Pyrexia, anorexia, lethargy
- **Diagnostics:**
  - ✓ SPO_2 and arterial blood gas
  - ✓ Radiographs:
    - ➢ 3 views: Inspiratory views, usually see interstitial/alveolar pattern with air bronchograms
  - ✓ Bronchoalveolar lavage:
    - ➢ Cytology, culture and sensitivity
  - ✓ +/- Haematology and biochemistry: Assess for systemic affects
  - ✓ Other:
    - ➢ Fungal titres, FeLV/FIV, serology for heartworm, toxoplasmosis, faecal flotation (baermann technique)
- **Treatment:**
  - ✓ Treatment of underlying disease process
  - ✓ Antibiotic therapy for at least 6 weeks:
    - ➢ Based on culture and sensitivity
    - ➢ Stable patients:
      - Amoxicillin clavulanic acid 25mg/kg PO BID or trimethoprim-sulpha 15mg/kg PO BID
      - Doxycycline 5mg/kg PO SID if suspect *Mycoplasma*
    - ➢ Systemically ill patients: Four quadrant antibiotic therapy
      - Cephalothin 22mg/kg IV TID, metronidazole 10mg/kg IV BID, enrofloxacin 5mg/kg IV SID
    - ➢ If hypoxaemic:
      - Oxygen therapy +/- mechanical ventilation
      - Prognosis for severe hypoxaemic respiratory failure is poor
  - ✓ Antifungals
  - ✓ Supportive therapy:
    - ➢ IV fluids to maintain systemic and airway hydration
    - ➢ Nebulisation: Sterile saline
    - ➢ Coupage
    - ➢ Turning of recumbent patients

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- **Pulmonary oedema (non-cardiogenic):**
- **Pathophysiology:**
  - ✓ Not as common as cardiac pulmonary oedema
  - ✓ Due to high protein fluid extravasation
  - ✓ Causes include:
    - ➢ Near drowning, aspiration, severe trauma, obstructive (upper airway obstruction – typically in young animals), neurological (seizures, electrocution), acute respiratory distress syndrome
- **Clinical signs:**
  - ✓ Cough soft
  - ✓ Dyspnoea: See inspiratory restrictive pattern that can progress to both an inspiratory and expiratory restrictive pattern when severe
  - ✓ Increased respiratory sounds: Crackles and wheezes
- **Diagnosis:**
  - ✓ History
  - ✓ Radiographs:
    - ➢ 3 views: Typically, dorsocaudal lobes affected first compared to perihilar in congestive heart failure
- **Treatment:**
  - ✓ Oxygen therapy
  - ✓ +/- Mechanical ventilation if develops hypoxaemia that is non-responsive to oxygen therapy
  - ✓ Diuretic therapy: Not effective as the fluid is high protein (compared to cardiogenic oedema)
  - ✓ Antibiotics: Not indicated unless concerned about infectious pathology
  - ✓ Supportive:
    - ➢ Conservative IV fluids, aim to maintain perfusion but avoid volume overload
    - ➢ General nursing care

- **Pneumothorax:**
- **Pathophysiology:**
  - ✓ Air within the pleural space
  - ✓ Open pneumothorax: Communication with the outside, usually due to trauma
  - ✓ Closed pneumothorax: No communication with the outside, due to air leaking from the airways or parenchyma
    - ➢ Spontaneous closed pneumothorax: Not due to trauma
    - ➢ Traumatic closed pneumothorax: Due to trauma but closed
- **Differentials:**
  - ✓ Open pneumothorax:
    - ➢ Traumatic penetration of the thoracic wall
  - ✓ Traumatic closed pneumothorax:
    - ➢ Tracheal tear, ruptured lung
  - ✓ Spontaneous (closed) pneumothorax:
    - ➢ Ruptured bullae
    - ➢ Bullous emphysema
    - ➢ Ruptured neoplasia, granuloma, abscess
    - ➢ Rough endotracheal intubation
    - ➢ Oesophageal perforation
- **Clinical signs:**
  - ✓ Inspiratory obstructive dyspnoea with tachypnoea
  - ✓ Reduced lung sounds dorsally
  - ✓ Hypoxemia and cyanosis
  - ✓ +/- Clinical signs of trauma

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- **Diagnostics:**
  - ✓ Radiographs:
    - ➢ 3 views: Retraction of lung lobes and parenchymal structures from thoracic wall
  - ✓ Thoracentesis
- **Treatment:**
  - ✓ Oxygen therapy and pain relief
  - ✓ Thoracentesis: Required if see signs of respiratory distress e.g. Tachypnoea, dyspnoea, hypoxaemia
  - ✓ Cover open wounds:
    - ➢ Make air tight seal with sterile lube and bandage (can use cling wrap)
    - ➢ Start IV antibiotics
  - ✓ +/- Chest tube placement and continual drainage
  - ✓ Surgery:
    - ➢ Exploration of open wounds +/- thoracotomy
    - ➢ Spontaneous closed pneumothorax, consult surgery text

- **<u>Pleural effusion:</u>**
- **Pathophysiology:**
  - ✓ Requires thoracentesis and analysis, see **"Effusions"** for analysis and differentials
  - ✓ Caused by alterations in hydrostatic and oncotic pressures, increased vascular permeability or lymphatic obstruction

Transudates:	
Causes: ▪ Reduced oncotic pressure e.g. hypoproteinaemia (albumin <15 g/l): ➢ PLN, PLE, liver disease ▪ Excess IV fluids (cats)	**Diagnostics:** ▪ Cytology and culture of fluid ▪ Haematology and biochemistry ▪ +/- Dynamic liver testing ▪ PLN: Urinalysis, UP:C, culture and sensitivity ▪ PLE = see **"Diarrhoea and Haematochezia"**
Modified transudate:	
Causes: ▪ Increased capillary hydrostatic pressure e.g. RHS CHF, LHS CHF (cats), pericardial disease ▪ Diaphragmatic hernia ▪ Neoplasia ▪ Lymphatic obstruction e.g. neoplasia, diaphragmatic hernia, abscess ▪ Increased permeability of vessels (blood and lymphatics) e.g. FIP	**Diagnostics:** ▪ Cytology and culture of fluid ▪ Haematology and biochemistry ▪ Cardiac auscultation ▪ Cardiac radiographs and ultrasound ▪ +/- CT
Non-septic exudate:	
Causes: ▪ Inflammation: FIP (can have high globulins), liver disease, lung torsion, hernia ▪ Neoplasia	**Diagnostics:** ▪ Haematology and biochemistry ▪ Cytology and culture of fluid ▪ +/- Ultrasound ▪ +/- CT ▪ FIP – see "Viral Disease and Vaccination"
Septic exudate:	
Causes: ▪ Ruptured abscess ▪ Foreign body inhalation or penetrating injury ▪ Fungal infection	**Diagnostics:** ▪ Haematology and biochemistry ▪ Cytology and culture of fluid ▪ +/- Ultrasound ▪ +/- CT

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Pyothorax:

- Septic exudate

Treatment for pyothorax:

- Pain relief
- IV antibiotics:
  - Four quadrant initially then change according to culture and sensitivity results
  - Cephalothin 22mg/kg IV TID, metronidazole 10mg/kg IV BID and enrofloxacin 5mg/kg SC SID
- Place a chest drain and drain as much as possible:
  - Perform saline lavage 5-10ml/kg BID, beware of fluid absorption and fluid overload
- Antifungals if indicated
- Surgery if see foreign body, penetration wound, fungal granuloma or consolidated lung lobe, consult surgery text for more information

Chyle:

- Opaque to pink

Causes:

- Rupture or obstruction of lymphatic flow:
  - Neoplasia, traumatic, idiopathic
- Secondary to heart failure (especially in cats)
- Pseudochyle (usually formed by lymphoma)

Diagnostics:

- CBC and biochemistry
- Cytology and culture of fluid
  - Fluid [TAG] > serum, large number of lymphocytes and other inflammatory cells
- +/- Ultrasound/CT

Treatment for chylothorax:

- Treatment of the underlying condition, difficult if idiopathic
- Medical therapy:
  - Repeat thoracentesis
  - Rutin 250-500mg PO TID:
    - Can increase removal of lymph from tissues
    - A benzopyrone, of unknown mode of action
  - +/- Octreotide (somatostatin analogue): Questionable efficacy, worth a try
- Surgery:
  - Thoracic duct ligation with pericardectomy, can be curative

Haemorrhage:

Causes:

- Trauma
- Neoplasia
- Coagulopathies
- Ruptured granuloma

Diagnostics:

- ACT, APTT, PT, blood smear and other coagulation tests, see **"Coagulopathy"**
- Blood smear:
  - True haemorrhagic i.e. not iatrogenic:
    - Not see platelets and sample should not clot
    - Presence of erythrophagocytosis = chronic
- PCV/TP:
  - Compare PCV/TP to venous PCV/TP
  - If PCV/TP is similar = recent bleed
  - If PCV is lower than peripheral blood, then chronic
- CBC and biochemistry
- +/- Ultrasound/CT

Treatment of haemothorax:

- Only remove enough to relieve clinical signs of dyspnoea
- +/- Auto-transfusion: Perform blood smear to ensure no bacteria present
- Trauma:
  - Symptomatic therapy: Oxygen and sedation
  - Intermittent thoracentesis to improve ventilation
- Coagulopathy:
  - Plasma transfusion, vitamin K+ therapy, see **"Coagulopathy"**

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Seizure Disorders

- **This chapter covers:**
  - ✓ General features and stages of a seizure episode
  - ✓ Differences in cats compared to dogs
  - ✓ Different causes of seizures
  - ✓ General diagnostic principles
  - ✓ Treatment of status epilepticus and long term management

- **Features:**

Generalised seizures:	▪ Aka Grand mal seizures, involves both cerebral hemispheres ▪ Loss of consciousness, incontinence, muscle activity: ➢ Tonic: Increased muscle tone ➢ Clonic: Rhythmic muscle contractions
Focal or partial seizures:	▪ Aka Petit mal, originates from one cerebral hemisphere ▪ No alterations in consciousness ▪ Repetitive twitching or movement of limbs, chewing body parts
Classification:	▪ **Epilepsy:** ➢ Condition of recurrent seizures ➢ ***Primary epilepsy:*** • No underlying disease process is identified e.g. inherited, idiopathic ➢ ***Symptomatic epilepsy:*** • Secondary to progressive underlying cause e.g. brain tumour, meningoencephalitis, head trauma • Can increase in frequency if untreated (due to kindling) ▪ **Cluster seizures:** More than one seizure in a 24 hour period ➢ Dogs: Reduced prognosis if presents with clusters first time ➢ Cats: No influence on prognosis ▪ **Isolated seizures:** Self-limiting, most common ▪ **Status epilepticus:** Continuous seizure activity lasting at least 5 mins ➢ Can cause neuronal necrosis if prolonged ➢ Can be associated with hypoxaemia, hyperthermia, lactic acidosis, hypoglycaemia, shock, aspiration pneumonia and neurogenic pulmonary oedema

- **Stages:**
  - ✓ **Pre-ictus:** Before actual seizure:
    - ➢ Variable time frame: Seconds to minutes
    - ➢ Feel a forthcoming seizure, may act strangely, become agitated, anxious, run around, hide, vomit
  - ✓ **Ictus:** Seizure event:
    - ➢ Clinical signs of seizure
    - ➢ Variable time frame, seconds to minutes
  - ✓ **Post-ictus:** Recovery phase:
    - ➢ Flat and lethargic, disorientated, blind
    - ➢ Variable, minutes to hours, possibly days

- **Seizures in cats:**
  - ✓ Not as common compared to dogs
  - ✓ Partial seizures more commonly than generalised seizures:
    - ➢ Stereotypic behaviours, bursts of activity

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- ✓ Multiple episodes a day
- ✓ Idiopathic epilepsy:
  - ➢ Usually seen between 1-4 years of age
  - ➢ Less common compared in cats ~50% compared to dogs ~70% i.e. more commonly due to intracranial pathology
- ✓ Infectious causes can occur at any age

Differentials for seizures:	
Intracranial:	**Extracranial:**
▪ Inflammatory (meningoencephalitis): ➢ Granulomatous meningoencephalitis ➢ Steroid responsive ➢ See **"Neurological Disease"** for differentials for inflammatory meningoencephalitis ▪ Viral: ➢ Distemper ▪ Metabolic: ➢ Storage disease ▪ Neoplasia ▪ Vascular accident: ➢ Clot or bleed ▪ Epilepsy (idiopathic) ▪ Hydrocephalus ▪ Traumatic accident	▪ Hepatic encephalopathy: ➢ Hepatic failure ➢ Portosystemic shunt ▪ Toxic: see **"Toxicity"** ➢ Plants ➢ Lead ➢ Mycotoxins (fungal – bread/compost) ➢ Pyrethroids (anti-parasitic) ➢ Organophosphate (anti-parasitic) ➢ Carbamate (anti-parasitic) ➢ Metaldehyde (snail bait) ➢ Rat bait ➢ Strychnine (poison) ➢ Chocolate and caffeine ➢ Ethylene glycol ▪ Metabolic: ➢ Hypoglycaemic ➢ Hypocalcaemia ➢ Ischaemic ➢ Thiamine deficiency
Species differences:	
▪ <u>Common causes in dogs:</u> ➢ **Less than 1 year old:** • Portosystemic shunts* • Inflammatory • Distemper • Hydrocephalus/storage disease • Toxicity ➢ **1 – 5 years old:** • Epilepsy* • Inflammatory • Toxicity • Cerebral neoplasia ➢ **>5 years old:** • Cerebral neoplasia* • Inflammatory • Toxicity • Epilepsy • Metabolic • Vascular	▪ <u>Common causes in cats:</u> ➢ Neoplasia ➢ FIP, FeLV, FIV ➢ Cryptococcus ➢ Toxoplasmosis ➢ Traumatic ➢ Toxins

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- **Diagnostic workup** of a dog or cat presented with seizures:
  - ✓ Aims:
    - ➢ Identify if there are indications that the disease process is intracranial
    - ➢ Rule out extracranial causes
    - ➢ Investigate intracranial causes

- **Indications that the disease process is intracranial:**
  - ✓ Wait until after patient recovers from post-ictal phase
    - ➢ Alterations in mental status, prior to seizure episodes
    - ➢ Blindness: Lack of menace, central blindness (PLRs should be normal)
    - ➢ Proprioceptive deficits, knuckling
  - ✓ **ANY of these features** → CT/MRI and CSF are indicated

- **History:**
  - ✓ Any altered behaviours or mental status (stupor/coma/depression)
  - ✓ Postural deficits are very important indicate intracranial lesion
  - ✓ Exposure to toxins, chemicals
- **Signalment:**
  - ✓ Age: See previous page
  - ✓ Breed:
    - ➢ Boxers: Intracranial tumours
    - ➢ Maltese: Inflammatory disease
- **Description of seizures** (type and frequency):
  - ✓ Confirm that seizures are actually seizures, not:
    - ➢ Syncope or collapse
    - ➢ Vestibular disease (can be transient)
    - ➢ Other behaviour disorder
- **General physical, neurological and fundic examination (bulging optic disc):**
  - ✓ Neurological exam tests that target the cerebral cortex
  - ✓ Wait until after patient recovers from post-ictal phase
  - ✓ See **"Neurological Disease"** for neurological examination
    - ➢ Alterations in mental status
    - ➢ Blindness: Lack of menace (central blindness = PLRs should be normal)
    - ➢ Proprioceptive deficits (knuckling)
- **Minimum database:**
  - ✓ Complete blood count, biochemistry and electrolytes, urinalysis
- **+/- Paired bile acids**
  - ✓ Assessment for PSS (signalment) and liver disease
- **Ultrasound:**
  - ✓ Assessment for PSS (signalment)
- **+/- Cerebrospinal fluid tap:**
  - ✓ Cytology
  - ✓ Culture and sensitivity and PCR panels
- **+/- CT or MRI**
- **Other tests:**
  - ✓ Titres: Viral titres in cats (FeLV, FIV, FIP), distemper/rabies virus, toxoplasma, neospora (dogs only), Cryptococcus
  - ✓ Blood lead levels if suspect lead toxicity
  - ✓ If coagulopathy is suspected: ACT, PT and APTT

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Treatment:

- **Treatment of status epilepticus → See status epilepticus flowchart following pages**
  - ✓ **Diazepam:**
    - ➢ Lipid soluble therefore enters brain rapidly
    - ➢ Binds to GABA receptors and enhances neuronal hyperpolarisation, reducing neuronal firing
    - ➢ Short duration of action
    - ➢ For status epilepticus:
      - 0.5-1mg/kg IV can repeat 3 times
      - Continuous rate infusion at 0.5-2mg/kg/hr:
        - o Dilution down to 1:40 in saline and prepare only 2 hours worth at a time, binds to plastic
        - o Once seizure activity has stopped for 4 hours then slowly decrease CRI over as many hours as the patient has been on the infusion
  - ✓ **Midazolam:**
    - ➢ Benzodiazepine like diazepam but water soluble, 0.2mg/k IV
    - ➢ Better for continuous rate infusions at 0.1-0.3 mg/kg/hr IV
    - ➢ Shorter duration of action but dosed the same as diazepam
  - ✓ **Phenobarbital:**
    - ➢ Used as a longer acting anticonvulsant, used after diazepam and as maintenance anticonvulsant
    - ➢ Potentiates the action of GABA
    - ➢ Diluted down and given slow IV (max 100mg/min)
    - ➢ For status epilepticus after control of seizures with diazepam or propofol:
      - If not on phenobarbitone maintenance therapy 6-8mg/kg IV
      - If already on phenobarbitone maintenance therapy 4mg/kg IV, must collect blood before administering to assess levels
      - Can repeat 4mg/kg IV doses till max cumulative dose is 18mg/kg IV
    - ➢ Maintenance therapy:
      - 2-2.5mg/kg PO BID
  - ✓ **Propofol:**
    - ➢ Rapid acting lipid soluble general anaesthetic
    - ➢ Likely to have GABA agonist activity
    - ➢ Does not stop seizure activity in the brain but does stop external manifestations
    - ➢ For status epilepticus to control muscle activity:
      - 2-4mg/kg IV bolus titrated to effect
      - Continued with a CRI at 0.2-0.5mg/kg/min, and continue for 6 hours then wean slowly over 6 hours
  - ✓ **Keppra (Levetiracetem):**
    - ➢ 20-40mg/kg IV TID
    - ➢ Not hepatotoxic, can be used in patients with porto-systemic shunts

- **Principles of chronic therapy:**
  - ✓ Oral medications can take several days to weeks to reach therapeutic levels
  - ✓ Treatment is usually for life
  - ✓ Start treatment when 2 seizures have occurred within 6 weeks
  - ✓ Aim to reduce frequency to less than one every 6-8 weeks
  - ✓ Start with **phenobarbitone** and increase dose as required until reach max dose (based on therapeutic range), then add **potassium bromide** until reach max dose (based on therapeutic range), then add either:
    - ➢ **Gabapentin, keppra** (Levetiracetem) **&/OR zonisamide** (can use as sole agent – but unlikely if phenobarbitone and potassium bromide combination is not working)

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- ✓ **Monitoring concentrations of anti-epileptic drugs:** Perform therapeutic drug concentration testing to ensure concentrations are within therapeutic range
  - ➢ 1 month after starting and after every increase and then every 6 months with patients with good seizure control
  - ➢ Aim for between 50% and 75% of therapeutic index, >75% increases the chance of hepatotoxicity
  - ➢ Therapeutic range: More of a "target range", does not mean that it should be working if it is within the range
  - ➢ Phenobarbitone: 70-170µmol/L
  - ➢ Potassium bromide: 10-20mmol/L
- ✓ **Liver Toxicity:**
  - ➢ Monitor liver profile every 6 months +/- paired bile acids
  - ➢ Clinical signs: Hypoalbuminaemia (ascites, pleural effusion, peripheral oedema), jaundice, vomiting, diarrhoea, inappetance, lethargy
  - ➢ What to do: Remove off phenobarbitone and start potassium bromide +/- another agent

- **Chronic therapy:** Aim to reduce the frequency of fits
- ✓ **Phenobarbitone: 4mg/kg/Day PO divided into 2 or 3 doses**
  - ➢ If oral loading 6-8mg/kg PO, then continue as above
  - ➢ Depresses activity of neurons by potentiating the inhibitory effects of GABA
  - ➢ Peak blood levels with oral dosing may NOT occur for 4-6 hour, can take 2 weeks to reach steady state, collect sample before next dose (trough)
  - ➢ Body develops both pharmacodynamic and pharmacokinetic tolerance
  - ➢ Side effects: Polyuria and polydipsia, polyphagia, sedation (reduces after 2 weeks), weight gain, ataxia
  - ➢ Liver damage will occur: Monitor liver function tests to see if need to stop
    - Monitor ALP for 2-6 x increase also ALT and GGT – if ALT starts to increase more so than ALP then may need to stop
    - Increase in bile acids or urine bile acids/creatinine ratio
  - ➢ If need/want to stop taper off gradually unless showing signs of liver damage
- ✓ **Potassium bromide:**
  - ➢ Dosage regimes:
    - Sole agent: 30mg/kg/day PO SID or divided
    - Combined with phenobarbitone: 20mg/kg/day PO SID or divided
    - Loading dose (GIT side effects and sedation): 400-600mg/kg PO divided
  - ➢ Slow to reach steady state (up to 4 months) - long half-life (24 hours)
  - ➢ **NOT used in cats** (pneumonitis)
  - ➢ OK for animals with liver damage
  - ➢ Beware has been associated with pancreatitis, feed a low fat diet at the same time
  - ➢ If need/want to stop therapy can stop at anytime
- ✓ **Gabapentin:** 10-20mg/kg PO TID
  - ➢ Can use in combination with phenobarbitone/potassium bromide
  - ➢ Can start to reduce phenobarbitone levels leaving potassium bromide and gabapentin
  - ➢ Not hepatotoxic
- ✓ **Keppra (Levetiracetem):** 20mg/kg PO/IV TID
  - ➢ Reaches steady state in couple of days
  - ➢ Can use in combination with phenobarbitone/potassium bromide/Zonisamide
  - ➢ Not hepatotoxic, can be used in patients with portosystemic shunts
- ✓ **Zonisamide:** If on phenobarbitone start 10mg/kg BID (phenobarbitone increases the levels of enzymes that metabolises Zonisamide), if **not** on phenobarbitone start 5mg/kg BID:
  - ➢ If good control on Zonisamide can try to wean off phenobarbitone and potassium bromide and reduce dose to 5mg/kg PO BID

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- **Prevention of cluster seizures:**
  - ✓ Given orally or rectally after a seizure episode to prevent the occurrence of cluster seizures
  - ✓ **Diazepam:** Give up to 3 times in a 24 hours post-seizure period:
    - ➢ Send home in a glass vial with rubber stopper (not plastic):
      - Rectally at onset of seizure episode: 2mg/kg via cannula 2 cm into rectum
      - Orally after seizure episode: 2mg/kg
      - Intranasal: 0.5mg/kg
  - ✓ **Clonazepam:** Give up to 3 times in a 24 hours post-seizure period:
    - ➢ Benzodiazepine with a longer duration of action than diazepam
    - ➢ Orally after seizure episode: Up to 4mg/dog, but 2mg/dog for an average size dog

- **Refractory seizures:**
  - ✓ Unacceptable seizure control when on a single agent
  - ✓ Measure drug therapeutic levels:
    - ➢ If low or there-is room to increase, then step-wise increase dosages by 10-25% then remeasure therapeutic ranges accordingly and monitor hepatic effects
    - ➢ If at high end of therapeutic range add another agent and remeasure therapeutic ranges accordingly
  - ✓ If still uncontrolled:
    - ➢ Reassess the initial diagnosis: Suspect a structural intracranial lesion or undiagnosed extracranial disease
    - ➢ Add in another agent

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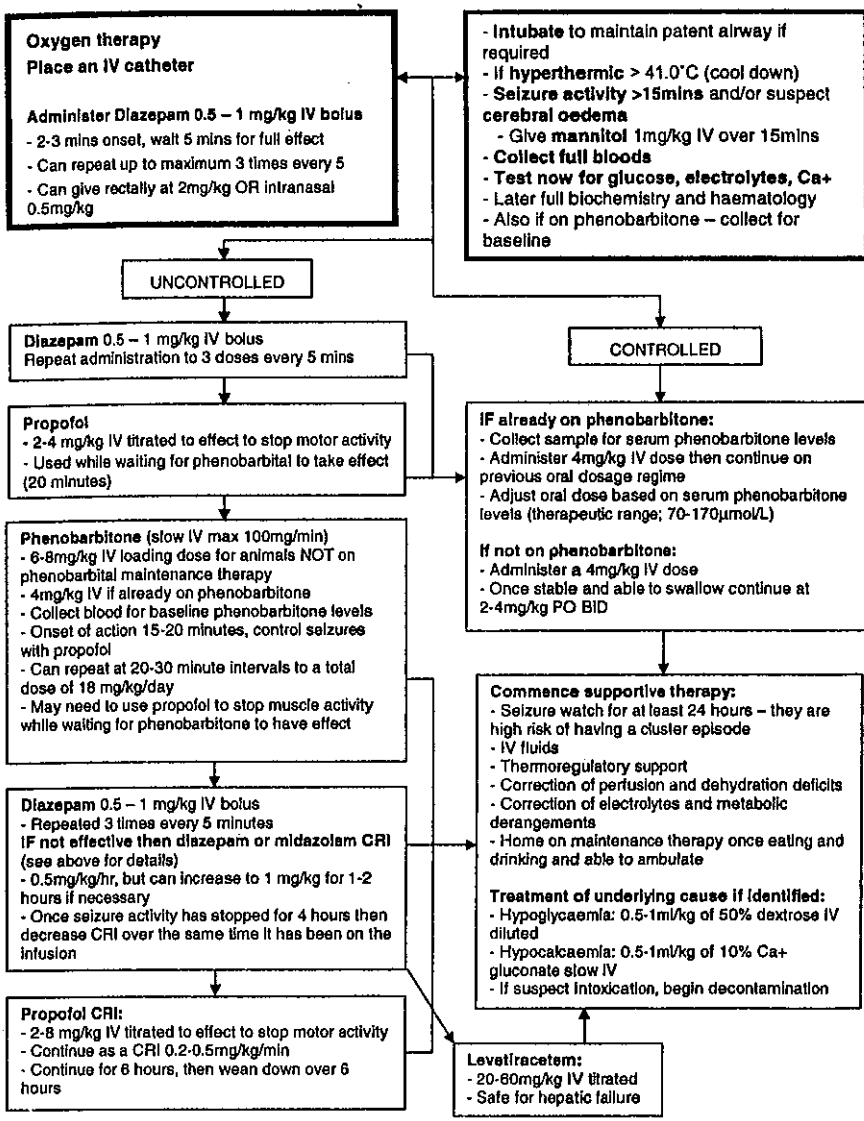
Status Epilepticus Flowchart

If in status epilepticus for a prolonged period, they may continue to slowly paddle this is not seizing (motor activity may take longer to stop). If the **eyelids are twitching, still seizing.**



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Shock and Anaphylaxis

- **This chapter covers:**
  - ✓ Different classifications of shock and clinical signs of shock
  - ✓ Treatment principles for the different types

- **Classification:**
  - ✓ Based on aetiology:
    - ➢ Hypovolaemic:
      - Haemorrhage, loss of water and electrolytes (renal, gastrointestinal)
    - ➢ Distributive (vasodilatory):
      - Due to severe inflammation, sepsis, neoplasia, burns, anaphylactic
    - ➢ Cardiogenic (reduced cardiac output):
      - Heart disease or arrhythmia:
        - o LHS heart disease: Pulmonary oedema, tachypnoea, cyanosis
        - o RHS heart disease: Ascites, jugular distension/pulses, pleural effusion
    - ➢ Obstructive:
      - Impairment to blood flow: GDV, pericardial effusion, pneumothorax

- **Clinical signs:**
  - ✓ Pale mucous membranes and slow CRT:
    - ➢ Slow capillary refill time + weak pulse = hypovolemia, poor peripheral perfusion
    - ➢ Normal/fast capillary refill time + normal bounding pulse = anaemia, pain OR **distributive shock**
  - ✓ Tachycardia plus weak pulses
  - ✓ Tachypnoea
  - ✓ Hypotension and cool peripheries
  - ✓ Hypothermia
  - ✓ Weakness/collapse

- **Treatment of shock:**
  - ✓ Aimed to support cardiac and organ function:
    - ➢ **A – Airway patency**
    - ➢ **B – Breathing** – oxygen support, bronchodilators
    - ➢ **C – Circulation** – control bleeding and IV fluids (not in cardiogenic shock)
    - ➢ **D – Drugs** (that exacerbate shock) – NO vasodilators, no α-agonists, no inhaled anaesthetics

Principles of circulatory support:

- **IV fluid resuscitation therapy:**
  - ✓ Bolus therapy:
    - ➢ Repeated doses of fluids administered intravenously, after each bolus the patient is reassessed for **end point resuscitation variables**
    - ➢ Can use a combination of crystalloids, colloids, blood products to achieve **end point resuscitation variables**
    - ➢ If responds and **end point resuscitation variables** are met, then continue with replacement/maintenance rates
  - ✓ Fluid choices:
    - ➢ Crystalloids:
      - Isotonic: 0.9% NaCl, Hartmanns etc.
        - o Dogs: 5-20ml/kg boluses IV
        - o Cats: 5-10ml/kg boluses IV
      - Hypertonic: 7% NaCl

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- Up to 5ml/kg administered at 1ml/kg/min
- Once off dose, not in dehydrated or hyponatraemic patients

➢ Synthetic colloids: Voluven, penta/hetastarch
- Given in 5ml/kg boluses, maximum dose varies between colloids, most recommendations are 20ml/kg/day but up to 40ml/kg/day for Voluven
- Must administer with crystalloids

✓ If more than a half a blood volume (45ml/kg for a dog and 30ml/kg for a cat) has been given and still not met **end point resuscitation variables**:

➢ Assess for cardiogenic shock: Assess for jugular pulses and cardiac disease **see below**

➢ Assess for obstructive shock: GDV, pericardial effusion, tension pneumothorax

➢ Assess for distributive shock: CVP if possible, trial colloids or vasopressor therapy **see below**

➢ Assess for ongoing losses: External and internal haemorrhage (consider abdominal counter pressure)

➢ **If still in shock and needs more fluids, consider:**
- TP if <45g/L or low oncotic pressure, consider colloids or whole blood
- PCV if <30% or anaemic/haemorrhagic patients, consider pRBC or whole blood and monitor PCV and lactic acid levels
- If clinically dehydrated, elevated PCV then continue with crystalloids

- **End point resuscitation variables:**

✓ Used to gauge adequate blood flow to vital organs, indicate when to stop shock fluid therapy and continue on maintenance therapy:

✓ Downstream parameters:
- Do not contribute to perfusion but rather depend on having adequate perfusion
- Provide early recognition of occult shock and be a source of feedback for resuscitation efforts and thus provide a guide for continued therapy

Resuscitation End Points	
Variables:	**Value:**
Mentation:	Alert
Heart rate (beats per minute):	Dog: 90–140 Cat: 180
Mucous membrane colour:	Pink
Capillary refill time (seconds):	1–2
Rectal temperature:	>37.5°C
Mean arterial Pressure (mm Hg):	70–80
Systolic blood pressure (mm Hg):	100
Urine output (mL/kg/h): **(not to be used in isolation as a marker of hypovolaemia)**	>1 >2 (if on fluids)
SpO2 (%):	>95
***SvO2 Mixed venous O_2 saturation (%):** ***PcvO2 (mmHg):**	>70 >35mmHg
***Lactate (mmol/L):**	<2
***Base deficit (mEq/L):**	> -4

***Downstream parameters**

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- **Treatment of hypovolaemic shock:**
  - ✓ **1. Oxygen therapy** and treatment of any causes of respiratory distress: e.g. thoracentesis, intubation and ventilation, airway suction, tracheostomy
  - ✓ **2. Increase circulating volume:**
    - ➢ See above **"Principles of Circulatory Support"**
  - ✓ **3. Analgesia** if pain is present; **see "Anaesthesia and Analgesia"**
  - ✓ **4. Re-assess PCV and TP:**
    - ➢ TP if <45g/L or low oncotic pressure, consider colloids or whole blood
    - ➢ PCV if <30% or anaemic/haemorrhagic patients, consider pRBC or whole blood and monitor PCV and lactic acid levels
  - ✓ **5. External warming to 37.5°C**
  - ✓ **6. Assess end point resuscitation variables**, if fulfilled then commence replacement therapy
  - ✓ **7. If end point resuscitation variables are not fulfilled:**
    - ➢ Trial more fluids and reassess
    - ➢ Stop ongoing haemorrhage:
      - Abdominal counter pressure to reduce internal haemorrhage
      - Emergency surgical intervention
    - ➢ Administer vasopressors to increase blood pressure:
      - Dopamine (vasopressor): 5-20µg/kg/min IV
      - Dobutamine (positive inotrope): 5-10µg/kg/min IV

- **Treatment of distributive shock:**
  - ✓ IV fluids as per **hypovolaemic shock**
  - ✓ If **septic shock:**
    - ➢ **Broad spectrum antibiotic therapy** – combination of:
      - Ampicillin or cephalexin + enrofloxacin +/- metronidazole
      - Cultures of blood, urine, tissue
      - Monitor development of multiple organ dysfunction syndrome
  - ✓ If **systemic anaphylactic shock:**
    - ➢ **Oxygen therapy**
    - ➢ **Adrenaline** for inotropic and vasopressor effects:
      - 0.005mg/kg IV:
        - 0.05ml/10kg of 1:1000
        - 0.5ml/10kg of 1:10,000
      - ↑ Heart rate and contractility
      - Vasoconstriction and splanchnic and skeletal vasodilation
      - Contraindicated in arrhythmias
    - ➢ +/- Atropine 0.04mg/kg IV:
      - To counteract bradycardia and hypotension
    - ➢ **Corticosteroids:** Prednisolone sodium succinate 2mg/kg IV TID
    - ➢ +/- Anti-histamine: Chlorpheniramine 0.5mg/kg IV

- **Treatment of cardiogenic shock:**
  - ✓ **Oxygen therapy**
  - ✓ Positive inotropic **cardiac support** if reduced cardiac motility:
    - ➢ Dobutamine: 5-20µg/kg/min (dogs) CRI
  - ✓ **Cats with hypertrophic cardiomyopathy** (not if reduced myocardial contractility):
    - ➢ Calcium channel blocker: 1.75-2.4mg/kg PO TID
    - ➢ β-blocker: Propranolol 0.05-0.1mg/kg IV

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- ✓ **Ventricular arrhythmias** (slow IV):
  - ➢ Lignocaine 2%: 2mg/kg IV, q10 minutes IV repeated up to 8mg/kg, followed by 40-80µg/kg/min CRI (cats use at 1/10th of dog dose and give slowly)
- ✓ **Diuretic therapy** if CHF and pulmonary oedema (not if pericardial effusion):
  - ➢ Furosemide: 2-6mg/kg IV (dog), 1-2mg/kg IV (cat) given to effect
- ✓ **Vasodilator therapy** – to reduce afterload
  - ➢ Sodium nitroprusside: 5-10µg/kg/min IV CRI
  - ➢ Nitroglycerine patches

- **Treatment of obstructive shock:**
  - ✓ IV fluids as per **hypovolaemic shock**
  - ✓ **Must relieve obstruction to blood flow:**
    - ➢ GDV: Relieving gastric distension with either the passage of a stomach tube or centesis
    - ➢ Tension pneumothorax: Thoracentesis
    - ➢ Pericardial effusion: Pericardiocentesis

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Skeletal Disease

- **This chapter covers:**
  - ✓ Osteoarthritis
  - ✓ Spinal, elbow, hip and stifle disease
  - ✓ Diagnosis and management
  - ✓ Fracture assessment and classification
- **See "Radiology" for radiographic features**

Osteoarthritis:

- **Features:**
  - ✓ Degenerative changes in the joints typically in older animals but also seen in younger animals that are obese, predisposed due to breed, sustained injury or skeletal abnormality
- **Clinical signs:**
  - ✓ Lameness or limb favouring
  - ✓ Reduced mobility, especially after laying down for a period of time or during cold weather
  - ✓ Reduced activity levels
  - ✓ Difficulty raising up, walking up stairs
  - ✓ Pain on palpation or manipulation of limbs
- **Differential diagnosis:**
  - ✓ Diagnosis of arthritis is important as it enables institution of appropriate therapy and help eliminate other causes of pain:
    - ➢ Neoplasia
    - ➢ Soft tissue damage
    - ➢ Fracture/dislocation
    - ➢ OCD
    - ➢ Osteomyelitis, joint inflammation/sepsis/immune mediated disease
- **Diagnostics:**
  - ✓ General physical examination: Including palpation and flexion and extension of joints
    - ➢ Pain and swollen inflamed joints and reduced range of motion
  - ✓ Radiographs: Secondary bony change e.g. osteophyte formation
  - ✓ Response to pain relief medication and anti-inflammatories
  - ✓ Haematology, biochemistry and urinalysis: Assess patient's suitability for pain medication (e.g. renal function) and systemic involvement
  - ✓ Joint fluid analysis: Normal to increased volume, macrophages may be prominent, few neutrophils
- **Follow-up:**
  - ✓ Repeat biochemistry and urinalysis every 6 months to monitor kidney function if on chronic anti-inflammatory therapy
- **Treatment options:**
  - ✓ Multi-modal therapy is more effective than single modal therapy
  - ✓ A combination of the options below tailored to the particular patient and the client's funds
  - ✓ **Fast acting treatment modalities for mild to moderate pain:**
    - ➢ NSAIDs
    - ➢ Tramadol up to 5mg/kg BID-TID, can use as an "escape drug", beware of constipation
  - ✓ **Fast acting treatment modalities for severe chronic pain, i.e. when uncontrolled with NSAIDs and Tramadol combination:**
    - ➢ NMDA-antagonists: Help reduce CNS wind-up (hypersensitivity to pain) due to chronic pain:
      - Amantadine 1-4mg/kg PO QID
        - Can use to bring chronic pain under control (2 – 4 weeks) but may need to be on indefinitely, use in conjunction with other analgesics
      - Gabapentin 3-5mg/kg PO SID-BID but up to 10mg/kg
        - For significant pain, increase dose in 2 week intervals until good pain relief

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- Opioids:
  - Methadone, fentanyl patch, buprenorphine
- Corticosteroids:
  - Intra-articular injections of long acting corticosteroids - last choice (can cause diabetes)
  - Must look at joint fluid under microscope if infectious/>10% neutrophils do not inject

✓ **Slower acting treatment modalities:**

- **Weight loss:**
  - Very important, can alter the clinical picture dramatically
- **Sensible exercise:**
  - Very important, maintain muscular support of the joints
  - Regular low intensity exercise, hydrotherapy
- Disease Modifying Osteoarthritis Drugs:
  - Pentosan polysulfate sodium
  - Loading phase then a maintenance phase:
    - Increase cartilage production, increase volume and quality of synovial fluid
- Nutraceuticals:
  - Glucosamine and chondroitin: Has a 4 week loading phase then a maintenance phase:
    - Example dosages: Dogs: 15-30mg/kg/day, Cats: 300mg/day
    - Provides the building blocks for cartilage repair
  - Omega 3 fatty acids:
    - 20mg/kg PO SID
  - Eicosapentaenoic acid (EPA): Helps reduce inflammation
    - Fish oils and joint diets
- Diet:
  - EPA containing diets "joint diets", should see effect within 2 months
- Acupuncture
- Other:
  - Physiotherapy: Slow manipulation of joints to relax stiff muscles and gentle kneading of muscles combined with warm wet towels/hot water bottle to improve circulation
  - Elevate food bowel
  - Sideless kitty litter trays: If can't get into kitty litter

- **Presentations:**
- **<u>Acute</u> presentation AND <u>chronic low-grade</u> presentation:**

✓ Rescue treatment required to alleviate pain and reduce inflammation whilst longer term therapy is instituted and given time to work:

- NSAIDs +/- tramadol up to 5mg/kg BID-TID
- Start pentosan polysulfate sodium and nutraceuticals
- Rest then sensible exercise and weight loss

- **Chronic low-grade <u>intermittent</u> presentation:**

✓ Rescue treatment is not generally required, long term therapy is instituted and usually sufficient to keep comfortable:

- Start pentosan polysulfate sodium and nutraceuticals
- Rest then sensible exercise and weight loss
- NSAIDs +/- tramadol up to 5mg/kg BID-TID
  - When needed i.e. intermittent pulse therapy when presents lame or uncomfortable
  - In anticipation of high amounts of activity i.e. going to the beach, give NSAIDs the day before, the day of and the day after going to the beach

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Spinal Disease:

- **Differentials for spinal pain:** See also **"Neurological Disease"**
  - ✓ Inflammatory CNS disorders **(see previous pages)**
  - ✓ Discospondylitis
  - ✓ Vertebral malformation
  - ✓ Polyarthritis or osteoarthritis of joint facets
  - ✓ Intervertebral disc disease
  - ✓ Caudal cervical spondylomyelopathy (Wobbler syndrome)
  - ✓ Cauda equine syndrome (Lumbosacral stenosis)
  - ✓ Neoplasia
  - ✓ Vascular: Fibrocartilaginous emboli, infarct
  - ✓ Trauma: Fracture/luxation
  - ✓ Polyradiculoneuritis (idiopathic immune mediated) inflammation of spinal nerve roots
  - ✓ Parasitic migration: e.g. *Anglostrongylus cantonensis* causing eosinophilic meningitis

- **Localising site of damage:**

Segments:	Functions:
Cervical C1-C5	▪ UMN to all limbs ▪ Urinary incontinence
Cervicothoracic C6-T2 **Brachial plexus**	▪ LMN to forelimb and UMN to hindlimbs ▪ Horner's syndrome ▪ Root signature
Thoracolumbar T3-L3	▪ UMN to hindlimbs ▪ Urinary incontinence (UMN bladder) ▪ ***Schiff Sherrington posture*** – rigid extension of forelimb and flaccid paralysis of hindlimbs
Lumbosacral L4-S3 **Lumbosacral plexus**	▪ LMN loss to hindlimbs ▪ Loss of perineal reflexes/sensation ▪ Urinary and faecal incontinence (LMN bladder) ▪ Root signature
Sacral S1-S3	▪ Normal all limbs ▪ Loss of sciatic function ▪ Loss of perineal reflexes/sensation

- **Depth of damage and prognosis:**

Loss Of:	Depth of damage:	Prognosis:
Proprioception	▪ Superficial	▪ Good
Voluntary motor	▪ Superficial	▪ Fair
Above and **cutaneous pain**	▪ Middle	▪ Moderate
Above and **deep pain**	▪ Deep	▪ Poor

- **Principles of spinal disease:**
- **Diagnosis:**
  - ✓ Physical and neurological examination
  - ✓ Radiographs:
    - ➢ Plain radiographs: Narrowing of IVD space, tilting of vertebrae
    - ➢ Myelogram: To identify site of compression
  - ✓ CSF tap and
  - ✓ Advanced imagery e.g. CT / MRI

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- **Management:**
  - ✓ Medical management:
    - ➢ Can be trialled for patients with mild proprioceptive deficits, weakness, ataxia:
      - Strict rest (cage confinement)
      - Pain relief combination of anti-inflammatories and opioids
      - Supportive care:
        - o +/- Bladder expression
        - o Physiotherapy
  - ✓ Surgery:
    - ➢ If severe neurological deficits (no deep pain), repeat episodes, failure to manage conservatively

Specific conditions:

- **Intervertebral disc disease (IVDD):**
- **Pathophysiology:**
  - ✓ Type 1: Chondroid metaplasia
    - ➢ See in young to middle aged 2-7 years
    - ➢ In chondrodystrophic breeds, changes in the IVD cause it to become less shock absorbent
    - ➢ Sudden extrusion of nuclear disc material through a ruptured dorsal annulus, usually triggered by an acute event, but is predisposed to by chronic changes affecting the IVD
    - ➢ Can get IVD mineralisation
    - ➢ Hansen type I disc protrusion: High velocity large mass
  - ✓ Type 2: Fibroid metaplasia
    - ➢ Age related change seen when older. See in older animals 8-10years
    - ➢ Progressive fibroid changes in the IVD cause the nucleus to become more soft leading bulging but no release of disc material
    - ➢ Rarely get IVD mineralisation
    - ➢ Hansen type II disc protrusion: Low velocity small mass
- **Cervical disc disease:**
  - ✓ Features:
    - ➢ Type I: Dachshunds, poodles, beagles, spaniels
    - ➢ Type II: Doberman pinschers
    - ➢ Most commonly at C2-C4
  - ✓ Clinical signs:
    - ➢ Severe neck pain and altered stiff gait
    - ➢ Lower head carriage and neck guarding
    - ➢ Muscle spasm of neck and shoulder muscles
    - ➢ Forelimb root signature pain: Neuropathic pain in a limb caused by compression of nerve roots to the affected limb, indicates C4-C7 lesion
    - ➢ Can get paresis and paralysis, worse in hindlimbs and can be unilateral in forelimbs
  - ✓ See above for diagnosis and treatment

Thoracolumbar disc disease:

- ✓ Features:
  - ➢ Most common cause of intervertebral disc disease (80%)
  - ➢ Most common at T11-12
- ✓ Clinical signs:
  - ➢ Neck and back pain
  - ➢ Rigid back with "saw horse" appearance
  - ➢ Hindlimb paresis with altered proprioception
  - ➢ +/- Urinary incontinence:
    - UMN: Firm, hard to express, LMN: Soft, easy to express
- ✓ See above for diagnosis and treatment

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- **Cauda equine syndrome (lumbosacral stenosis):**
- **Pathophysiology:**
  - ✓ Compression of the spinal nerve roots at the cauda equine region usually the lumbosacral joint (L7-S1) due to stenosis of spinal canal or nerve root canal either due to bony or soft tissue changes:
    - ➢ L4 vertebrae houses L5-L7 spinal segments
    - ➢ L5 vertebrae houses S1-S3 spinal segments
    - ➢ L6 vertebrae houses coccygeal spinal segments
- **Clinical signs:**
  - ✓ Lumbosacral pain
  - ✓ Hindlimb weakness: Slow to rise, does not want to jump or go up stairs
  - ✓ Tail pain, weakness, paralysis
  - ✓ +/- Incontinence: Urinary or faecal incontinence +/- LMN bladder
- **Diagnostics:** See previous page
  - ✓ Radiographs +/- myelogram:
    - ➢ Narrowing of the intervertebral disc space
    - ➢ Ventral spondylosis: If present then likely to be clinically relevant if consistent clinical signs (but require further diagnostics)

- **Caudal cervical spondylomyelopathy (Wobbler syndrome):**
- **Pathophysiology:**
  - ✓ Compression of the spinal cord and spinal nerve roots typically at C4-5, C5-6, C6-7
  - ✓ Usually seen in large dogs - Doberman pinschers, Great Danes, Rottweiler, Poodles
  - ✓ Can due to one or a combination of:
    - ➢ Congenital osseous malformation – Great Danes
      - Malformation and malarticulation of the vertebrae and articular facets
    - ➢ Cervical vertebral instability and vertebral tipping – Dobermans
      - Malposition of the vertebra caudal to the affected disc
    - ➢ Thickening of the ligamentum flavum and disc protrusions – Great Danes
      - Thickening of the soft tissue structures leads to compression
- **Clinical signs:**
  - ✓ Progressive development of cervical pain leading to paresis and ataxia of the hindlimbs but usually normal forelimbs, can progress to tetraparesis and proprioceptive deficits
  - ✓ Cervical pain and guarding, lowered head carriage
- **Diagnostics:** See above
  - ✓ Radiographs and myelogram:
    - ➢ Narrowing or wedging of the intervertebral disc space, vertebral tipping C4-C7
    - ➢ Ventral spondylosis: If present then likely to be clinically relevant if consistent clinical signs (but require further diagnostics)

- **Discospondylitis:**
- **Pathophysiology:**
  - ✓ Infection of the intervertebral disc: Either due to bacteria or fungi (female German Shepherds)
  - ✓ *Staphylococcus spp* is the most commonly isolated organism
  - ✓ Usually large dogs especially German Shepherds
- **Clinical signs:**
  - ✓ Neck or back pain
  - ✓ Lameness and altered gait
  - ✓ Lethargy, Inappetance and pyrexia

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- **Diagnostics:**
  - ✓ Radiographs:
    - ➢ Lysis of vertebrae and end plates and sclerosis of adjacent bone
    - ➢ Narrowing of the intervertebral disc space due to collapse
    - ➢ +/- Ventral spondylosis
  - ✓ CSF tap, blood and urine culture
- **Treatment:**
  - ✓ Long term antifungals or antibiotics several months

Elbow disease:

- **Causes:**
  - ✓ Elbow incongruity (developmental malarticulation)
  - ✓ Osteochondrosis dissecans of medial humeral condyle
  - ✓ Ununited anconeal process (UAP)
  - ✓ Fragmented medial coronoid process (FCP)
- **Signalment:**
  - ✓ Young, usually 4-8 months old (can be later 2-3 years)
  - ✓ Large to giant breed
  - ✓ Male > female
- **Clinical signs:**
  - ✓ Forelimb lameness
  - ✓ Worse with strenuous exercise or after rest
  - ✓ May be bilateral
- **Physical examination findings:**
  - ✓ Reduced range of motion
  - ✓ Effusion palpable between epicondyles and olecranon
  - ✓ +/- Crepitus
  - ✓ +/- Periarticular thickening
- **Diagnostics:**
  - ✓ Radiographs:
    - ➢ Both elbows
    - ➢ 3 standard views:
      - Relaxed mediolateral view with an inside angle of approximately 120°
      - Flexed mediolateral view with an inside angle of 45°
      - Craniocaudal view
    - ➢ Other views:
      - Craniocaudal view with 15° pronation (highlights medial coronoid process)
      - 90° flexed lateral – best for assessing elbow incongruity
  - ✓ Advanced imagery: CT/MRI

- **Elbow incongruity (developmental malarticulation):**
- **Pathophysiology:**
  - ✓ Due to uneven growth between the radius and the ulna
  - ✓ Physeal trauma or congenital disease
  - ✓ Increase pressure on the medial coronoid process, anconeal process and medial condyle of the humerus, potentially leading to:
    - ➢ Ununited anconeal process (UAP)
    - ➢ Fragmented medial coronoid process (FCP)

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- **Osteochondrosis dissecans of medial humeral condyle:**
- **Pathophysiology:**
  - ✓ Disturbance in endochondral ossification leads to the retention of epiphyseal cartilage, weakness in the subchondral bone leading causing a dissecting flap of articular cartilage and secondary inflammatory joint changes
  - ✓ Possible causes: Hereditary, trauma, rapid growth, ischaemia, nutrition
- **Specific features:**
  - ✓ +/- Valgus of the carpus
  - ✓ Pain on deep palpation over the medial collateral ligament or by stressing the ligament by flexing the carpus 90 degrees and rotating the foot laterally
- **Radiographic features:**
  - ✓ Radiolucent defect on the medial humeral condyle
  - ✓ Secondary osteoarthritic change
- **Treatment:**
  - ✓ Conservative:
    - ➢ If diagnosed early (<6 months) may respond to conservative therapy
    - ➢ Strict rest, physical therapy
    - ➢ NSAIDs, pentosan polysulfate sodium and glucosamine and chondroitin sulphate
    - ➢ Weight reduction
  - ✓ Surgery:
    - ➢ If lame after 7 months of age may benefit from surgery, can speed recovery and minimise development of osteoarthritis
    - ➢ Older animals with significant osteoarthritis, surgery may be unrewarding

- **Ununited anconeal process:**
- **Pathophysiology:**
  - ✓ Failure of osseous union between the anconaeus and the proximal ulnar metaphysis by 20 weeks of age, larger dogs may take up to 24 weeks
  - ✓ Possible causes: OCD lesion, trauma (hyperextension), developmental joint incongruity (short ulna, elliptical trochlea notch)
  - ✓ Can have concurrent FCP
- **Specific features:**
  - ✓ Front paws point laterally and elbows abducted
  - ✓ +/- Pain over the anconeal process with the elbow in flexion with deep digital flexion
- **Radiographic features:**
  - ✓ Best visualised on flexed lateral view
  - ✓ Separation between anconaeus and ulna can be seen +/- osteoarthritic changes
- **Treatment:**
  - ✓ Conservative:
    - ➢ Weight reduction, strict rest, physical therapy
    - ➢ NSAIDs, pentosan polysulfate sodium and glucosamine and chondroitin sulphate
  - ✓ Surgery: Indicated if not firmly attached (i.e. anchored with cartilage) or completely loose

- **Fragmentation of the medical coronoid process:**
- **Pathophysiology:**
  - ✓ Fragmentation of the medical coronoid process
  - ✓ Possible causes: OCD lesion, trauma (hyperextension), developmental joint incongruity (short radius, elliptical trochlea notch)
- **Specific features:**
  - ✓ Usually older, 8-10 months old but up to 3 years
  - ✓ Front paws point laterally and elbows adducted

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- ✓ Painful especially on external rotation of the paw with the elbow in hyperextension also painful over the medical collateral ligament
- ✓ Diagnosis of exclusion, rule out UAP and OCD

- **Radiographic features:** Only moderate to severe cases
  - ✓ +/- Secondary osteoarthritic changes, associated with the medial coronoid process and anconeal process
  - ✓ +/- Sclerosis of the trochlear notch
  - ✓ +/- Abnormal contour or lack of visualisation of the medial coronoid process
- **Treatment:**
  - ✓ Conservative:
    - ➢ Strict rest, physical therapy
    - ➢ NSAIDs, pentosan polysulfate sodium and glucosamine and chondroitin sulphate
    - ➢ Weight reduction
  - ✓ Surgery:
    - ➢ Surgery is treatment of choice for most young dogs without advanced osteoarthritis

Hip Disease:

- **Causes:**
  - ✓ **Avascular necrosis**
  - ✓ **Hip dysplasia**
  - ✓ **Dislocation**
  - ✓ **Femoral capital physeal fractures**
- **See "Radiology" for radiographic features**

- **Avascular necrosis aka Legg-calve-perthes disease:**
- **Pathophysiology:**
  - ✓ Young, usually <10 months, small breed dogs
  - ✓ Progressive lameness, usually unilateral but can be bilateral
  - ✓ Trabecular bone of the femoral head degenerates and collapses on itself leading to lameness and secondary degenerative changes in the joint
  - ✓ Radiographic signs:
    - ➢ Lack of head congruence
    - ➢ Lack of parallelism between head and acetabula and squaring of the femoral head
    - ➢ Thickening of the neck of the femur
    - ➢ Secondary arthritic changes
- **Treatment:**
  - ✓ Pain relief (NSAIDs) and strict rest, until surgery
  - ✓ Femoral head ostectomy (definitive)
  - ✓ Physiotherapy

- **Hip dysplasia:**
- **Pathophysiology:**
  - ✓ Abnormal development and confirmation of the coxofemoral joint, leading to the development of secondary degenerative joint changes
  - ✓ Usually large breed dogs, especially Labradors, Golden Retrievers, Rottweilers, German Shepherds
  - ✓ Rapid growth and weight gain can lead to earlier and more severe expression
- **Diagnostics:**
  - ✓ Tests: Ortolani laxity test
  - ✓ Radiographs:
    - ➢ Lack of head congruence between the femoral head and acetabulum
    - ➢ Femoral head squaring, femoral neck thickening, secondary degenerative joint changes

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- **Treatment:**
  - ✓ Medical:
    - ➢ Pain relief (NSAIDs) +/- opioids (Tramadol)
    - ➢ Strict rest
    - ➢ Weight loss
    - ➢ Physiotherapy and hydrotherapy to maintain joint movement and muscle tone
    - ➢ Chondroprotective agents:
      - Pentosan polysulfate sodium: Weekly for 4 weeks then monthly for 3 months
      - Glucosamine and chondroitin sulphate
  - ✓ Surgical:
    - ➢ Procedures performed in young dogs to improve hip structure and joint congruency:
      - Juvenile pubic symphysiodesis:
        - Artificial premature fusion of the pubic symphysis
        - Must be performed early for maximum effect <4-5 months but can be difficult to assess the joint
      - Triple pelvic osteotomy:
        - Dogs younger than 12 months with no degenerative joint disease
    - ➢ Procedure performed in older dogs as salvage procedures:
      - Femoral head ostectomy:
        - Best if dogs under 20kg
        - Improved outcome if limb use is encouraged combined with active physiotherapy
      - Dorsal acetabular neurectomy:
        - Desensitisation of the coxofemoral joint by cutting sensory nerve supply (Cranial gluteal nerve)
        - Sensation can return over time
      - Total joint replacement

- **<u>Hip dislocation:</u>**
- **Pathophysiology:**
  - ➢ Craniodorsal dislocation is significantly more common compared to caudoventral
  - ➢ Seen typically in mature dogs, usually due to traumatic injury
- **Clinical signs:**
  - ➢ Acute onset non-weight bearing lameness
  - ➢ Affected limb is held off the ground, usually toe point in
  - ➢ Leg length discrepancy, distance between greater trochanter and ischium is greater if hip forward
- **Diagnosis:**
  - ➢ Radiographs (lateral and ventrodorsal views)
- **Treatment:**
  - ➢ Closed reduction:
    - The more easily the limb re-luxates the less likely it will remain stable once reduced
    - Place limb in an Ehmer sling to allow for join to heal, place for at least 2-3 weeks
    - Repeat radiographs after placing a sling to ensure the hip is correctly reduced
    - Limb may luxate again
  - ➢ Open reduction:
    - Surgical reduction, for limbs that easily re-luxate
    - Numerous techniques and some used in combination e.g. Toggle-pin fixation, capsular repair, femoral head ostectomy etc.

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Stifle Disease:

- **Causes:**
  - ✓ **Cranial cruciate ligament rupture**
  - ✓ **Patella luxation**
  - ✓ **Osteochondrosis dissecans of the femoral condyle (not covered)**
- **See "Radiology" for radiographic features**

- **Cranial cruciate ligament rupture:**
- **Pathophysiology:**
  - ✓ Loss of support results in the tibia moving cranially (in relation to the femur)
  - ✓ Commonly medial meniscus is damages as well
  - ✓ Partial tears are common, and are a common cause of chronic lameness without instability
  - ✓ Rupture can occur acutely due to increased activity, or progressively over time due to degeneration leading weakness
- **Clinical signs:**
  - ✓ Acute onset non-weight bearing lameness with toe out
  - ✓ Chronic intermittent lameness, then sudden lameness
- **Diagnosis:**
  - ✓ Orthopaedic exam:
    - ➢ Cranial drawer test: Best done under anaesthesia or heavy sedation
    - ➢ Tibial thrust: Best done under anaesthesia or heavy sedation
    - ➢ Joint effusion
    - ➢ Chronic changes include periarticular thickening on the medial aspect of the tibial head, muscle atrophy and reduced range of motion
  - ✓ Radiographs:
    - ➢ Joint effusion, +/- osteophytosis (if chronic)
- **Treatment:**
  - ✓ See table below to help decision making process
  - ✓ Conservative:
    - ➢ <5kg can trial conservative therapy, 50% return to normal activity after 6 months
    - ➢ Strict rest 1-2 months
    - ➢ Physiotherapy to maintain range of motion
    - ➢ Anti-inflammatories
    - ➢ Chondroprotective agents:
      - Pentosan polysulfate sodium: Weekly injections for 4 weeks
      - Glucosamine and chondroitin sulphate: Daily supplementation
  - ✓ Surgical:
    - ➢ Return to normal function faster
    - ➢ Reduced progression of degenerative joint disease
    - ➢ Lateral stabilisation technique:
      - De Angelis suture - mimics cranial cruciate function
    - ➢ Osteotomies: Tibial plateau levelling techniques +/- tibial crest advancement
      - Tibial plateau levelling osteotomy (TPLO)
      - Triple tibial osteotomy (TTO)
      - Tibial wedge ostectomy (TWO)

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	De Angelis:	Osteotomies:
Features:	▪ Simple surgery ▪ Simple equipment ▪ Relatively inexpensive	▪ Complicated procedure ▪ Need special equipment ▪ Costly
Suitability:	▪ Dogs less than 20kg ▪ Minimal degenerative joint disease ▪ Minimal tibial slope	▪ Any breed ▪ Any size ▪ Any tibial slope ▪ Any degree of degenerative joint disease
Outcome:	▪ Reduces cranial drawer signs but tibial thrust not as much ▪ Can rupture the suture in larger or very active dogs ▪ Reduced progression of degenerative joint disease compared to conservative but not as good as osteotomy	▪ Reduces tibial thrust but not cranial drawer sign as much ▪ Reduced progression of degenerative joint disease ▪ Faster return to weight bearing ▪ Better long term outcome and function

- **Patella luxation:**
- **Pathophysiology:**
  - ✓ Displacement of the patella:
    - ➢ Small breeds most commonly medially, large breeds laterally
  - ✓ Usually middle-aged small breed dogs
  - ✓ Can have concurrent cranial cruciate rupture
  - ✓ Progressive disease, can get worse with age
  - ✓ Due to a combination of bone deformities:
    - ➢ Lateral rotation and bowing of femur
    - ➢ Femoral epiphyseal dysplasia
    - ➢ Shallow trochlear groove
    - ➢ Hypoplasia of medial trochlear ridge
    - ➢ Tibial tuberosity displaced medially
    - ➢ Proximal tibial epiphyseal dysplasia
    - ➢ Medial torsion and bowing of proximal tibia
- **Clinical signs:**
  - ✓ Intermittent skipping or leg locking
  - ✓ +/- Bowed legged (genu varum)
  - ✓ +/- Lameness
- **Diagnostics:**
  - ✓ Physical examination:
    - ➢ Palpable luxation of the patella on flexion of the leg
    - ➢ Assess for concurrent cruciate rupture
  - ✓ Radiography:
    - ➢ Not required for diagnosis, but can be used to determine severity or presence of concurrent disease

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✓ **Grading:**

➢ Based upon position of patella, how mobile it is, degree of bony deformity

Grade:	Palpable abnormalities:
I	▪ Hypermobile ▪ **Immediately returns after displacement** ▪ Minimal tibial tuberosity deviation ▪ Asymptomatic
II	▪ **Luxates when walking** ▪ Relocates with extension of stifle ▪ Tibial tuberosity is deviated up to 30 degrees medially ▪ Mildly bow legged ▪ Intermittent non-weight bearing
III	▪ **Luxated most of time** ▪ **Requires digital pressure to reduce but luxates immediately** ▪ Tibial tuberosity is deviated up to 60 degrees medially ▪ Moderate bow legged +/- flexed stifles ▪ Frequently non-weight bearing
IV	▪ **Permanently luxated** ▪ Unable to be reduced with digital pressure ▪ Tibial tuberosity is deviated >60 degrees medially ▪ Severe bow legged and flexed stifle

- **Treatment:**

✓ Conservative:

➢ Grade 1, without lameness

➢ Monitor every 6 months, surgery if increases in grade

➢ Pain relief: NSAIDs +/- tramadol up to 5mg/kg BID-TID

➢ Strict rest

➢ Weight loss

➢ Physiotherapy and hydrotherapy to maintain joint movement and muscle tone

➢ Chondroprotective agents:

- Pentosan polysulfate sodium: Weekly injections for 4 weeks
- Glucosamine and chondroitin sulphate: Daily supplementation

✓ Surgical:

➢ Combination of a number of the following techniques

➢ Soft tissue reconstruction:

- Grade 1 and used in all other grades with another technique
- Lateral joint capsule and fascia lata imbrications: Tightening of the lateral joint capsule to reduce medial movement
- +/- Medial releasing desmotomy: Reduce tension on the patella on the medial side

➢ Bone reconstruction:

- Grades 2-4
- Sulcoplasty: Deepening of the trochlear groove by removing bone
- Chondroplasty: Deepening of the trochlear groove by cartilage, only if less than 5 month old
- Tibial tuberosity translocation: Transposition of the patella ligament insertion so that it runs in line with the trochlear groove
- +/- Corrective osteotomy of femur/ tibia: Only when severe bone deformity

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Fracture assessment:

- **General physical examination:**
  - ✓ Assess the patient's stability and stabilise accordingly:
    - ➢ Commence oxygen therapy
    - ➢ Commence IV fluids for shock
    - ➢ Administer pain relief
    - ➢ Stop any obvious bleeding
  - ✓ Assess for signs of trauma to the vital areas such as the chest, abdomen and central nervous system
  - ✓ Assess soft tissue injuries:
    - ➢ Lavage and place temporary moist sterile dressings until able to definitively assess
    - ➢ Administer IV antibiotics
- **Diagnostics:**
  - ✓ Haematology, biochemistry, blood gas: Assessment of systemic compromise
  - ✓ Radiographs:
    - ➢ Thoracic: Assess for pulmonary contusions, fractures, diaphragmatic hernia, pleural disease
    - ➢ General fracture assessment
  - ✓ Abdominal ultrasound: Free abdominal fluid, uroabdomen, haemabdomen, pleural effusion
- **Principles of fracture repair:**
  - ✓ Debridement: Only conservative debridement of bones, ligaments and tendons
  - ✓ Appropriate reduction of the fracture, accurate anatomical alignment and stabilisation
  - ✓ Minimise soft tissue trauma and ensure accurate aseptic technique
- **Principles of fracture healing needs:**
  - ✓ Provision of adequate blood supply
  - ✓ Elimination of overwhelming infection
  - ✓ Accurate anatomical alignment and stabilisation

- **Classification of fractures:**
  - ✓ According to type of fracture:
    - ➢ Pathological/non-pathological:
      - Pathological fractures are associated with regions of bone weakness, i.e. Lysis secondary to neoplasia or nutrition
  - ✓ According to stability stable/unstable
  - ✓ According to accompanying wound:
    - ➢ Closed fracture: If there is associated skin trauma even if not full thickness then treat as an open fracture
    - ➢ Open fracture
  - ✓ According to the extent of bone damage:
    - ➢ Complete fractures, incomplete
  - ✓ According to the direction of the fracture:
    - ➢ Transverse, oblique, spiral, comminuted
  - ✓ According to the location of the fracture:
    - ➢ Diaphyseal: Proximal, midshaft and distal thirds
    - ➢ Metaphyseal
    - ➢ Epiphyseal
    - ➢ Physeal fracture: Possible premature growth place closure and bone growth deformity
    - ➢ Salter–Harris classification of physeal fractures, see **"Radiology"**
  - ✓ According to fracture displacement:
    - ➢ Displaced and non-displaced

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Toxicology

- **This chapter covers:**
  - ✓ Common toxins, clinical signs and organ systems affected
  - ✓ Management of an intoxication

Clinical Sign:	Possible Intoxicants:
• **Seizures:**	• Cane toad • Organophosphate /Carbamate • Metaldehyde • Synthetic pyrethroids • Strychnine • Mycotoxins • Yesterday, today and tomorrow (*Brunfelsia* spp.) • Tobacco • *Duranta erecta* • Lead • Cyanobacteria • Chocolate • Cycads
• **Tremors:**	• Cane toad • Organophosphate / Carbamate • Synthetic pyrethroids • Tremorgenic mycotoxin • Macrocyclic lactone anthelmintic • Tobacco • Yesterday today and tomorrow (*Brunfelsia* spp.) • Cannabis • Macadamia nuts
• **Depression:**	• Lead • Cannabis
• **Paresis or paralysis:**	• Botulism • Ciguatoxin • Tetrodotoxin • Macadamia (due to polyarthritis)
• **Hyperaesthesia:**	• Chocolate • Tobacco
• **Hypersalivation:**	• Cane toad • Family *Araceae* (arum family) • Organophosphate/Carbamate • Synthetic pyrethroids • *Duranta erecta* • Tobacco • Macrocyclic lactone anthelmintic
• **Hepatotoxicity:**	• Paracetamol (cats) • Aflatoxins • Cycads
• **Haemolysis and or Haemoglobinuria:**	• Paracetamol (cats) • Onions and garlic • Snake envenomation • Lead
• **Coagulopathy:**	• Anti-coagulant rodenticides • Aflatoxins (due to liver failure) • Cycad seeds
• **Renal failure:**	• Ethylene glycol • Grapes • Lilies

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- **Common Poisons:**

Family Araceae **(arum family):** • Dieffenbachia spp. (dumb cane) • Zantedeschia spp. (arum lily, calla lily) • Alocasia brisbanensis (cunjevoi) • Monstera deliciosa (Oxalate raphide crystals)	
Organs systems: • Oral irritation	**Clinical Signs:** • Congestion +/- swelling of buccal mucosa/tongue • Excessive salivation/ drooling • Pain reactions (e.g. pawing at mouth)
Avocado (*Persea Americana*): • Persin	
Organs systems: • Respiratory • Gastrointestinal	**Clinical Signs:** • Dyspnea and coughing • Head and neck swelling • Brisket oedema • +/- Sudden death
Cannabis: • Signs may persist up to 48 hours	
Organs systems: • Neurological	**Clinical Signs:** • Depression but rapid arousal • Ataxia • Incoordination • +/- Hallucinations • +/- Tremors • +/- Vomiting
Cane toad:	
Organs systems: • Gastrointestinal • Cardiac • Neurological	**Clinical Signs:** • Hypersalivation and vomiting • Buccal mucosa hyperaemia • Dysrhythmia: ➢ Bradycardia ➢ Tachycardia • Ataxia • Tetany and seizures
Carbamate (snail bait) and Organophosphate (insecticides): • Due to acetylcholine esterase inhibition → ↑ Parasympathetic tone	
Organs systems: • Neurological: ↑ Parasympathetic tone	**Clinical Signs:** • Hypersalivation • Vomiting • Diarrhoea • Bradycardia • Dyspnoea (pulmonary oedema) • Miosis • Muscle tremors and tetany • Paralysis • Seizures

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Chocolate:

- Theobromine (part of the methylxanthines toxins)

Organs systems:

- Gastrointestinal
- Respiratory
- Neurological

TOXIC DOSE:

Type:	Dose:
Milk	50gm/10kg
Dark	20gm/10kg
Cooking	5gm/10kg

Clinical Signs:

- Vomiting
- Hyperthermia
- Hyperexcitability
- Tachycardia/pnoea
- Muscle rigidity and hyperreflexia
- Ataxia
- Seizures
- Can lead to cardiac failure if severe

Ciguatoxin:

- Tropical fish poisoning (Tetrodotoxin)

Organs systems:

- Gastrointestinal
- Neurological

Clinical Signs:

- Muscle and joint pain
- Vomiting
- Diarrhoea
- Weakness
- Paresis to paralysis

Cycad seeds (*Cycas revoluta*):

- All parts are toxic

Organs systems:

- Hepatic
- Gastrointestinal
- Coagulation

Clinical Signs:

- Inappetance
- Vomiting within couple hours
- Diarrhoea can be haemorrhagic
- Abdominal pain
- Bleeding
- Ataxia
- Seizures

Cyanobacteria:

Organs systems:

- Gastrointestinal
- Hepatic
- Renal
- Neurological

Clinical Signs:

- Vomiting and diarrhea (minutes)
- Abdominal pain
- Neurological signs (minutes to hours after exposure):
  - Salivation
  - Weakness/ataxia
  - Dyspnea
  - Seizures
  - Coma

Duranta erecta:

- Golden dewdrop, pigeonberry, skyflower

Organs systems:

- Gastrointestinal
- Neurological

Clinical Signs:

- Depression (drowsiness, drooped eyelids)
- Hypersalivation
- Bradycardia
- Melaena
- Seizures
- +/- Coma

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Ethylene glycol:
- Anti-freeze, cold packs

Organs systems:	Clinical Signs:
• Neurological • Gastrointestinal • Renal	• Depression and stupor 30 minutes • Ataxia and paresis • Anorexia • Vomiting • Polyuria/polydipsia • Anuria 12-72 hours • Seizures

Grapes ***(Vitis vinifera)*:**

Organs systems:	Clinical Signs:
• Gastrointestinal • Renal • **TOXIC DOSE** ➢ Reported 20gm/kg grapes BUT varies significantly between individuals ➢ 3mg/kg raisins	• Vomiting • Diarrhoea • Anorexia • Abdominal pain • +/- Polydipsia • Anuria

Lead:
- Paint (renovations), batteries

Organs systems:	Clinical Signs:
• Gastrointestinal • Neurological • Haematological	• Vomiting/diarrhoea • Altered behaviour – hyperaesthesia • Ataxia and tremors • Seizures • Blindness • Anaemia

Lilies (*Lilium* spp.):
- All parts are toxic
- Cats are most sensitive progress to renal failure, dogs primarily gastrointestinal signs

Organs systems:	Clinical Signs:
• Gastrointestinal • Renal	• Lethargy and depression • Vomiting • Anorexia • Polyuria • Anuria – 24-48 hours

Macadamia:

Organs systems:	Clinical Signs:
• Musculoskeletal (polyarthritis) • Gastrointestinal • Neurological **TOXIC DOSE:** • Average 4gm/kg • 5-20 kernels for a 10 kg dog	• Depression • Vomiting • Hyperthermia • +/- Joint pain and swelling • Posterior paresis • Ataxia and muscle tremors – 12 hours

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Macrocyclic lactone anthelmintic:

- Anthelmintic and insecticide/acaricide
- Preparations e.g. any preparation ending with "ectin" e.g. ivermectin
- Collies and Old English Sheepdogs increased sensitivity

Organs systems:	Clinical Signs:
• Neurological	• Hypersalivation • Ataxia • Tremors • Diarrhoea • Blindness

Metaldehyde:

- Snail baits

Organs systems:	Clinical Signs:
• Neurological • Musculoskeletal • Cardiovascular	• Tachycardia • Hyperthermia • Ataxia • Muscle tremors • Hyper-excitability • Nystagmus (cats) • Seizures

Mycotoxins:

- Aflatoxins
- Tremorgenic

Organs systems:	Clinical Signs:
• Aflatoxins: ➢ Gastrointestinal ➢ Hepatic ➢ Coagulation • Tremorgenic: ➢ Neurological	• Aflatoxins: ➢ Sudden death ➢ Anorexia ➢ Vomiting ➢ Jaundice ➢ Liver failure ➢ Coagulopathy • Tremorgenic: ➢ Panting ➢ Hyperexcitability ➢ Ataxia ➢ Muscle tremors ➢ Hyperthermia ➢ Seizures

Oleanders (Allamandas):

- Cardiac glycosides

Organs systems:	Clinical Signs:
• Cardiovascular • Gastrointestinal • Respiratory	• Sudden death • Diarrhoea +/- haemorrhagic • Dysrhythmias ➢ Tachycardia/bradycardia • Vomiting

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Onions and garlic:
- S-methylcysteine sulphoxide (SMCO)

Organs systems:	**Clinical Signs:**
• Haematological → Heinz body formation → haemolysis **TOXIC DOSE:** • Dogs: ➢ >0.05% of body weight (single dose) ➢ >10gm/kg daily over several days → anaemia • Cats: ➢ 25gm/kg daily over several days → anaemia	• Lethargy • Anorexia • Pigmenturia - haemoglobinuria • Pale mucous membranes • Jaundice

Organophosphates – see Carbamate toxicity above:
- Due to acetylcholine esterase inhibition → ↑ Parasympathetic tone

Paracetamol (cats)
- Acetaminophen

Organs systems:	**Clinical Signs:**
• Haematological: ➢ Heinz body formation → haemolysis ➢ Methaemoglobinaemia • Hepatic • Dogs: 70mg/kg • Cats: 10mg/kg	• Lethargy • Dyspnoea, tachypnoea • Tachycardia • Methaemoglobinaemia • Pale mucous membranes, cyanosis • Pigmenturia – Haemoglobinuria, haematuria • Oedema of face and paws • Jaundice (in 2-7 days)

Rodenticide: see "Coagulopathy"
- Reduced vitamin K → essential for activation of vitamin K dependant clotting factors
- Can get intoxication from eating poisoned rat

Organs systems:	**Clinical Signs: 1-5 days**
• Coagulation	• Pale mucous membranes • Haematomas • Dyspnoea +/- haemoptysis • Haemorrhaging from cuts, nose and mouth, into faeces and urine • +/- Lameness

Pyrethroids:
- Cats deficient in hepatic glucuronidation

Organs systems:	**Clinical Signs:**
• Neurological	• Tremors • Hypersalivation • Ataxia • Seizures

Tobacco (*Nicotiana tabaccum*):
- Part of the methylxanthines toxins

Organs systems:	**Clinical Signs:**
• Gastrointestinal • Neurological **TOXIC DOSE** for a 20 kg dog: • 1 cigarette (very sick), 10 cigarettes (death) • No antacids – acids inhibit absorption	• Vomiting • Diarrhoea • Muscle tremors • Hypersalivation • Constricted pupils • Hyperexcitability • Seizures

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Strychnine:	
Organs systems: ▪ Neurological	**Clinical Signs:** ▪ Muscle rigidity ▪ Tetany ▪ Seizures (tiranic) – triggered by stimuli ▪ Mydriasis
Yesterday, today, tomorrow *(Francisia Brunfelsia australis)*: ▪ Flowers change from violet to white in a couple days	
Organs systems: ▪ Gastrointestinal ▪ Neurological	**Clinical Signs:** ▪ Vomiting and diarrhoea ▪ Ataxia ▪ Muscle tremor and rigidity ▪ Opisthotonus ▪ Seizures ▪ Anisocoria and nystagmus

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- **Management of Poisoning:**

Telephone advice:

Owner protection:	▪ Patient may become aggressive ▪ They may bite unintentionally
Bring down immediately:	▪ Respiratory distress ▪ Weakness or neurological ▪ Severe vomiting ▪ Bleeding ▪ Mucous membrane pallor
Decontamination:	▪ **Oral:** ➢ Use a damp rag to wipe the mouth and gums ➢ Avoid using hose as can lead to aspiration and drowning ▪ **Ingestion:** ➢ Corrosive: • Dilute with milk or water, **do not** induce emesis ➢ Non-corrosive: • Recommend owner to bring down, emesis in veterinary clinic is safer and more effective • Attempt induction of emesis, provided that there are no contraindications o Salt slurry: 3 teaspoons and water o Syrup of ipecac: Dogs and cats 2-4ml PO o Washing soda crystals: Small amount into mouth o Liquid hand washing soaps: 2 teaspoons into mouth ▪ **Topical:** ➢ Owners should protect themselves from contact with the toxin before helping their pet animal ➢ Dry: e.g. Powders • Try to dust off or vacuum ➢ Wet: e.g. Anti-parasite preparations • Wash with copious amounts of water and hand washing soap or shampoo ▪ **Ocular:** ➢ Immediately flush eye for 20 minutes with copious amounts of clean water
Other instructions:	▪ Bring any vomited material and any topically applied or ingested products into the clinic

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B. Emergency assessment of a suspected intoxication:

Assess and stabilize vital signs:	▪ See **"Cardiopulmonary Resuscitation"** ▪ Airway: ➢ Provide oxygen supplementation ➢ Check airway patency ➢ Intubate if required +/- ventilation ➢ +/- Tracheostomy ▪ Breathing: ➢ +/- Commence manual ventilation ▪ Cardiovascular: ➢ Assess heart rate, rhythm and pulses ➢ Commence compressions if required ➢ Place IV catheter ➢ Administer cardioactive drugs: Adrenaline, atropine ➢ Administer IV fluids, bolus therapy titrated to effect ▪ Drugs: ➢ Neurological signs (seizures): • Diazepam 0.5m/kg IV can repeat ▪ **Other:** ➢ Assess body temperature: • Active cooling/warming
History:	▪ Toxin: ➢ Type, amount, route of exposure ▪ Clinical signs: ➢ What kind of clinical signs, and for how long? ▪ Prior heath issues • Concurrent medication
Physical examination:	▪ Perform a more thorough physical examination ▪ Collect blood/urine for diagnostics
Decontaminate:	▪ **Topical toxic agents:** ➢ Removal of topically applied agents e.g. pyrethroids ➢ Thorough washing and bathing with mild shampoo or hand washing liquid ▪ **Ingested toxic agents:** ➢ **Emesis induction:** • Contraindications: o Neurological derangements or obtunded o Reduced ability to protect airways from aspiration: E.g. Reduced gag reflex, laryngeal paralysis o Caustic substances: E.g. Petroleum products (petrol, kerosene, turpentine), dishwashing tablets, alkaline and acids • Agents: o Apomorphine (dogs only): 0.03mg/kg IV/IM o Xylazine (cats) but variable efficacy: 0.4-1mg/kg IM/SC o Reverse with yohimbine 0.25-05mg/kg IM ➢ **Gastric lavage:** • Life-threatening intoxications e.g. chocolate, snail baits • When emesis is contraindicated or has not been effective o Note: Gastric lavage is less effective than emesis • Contraindicated with hydrocarbons, corrosive substances • How: o Anaesthesia and placement of a cuffed endotracheal tube o Measure and mark a large bore tube from nose to last rib

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	○ Pass into oesophagus and confirm placement ○ Lavage warm water up to 10ml/kg into tube until moderately distended and then repeat until clear fluid returns ○ Administer activated charcoal 1-2gm/kg or equivalent ○ Kink tube before removal ➢ **Cathartics** (increase speed of passage through gastrointestinal tract and reduce absorption): • Sorbitol (usually in combination with activated charcoal): ○ Monitor for hypernatraemia and diarrhoea • Sodium sulphate (Glauber's salts): Avoid if have CHF • Magnesium sulphate (Epsom salts): Avoid if have CHF • Mineral oil (paraffin oil): not vegetable oil ➢ **Toxin immobilization** (reduce further toxin absorption): • Adsorbents: ○ **Activated charcoal** at 1-3g/kg every 6 hours: ○ Give repeat doses as most toxins undergo enterohepatic recycling ○ Combination product with a cathartic (e.g. Sorbitol) have added benefit ○ NOT useful against small molecules (alcohols, caustic substances, nitrates, petroleum distillates or heavy metals etc.) • Chelating/precipitating agents: ○ Sodium bicarbonate: Iron ○ Magnesium sulphate: Lead ○ Calcium salts: Fluoride ○ Tannic acid (tea): Alkaloids – use up to 2 liters of tea for gastric lavage
Specific antidote:	▪ If a specific antidote is available ▪ Specific enzyme inhibitors (e.g. 4-methylpyrazole for ethylene glycol) ▪ Specific antibodies (e.g. digitalis)
Detoxification and toxin excretion:	• Support and promote renal excretion: ➢ Induce diuresis with IV fluids ➢ Reduce urinary reabsorption (urinary catheter) e.g. Chocolate toxicity ▪ Reduce enterohepatic recycling of toxins: ➢ Repeat administration of adsorbents such as activated charcoal
Supportive care:	▪ Maintenance of hydration, normotension and normothermia ▪ Control of cardiovascular derangements • Control of acid-base derangements • Symptomatic treatment of neurological and gastrointestinal signs ▪ Promotion of normal urinary function

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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

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- **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

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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, i.e. 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

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- Results:
  - ✓ If either of the controls agglutinate, then cannot interpret the cross match
  - ✓ Cats:
    - ➢ Type A and B recipients: Both major and minor must be negative
    - ➢ If type AB recipient receiving type A blood: Major must be negative but if the minor is slightly positive that is expected
  - ✓ Dogs:
    - ➢ Ideally both major and minor cross matches are negative
    - ➢ Or select the donor with the least positive minor cross match
  - ✓ IF positive minor cross match: The blood is best given as "washed RBC" or packed red blood cells to remove as much of the plasma as possible

- **Administration of blood products:**
  - ✓ Premedication:
    - ➢ Chlorpheniramine 0.5mg/kg IM/SC
    - ➢ +/- Solu-delta cortef 10mg/kg IV
  - ✓ How much:
    - ➢ PCV after transfusion at least 25-30%
    - ➢ Rough guide = to increase PCV by 1 need 2ml/kg of whole blood or 1ml/kg of packed RBC

$$\text{Volume (ml)} = \frac{\text{Body Wt (kg)} \times 90 \text{ (dogs) or } 66 \text{ (cats)} \times (\text{target PCV} - \text{recipient PCV})}{\text{Donor PCV}}$$

  - ✓ How to administer:
    - ➢ Use a 170µm blood filter, can administer via peripheral catheter
    - ➢ Dilute packed RBC with at least 100mls of saline
    - ➢ **Avoid Ca+ containing fluids:**
      - Run with 0.9% saline or PlasmaLyte 148
    - ➢ To reduce risk of bacterial contamination should transfusion within 4 hours
    - ➢ Recheck PCV 1 hour post-transfusion
  - ✓ **Rate:**
    - ➢ **Start slow at 2ml/kg/hr for 15 minutes and monitor for reactions, then progressively double the rate until the desired rate is reached**
    - ➢ Suggested rates:
      - Cardiac disease/failure: 3ml/kg/hr
      - Normovolemic: Up to 10ml/kg/hr
      - Haemorrhagic shock: Can administer blood as a bolus at 5–10ml/kg IV repeat if necessary

Transfusion reactions:

- ✓ **Clinical signs:**
  - ➢ Collapse and shock: Pale mucous membranes, slow capillary refill time, hypotension, tachycardia
  - ➢ Vomiting, pyrexia, hypersalivation, coughing, angioedema, urticaria, haematuria, incontinence
  - ➢ Acute immunological reactions (<6 hours post-transfusion):
    - Intravascular haemolysis: Can lead to SIRS and DIC, renal failure
    - Anaphylactic reactions: Collapse and shock
    - Acute lung injury: Respiratory distress, bilateral pulmonary infiltrates and hypoxaemia
    - Urticaria: Due to release of histamine stimulated by mediators in donor plasma
  - ➢ Acute non-immunological reactions:
    - Old/damaged RBC's: ↑ K+, ↑ ammonia, ↑ H+ ions → hyperkalaemia, encephalopathy, acidosis
    - Bacteria contamination

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- Citrate toxicity: Leads to hypocalcaemia, see vomiting, tremors, seizures
- Volume overload: Tachypnoea, jugular distension/pulses, watery nasal discharge, pulmonary disease

➢ Delayed immunological reactions:
- Haemolysis

➢ Delayed lung injury (6-96 hours post-transfusion)

✓ **Treatment and investigation:**

➢ Stop transfusion:
- Baseline measurements: Capillary refill times, mucous membrane colour, heart rate, temperature, heart rate, blood pressures, SPO_2, ECG

➢ Supportive therapy:
- Treat distributive shock, see **"Shock and Anaphylaxis"**:
  - IV fluid shock therapy
  - Prednisolone sodium succinate up to 10mg/kg IV
  - Diphenhydramine 2mg/kg IV OR chlorpheniramine 0.5mg/kg IV
  - +/- Adrenalin 0.005mg/kg IV if severe shock:
    - 0.05ml/10kg of 1:1000
    - 0.5ml/10kg of 1:10,000
- Oxygen therapy

➢ Assess for haemolysis:
- Spin down samples from both the donor blood and the recipient
- Both samples: Acute non-immunological (suggestive of)
- Patient only: Acute immunological

➢ Assess for bacterial contamination of blood products → cytology

➢ If large volumes administered:
- Check patient K+ and Ca+ levels: Treat abnormalities, see **"Fluid Therapy"**
- If suspect circulatory overload: Diuretics, furosemide 1-2mg/kg IV

➢ Monitor urine:
- If haemoglobinuria: IV fluids, mannitol CRI to reduce risk acute kidney injury due to pigmenturia

➢ Urticaria and pyrexia (with no haemolysis or bacteria in donor blood):
- Administer anti-histamines, restart slowly when clinical signs resolve

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Urinalysis

- **This chapter covers:**
  - ✓ Collection of urine
  - ✓ Interpretation of findings on urinalysis, dipstick test
  - ✓ Interpretation of proteinuria

- **Collection:**
  - ✓ Maybe refrigerated (not frozen) for up to 12 hours and warmed to room temp before testing
  - ✓ Cystocentesis: Best for assessing urine especially if wanting to culture

- **Gross examination:**
  - ✓ Volume
  - ✓ Turbidity
  - ✓ Colour:
    - ➢ Red/cloudy: Blood
    - ➢ Yellow/brown: Bilirubin
    - ➢ Red/brown: Haemoglobin

- **Urine concentration:**
  - ✓ Morning sample is best, it is most concentrated
  - ✓ Repeat samples are required to accurately determine urine specific gravity

Urine specific gravity:	Differentials (proposed mechanisms):
<1.008 = Hyposthenuria Actively diluting	• Hyperadrenocorticism (↓ *ADH secretion and reduced activity*) • Hypercalcaemia (↓ *NaCl reabsorption, ↓ ADH sensitivity*) • Hepatic disease (↓ *Urea → medullary washout*) • Pyelonephritis (↓ *sensitivity to ADH due to endotoxins*) • Diabetes insipidus (↓ *ADH production or ↓ ADH activity at kidneys*) • Pyometra (↓ *sensitivity to ADH due to endotoxins*) • Hyperthyroidism (↑ *GFR – medullary washout, polydipsia*) • Psychogenic polydipsia (*primary polydipsia*) • IV fluids • Diuretics
1.008-1.013 = Isosthenuria Not concentrating	• Renal disease (↑ *GFR, osmotic diuresis and medullary washout*) And above
1.014-1.029 = Hypersthenuria but minimally concentrated (Inappropriate if concurrent dehydration)	• Hypoadrenocorticism (↓ *Aldosterone → ↓ NaCl reabsorption*) • Diabetes mellitus/renal glycosuria (*osmotic diuresis*) • Renal disease (↑ *GFR, osmotic diuresis and medullary washout*) • Hepatic disease (↓ *Urea → medullary washout*) • Hyperadrenocorticism (↓ *ADH secretion and reduced activity*)
1.030-1.045 = Hypersthenuria	• Normal or could be acute kidney injury
>1.045: Hypersthenuria	• Dehydration in dogs, normal in cats

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- **Dipstick test:**

pH:

- **Increased pH** (alkalinity):
  - ➢ Alkalosis, urinary tract infection, urine retention
- **Decreased pH** (acidity):
  - ➢ Fever, starvation, high-protein diet, acidosis, excessive muscular activity

Protein:

- Assess in conjunction with urinary specific gravity and sediment examination
- Small amounts in concentrated urine is ok (e.g. 1+ with >1.030, 2+ with >1.040)
- Only detects > 30mg/dl of protein
- Does not detect globulins and Bence-Jones proteins (plasma cell myeloma)
- UP/C ratio is a more accurate quantitative estimate of proteinuria – **See below**

- ***False negative:*** Dilute urine, acidic urine, albumin concentrations 1-30mg/dl
- ***False positive:*** Alkaline urine

- ***Causes of proteinuria:*** 2+ or more or >1+ in dilute urine <1.030 is significant
  - ➢ **Haemorrhage** (>5 RBC per HPF):
    - Trauma, inflammation, neoplasia
  - ➢ **Inflammation in the urinary tract** (>5 WBC per HPF):
    - Inflammation, neoplasia, infection
  - ➢ **Renal disease:**
    - Usually NO blood or significant cellular sediment
    - *Primary glomerular disease:* Significant 3-4+ dipstick protein: Amyloidosis, glomerulonephritis
    - *Primary tubular disease:* Mild to moderate <2+ dipstick protein
  - ➢ **Pre-renal proteinuria:**
    - Mild <2+ dipstick protein: fever, cardiac disease, shock, muscular exertion, seizures

Glucose:

- Present in urine if exceeds renal reabsorption capacity:
  - ➢ Cats: Blood glucose >15mmol/L
  - ➢ Dogs: Blood glucose >10mmol/L

- Diabetes mellitus: Concurrent hyperglycaemia
- Fanconi syndrome: Glucosuria without hyperglycaemia, due to renal tubular pathology
- Stress hyperglycaemia (cats): Blood glucose has exceeded renal threshold
- Hyperadrenocorticism

Ketones:

- See ketonuria before ketonaemia

- High-fat diet: Fat breakdown
- Starvation/anorexia: Catabolism
- Diabetes mellitus/ketoacidosis: Ketoacidosis due to uncontrolled diabetes
- Very young

Bilirubin:

- Will see bilirubinuria before hyperbilirubinaemia
  - ➢ Dogs: Small amounts in concentrated urine is ok (e.g. 1+ with >1.030, 2+ with >1.040)
  - ➢ Cats: Trace amounts are significant in cats (hepatobiliary disease)

- Hepatobiliary disease:
  - ➢ Cholestasis, hepatic disease
- Haemolysis:
  - ➢ Haemolytic anaemia

Urobilinogen:

- No clinical value

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Blood/Haemoglobin:

- Spin down the urine to differentiate

- **Haematuria:**
  - ➢ Lower urinary tract infection/inflammation, trauma, neoplasia, coagulopathies
  - ➢ Spin down urine: If RBC pellet with clear supernate = haematuria
- **Pigmenturia:**
  - ➢ **Haemoglobinuria:**
    - Haemolytic disease e.g. IMHA, snake bites etc.
    - Spin down urine: No pellet with red/brown supernate
    - Serum: Haemolysis and anaemia
  - ➢ **Myoglobinuria:**
    - Muscle damage (↑ CK)
    - Spin down urine: No pellet with red/brown supernate
    - Serum: Clear and no anaemia

- **Sediment examination:**
  - ✓ Slow centrifugation over a longer period of time. E.g. 1500rpm over 5-10 minutes

Erythrocytes:

- Bleeding, coagulopathy, inflammation, neoplasia

Leucocytes:

- Inflammation
- Infection
- Neoplasia

Microorganisms:

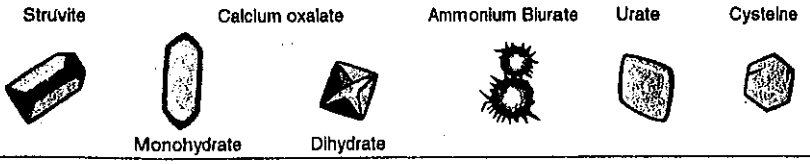
- Bacteria:
  - ➢ Primary or secondary (neoplasia, hyperadrenocorticism)
  - ➢ Culture is the only way to rule out infection, lack of haematuria, pyuria, proteinuria does **not** rule out infection as patient could be immunosuppressed or polyuric
- Fungi
- Protozoa
- Parasites

Casts:

- Increased in acidic urine
- Decreased in alkaline urine (dissolve)
- Increased hyaline or granular casts indicate nephron damage or kidney damage, inflammation

Crystals:

- Types depend on urine pH, concentration, temperature
- See **"Urinary Tract Disease"** for more information



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- **Urine Protein:Creatinine (UPC):**
  - ✓ Used to quantify proteinuria
  - ✓ Only if **no** evidence of inflammation or haematuria on sediment exam

Type of proteinuria:	Differentials:	UP/C and diagnostics:
Physiologic/benign:	▪ Exercise, seizing, fever, extreme temperature, stress	▪ Assess general physical exam and history ▪ **UP/C: usually < 0.5** – also intermittent
Pathological:		
Pre-renal:	▪ Congestive heart failure ▪ Haemoglobin/myoglobin	▪ Assess general physical exam and history ▪ **UP/C varies** ▪ Urine sediment: Sample remains red after spinning down
Renal:	▪ Parenchymal inflammation: ➢ Pyelonephritis ➢ Neoplasia	▪ **UP/C varies** ▪ Urine sediment: Inflammatory but could be inactive ▪ Ultrasound +/- radiographs
	▪ Tubular proteinuria	▪ **UP/C: 0.5 – 1** usually <3 ▪ +/- Associated with glucosuria (with normoglycaemia) and loss of urinary electrolytes
	▪ Glomerular proteinuria: ➢ Glomerulonephritis, amyloidosis (usually > 8)	▪ **UP/C: >1 often >3** ▪ Urine sediment: Usually inactive +/- hyaline casts
Post-renal:	▪ Urinary tract inflammation: ➢ Infection, neoplasia, crystals ▪ Genital tract inflammation: ➢ Infection, neoplasia, bleeding	▪ **UP/C variable** ▪ Urine sediment: Active ▪ Ultrasound ▪ +/- Radiographs, +/- contrast studies

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Urinary Tract Disease

- **This chapter covers:**
  - ✓ Commonly seen urinary tract disease:
    - ➢ **Urinary tract infection**
    - ➢ **Urolithiasis**
    - ➢ **Feline lower urinary tract disease (FLUTD):**
      - Non-obstructed FLUTD
      - Obstructed FLUTD
    - ➢ **Urinary incontinence**
  - ✓ Clinical signs, diagnostic and treatment principles

Urinary tract infection:

- **Causes:**
  - ✓ Urinary system is usually resistant to infection therefore pathogens are usually secondary to predisposing factors:
    - ➢ Abnormal micturition or urine properties (pH, USG, glucose, crystals), anatomical and mucosal defects, systemically immunocompromised
  - ✓ Usually bacteria, other rare causes are fungal and viruses
  - ✓ Organisms:
    - ➢ Commonly isolated: *E. coli* > *Staphylococcus* > *Proteus* > *Klebsiella*
    - ➢ Other isolated: *Enterobacter, Pseudomonas*
- **Clinical signs:**
  - ✓ Bladder (Cystitis):
    - ➢ Pollakiuria, dysuria, haematuria, incontinence, stranguria
    - ➢ Usually not systemically affected
  - ✓ Kidneys (Pyelonephritis):
    - ➢ Fever, lumbar pain, anorexia, vomiting, PU/PD
    - ➢ Systemically ill
  - ✓ Prostate:
    - ➢ Urethral discharge (independent of micturition), dysuria, dyschezia, enlarged and painful prostate on rectal palpation (if acute)
- **Diagnostics:**
  - ✓ **Urinalysis:** see "**Urinalysis**" for more information:
    - ➢ Best collected via cystocentesis
    - ➢ Must always perform sediment exam, best within 30 minutes
    - ➢ Culture is the only way to definitively rule out infection
    - ➢ Absence of haematuria, pyuria, proteinuria does **not** rule out infection as the patient could be immunosuppressed or polyuric
  - ✓ **Haematology and biochemistry:**
    - ➢ Usually indicated if systemically ill or suspect renal compromise or predisposing diseases e.g. Hyperadrenocorticism or diabetes mellitus
  - ✓ **Culture and sensitivity:**
    - ➢ Qualitative: Identification of the pathogen
    - ➢ Quantitative: Indication of numbers of bacteria and therefore significance of what was cultured

Significance:	Method of collection:	Dog (cfu/ml):	Cat (cfu/ml):
Significant if	Cystocentesis	>1000	>1000
	Catheterization	>10,000	>1000
	Voided	>100,000	>10,000

  - ✓ **Imagery:**
    - ➢ Ultrasound, radiographs +/- contrast studies

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- **Treatment:**
  - ✓ Antibiotic selection is best based on culture and sensitivity results:
    - ➢ Amoxicillin 12.5mg/kg IV TID, ampicillin 10-20mg/kg IV TID, cephalexin 22mg/kg IV TID
    - ➢ Trimethoprim-sulfa 15mg/kg IV/PO BID, amoxicillin clavulanic acid 12.5-25mg/kg PO BID
  - ✓ **Simple non-complicated UTI:**
    - ➢ If first occurrence/infrequent, no predisposing factors
    - ➢ 2 weeks of antibiotics, clinical signs and urinalysis should improve in 48 hours
    - ➢ Culture after treatment (see **"follow up"** below)
  - ✓ **Recurrent or relapsing:**
    - ➢ Identification and correction of predisposing cause e.g. hyperadrenocorticism or diabetes mellitus
    - ➢ Recurrence can also indicate nidus of infection somewhere e.g. Urinary calculi, pyelonephritis or prostate
    - ➢ Antibiotics: Select based on culture and sensitivity results, 4-6 week course
    - ➢ Long term (months) once daily low dose treatment (at night after last urination before bedtime) may be required, selection based on culture and sensitivity and at 1/3 of normal dose
    - ➢ Culture before, during and after treatment
    - ➢ Beware long term antibiotic therapy and risks of toxicity e.g. KCS with trimethoprim-sulpha
- **Follow up:**
  - ✓ Culture and sensitivity, timing depends on goal:
  - ✓ To see if antibiotic is effective: 3-4 days after starting, negative culture indicates it is working
  - ✓ To see if treatment has eliminated infection: 7-10 days after finishing
  - ✓ To follow up on chronic long term therapy: Every 1-2 months

Urolithiasis:

- **Pathogenesis: (See below)**
  - ✓ More common in dogs than cats and small breed dogs rather and large breed
  - ✓ Males can present with urinary tract obstruction
  - ✓ Factors that cause crystalluria due to oversaturation of urine and then factors that cause aggregation leading to discrete stones:
    - ➢ Crystalluria can be normal in both dogs and cats
    - ➢ Does not always indicate urolithiasis
    - ➢ Does not always indicate that treatment is required
  - ✓ Location:
    - ➢ 5% in the renal pelvis, usually struvite (*Mg ammonium phosphate = MAP*) and calcium oxalate/phosphate
    - ➢ Bladder in females
    - ➢ Bladder and urethra in males
  - ✓ Calculi are classified by the major crystalline component
  - ✓ **Feline Urolithiasis:**
    - ➢ See next page **"Feline lower urinary tract disease"** for more information
    - ➢ 90% of uroliths are struvite or calcium oxalate
    - ➢ Obstructive FLUTD usually caused by a urethral plug that can be comprised of mineral and matrix
- **Clinical signs:**
  - ✓ Renal calculi: Commonly no clinical signs, but obstruction of urine can lead to renal atrophy which can lead to renal failure (especially if bilateral), but can also develop pyelonephritis
  - ✓ Cystic calculi: Clinical signs of cystitis, pollakiuria, dysuria and haematuria
  - ✓ Urethral calculi: Clinical signs of urinary tract obstruction, stranguria, large firm bladder

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- **Diagnostics:**
  - ✓ Definitive diagnosis of the type of crystal may require laboratory analysis of a sample of the stone
  - ✓ Urinalysis and sediment examination, always submit urine for culture and sensitivity:
    - ➢ pH, type of crystal, presence of concurrent urinary tract infection
    - ➢ Maybe able to collect small stones via endoscope or voiding urohydropulsion
  - ✓ Imagery:
    - ➢ Radiographic density:
      - Good: Struvite, Calcium oxalate and phosphate, Silicate
      - Poor: Urate, cysteine, xanthine
    - ➢ Ultrasound: Can see all types of stones, see hyperechoic interface with acoustic shadowing
  - ✓ Signalment: Presumptive diagnosis can be based on signalment
- **Treatment:**
  - ✓ Medical management: **See below** can take months to dissolve and may require months of antibiotics to treat secondary infections
  - ✓ Surgical removal: Required for urethral obstructions and large stones, must pass urinary catheter to ensure no stones remain within the urethra

	Struvite (MAP):	**Calcium Oxalate:**	**Ammonium Urate:**	**Cysteine:**
Sex/breed:	▪ Common both sexes (causes ~50% of lower UT uroliths) ➢ Females (90%) due to UTI ➢ Males (<50%)	▪ Second most common (33%) ▪ Males (>80%)	▪ Males (90%) ▪ Dalmatians	▪ Males (90%)
Age:	▪ All ages	▪ Older	▪ Younger (1 – 4 years)	▪ Younger (1 – 7 years)
Pathogenesis:	▪ Secondary to UTI ➢ Microbial urease ➢ *Staphylococcus, Proteus* ▪ Sterile (not as common)	▪ Unknown ▪ Usually multiple small stones ▪ Possibly related to hyperadrenocorticism, hypercalcaemia	▪ Dalmatians ➢ Defective transport mech ▪ PSS and older dogs with liver disease	▪ Defective reabsorption of cysteine
Treatment:	▪ Dietary dissolution ▪ Tx concurrent lower UTI – **UNTIL** no crystals and no UTI ▪ ↓ pH	▪ Surgical removal ▪ No dietary dissolution	▪ Dietary dissolution ▪ Treatment of liver dysfunction ▪ Allopurinol	▪ Dietary dissolution ▪ Potassium citrate ➢ ↑ pH to 7.5
Prevention:	▪ Dietary management ▪ Preventing UTI	▪ Dietary management ▪ +/- Thiazide diuretic ▪ Potassium citrate ➢ ↑ pH to 6.5- 7.5	▪ Dietary management	▪ Dietary management
Microscopy:		 Monohydrate  Dihydrate	 Ammonium biurate  Urate	

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Feline lower urinary tract disease (FLUTD):

1. **Non-obstructed FLUTD**

- **Features:**
  - ✓ Age: Young, 2-6 years old, male cats most common
  - ✓ Risk factors: Neutering, obesity, dry diets, sedentary lifestyle
  - ✓ Many causes of FLUTD are idiopathic, i.e. can't find attributable cause
- **Clinical signs:**
  - ✓ Dysuria, pollakiuria, stranguria, haematuria, vocalisation
  - ✓ "Straining to defecate" or "constipated"
  - ✓ Abdominal pain, inappropriate urination, lethargy and vomiting
  - ✓ Thickened, firm, contracted bladder wall
  - ✓ Large firm bladder = obstructed
- **Causes:**
  - ✓ Idiopathic (most common): Interstitial cystitis
  - ✓ Crystalluria, urolithiasis
  - ✓ Neurological: Urethral spasms, reflex dyssynergia
  - ✓ Neoplasia
  - ✓ Infectious: Bacteria 5-10% of cases
  - ✓ Iatrogenic
- **Diagnostics:**
  - ✓ General physical examination:
    - ➢ Small or empty bladders with increased frequency or straining to urinate
    - ➢ The penis or vulva should be assessed for discharge
  - ✓ Urinalysis and sediment:
    - ➢ Sediment examination: WBC, RBC, +/- crystals, +/- bacteria
    - ➢ Urine culture and sensitivity: Indicated if see bacteria or recurrent issue
  - ✓ Biochemistry:
    - ➢ +/- Azotaemia, hyperphosphataemia
  - ✓ Electrolytes:
    - ➢ +/- Hyperkalaemia
  - ✓ Imaging:
    - ➢ Radiographs +/- contrast studies: Neoplasia, strictures, calculi, recurrent disease
    - ➢ Ultrasound: Radiolucent calculi, urachal diverticulum or neoplasia
- **Treatment for non-obstructed or idiopathic FLUTD:**
  - ✓ Usually experience recurrent bouts of disease, 40-65% of cats will have a recurrence within 1 to 2 years
  - ✓ Recurrence **is not always** recurrence of the original disease
  - ✓ Treatment:
    - ➢ Benign neglect: Most (up to 90%) cats with idiopathic FLUTD exhibit remission of clinical signs in 5-7 days without therapy
    - ➢ Anti-inflammatories: Check renal parameters first, but neither NSAIDs or corticosteroids alter the course of disease
    - ➢ Opioids: Buprenorphine, butorphanol, tramadol
    - ➢ Antibiotics: Not usually required as only <5% are bacterial, evidence of pyuria and bacteria
    - ➢ Amitriptyline 5-10mg/kg SID at night, try in chronic cases, multiple modes of action
    - ➢ Smooth muscle anti-spasmodic: Propantheline 0.25-0.5mg/kg, help reduce urge incontinence, monitor for urine retention
    - ➢ Environmental enrichment: Can be very important
    - ➢ Dietary management: See below

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- **Discharge:**
  - ✓ When fluid and electrolyte disturbances are corrected, renal enzymes are normal, urine sediment and urination patterns are normal
- **Prevention:**
  - ✓ Dietary management:
    - ➢ Crystal dissolution diet
    - ➢ **Wet food:** Probably most important factor, add water and blend, offer lactose free milk
  - ✓ Reduce stress: Minimise changes at home, providing safe places to hide/rest, extra litter trays and proper litter box hygiene, litter trays in quite places (i.e. not next to washing machine), different types of litter
  - ✓ Behaviour drugs: Amitriptyline (5-10mg/cat SID at night) during times of stress, Feliway ® spray/diffuser, fluoxetine
  - ✓ Pentosan polysulfate sodium (glycosaminoglycan): As per treatment regime for osteoarthritis

2. Obstructed FLUTD – Emergency

- **Causes:**
  - ✓ Obstructive FLUTD usually caused by a urethral plug that can be comprised of mineral and matrix (which can attribute to 50-100% of the plug)
  - ✓ 90% of uroliths are struvite (most common) or occasionally calcium oxalate
- **Clinical signs:**
  - ✓ Dysuria, pollakiuria, stranguria, haematuria, urethral obstruction, vocalisation +/- uraemia
  - ✓ Owner may report – "straining to defecate" "constipated"
- **Physical examination:**
  - ✓ Large firm bladder, abdominal discomfort, +/- bradycardia, collapse/recumbency
  - ✓ Urethral plugs/sludge on tip of penis
- **Diagnostics:**
  - ✓ History: Repeated unproductive attempts in the litter tray
  - ✓ Clinical signs and general physical examination: Full/hard bladder on palpation
  - ✓ Urinalysis and sediment examination: Assess for crystalluria, bacteria
  - ✓ CBC and serum biochemistry: Assessment of systemic affects – e.g. Azotaemia
  - ✓ Electrolytes: Hyperkalaemia, hyperphosphataemia
- **Treatment for <u>BLOCKED CAT</u>:**
  - ✓ Goals of treatment:
    - ➢ Relieve obstruction (but must be stabilized prior to anaesthesia and sedation)
    - ➢ Correct biochemical and electrolyte disturbances, e.g. hyperkalaemia and azotaemia
    - ➢ Force post-obstructive diuresis and provide required fluid therapy
    - ➢ Monitor renal parameters and electrolyte levels
    - ➢ Provide supportive care during post-obstructive phase
  - ✓ Examine tip of penis for urethral plugs
  - ✓ Place a catheter and collect blood for haematology, biochemistry, electrolytes and blood gas analysis
  - ✓ Administer pain relief e.g. partial or pure opioid agonist
  - ✓ Can try to temporarily relieve obstruction by flushing urethra with a 22g catheter and saline (pass urinary catheter after)
  - ✓ Fluid therapy
    - ➢ Correct perfusion deficits, then start to replace hydration deficits
  - ✓ Correct hyperkalaemia if present, see **"Fluid Therapy"**:
    - ➢ Slow IV bolus of 10% calcium gluconate (1ml/kg) whilst monitoring heart (auscultation/ECG)
      - Cardiopulmonary effects last 20 minutes
    - ➢ Slow IV bolus of regular insulin (e.g. Actrapid) 0.25U/kg IV plus 4mls 50% dextrose per 1 unit of insulin IV, then continue with a 2.5% dextrose CRI

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- ✓ Can empty the bladder by cystocentesis using a 22g needle with extension set
  - ➢ **Risk bladder rupture**
  - ➢ Keep urine for urinalysis
  - ➢ Empty the bladder completely
- ✓ Once stabilised and corrected electrolyte abnormalities, anaesthetise for urinary catheter placement
- ✓ **Urinary catheterisation:**
  - ➢ Clip and surgical prepare the prepuce/perineal area → use sterile kit
  - ➢ 14cm, 3.5 – 5.0 french soft urinary catheter
  - ➢ Using a 22g catheter, flush urethra gently with either saline or mix of saline and KY jelly mix (5:1)
  - ➢ Flush with a twisting and rolling motion to clear blockage
  - ➢ Once the catheter is in the tip of the penis, pull the prepuce towards the tail to straighten out the urethra, this helps pass the urinary catheter
  - ➢ If suspect urethral spasm flush with lignocaine (0.25-0.5ml)
  - ➢ Then pass urinary catheter
  - ➢ Otherwise can try stiffer catheter (i.e. open or closed ended tom-cat catheter) with the same saline/KY jelly mix to flush obstruction
  - ➢ Empty and flush the bladder a couple times with sterile saline to reduce accumulated sediment
- ✓ Continue fluids until fluid deficits are corrected and to cover for post-obstructive diuresis
- ✓ Monitor:
  - ➢ Urine output, match IV fluids and urine output to cover for post-obstructive diuresis
  - ➢ Electrolytes (q4-6 hours
  - ➢ Renal enzymes q6-12 hours
  - ➢ Urine characteristics: Colour, USG, sediment exams
- ✓ Removal of urinary catheters:
  - ➢ Generally, trial when urine sediment clears, after 24 – 48 hours
- ✓ **Medication:**
  - ➢ **Place all ingredients as below in a gelatine capsule or give individually**
  - ➢ Start after catheter removal:
  - ➢ Combination of medications to help support normal urination (counter detrusor atony and urethral spasm)
  - ➢ Increase detrusor contractions: Bethanechol 2.5mg PO TID, must ensure urethral patency before giving and use in conjunction with smooth muscle relaxant
  - ➢ Relax internal (smooth muscle) urethral sphincter: Phenoxybenzamine 0.5mg/kg PO BID **OR** prazosin 0.25-1mg/cat PO BID
  - ➢ Relax external (skeletal muscle) urethral sphincter: Dantrolene (12.5mg) & diazepam (2.5mg) BID
  - ➢ +/- Antibiotics: e.g. Amoxicillin clavulanic acid 25mg/kg PO BID

Urinary Incontinence:

- **Pathogenesis:**
  - ✓ Inability to voluntarily control micturition
  - ✓ Neurological dysfunction:
    - ➢ Pudendal nerve S1 – S3 innervates anal and urethral sphincters
    - ➢ Upper motor neuron lesion:
      - Lesion cranial to S1 – S3 leads to ↑ sphincter tone and intact perineal reflex
      - Bladder is distended and difficult to express leads to overflow incontinence when bladder pressure exceeds sphincter tone, never completely empties
    - ➢ Lower motor neuron lesion:
      - Lesion at S1 to S3 leads to ↓ sphincter tone and absent perineal reflex
      - Bladder is easy to express, leaks urine when moves and has overflow incontinence
    - ➢ Detrusor areflexia:
      - Over distension of the bladder leads to damage to tight junctions resulting in defective contractions

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- ✓ Urethral incompetence:
  - ➢ Most commonly hormone responsive, 20% of speyed bitches
  - ➢ Pathogenesis not completely understood but possibly due to reduced resting urethral tone or possibly loss to broad ligament support leading to an intrapelvic bladder
- ✓ Lower urinary tract inflammation, **see above**:
  - ➢ Hyperreflexia of the detrusor muscle due to inflammation of the bladder usually due to infection
- ✓ Paradoxical urinary incontinence:
  - ➢ Caused by partial obstruction of bladder neck/urethra
  - ➢ Prostatic disease (abscess, cyst, neoplasia, hyperplasia), urolithiasis, tumours (carcinomas)
- ✓ Congenital abnormalities: E.g. ectopic ureter
  - ➢ Incontinence from couple months old

Diagnostics:

- ✓ History: Puddles of urine after sleeping/lying down, blood in urine, drinking habits/volume
- ✓ Clinical signs: Urge incontinence then most likely inflammatory disease e.g. FLUTD, urinary tract infection
- ✓ Physical examination: Palpation of bladder, ease of expression of urine, rectal to palpate the prostate
- ✓ Neurological examination: Assessment of pudendal nerve function and hindlimb reflexes, e.g. perineal reflex, rectal tone, if able to initiate urination pudendal nerve is most likely intact
- ✓ Urinalysis and sediment examination +/- culture and sensitivity
- ✓ Haematology and biochemistry
- ✓ Imagery:
  - ➢ Radiographs +/- contrast: Excretory for ectopic, retrograde positive/negative contrast for uroliths and neoplasia
  - ➢ Ultrasound (kidneys, prostate, bladder)
- ✓ Prostatic fluid cytology

- **Treatment:**
  - ✓ Neurological dysfunction:
    - ➢ Expression to keep bladder small – reduce risk of detrusor areflexia/UTI
    - ➢ Treat urinary tract infections
    - ➢ **Upper motor neuron lesion:**
      - Relax internal urethral sphincter:
        - o Phenoxybenzamine 0.2mg/kg PO BID for dogs or 0.5mg/kg PO BID for cats **OR**
        - o Prazosin 0.5mg/kg PO BID for dogs or 0.25-1mg/cat PO BID
      - Improve bladder contraction: Ensure patent urethra
        - o Bethanechol 5-20mg/dog PO TID or 2-4mg/cat PO TID
      - Relax external urethral sphincter: Diazepam 0.5mg/kg PO TID
    - ➢ **Lower motor neuron lesion and detrusor areflexia:**
      - Improve bladder contraction: Bethanechol 5-20mg/dog PO TID or 2-4mg/cat PO TID
  - ✓ Urethral incompetence:
    - ➢ Treat urinary tract infections
    - ➢ Increase internal urethral sphincter tone: Phenylpropanolamine (e.g. Propaline) 1mg/kg PO BID
    - ➢ Increases responsiveness of urethral sphincter muscles: Oestrogen (diethylstilboestrol) 0.2mg/kg SID PO for 5 days then once every 3-7 days as needed
      - Avoid cumulative doses of >1mg/week
      - Side effects: Bone marrow suppression, alopecia, behaviour changes
    - ➢ GnRH implant (Suprelorin ®): Mode of action is not confirmed, use is off-label
  - ✓ Lower urinary tract inflammation: Treatment for **urinary tract infections**, see above
  - ✓ Paradoxical urinary incontinence:
    - ➢ Secondary to outflow obstruction, get overflow incontinence when pressure exceeds sphincter
    - ➢ Identification and treatment of underlying cause
  - ✓ Congenital abnormalities e.g. ectopic ureter: Surgical correction of the ectopic ureter

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Viral Disease and Vaccination

- **This chapter covers:**
  - ✓ Common viral diseases
  - ✓ General information and diagnostics
  - ✓ Principles of treatment and prevention
  - ✓ Outline common causes of feline upper respiratory tract disease (non-viral included)
  - ✓ Vaccination regimes and vaccine reactions

Viral agents:

- **Feline infectious peritonitis:**
- **Features:**
  - ✓ Mutant form of feline coronavirus which usually causes mild enteritis
  - ✓ During coronavirus infection a mutation occurs, enabling it to infect macrophages
  - ✓ Typically, a disease of young cats (5 months to 3 years) as infected when young, especially those in a cattery or multicat household
  - ✓ The coronavirus (not the mutated form) is shed in the faeces (also saliva and respiratory secretions) of carriers who can shed it for years without clinical signs
  - ✓ Immunity to coronavirus is transient and recurrent infections are common
  - ✓ The host immune response to the virus leads to the clinical signs, causes an immune-mediated vasculitis due to antigen-antibody complex deposition within the vessel epithelium:
    - ➢ Two forms and can develop both at the same time
    - ➢ Effusive form "Wet" form:
      - Develops within weeks due to accumulations of inflammatory exudate
    - ➢ Non-effusive "Dry" form:
      - Can take months, pyogranulomatous inflammation in various tissues
- **Clinical signs:**
  - ✓ Early presenting signs are non-specific:
    - ➢ Pyrexia of unknown origin
    - ➢ Anorexia, weight loss, vomiting and diarrhoea
  - ✓ Wet form (effusive):
    - ➢ High protein effusions
    - ➢ Usually abdominal >50%, but can occur in the pleural space or in both
    - ➢ +/- involvement of abdominal organs
    - ➢ Liver involvement can lead to hyperbilirubinaemia
    - ➢ Gastrointestinal tract leads to diarrhoea and vomiting
    - ➢ Respiratory distress from pleural effusions
    - ➢ Scrotal swellings in intact males
  - ✓ Dry from:
    - ➢ Pyogranulomatous inflammation in affected organs
    - ➢ Most common infectious cause of neurological signs in Australian cats
    - ➢ Nodular kidneys and eventually renal failure
    - ➢ Hepatic failure
    - ➢ Ocular: Bilateral uveitis
    - ➢ Central nervous system: Any neurological signs due to meningoencephalitis

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- **Diagnostics:**
  - ✓ No definitive diagnostic test
  - ✓ Diagnosis is from a combination of history, clinical signs and diagnostic tests
  - ✓ Analysis of effusions:
    - ➢ High protein, pyogranulomatous exudate
    - ➢ Cerebrospinal fluid: protein >2g/L, neutrophilia
  - ✓ Rivalta's test:
    - ➢ See if clots in water and acetic acid
    - ➢ 3% chance that it is wrong
  - ✓ Biochemistry:
    - ➢ Increased globulins, and low A:G ratio
  - ✓ Haematology:
    - ➢ Non regenerative anaemia, neutrophilia, lymphopenia
  - ✓ Serology:
    - ➢ Serum coronavirus antibody:
      - Positive only indicates prior exposure, high titre provides support
      - Negative rules out FIP
  - ✓ Histopathology and immunohistochemistry:
    - ➢ Detection of coronavirus antigens in macrophages from affected tissues
    - ➢ Confirmatory of FIP
- **Treatment:**
  - ✓ Palliative only
  - ✓ Corticosteroids: immunosuppressive dose may reduce the inflammation
  - ✓ Antibiotics: +/- Broad-spectrum to reduce to risk of secondary infections
- **Prevention:**
  - ✓ Vaccination:
    - ➢ No effective vaccination and will test positive to coronavirus anti-body tests
  - ✓ Improve nutrition and reduce stress:
    - ➢ Reduce overcrowding
    - ➢ Improved hygiene, only survives in environment for 48 hours
    - ➢ Reduce faecal contamination, clean litter trays, dishes and bedding
  - ✓ Separating kittens from mother at 5 weeks of age before maternal antibodies wane
  - ✓ Antibody testing cats:
    - ➢ All new cats
    - ➢ Seropositive cats in the household should be removed – constant source of infection
  - ✓ Consider desexing animals that produce kittens that die of FIP possibly carry the gene responsible for mutating the virus

- **<u>Feline immunodeficiency virus:</u>**
- **Features:**
  - ✓ Retrovirus that leads to interference of normal immune system function
  - ✓ Targets immune cells and leads to impaired cell mediated immune more so than humoral responses
  - ✓ Usually leads to immunosuppression, death occurs to secondary disease processes
  - ✓ Predisposes to:
    - ➢ Oral, respiratory tract, gastrointestinal, dermatological, renal, neurological and neoplastic disease
  - ✓ Shed in saliva therefore transmission by fighting:
    - ➢ Male entire outdoor cats are at high risk

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- **Clinical signs:**
  - ✓ Acute primary infection phase:
    - ➢ About 1 month after infection
    - ➢ Usually fever, lethargy
    - ➢ Lymphadenopathy
  - ✓ Asymptomatic phase:
    - ➢ Months to years
    - ➢ No specific clinical signs
    - ➢ +/- Lymphadenopathy
  - ✓ AIDS-related complex (ARC):
    - ➢ Immunodeficiency leads to opportunistic infections and chronic illness
    - ➢ Weight loss, recurrent infections and pyrexia, +/- lymphadenopathy
    - ➢ Clinical signs of oral, renal, respiratory, dermal, gastrointestinal disease
- **Diagnostics:**
  - ✓ Detection of antibodies to FIV via ELISA:
    - ➢ Antibodies takes up to 12 months to develop
    - ➢ May need to repeat tests every couple months for 12 months to prove not infected
    - ➢ Recent vaccination interferes with ELISA, PCR test is required but can lack sensitivity
    - ➢ Kittens less than 6 months can be false positive due to persistent maternal antibodies
  - ✓ Haematology:
    - ➢ Non-regenerative anaemia, lymphopenia, neutropenia
  - ✓ Biochemistry:
    - ➢ Hyperglobulinaemia
- **Treatment:**
  - ✓ No specific treatment
  - ✓ Antiviral therapy: Poor response
  - ✓ Aggressive treatment of secondary disease processes during the AIDS-related complex phase
  - ✓ Optimise health: Good husbandry and nutrition
- Prevention:
  - ✓ Keep indoors
  - ✓ Desex all cats
  - ✓ Test introduced cats before allowing them to mix into cattery/multicat house situation
  - ✓ Vaccination: Variable efficacy, generally not considered a core vaccination

- **<u>Feline leukaemia virus:</u>**
- **Features:**
  - ✓ Retrovirus infection
  - ✓ Transmission may be in-utero or via milk, prolonged cat to cat contact, multicat households
  - ✓ Virus is constantly shed in saliva, respiratory secretions and urine by viraemic cats
  - ✓ Low prevalence 1-2%
- Consequences of infection:
  - ✓ Elimination of the virus by immune response, but can become latent carriers don't shed
  - ✓ Persistently infected:
    - ➢ Failure to eliminate the infection leads to infection of the bone marrow and lymphoid system
    - ➢ Immunosuppression (60%): Seen as bone marrow suppression, anaemia, lymphopenia, neutropenia
    - ➢ Neoplasia (30%): Seen as lymphoma and leukaemia, infiltration of the bone marrow, spleen and liver

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- **Clinical signs:**
  - ✓ Immunosuppression: (60%)
    - ➢ Weight loss, fever
    - ➢ Ocular and nasal discharge, diarrhoea, stomatitis, and lymphadenopathy
  - ✓ Neoplasia: (30%)
    - ➢ Lymphadenopathy
    - ➢ Lymphoma: Gastrointestinal tract, liver, spleen, skin, eyes
    - ➢ Leukaemia: Anaemia, leucocytosis, mild lymphadenopathy, hepato-splenomegaly
- **Diagnostics:**
  - ✓ Detection of FeLV antigens by ELISA:
    - ➢ Highly sensitive test
    - ➢ Does not detect latent infections (infected but not shedding)
    - ➢ Not affected by vaccination
  - ✓ Immunofluorescent antibody test:
    - ➢ Highly specific test
    - ➢ Use as a confirmatory test in any FeLV ELISA positives
    - ➢ 85% of persistently viraemic cats die within 3 years
  - ✓ Haematology:
    - ➢ Normochromic-normocytic or macrocytic non-regenerative anaemia
    - ➢ Leukopenia, thrombocytopenia
  - ✓ Blood smears: Abnormal blast cells in peripheral circulation
  - ✓ Bone marrow biopsy: Abnormal blast cells
- **Treatment:**
  - ✓ No specific treatment
  - ✓ Antiviral therapy: Not eliminate virus but can improve clinical signs
  - ✓ Aggressive treatment of secondary disease processes
  - ✓ Optimise health: Good husbandry and nutrition
- **Prevention:**
  - ✓ Prevent contact or breeding with infected cats
  - ✓ Test all incoming cats
  - ✓ Remove and isolate all ELISA positive cats from breeding colony
  - ✓ Disinfect cages, food and water bowls and litter boxes
  - ✓ Vaccination:
    - ➢ Variable efficacy but can reduce the risk of infection
    - ➢ Recommended for at risk situation
    - ➢ Otherwise not recommended as a core vaccine

- **Feline upper respiratory tract infectious agents:**
  - ✓ Causes upper respiratory tract and ocular infections:

Feline herpes virus:	Feline calicivirus:	*Chlamydophila felis:*	*Mycoplasma felis / gatae:*
▪ Shed in ocular, oral and respiratory secretions ▪ Lifelong carrier states shed intermittently secondary to stress ▪ Reactivation of carrier states, reoccurrence of clinical signs ▪ Carrier states are source of outbreaks	▪ Shed in ocular, oral and respiratory secretions ▪ FCV shed continuously ▪ Carriers state can persist for years ▪ Carrier states are typically the source of outbreaks	▪ Intracellular bacteria ▪ Breeding colonies and multicat households high risk ▪ Causes chronic infections lasting a couple months unless treated	▪ Commensal organism ▪ Infections are often secondary to viral ▪ If detected by PCR in ocular swabs, then significant

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Clinical signs:

- **Chronic snufflers:**
  - Chronic uni-bilateral mucopurulent nasal discharge due to bacterial infection secondary to viral infection

• Typically resolve after 10 days
• Anorexia, lethargy
• Pyrexia (usually)
• Severe sneezing with profuse discharge
• Conjunctivitis (severe)
• Corneal ulcers, sequestrum
• Abortion in pregnant cats
	• Anorexia, lethargy
	• Pyrexia (variable)
	• Mild sneezing and discharge
	• Conjunctivitis (mild)
	• Oral ulcers: Can lead to stomatitis, gingivitis
	• Lameness
	• +/- Vomiting and diarrhoea
		• Conjunctivitis (most commonly seen):
		  ➢ Unilateral or bilateral
		  ➢ Hyperaemic and chemotic
		• No corneal ulcers
		• Chronic infections lead to follicular conjunctivitis
		• +/- Mild sneezing and nasal discharge
			• Conjunctivitis:
			  ➢ Unilateral or bilateral
			  ➢ Hyperaemic and chemotic

Diagnosis:

- Virus isolation – can detect both viruses (best for FCV)
- PCR on conjunctival swab – can detect both viruses (best for FHV), also detect *Mycoplasma* and *Chlamydophila*
- Culture of conjunctival/nasal swabs for *Chlamydophila* and *Mycoplasma*, can take weeks
- **Chronic snufflers:** Radiographs, fungal titres, scoping to rule out other causes of chronic mucopurulent discharge. See **"Nasal and Nasopharyngeal Disease"**

Treatment:

- Antiviral therapy:
  - Not proven to be effective, but can use drops for eye infections
- Antibiotics:
  - Doxycycline 5mg/kg PO BID
  - Amoxicillin clavulanic acid 20mg/kg PO BID
- Topical eye medications for ulcers
- L-lysine:
  - Inhibits FHV growth by competitive inhibition
  - 250mg PO BID kittens, 500mg PO BID cats
- Supportive:
  - Fluids to maintain hydration or rehydrate
  - Airway humidification via nebulisation
  - Frequent cleaning of secretions and encourage eating
  - Vaseline on nose

- **Chronic snufflers:**
- Chronic bacterial infection secondary to primary viral infection due to destruction of mucosa
- Bacterial infection leads to damage of the underlying bone
- Antibiotics:
  - 8 weeks of doxycycline or clindamycin
  - May require short courses for relapses
  - Need good bone penetration
- +/- Saline nebulisation
- Anti-viral medication

Prevention:

- FHV is susceptible to disinfectants and survives for up to 24 hours in the environment
- FCV survives in environment for up to 10 days
- Frequent and thorough cleaning to reduce fomite spread
- Reduce overcrowding
- Vaccination: All new cats, reduce severity of infections, does not prevent infections or eliminate carrier states

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- **Canine distemper virus:**
- **Features:**
  - ✓ Transmission is via respiratory secretions
  - ✓ The virus spreads to the respiratory, gastrointestinal and urinary tracts and the central nervous syste
  - ✓ Degree of immune response to infection determines severity of clinical signs. Failure to mount an immune response can lead to death within a couple weeks.
- **Clinical signs:**
  - ✓ Acute signs: Fever, lethargy, anorexia
  - ✓ Respiratory tract: Nasal and ocular discharge, coughing and dyspnoea
  - ✓ Gastrointestinal tract: Vomiting and diarrhoea
  - ✓ Ocular signs: Conjunctivitis, uveitis
  - ✓ Neurological signs: Ataxia, paresis, hyperaesthesia, vestibular signs, seizures
    - ➢ Signs can develop a couple weeks after infection
  - ✓ Dermatological signs:
    - ➢ Hyperkeratosis of nasal planum and footpads
- **Diagnostics:**
  - ✓ Blood smear: May see viral inclusion bodies in white or red blood cells
  - ✓ PCR Assay: Identification and quantification, if high enough can be diagnostic
  - ✓ Serology: Elevated IgM and IgG levels difficult to determine if active infection, prior infection or vaccination
  - ✓ Post-mortem via histopathology and immunofluorescence
- **Treatment:**
  - ✓ No specific treatment
  - ✓ Symptomatic management of clinical signs
  - ✓ Aggressive treatment of secondary bacterial infections
  - ✓ Antiviral therapy: Unknown efficacy
  - ✓ Optimise health: Good husbandry and nutrition
- **Prevention:**
  - ✓ Vaccination: Highly efficacious
  - ✓ Appropriate disinfecting and isolation procedures and protocols

- **Canine parvovirus and feline panleucopenia virus:**
- **Features:**
  - ✓ Highly contagious viruses that are spread primarily by ingestion of affected animal's faecal material:
    - ➢ Dogs: Common especially between weaning to one year old
    - ➢ Cats: Rare
  - ✓ Resilient viruses, persist in the environment for up to 8 months, likely to be a major source of infection
  - ✓ Can occur in previously vaccinated animals
  - ✓ Both viruses target rapidly dividing cells:
    - ➢ Intestinal cells: Diarrhoea, vomiting, haematochezia
    - ➢ Bone marrow: Leukopenia
    - ➢ Growing foetuses: Abortion
- **Clinical signs:**
  - ✓ Anorexia, lethargy, pyrexia
  - ✓ Vomiting and diarrhoea, typically haemorrhagic
  - ✓ Severe dehydration leading to hypovolaemic shock
  - ✓ +/- Icterus
  - ✓ Abortion
  - ✓ Death from shock (hypovolaemic, endotoxic, septic)

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- **Diagnostics:**
  - ✓ Demonstration of viral antigens in faeces via ELISA:
    - ➢ False-positive results occasionally up to 5 to 12 days following vaccination with a modified live virus
  - ✓ Faecal testing: Intestinal worms, *Coccidia* and *Giardia*
  - ✓ Haematology:
    - ➢ Leukopenia can be severe
    - ➢ +/- Anaemia due to blood loss
  - ✓ Biochemistry:
    - ➢ Hypokalaemia, hypoalbuminaemia, hypoglycaemia
    - ➢ +/- Hepatopathy and prerenal azotaemia
- **Treatment:**
  - ✓ Isolation and barrier nursing
  - ✓ Supportive therapy: Keep warm and quiet
  - ✓ Fluid therapy: Aggressive IV fluid therapy, correction of perfusion and dehydration deficits and electrolyte abnormalities
  - ✓ Antiemetic: Metoclopramide CRI 1-2mg/kg/day IV, maropitant 1mg/kg SC SID for <5 days
  - ✓ Gastric protectants: Ranitidine 2mg/kg BID, sucralfate 0.5-1gm PO TID, proton pump inhibitors
  - ✓ IV antibiotics: Recommended to reduce the risk of bacterial translocation from the gastrointestinal tract
    - ➢ Broad spectrum bactericidal
    - ➢ If not leukopenia: Cephalothin 22mg/kg IV TID and metronidazole 10mg/kg IV BID
    - ➢ If leukopenia: Ticarcillin 50mg/kg IV QID
  - ✓ Transfusions:
    - ➢ Blood if anaemia develops
    - ➢ +/- Plasma or colloids for oncotic support if hypoalbuminaemia is present
  - ✓ Early enteral nutrition:
    - ➢ Important, start if anorexic >2 days
    - ➢ Micro-enteral feeding with electrolyte solutions via tube feeding (nasoesophageal tubes) then progress to food
- **Prevention:**
  - ✓ Vaccination is very effective, vaccinate all animals
  - ✓ Isolation of infected animal as they can shed virus for up to 40 days after recovery
  - ✓ Beware the virus can remain in the environment for up to 8 months do not allow unvaccinated animals access to that environment
  - ✓ Isolation of puppies away from other unvaccinated animals at least 2 weeks after final vaccination
  - ✓ Clean and disinfect the environment

- **Canine parainfluenza virus:**
  - ✓ Plays important role along with *Bordetella bronchiseptica* in "kennel cough" or canine infectious tracheobronchitis
  - ✓ See **"Respiratory Disease"** for more information

- **Other viral gastrointestinal viruses:**
- **Canine and feline coronavirus and rotavirus and other:**
  - ✓ Typically, all are of minor clinical relevance:
    - ➢ Canine coronavirus can last for up to a month
    - ➢ Feline coronavirus can mutate and cause feline infectious peritonitis
  - ✓ Transmission is faecal-oral route
- **Clinical signs:**
  - ✓ Cause mild self-limiting diarrhoea can be watery and contain mucus
  - ✓ Diarrhoea can persist up to a week

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- **Diagnostics:**
  - ✓ Rule out parvovirus and panleukopenia virus
  - ✓ Other causes of gastroenteritis: Intestinal worms, *Coccidia* and *Giardia*
  - ✓ Positive coronavirus tests: indicate exposure not definitive cause
- **Treatment:**
  - ✓ Supportive, IV fluids, bland highly digestible food
  - ✓ Vaccinations are available to for canine and feline coronavirus but efficacy is limited

Vaccination:

- **MUST READ THE PRODUCT LEAFLET OF YOUR BRAND OF VACCINE**
- The following information is based on the WSAVA recommendations (2006)

- **Vaccinations:**
- **Dogs:**
  - ✓ Core vaccinations, i.e. C3:
    - ➢ Canine parvovirus: Fatal bloody diarrhoea
    - ➢ Canine distemper virus: Coughing, diarrhoea, seizure, loss of balance, blindness
    - ➢ Canine adenovirus: Vomiting, diarrhoea, abdominal pain, liver failure
    - ➢ Rabies (non-core in Australia)
  - ✓ Examples of non-core:
    - ➢ These vaccines are recommended if regionally present or travelling into an endemic region
    - ➢ Parainfluenza virus, *Bordetella bronchiseptica*, Rabies, *Leptospira* etc.
- **Cats:**
  - ✓ Core vaccinations, i.e. F3:
    - ➢ Feline panleukopaenia: Gastroenteritis, fever, vomiting, diarrhoea, liver failure or sudden death
    - ➢ Feline calicivirus: Cat flu - sneezing, lethargy, conjunctivitis, mouth ulcers
    - ➢ Feline herpesvirus: Cat flu - sneezing, lethargy, conjunctivitis, inappetance or permanent nasal and sinus infection
  - ✓ Examples of non-core:
    - ➢ These vaccines are recommended if regionally present or travelling into an endemic region
    - ➢ Feline leukaemia virus, feline immunodeficiency virus, rabies, feline infectious peritonitis virus, *Chlamydophila felis*, *Bordetella bronchiseptica* etc.

- **Core vaccination regimes:**
- For puppy:
  - ✓ Start at 6 to 8 weeks of age, then give booster every 2-4 weeks (depending on environmental risk) until 16 weeks of age or older
    - ➢ Aim to give last booster no earlier than 16 weeks of age
  - ✓ At 6 to 12 months of age receive a booster OR
  - ✓ Perform serology testing (CDV/CAV/CPV-2) 4 weeks after the 16 week vaccination:
    - ➢ If seropositive, then a 6 to 12 month booster is not required, continue with adult regime, repeat core vaccine booster 3 years later
    - ➢ If seronegative, then revaccinate and re-test the serology 4 weeks later
    - ➢ If again seronegative, consider the puppy a non-responder (possibly incapable of developing protective immunity)
- For adults dogs:
  - ✓ Core vaccines can confer up to 7 years protection, but current recommendation is 3 yearly booster
  - ✓ If never vaccinated or unknown history, then give 2 doses 2 to 4 weeks apart
- For kittens:
  - ✓ Caution with live vaccines as spillage can lead to clinical signs

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- ✓ Start at 6 to 8 weeks of age, then give booster every 2 to 4 weeks (depending on environmental risk) until 16 weeks of age or older
  - ➢ Aim to give last booster no earlier than 16 weeks of age
- ✓ At 6 to 12 months of age receive a booster OR
- ✓ Perform serology testing (FPV/FCV/FHV-1) 4 weeks after the 16 week vaccination:
  - ➢ If seropositive, then a 6 to 12 month booster is not required, continue with adult regime, repeat core vaccine booster 1-3 years later depending on environmental risk
  - ➢ If seronegative, then revaccinate and re-test the serology 4 weeks later
  - ➢ If again seronegative, consider the kitten a non-responder (possibly incapable of developing protective immunity)

- For adult cats:
  - ✓ Protection conferred by FCV and FHV-1 core vaccines are not as robust as the FPV and canine core vaccines
  - ✓ Revaccinate at least every 3 years unless high risk e.g. visits cattery, outdoor cat etc.
    - ➢ If high risk, then yearly or strategic vaccinations e.g. 3 months before visiting a cattery
- For pregnant animals:
  - ✓ Must read the manufactures product information
  - ✓ +/- Avoid modified live vaccinations

- **Vaccination regimes for non-CORE vaccines (see below):**
- Read and follow the recommended regime by the manufacture
- These vaccines are recommended to be given if regionally present or travelling into an endemic region

Vaccine reactions:

- **Features:**
  - ✓ Anaphylaxis can develop within minutes
  - ✓ Dermatological signs can develop within hours of vaccination
  - ✓ Unless severe anaphylaxis, vaccine reactions are not a reason not to revaccinate, administer an anti-histamine prior and monitor after vaccination, a short course of anti-histamines may be indicated
- **Clinical signs:**
  - ✓ Dermatological or localised:
    - ➢ Usually see urticaria (welts over body) and facial angioedema
  - ✓ Systemic:
    - ➢ Uncommonly systemic anaphylaxis
    - ➢ Collapse, pale mucous membranes, slow capillary refill time
    - ➢ Can start as pruritus, urticaria and progress to loss of bladder and bowel control
- **Treatment:**
  - ✓ Dermatological or localised:
    - ➢ Antihistamine:
      - Chlorpheniramine 0.5mg/kg SC/IM, +/- oral course to cover for delayed hypersensitivity
    - ➢ Corticosteroids:
      - Dexamethasone 0.2mg/kg SC
  - ✓ Systemic:
    - ➢ See **"Shock and Anaphylaxis"**
    - ➢ Combination:
      - Adrenalin 0.005mg/kg IV
      - Corticosteroids: Prednisolone sodium succinate 2mg/kg IV
      - Antihistamine: Chlorpheniramine 0.5mg/kg IM/IV
      - IV fluids:
        - o Boluses and then high rates of fluids
        - o Monitoring **"end point resuscitation variables"**

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Vomiting and Regurgitation

- **This chapter covers:**
  - ✓ Differentiation between vomiting and regurgitation
  - ✓ General diagnostic principles
  - ✓ General treatment and management options

- **Questions:**
  - ✓ **Is it vomiting or regurgitation:**

Regurgitation:	**Vomiting:**
▪ Passive – no heaving ▪ No nausea or retching ▪ +/- Undigested food ▪ +/- No bile	▪ Active – heaving ▪ Usually nausea or retching ▪ Usually digested food with bile

- **History:**
  - ✓ **Rule out regurgitation:** Heaving, retching, bile, digested food
  - ✓ When?: Associated with eating or not (if yes then most likely gastrointestinal related)
  - ✓ Frequency?
  - ✓ Duration: Acute/chronic
  - ✓ Appetent or inappetent
  - ✓ Water intake: Normal or altered
  - ✓ Concurrent diarrhoea
  - ✓ Parasite control: Hookworm
  - ✓ Current medications: e.g. NSAIDS or antibiotics
  - ✓ Diet: Currently fed, recent changes, how much, treats/scraps
  - ✓ Access to toxins: e.g. Toxic plants – Cycads, *Brunfelsia sp.*, compost or decaying vegetable matte
  - ✓ Access potential foreign bodies: Bones, toys, string

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- **Vomiting:**

Differentials for vomiting:

- **Mechanisms of vomiting:**
  - Activation of peripheral receptors within the gastrointestinal tract
  - Activation of central vomit centre by CNS disease e.g. Motion sickness, neoplasia etc.
  - Vestibular disease
  - Activation of chemoreceptor trigger zone by chemicals/toxins, e.g. Uraemia, diabetic ketoacidosis

Gastrointestinal:	Extra-gastrointestinal:
• **Gastritis/enteritis:** ➢ Acute gastritis: • Most common cause - not seriously ill • Garbage ingestion, high fat content, allergy, specific protein intolerance, viruses, parasites, plants • Bacterial: *Helicobacter sp.* (difficult to Dx) ➢ Haemorrhagic gastroenteritis (HGE): • *Clostridium perfringens* hypersensitivity, parvovirus, parasites ➢ Chronic gastritis: • Sporadic vomiting • Endoscope and biopsy to assess for inflammatory bowel disease and neoplasia • **Gastric/intestinal foreign body/outlet obstruction:** ➢ Signs of obstruction: • Foreign body, intraluminal masses or intussusception • Muscular proliferative or infiltrative disease • Compression of outflow tract e.g. neoplasia, granulomatous disease • Malposition of stomach e.g. GDV • **Gastric/intestinal ulceration and erosion:** ➢ Signs of haematemesis, haematochezia, abdominal pain, melaena, anaemia and hypoproteinaemia: • Drugs e.g. NSAIDs, corticosteroids • Hypotensive states • Severe hepatic or liver disease • Neoplasia – mast cell tumours • Stress e.g. severe illness • Bacterial, viral and parasites • **Gastric motility disorders:**	• **Systemic diseases:** ➢ Endocrine disease: • Hypoadrenocorticism • Hyperthyroidism • Diabetes mellitus/ketoacidosis • Hypercalcaemia ➢ Metabolic disease: • Hepatic disease • Renal disease • Electrolyte abnormalities ➢ Pancreatitis ➢ Neoplasia ➢ Etc. ➢ See **"Hepatobiliary Disease"** and **"Pancreatic Disease"** and **"Renal Disease"** • **Vestibular disease:** ➢ +/- Head tilt, nystagmus ➢ See **"Neurological Disease"** • **CNS disease and activation of CRTZ:** ➢ CNS disease: Any can cause vomiting ➢ Motion sickness ➢ Chemicals or toxins e.g. uraemia, diabetic ketoacidosis

Diagnostics for vomiting:

- Good history and general physical examination (abdominal palpation and rectal examination)
- Radiographs: Rule out indications for surgery e.g. Foreign bodies and obstructive patterns
- Haematology, biochemistry and electrolytes
- Urinalysis
- Abdominal ultrasound
- Faecal analysis: Smears and float, *Giardia* SNAP test
- Viral testing: Parvovirus, Coronavirus, FeLV, FIV
- Other tests: Total T4 (cats), ACTH stimulation test, cPLI, fPLI, TLI, heartworm (cats)
- Endoscopy and or laparotomy + biopsy

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- **General treatment plans:**

Clinical signs:	Plans:
• **Chronic but infrequent** • **Bright, alert and responsive** • Normal general physical examination • No diarrhoea or weight loss • Eating	• Dietary investigation: ➢ History of indiscretion/related to eating ➢ Nil per os for 12 hours ➢ Dietary modification: • Highly digestible bland food (Hills I/D® or chicken and rice) • If not better in 2 weeks, trial Hills Z/D® • If not better in further 2 weeks, continue along diagnostic path ➢ Symptomatic medications: • H_2 receptor antagonists: E.g. Ranitidine 2mg/kg BID • Proton pump inhibitors: E.g. Omeprazole 1mg/kg SID • Gastrointestinal protectants: E.g. Sucralfate 0.5-1gm PO BID • Metronidazole 10mg/kg BID, immune modulation/anti-inflammatory • Parasite and *Giardia* control +/- faecal analysis • IF >7 years old: ➢ Haematology, biochemistry, urinalysis ➢ Endocrine testing ➢ If labs are normal, then dietary modification • **If continued vomiting:** ➢ Survey radiograph +/- ultrasound ➢ Endoscope and biopsy
• **Acute and frequent** • **Bright, alert and responsive** • Normal general physical examination • No diarrhoea • Eating	• **Survey radiograph +/- ultrasound,** assess for obstruction ➢ If older: Haematology, biochemistry, urinalysis ➢ If young: Faecal testing, *Giardia* testing • +/- Hospitalise and supportive therapy whilst NPO: ➢ IV fluids and correction of metabolic abnormalities ➢ Symptomatic medications, as above • Follow plan above – i.e. Diet modification
• **Acute and frequent** • **Looks sick, depressed** • Abnormal general physical examination • Dehydrated, weight loss, inappetant	• **Hospitalise and supportive therapy:** ➢ IV fluids, correction of perfusion, hydration and metabolic abnormalities ➢ Symptomatic medications, as above • **Full diagnostic work-up:** ➢ Full work-up including imagery, blood tests and faecal testing
• Any of the above **but also diarrhoea**	• **Follow appropriate plan above, plus:** ➢ Differentiate small intestinal diarrhoea from large intestinal diarrhoea ➢ Faecal analysis ➢ Parvovirus and *Giardia* testing

- **Treatment of specific conditions:**
  - ✓ **See "Diarrhoea and Haematochezia"**
  - ✓ **See "Hepatobiliary Disease"**
  - ✓ **See "Pancreatic Disease"**
  - ✓ **See "Neurological Disease"**

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- **Regurgitation:**

Differentials for regurgitation:

- **Mechanisms of Regurgitation:**
  - ➢ Altered motility
  - ➢ Obstruction of flow:
    - External compression
    - Internal obstruction
  - ➢ Inflammation

- **Altered motility:**
  - ➢ Megaoesophagus:
    - Congenital
    - Acquired (adult onset):
      - o Primary megaoesophagus (idiopathic): Diagnosis of exclusion (i.e. causes of secondary)
      - o Secondary megaoesophagus: Myasthenia gravis, myopathy/neuropathy, toxicity, dysautonomia, tick paralysis, snake envenomation etc.
- **Obstruction of flow:**
  - ➢ **External compression of oesophagus:**
    - Vascular ring abnormalities: Most commonly persistent right aortic arch
      - o Neoplasia within the cervical region or thoracic cavity
  - ➢ **Internal obstruction of oesophagus:**
    - Strictures: Secondary to inflammation
    - Oesophageal foreign bodies
    - Neoplasia (rare)
- **Inflammation of the oesophagus:**
  - ➢ Chronic vomiting:
    - Topical irritants: Oral antibiotics (Doxycycline and clindamycin), ingested caustic agents, foreign body

Diagnostics for regurgitation:

- Should assess for concurrent diseases such as laryngeal paralysis as may indicate primary neurological disease
- Good history and general physical examination
- Radiographs:
  - ➢ +/- Barium swallow
  - ➢ Assess for aspiration
- Endoscopy and biopsy
- Other see below under **"Megaoesophagus"**

- **Megaoesophagus:**
- **Features:**
  - ✓ Common in dogs, rare in cats
  - ✓ Persistent reduced motility of the oesophagus
  - ✓ If have laryngeal paralysis, can have megaoesophagus
  - ✓ Must assess for aspiration pneumonia
- **Causes:**
  - ✓ Congenital:
    - ➢ Clinical signs develop when weaning onto solid food
  - ✓ Acquired (adult onset):
    - ➢ Primary megaoesophagus (idiopathic):
      - Diagnosis of exclusion (i.e. causes of secondary)
      - Typically, large breed dogs

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- Secondary megaoesophagus:
  - Myasthenia gravis (20% of cases)
    - Immune response against acetylcholine receptors
    - +/- Can be focal or generalised (systemic weakness)
  - Other: Myopathy, neuropathy (tick paralysis, snake envenomation), toxicity, dysautonomia, systemic lupus erythematosis, hypoadrenocorticism, oesophagitis (*Spirocerca lupi*), thymoma etc.

- **Diagnostics:**
  - ✓ Radiographs:
    - +/- Barium swallow
  - ✓ Haematology, biochemistry, urinalysis
  - ✓ Endoscopy
  - ✓ Faecal float – *Spirocerca lupi*
  - ✓ ACh receptor anti-body test
  - ✓ Adrenal functioning tests
  - ✓ Systemic lupus erythematosus anti-nuclear antibody tests
  - ✓ If central nervous system signs, +/- distemper virus testing, CSF, CT
- **Treatment:**
  - ✓ Congenital: No treatment, poor prognosis
  - ✓ Acquired: Treat underlying cause, prognosis depends on cause
  - ✓ Other: Feeding height, vary food consistency (variable response between dogs)

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Wound Management

- **This chapter covers:**
  - ✓ Steps in assessing and managing a wound

- **4 Categories:**
  - ✓ Clean: Atraumatic, surgical wound, aseptic conditions
  - ✓ Clean contaminated: Minor break in asepsis e.g. Controlled entry into gastrointestinal tract
  - ✓ Contaminated: Recent wound with contamination with exposure to elements (abrasions) or major breaks in asepsis
  - ✓ Dirty or infected: Old wound with exudate and obvious infection e.g. Wound with abscess

- **Wound management:**
  1. Triage and patient stabilisation:
     - ➢ A: Airway, B: Breathing, C: Circulation
  2. Control any obvious bleeding:
     - ➢ Pressure bandage or tourniquet (<1 hours)
  3. Analgesia and antibiotics:
     - ➢ Systemic analgesia or local or regional analgesia
     - ➢ Broad spectrum antibiotics
  4. Prevention of further contamination:
     - ➢ Temporary sterile dressing: Moist gauze, cotton wool, elastoplast
  5. Assessment for other injuries:
     - ➢ Radiographs (chest - contusions, spine) and ultrasound (free abdominal fluid)
  6. Cleaning and preparing site for definitive management:
     - ➢ Clip surrounding hair, surgical scrub of surrounding skin (not wound)
  7. Debridement:
     - ➢ Lavage with Hartmanns (best):
       - 60ml syringe with 18g needle and 3 way tap
     - ➢ Surgical removal of foreign material and necrotic / non-viable tissue:
       - Exploration of all wounds for dead space
       - En-bloc debridement
     - ➢ Minimal debridement:
       - Distal limbs, bone and tendons aim to conserve as much as possible
  8. Drainage:
     - ➢ If excessive dead space that cannot be removed
     - ➢ Drains exit from most dependant site
     - ➢ Type:
       - Passive: Gravity
       - Active: Negative pressure
  9. Promotion of wound granulation (if left open):
     - ➢ Proper selection of bandaging material
  10. Selection of an appropriate method of closure:
     - ➢ **Primary closure:**
       - Clean, surgical wounds
       - Infected wounds that can be removed en-bloc
     - ➢ **Delayed primary closure:** 2 – 5 days post injury
       - Contaminated wound with unsure viability

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- Close when healthy granulation tissue has developed
- Bandage appropriately while waiting

➢ **Secondary closure:** > 5 days post injury
- Dirty or infected wounds
- Primary closure after granulation and epithelisation:
  - Usually excision of granulation and epithelised tissue

➢ **Secondary intention:** Not closed primarily
- Large wounds that cannot be closed primarily
- Healing by contraction of wound over granulation tissue bed and epithelialisation
- Can be very time consuming and expensive
- Not all wounds will heal properly

- **Wound dressing and bandaging:**

✓ Functions:
➢ Reduce dead space, oedema and haemorrhage by applying pressure
➢ Environment to facilitate debridement and absorb exudates
➢ Protection and immobilise the wound

✓ Three layers:
➢ Primary: Dressing applied directly to wound
- Adherent or non-adherent

➢ Secondary: Padded material that aids absorption
- Several layers of rolled cotton held with rolled gauze
- Moisture absorption and padding

➢ Tertiary: Protective layer that holds it in place
- Vet wrap or Elastoplast

✓ Adherent verses non-adherent:
➢ Adherent:
- Mechanical debridement when removed - necrotic tissue adheres to dressing
- Wet to dry dressing – wet sponges applied to wound with dry ones on top to absorb moisture
- Change regularly TID-SID when strikes through

➢ Non-adherent:
- When granulation tissue has formed
- Promote wound healing: Retain moisture, promote epithelisation, prevent desiccation
- Early non-adherent dressing:
  - Wounds that are still granulating e.g. Jelonet
- Late non-adherent:
  - Wounds are beginning to epithelialize and have already developed granulation tissue
  - E.g. Melonin, Cutilin, Solosite (wound gel)

✓ Autolytic debridement:
➢ Advantages:
- Selective debridement of only non-viable tissues
- Facilitates healing and granulation and epithelialization

➢ Disadvantages:
- Slow, moist and malodorous
- Require frequent and potentially expensive dressing changes

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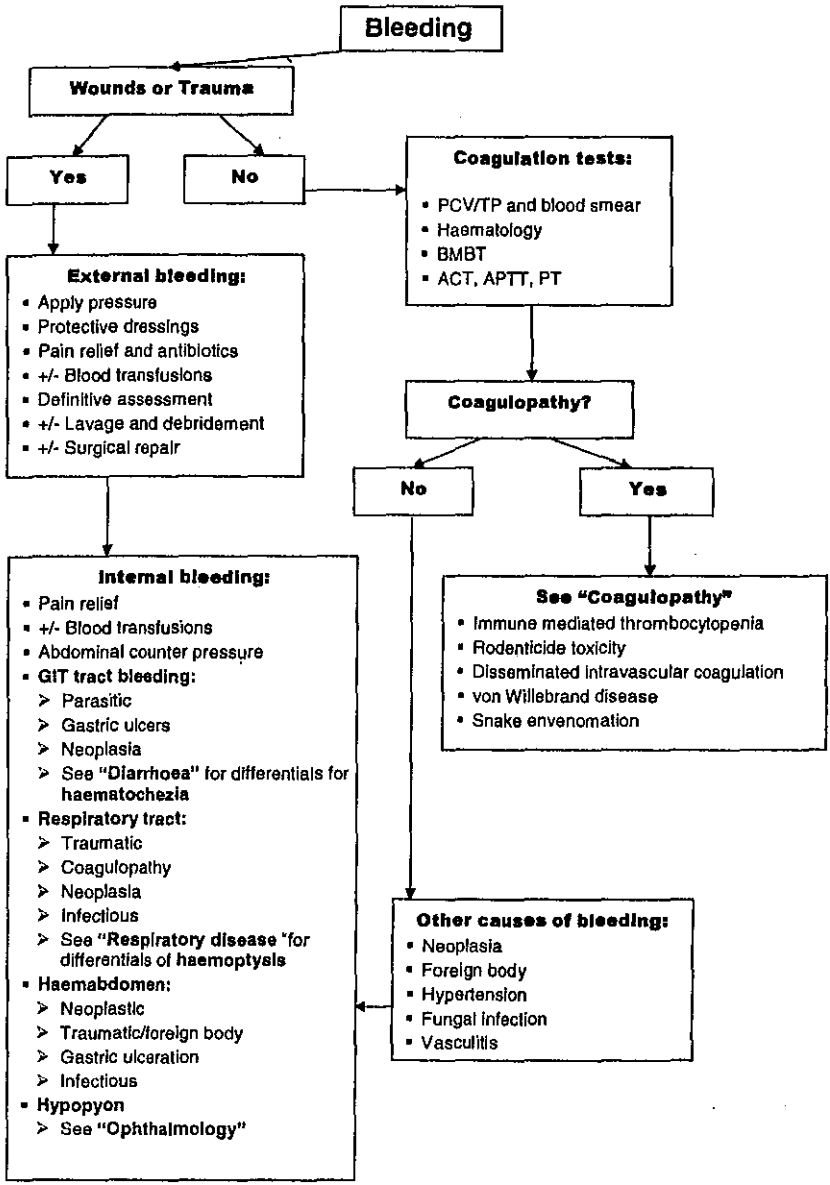

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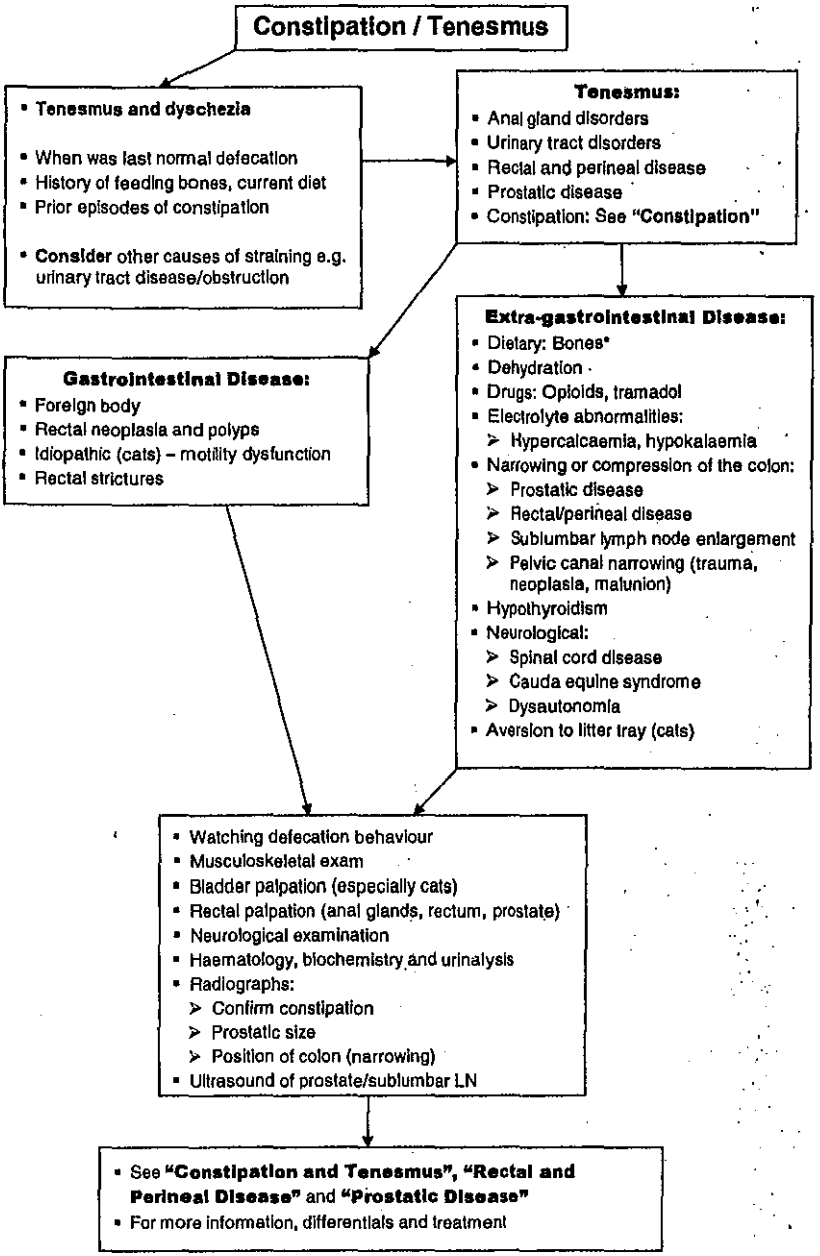

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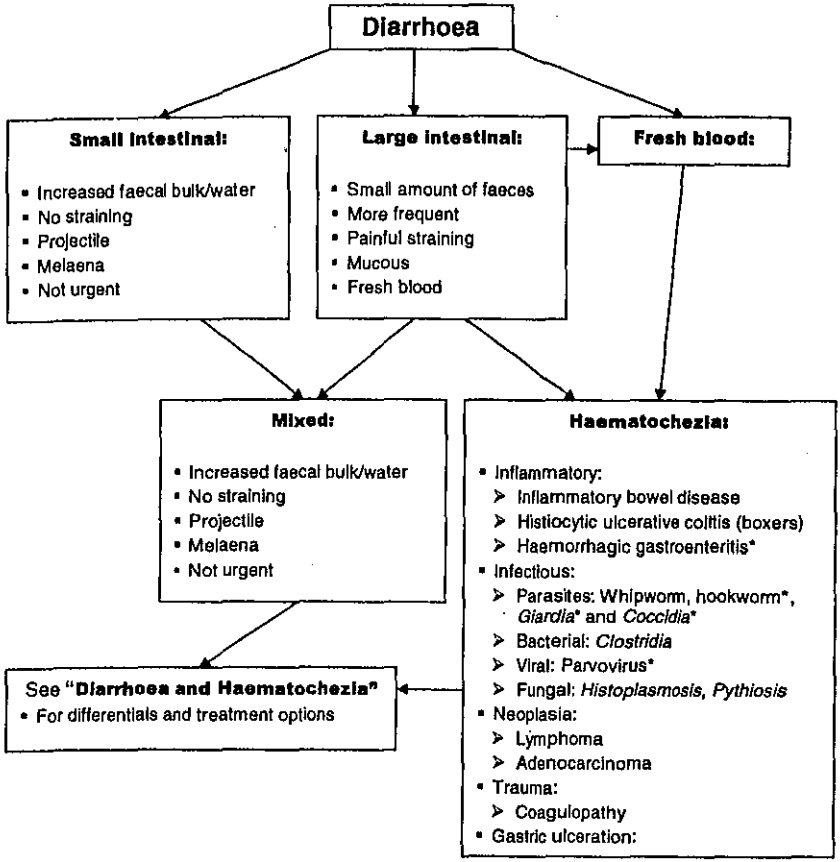

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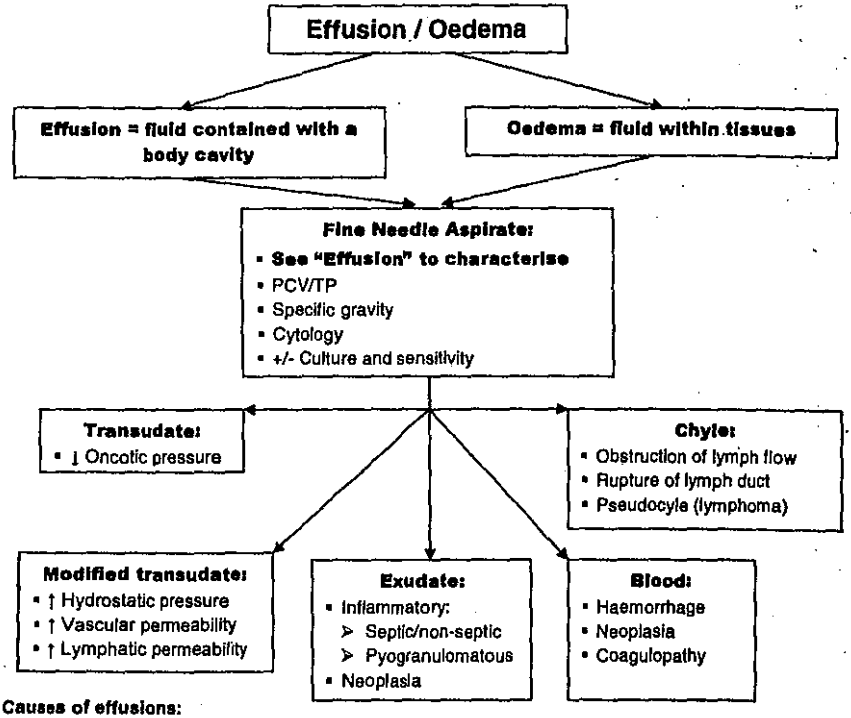

- **Causes of effusions:**

	Pleural:	Abdominal:
Transudates:	• Reduced oncotic pressure e.g. hypoproteinaemia (albumin <15gm/L) ➢ PLN, PLE, liver disease • Excess IV fluids (cats)	• Reduced oncotic pressure e.g. hypoproteinaemia (albumin <15gm/L) ➢ PLN, PLE, liver disease
Modified transudate:	• Increased capillary hydrostatic pressure e.g. RHS CHF, LHS CHF (cats), pericardial disease • Diaphragmatic hernia • Neoplasia • Lymphatic obstruction e.g. neoplasia, diaphragmatic hernia, abscess • Increased permeability of vessels (blood and lymphatics) e.g. FIP	• Increased capillary hydrostatic pressure: e.g. Portal hypertension: pre/intra/post-hepatic. RHS CHF • Increased permeability of vessels (blood and lymphatics) e.g. FIP
Non-septic Exudate:	• Inflammation: FIP (can have high globulins), liver disease, lung torsion, hernia • Neoplasia	• Neoplasia • Inflammation: Bile (increased [bilirubin] compared to serum), FIP, pancreatitis, torsion, uroabdomen
Septic Exudate:	• Ruptured abscess, foreign body inhalation, penetrating thoracic injury, severe pulmonary infection – especially fungal, oesophageal perforation	• Ruptured abscess (splenic, hepatic, urinary, prostatic), foreign body perforation, penetrating injury, bowel rupture

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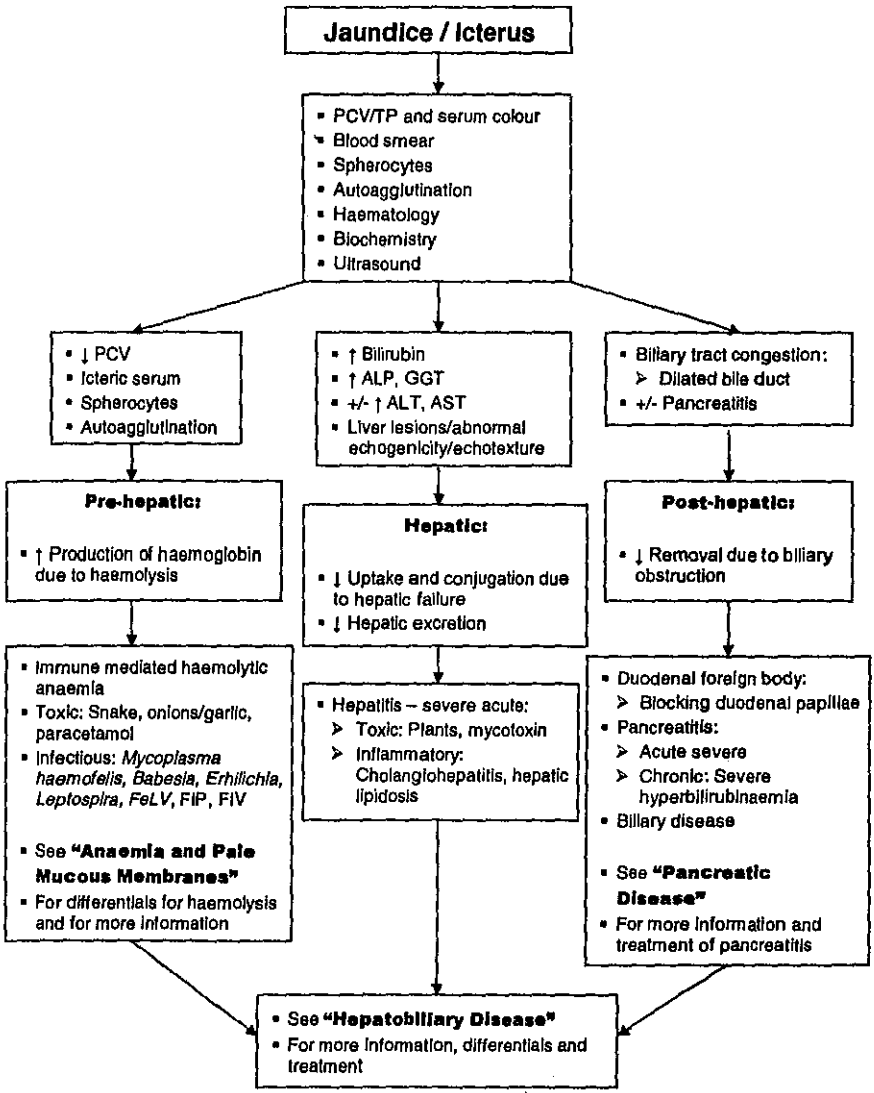

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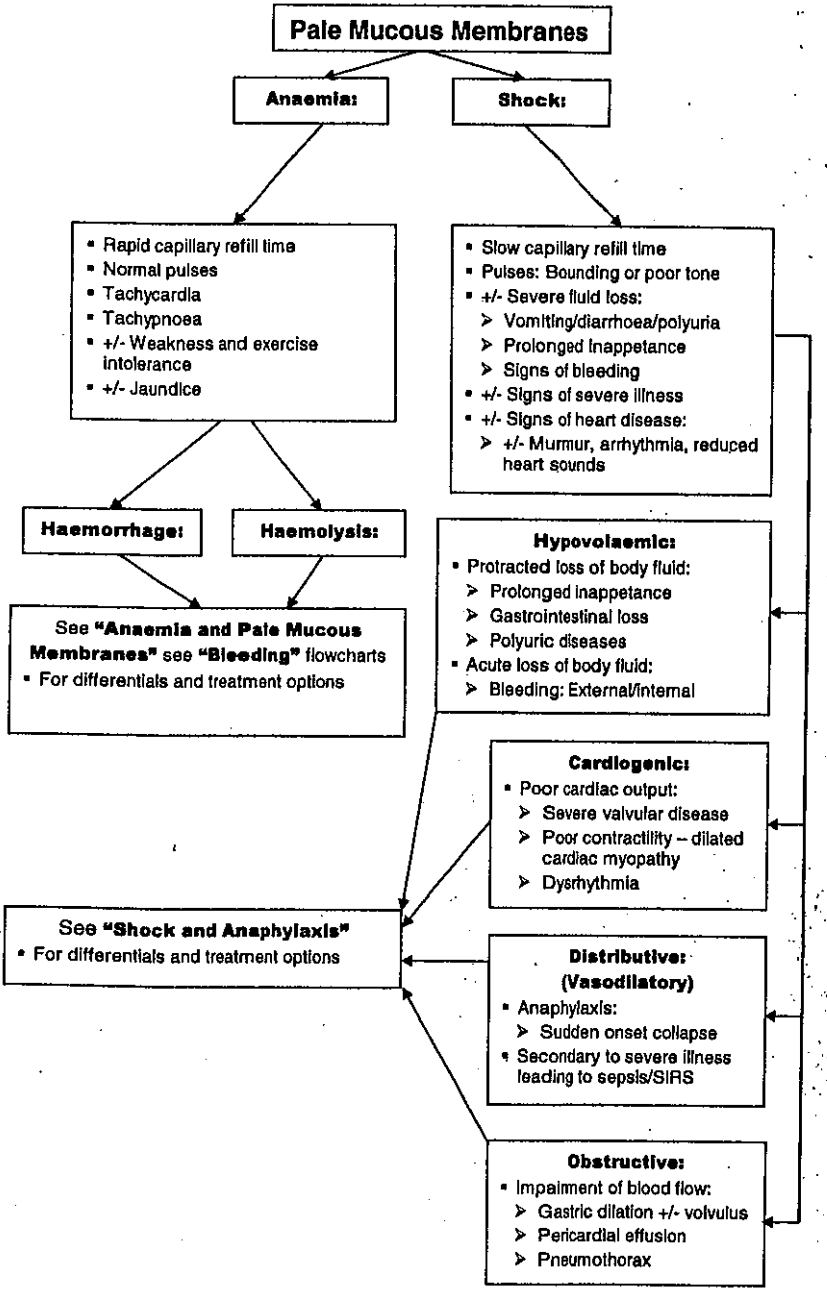

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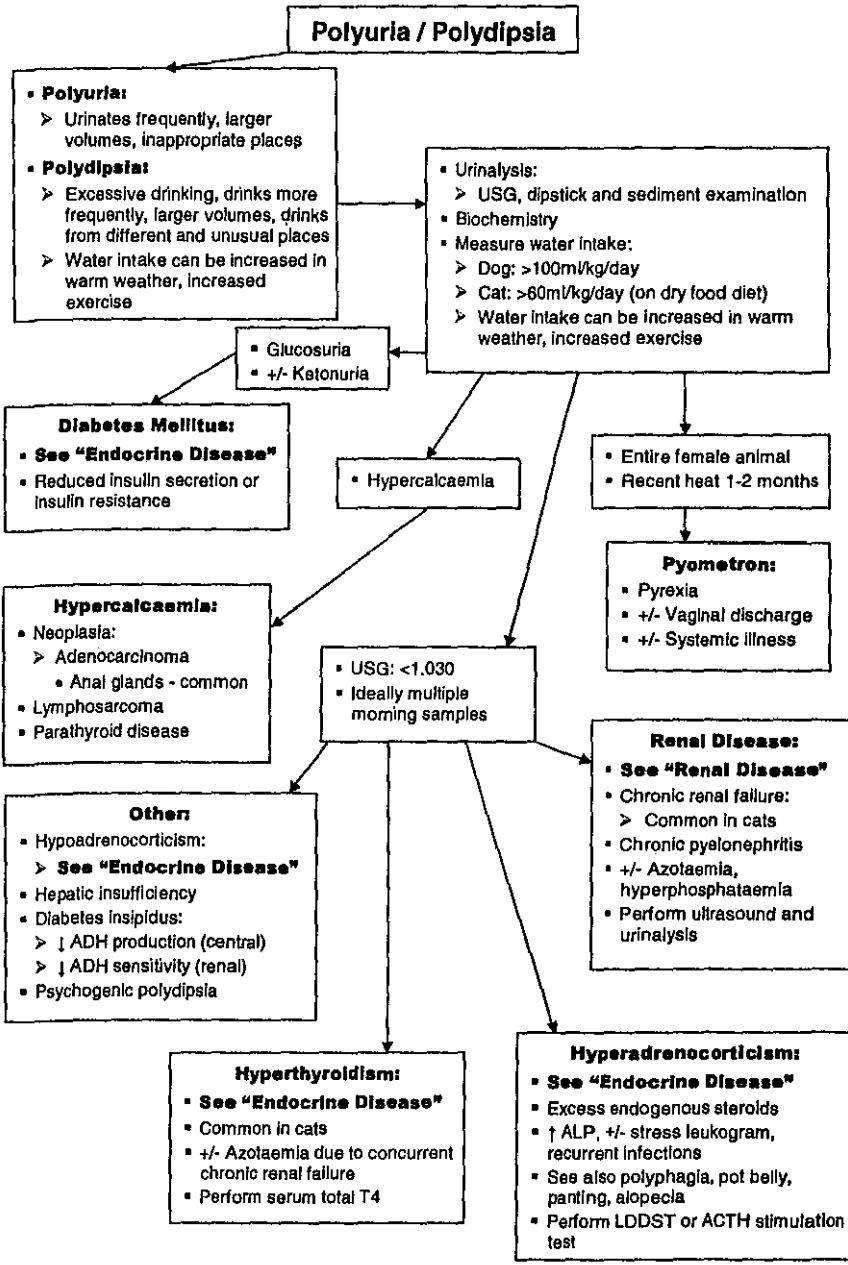

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Pyrexia / Fever of Unknown Origin

- **Persistent fever**
- **Severity:**
  - <40°C = mild
  - 40-40.5°C = moderate
  - >40.5°C = severe
- **NOT hyperthermia:**
  - ↑ Environmental temperature
  - Recent exercise
  - Reduced cooling – brachycephalic and laryngeal paralysis

- **General physical examination:**
  - Repeat every 8 to 12 hours
  - Assess all body systems: Oral, ocular, ear, skin, respiratory, cardiovascular, gastrointestinal, hepatic, renal, urinary, prostatic, lymph nodes, musculoskeletal, reproductive
  - Neurological examination
- **Diagnostics:**
  - PCV/TP and blood smear, biochemistry, haematology, urinalysis
  - Cytology and culture of effusions, blood, CSF and urine
  - Serology
  - Radiographs and ultrasound

Infectious:

- **Bacterial:**
  - Foreign body
  - Peritonitis: Abdominal pain
    - Ultrasound
  - Discospondylitis: +/- Back pain
    - Radiographs, urine culture, +/- fungal
  - Endocarditis: +/- Murmur
    - Blood cultures, echocardiogram
  - Pneumonia/pyothorax: +/- Respiratory distress
    - Radiographs and BAL/FNA
  - Atypical: +/- Non-healing wounds
    - Mycobacterial, culture
  - Cat fight abscesses: Clip cat
  - *Mycoplasma haemofelis*: Smears, PCR
- **Protozoa:**
  - See **"Neurological Disease"** and **"Parasitic Disease"**
  - Neospora
  - Toxoplasma
- **Fungal:**
  - See **"Nasal and Nasopharyngeal disease"**
  - Aspergillosis:
    - Upper respiratory tract and disseminated (anywhere – eyes, brain, spine, bone, kidneys)
    - Beware German Shepherd
  - Cryptococcus: Mainly cats
    - Upper respiratory tract and neurological signs
- **Viral:**
  - See **"Viral Disease and Vaccination"**
  - FIP, FIV, FeLV: Systemic illness
  - FHV, FCV: Oral, upper respiratory tract and ocular signs

Neoplastic:

Inflammation:

- **Inflammatory conditions:**
  - Pancreatitis
  - Prostatitis
  - Cholangiohepatitis
  - Panniculitis

Immune mediated:

- **Immune mediated conditions:**
- IMHA:
  - See **"Anaemia and Pale Mucous Membranes"**
  - Jaundice, anaemia, agglutination, spherocytes
- IMTP:
  - See **"Coagulopathy"**
  - Thrombocytopenia, bleeding
- Immune mediated polyarthritis:
  - Shifting, multiple limb lameness
  - Hock and carpus usually
- Inflammatory meningitis:
  - See **"Neurological Disease"**
  - +/- Neck pain, neurological

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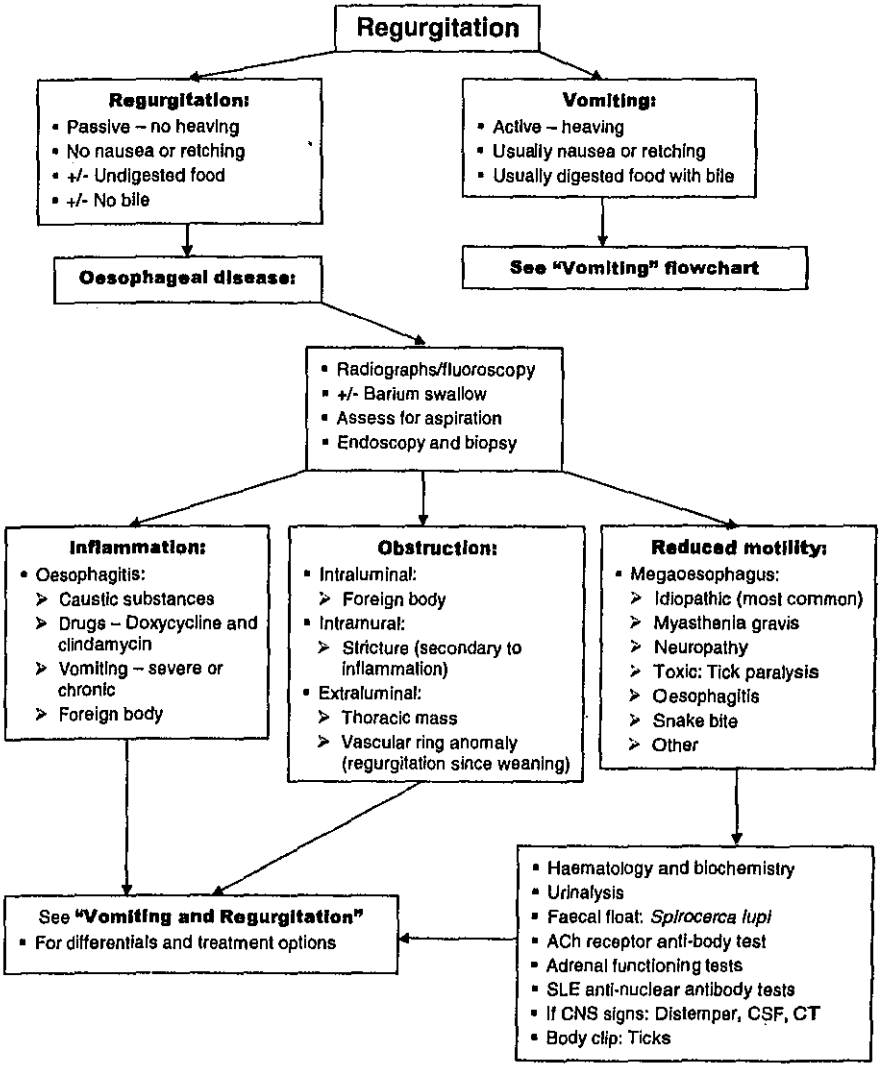

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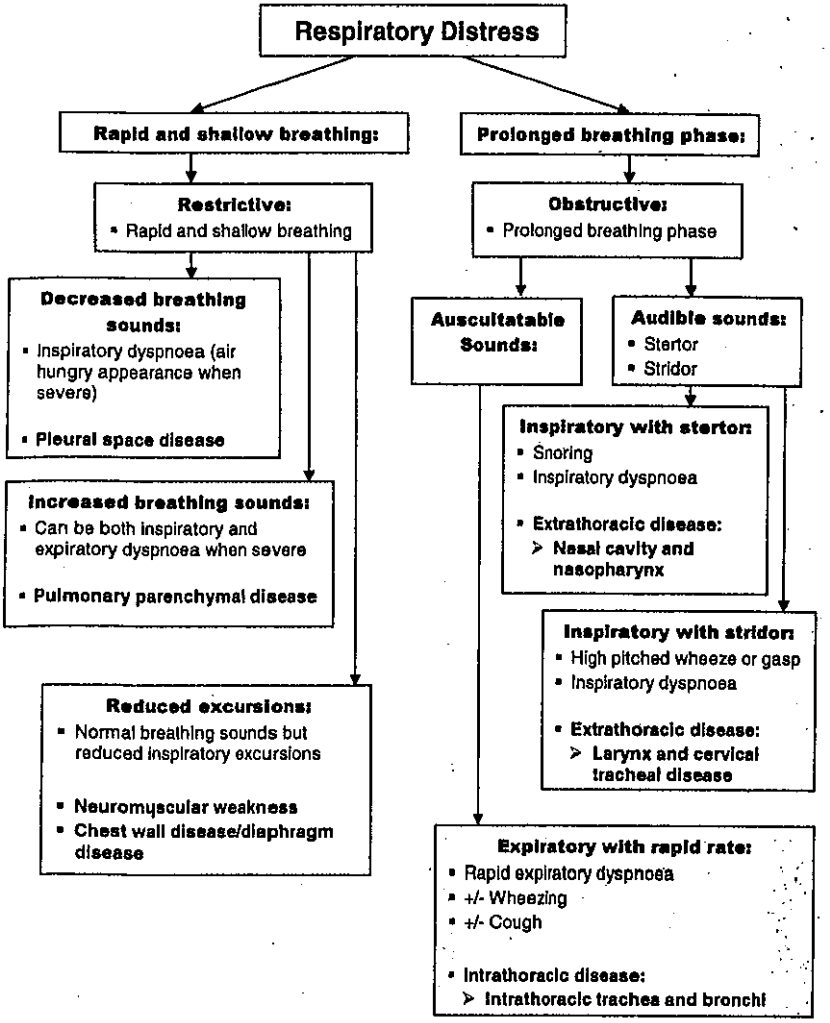

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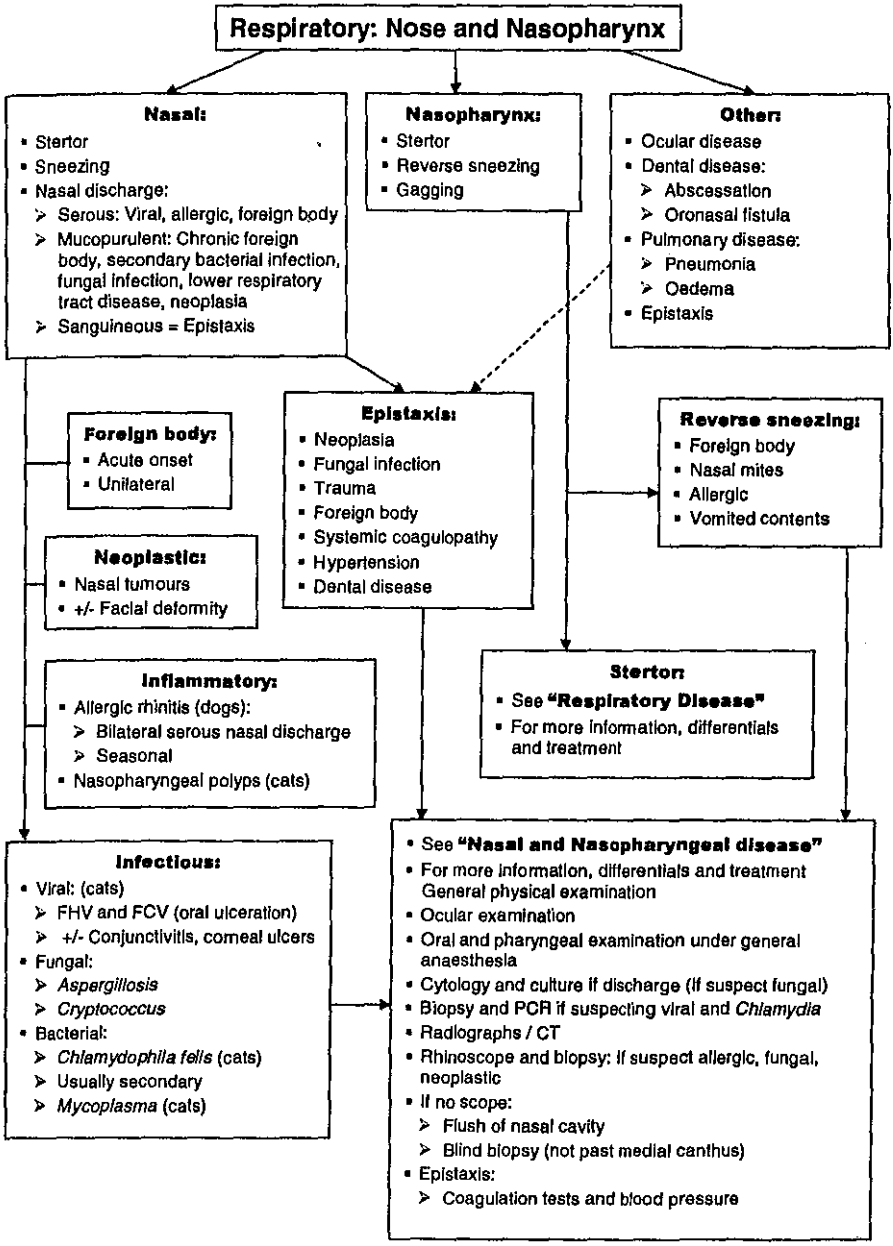

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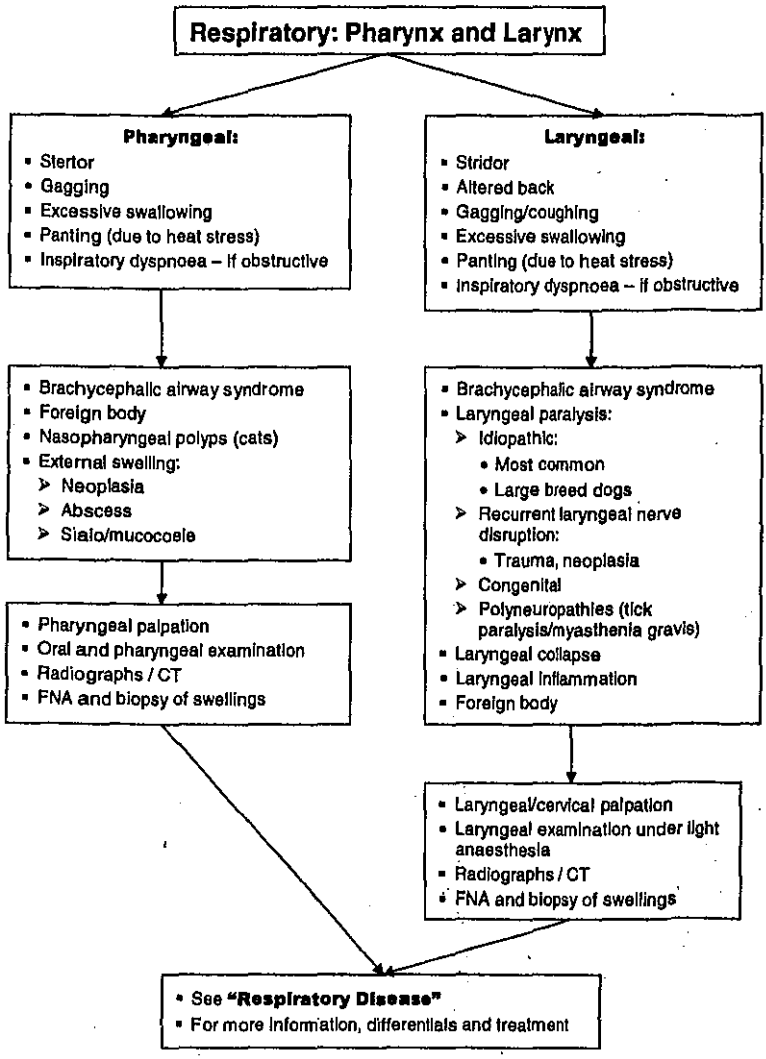

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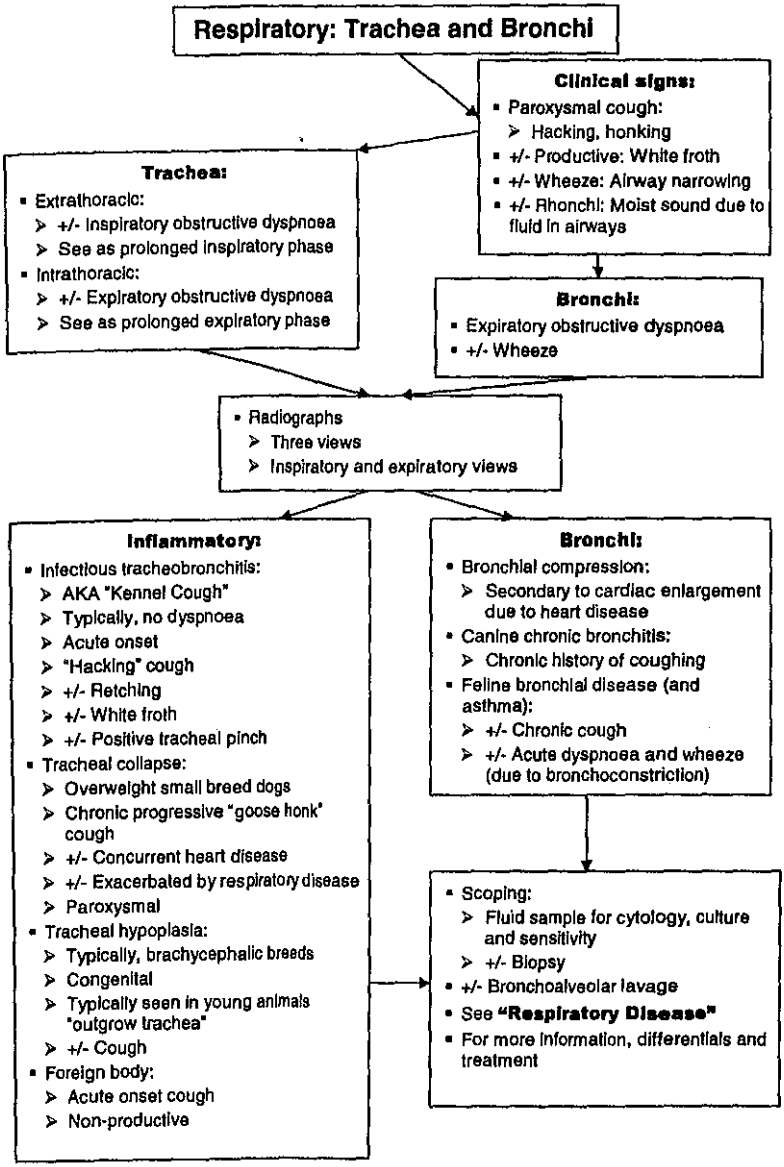

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Respiratory: Pulmonary, Pleural and Chest Wall

- **Restrictive dyspnoea**
- See = rapid, shallow breathing

- Reduced lung sounds

Pleural disease:
- Pneumothorax
- Diaphragmatic hernia
- Pleural effusion:
- Transudate/modified transudate:
  - RHS CHF, hypoalbuminaemia
- Haemorrhage:
  - Trauma, coagulopathy
- Inflammation:
  - Pyogranulomatous: FIP
  - Pus: Pyothorax
- Neoplastic:
- Chylous:
  - Obstruction or rupture of thoracic duct

- Cough:
  - Soft, moist
- +/- Productive
- Increased respiratory sounds: Crackles
- Exercise intolerance

- Reduced movement of chest wall with breathing
- Normal chest sounds

Chest wall disease:
- Trauma
- Tick paralysis
- Snake envenomation
- Botulism
- Neuromuscular disease – polyradiculoneuritis
- Neoplasia

Cardiac disease:
- Pulmonary oedema:
  - LHS congestive heart failure
  - Typically see a tachycardia if heart disease is causing the respiratory distress
  - Can also get bronchial compression causing coughing
- See **"Cardiovascular Disease"**:
  - Myxomatous mitral valve disease
  - Dilated cardiomyopathy

- Radiographs:
  - Three views
  - Inspiratory views
- PCV/TP, SPO_2
- Mucous membrane colour
- Colour of blood (methaemoglobin)
- Fine needle aspirate
- See **"Effusions"** to characterise and for more information

Pulmonary disease:
- Pneumonia:
  - Usually secondary – aspiration
- Pulmonary oedema (non-cardiogenic):
  - Near downing, obstructive, neurogenic (seizure, electrocution)
- Pulmonary neoplasia
- Inflammatory:
  - Eosinophilic bronchopneumonia
  - Acute respiratory distress syndrome
- Infectious:
  - Parasitic:
    - Toxoplasma
    - *Aelurostrongylus abstrusus*
    - Heartworm disease
  - Fungal
  - Bacterial – primary pneumonia = rare
- Haemorrhage:
  - Contusions
  - Coagulopathy

- See **"Respiratory Disease"**
- For more information, differentials and treatment

Other causes of dyspnoea:
- Reduced haemoglobin:
  - Anaemia
- Haemoglobin problem:
  - E.g. Paracetamol toxicity (cats)

Haemoptysis:
- Heart worm disease
- Neoplasia
- Coagulopathy
- Pneumonia
- Thromboembolism
- Pulmonary contusions
- CHF

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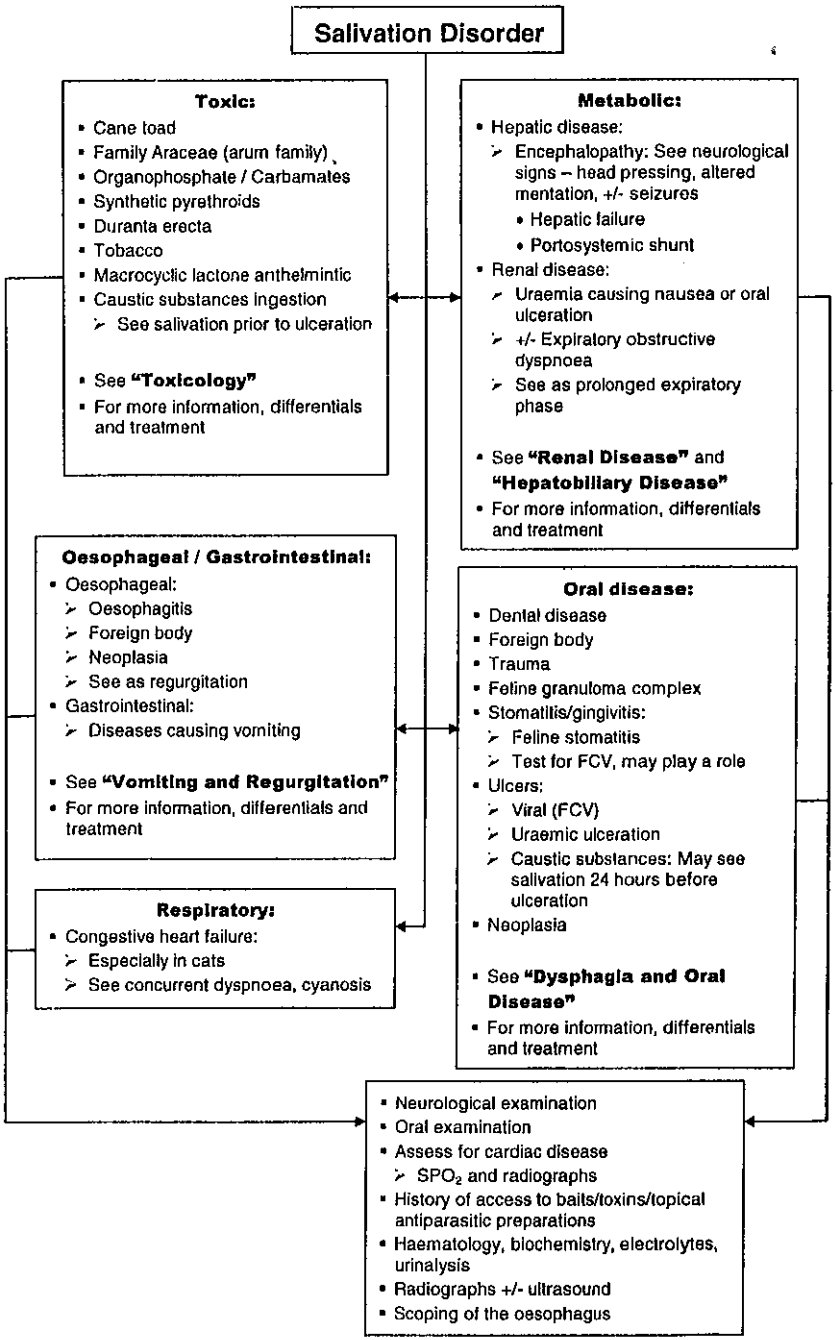

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Seizure Disorder

Seizure:

- **Generalised:**
  - Mass uncontrolled muscular activity
  - Loss of consciousness
  - Unresponsive to stimuli
  - +/- Urinary/faecal incontinence
  - Typically altered mentation prior to and after a seizure episode "disorientated, ataxic"
- **Partial:**
  - Repetitive twitching or movement of limbs
  - **More common in cats**

Intracranial:

- Inflammatory (meningoencephalitis):
  - GME
  - Steroid responsive
- Viral:
  - Distemper
- Metabolic:
  - Storage disease
- Neoplasia
- Vascular accident:
  - Clot or bleed
- Epilepsy (idiopathic)
- Hydrocephalus

Extracranial:

- Hepatic encephalopathy:
  - Hepatic failure
  - Portosystemic shunt
- Toxic:
  - Plants
  - Lead
  - Mycotoxins
  - Pyrethroids
  - Carbamate
  - Metaldehyde
  - Strychnine
- Metabolic:
  - Hypoglycaemic
  - Hypocalcaemia
  - Ischaemic
  - Thiamine deficiency

- Neurological examination
- Ocular examination
- Assess for extra-cranial disease:
  - History of access to baits/toxins
  - Haematology, biochemistry, electrolytes, urinalysis
  - If normal
- Assess for intracranial disease:
  - Cerebrospinal fluid: Cytology, culture, PCR
  - CT or MRI

- See **"Neurological Disease"** and **"Seizure Disorders"** and **"Toxicology"**
- For more information, differentials and treatment

Syncope:

- Collapse and loss of consciousness
- Generally, no muscular movement
- Recover quickly
- No altered mentation

- **Many differentials:**
- Cardiac (due to ↓ cardiac output):
  - Arrhythmia/Vasovagal
  - Heart disease
  - Cardiac tamponade
- Respiratory:
  - Hypoxia
  - Cough induced
- Systemic hypertension
- Glucose and electrolyte abnormalities
- Anaemia
- Vascular accident

- Haematology, biochemistry, electrolytes
- Urinalysis
- Resting ECG
- Thoracic radiographs
- Echocardiogram
- Systolic BP

Common causes in dogs:

- **Less than 1 year old:**
  - **Portosystemic shunts**
  - Inflammatory
  - Distemper
  - Hydrocephalus/storage disease
  - Toxicity
- **1 – 5 years old:**
  - **Epilepsy**
  - Inflammatory
  - Toxicity
  - Cerebral neoplasia
- **>5 Years:**
  - **Cerebral neoplasia**
  - Inflammatory
  - Toxicity
  - Epilepsy
  - Metabolic

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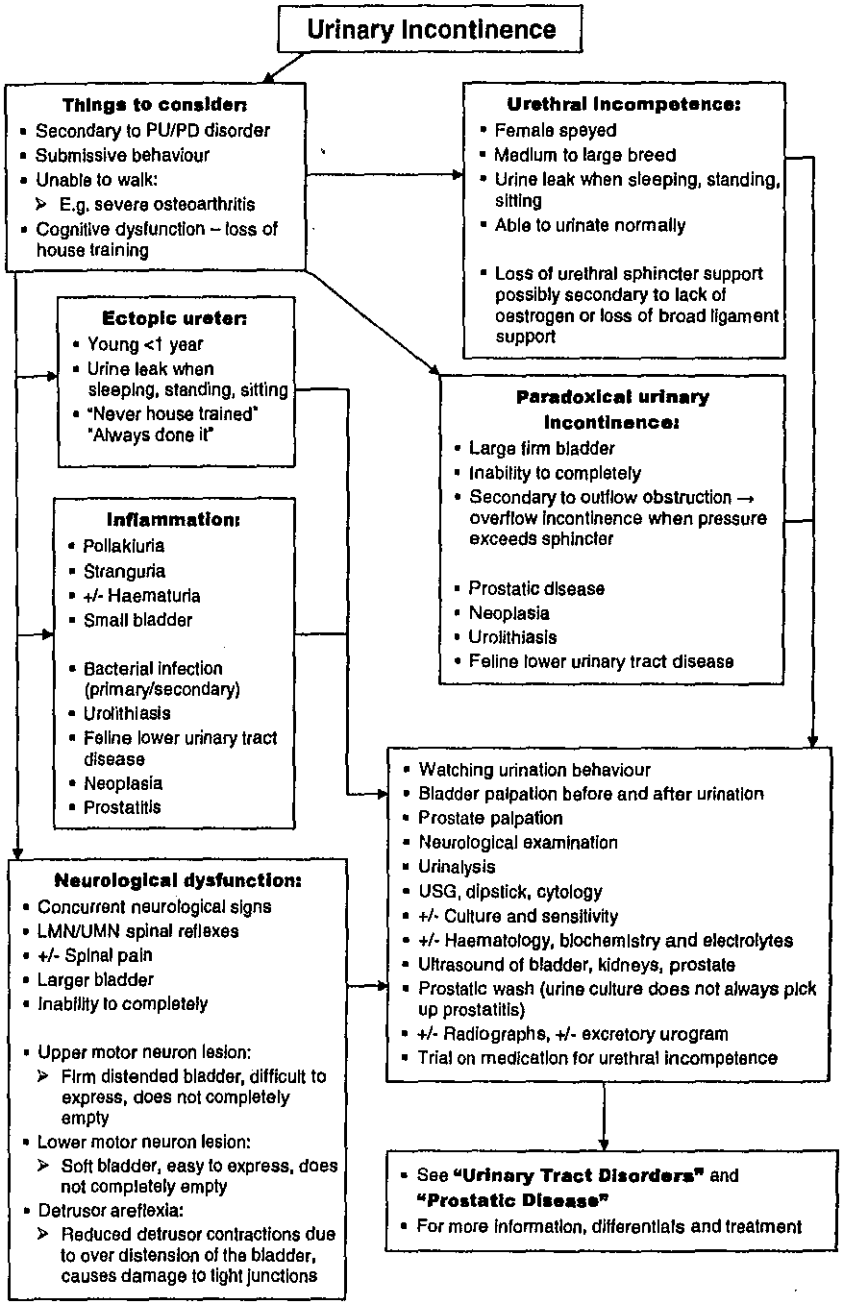

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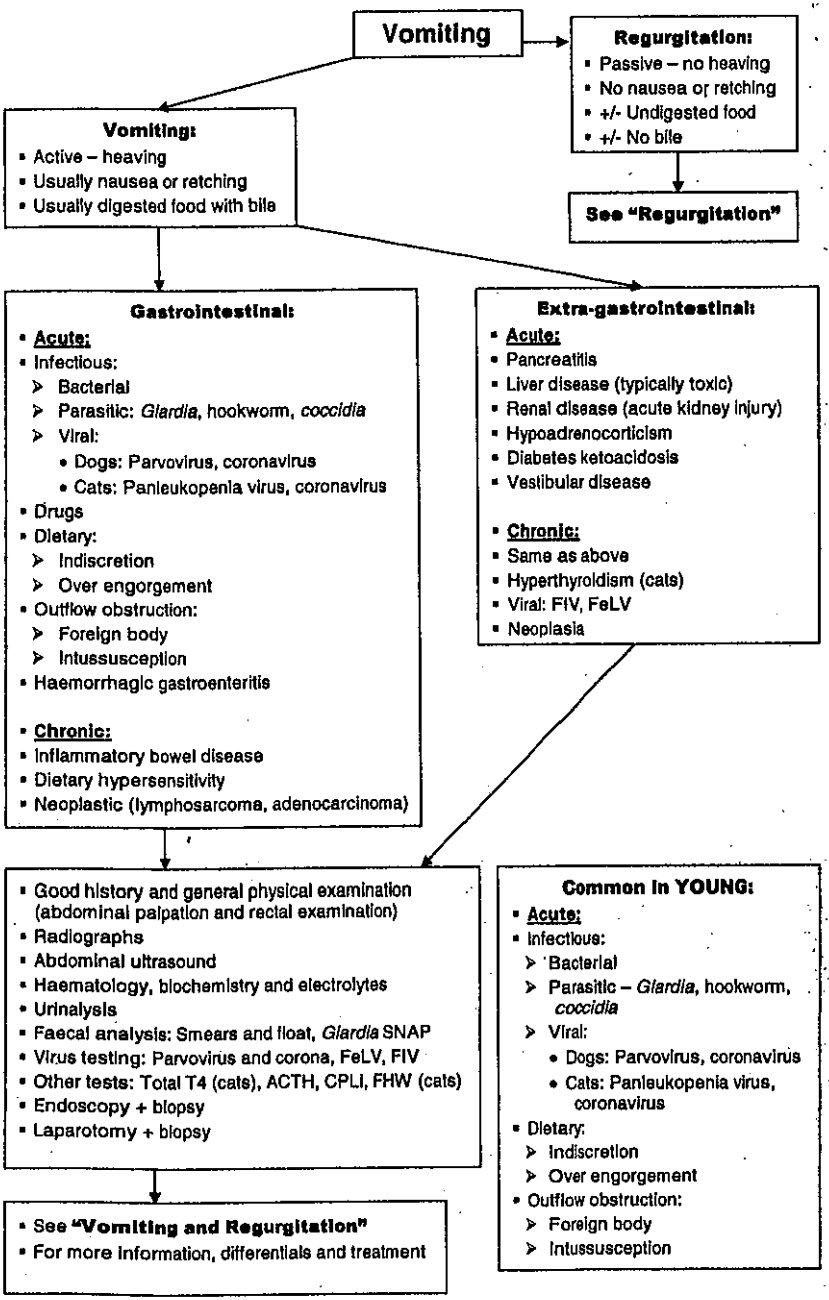

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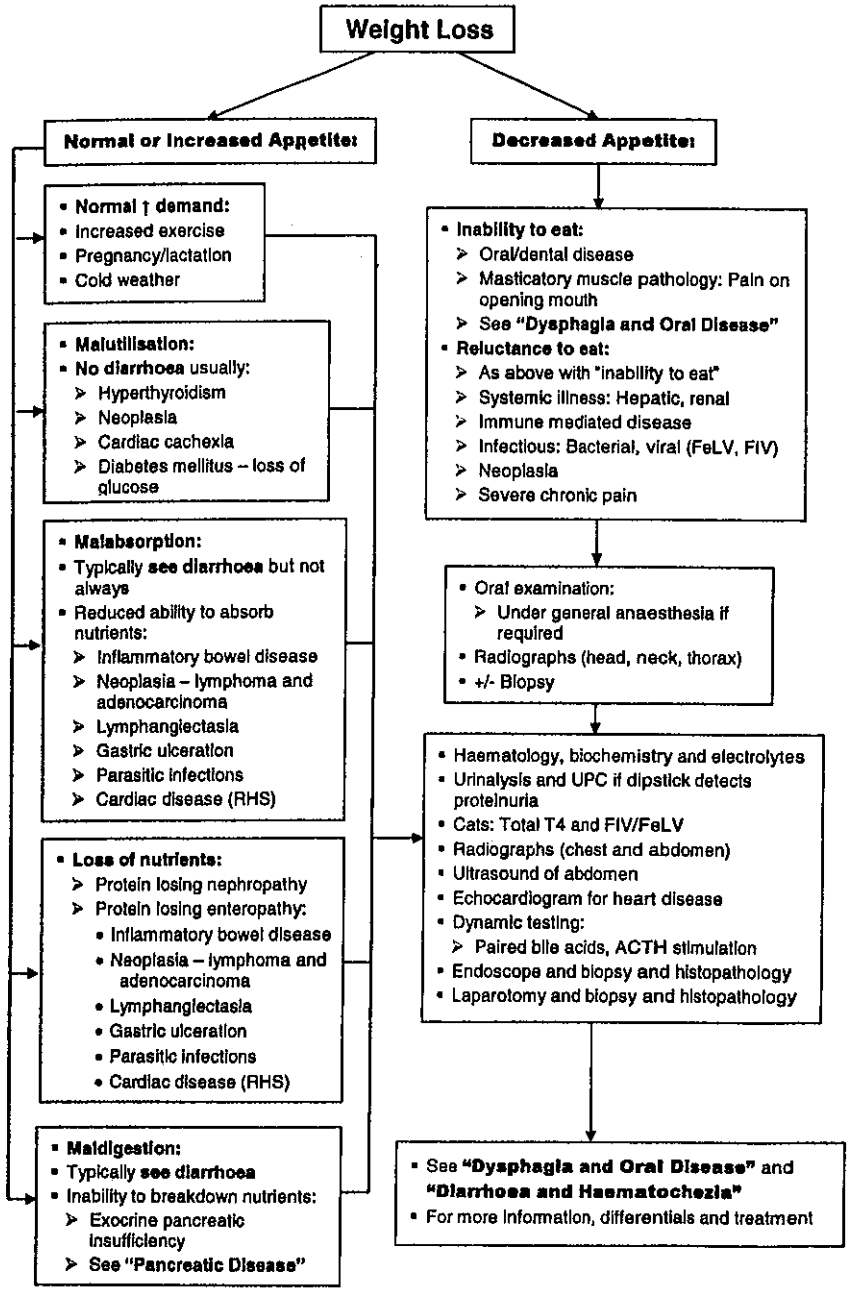

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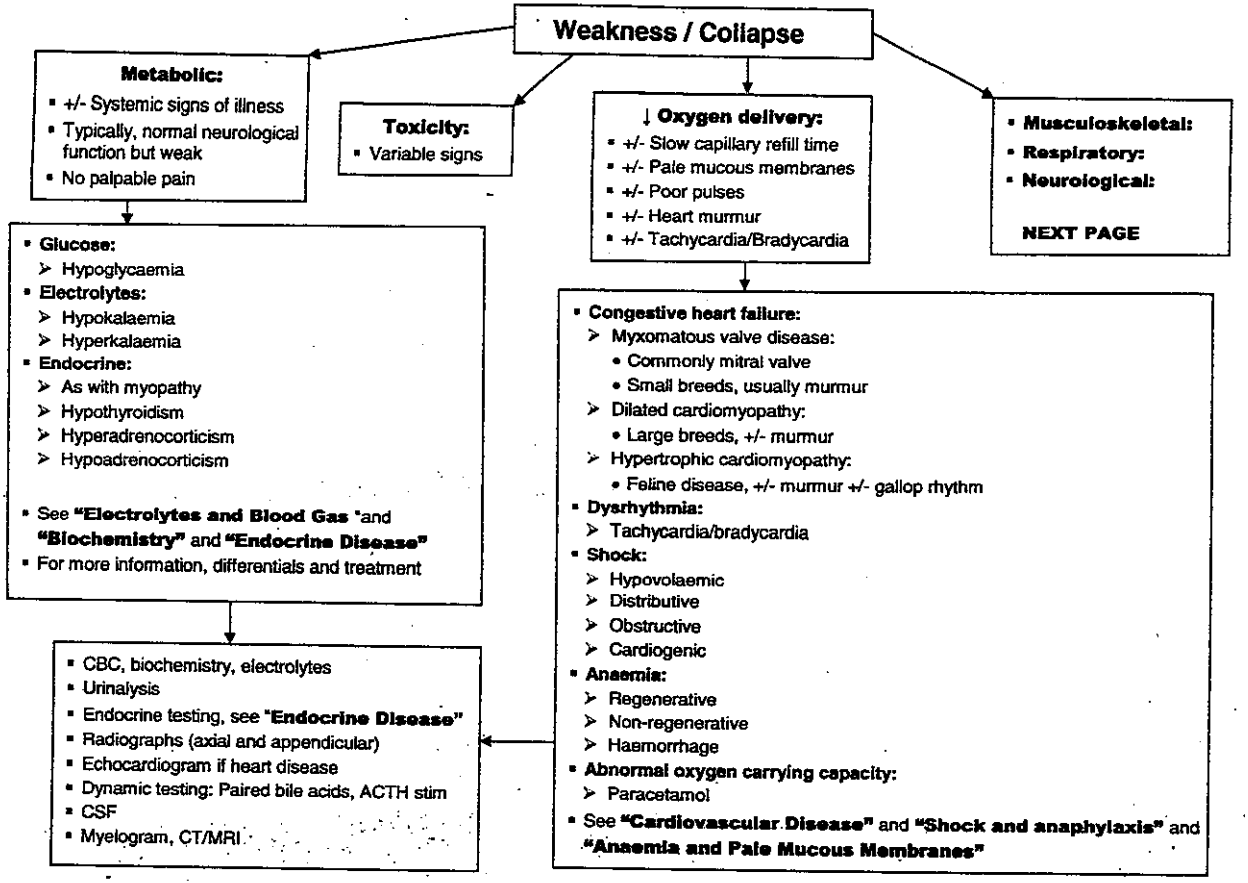
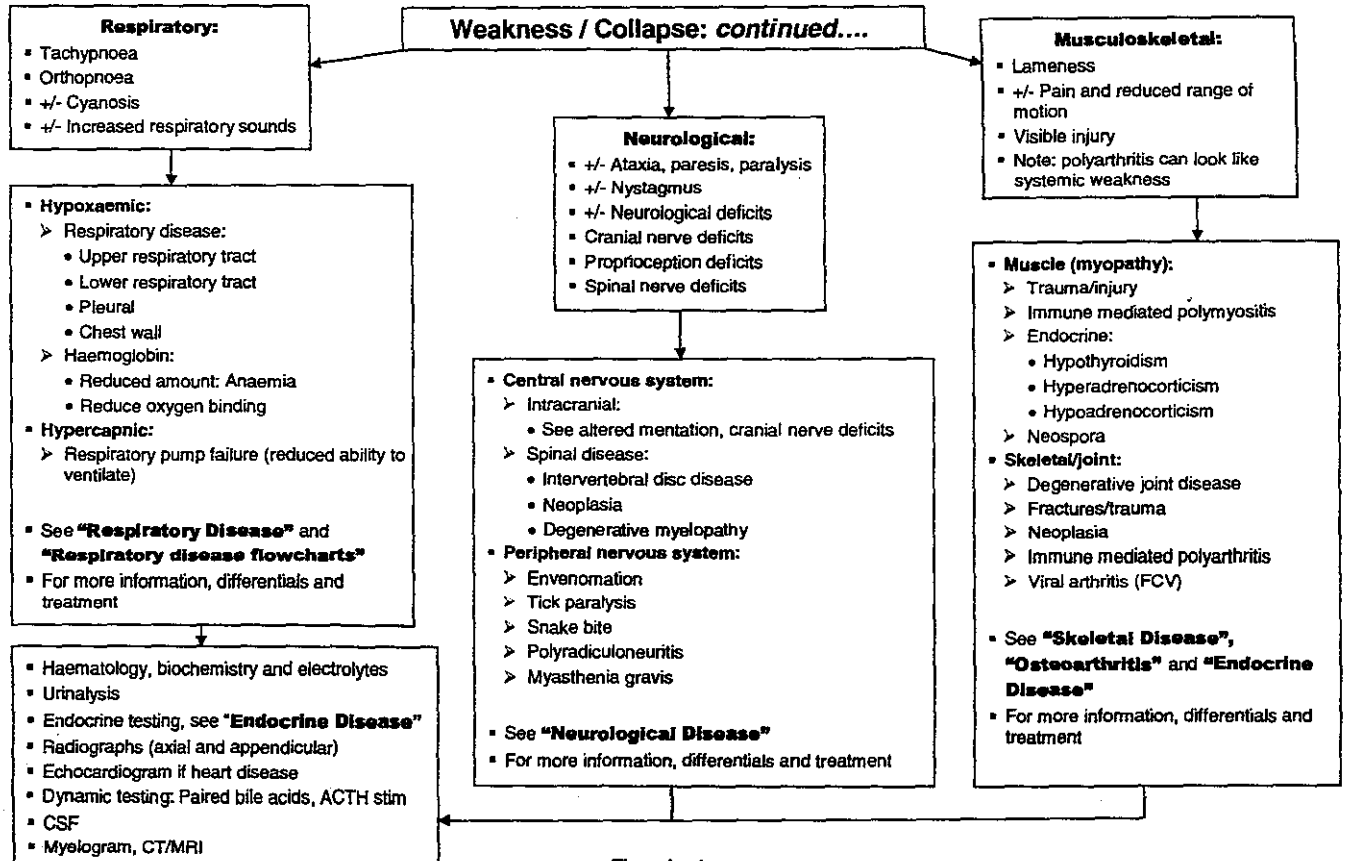

Weakness / Collapse
Metabolic:
+/- Systemic signs of illness
Typically, normal neurological function but weak
No palpable pain
Toxicity:
Variable signs
↓ Oxygen delivery:
+/- Slow capillary refill time
+/- Pale mucous membranes
+/- Poor pulses
+/- Heart murmur
+/- Tachycardia/Bradycardia
Musculoskeletal:
Respiratory:
Neurological:
NEXT PAGE
Glucose:
Hypoglycaemia
Electrolytes:
Hypokalaemia
Hyperkalaemia
Endocrine:
As with myopathy
Hypothyroidism
Hyperadrenocorticism
Hypoadrenocorticism
See "Electrolytes and Blood Gas" and "Biochemistry" and "Endocrine Disease"
For more information, differentials and treatment
Congestive heart failure:
Myxomatous valve disease:
Commonly mitral valve
Small breeds, usually murmur
Dilated cardiomyopathy:
Large breeds, +/- murmur
Hypertrophic cardiomyopathy:
Feline disease, +/- murmur +/- gallop rhythm
Dysrhythmia:
Tachycardia/bradycardia
Shock:
Hypovolaemic
Distributive
Obstructive
Cardiogenic
Anaemia:
Regenerative
Non-regenerative
Haemorrhage
Abnormal oxygen carrying capacity:
Paracetamol
See "Cardiovascular Disease" and "Shock and anaphylaxis" and "Anaemia and Pale Mucous Membranes"
CBC, biochemistry, electrolytes
Urinalysis
Endocrine testing, see "Endocrine Disease"
Radiographs (axial and appendicular)
Echocardiogram if heart disease
Dynamic testing: Paired bile acids, ACTH stim
CSF
Myelogram, CT/MRI



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APPENDIX:

Neurological Examination

ORDER OF TESTS

1) History: Mental status and animal's behaviour in exam room
2) Gait and posture
3) Cranial nerves
4) Proprioception: Knuckling and hopping
5) Spinal reflexes: Patella, withdrawal, perineal reflexes
6) Cutaneous trunci reflex
7) Back pain: Palpation

**** MENTAL STATUS, GAIT & POSTURE SETS THE EXAMINATION ****

After these you will know if case is neurological or not and if the cause is intra- or extra-cranial

1. MENTAL STATUS

- ✓ GOOD history is very important
- ✓ Let the animal wander in the examination room and watch its behaviour
- ✓ Fear or excitement in clinic may mask behavioural changes that evident at home

- Observations to make/Questions to ask the owner:
  - ✓ Is there a tendency to circle?
  - ✓ Are there signs of disorientation?
  - ✓ Is there a change in the sleep/wake cycle?
  - ✓ Are there changes in social interaction?
  - ✓ Is it more or less affectionate, more or less aggressive?
  - ✓ Has it stopped greeting you when you come home?
  - ✓ Is it house soiling?

- If the mental status is **abnormal → Brainstem OR Cortex problem**
  - ✓ Cerebrum is intelligence
    - ➢ Behaviour changes
    - ➢ Depression
    - ➢ Disconnected from environment – "out to lunch"
  - ✓ Brain stem is arousal/wakefulness
    - ➢ Defect causes somnolence i.e. lack of arousal, stupor, coma.

  - ✓ Cerebral lesion - circles usually (9/10 of cases) to the side of the lesion
  - ✓ Mental signs due to extra-cranial causes e.g. metabolic dysfunction or a toxicity:
  - ✓ Should be bilaterally symmetrical
  - ✓ House training is last cognitive function → very severe damage if training is lost

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2. EVALUATION OF GAIT AND POSTURE

- Look for ataxia or lameness:
  - ✓ Walk the dog up and down
  - ✓ Let off the lead and watch free movement.
  - ✓ Circle the dog to the left and right → can see subtle changes when it turns

- Ask the questions:
  - ✓ Which legs are involved? Hindlegs/forelegs or both?
  - ✓ Is the dog ataxic? If yes what kind - proprioceptive, vestibular, cerebellar?

- Deciding if the dog is ataxic or not is extremely important as this designates the area of the neurological system affected

ATAXIA

- **ASCENDING PROPRIOCEPTIVE TRACT ATAXIA** (White matter)
  - ✓ Tendency to:
    - ➢ Cross limbs so that they interfere with one another
    - ➢ Walk on dorsal surface of paw
    - ➢ Longer stride with a prolonged supporting phase
    - ➢ Tendency to abduct the limb, especially on turns
  - ✓ +/– paresis depends if the motor system is involved
  - ✓ Sometimes the ataxia is very subtle and is only seen when the dog changes direction

- **CEREBELLAR ATAXIA**
  - ✓ No paresis → normal strength
  - ✓ Spasticity and ataxia → fall to the side or forward or backward
  - ✓ Inability to regulate the rate, range and force of the movement (dysmetria)
  - ✓ Delayed voluntary movement → initiation and cessation
  - ✓ Hypermetria → movements are exaggerated in all 4 planes
    - ➢ Limb is raised too high, forcefully returned to the ground → goose stepping

- **VESTIBULAR ATAXIA**
  - ✓ Loss of co-ordination between head, trunk and limbs resulting in imbalance
  - ✓ A head tilt towards side of lesion (except in paradoxical vestibular disease)
  - ✓ Trunk will tip, fall or roll toward the side of the lesion
  - ✓ Trunk may be flexed laterally, with the concavity toward the side of the lesion
  - ✓ Circling towards the side of lesion (circles with short radii)
  - ✓ +/– Mild hypertonia and hyperreflexia in the limbs on the opposite side to the lesion
  - ✓ Signs explained by loss of vestibulospinal tract ipsilateral to the side of the lesion

3. CRANIAL EXAMINATION

- **Menace**
  - ✓ Blindfold one eye with one hand
  - ✓ Make a flicking motion as though going to hit the dog to make it react by blinking.
  - ✓ Repeat
  - ✓ Repeat this procedure on the other eye.

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✓ **Menace Reaction Tests:**
- CN II - vision, the whole visual pathway. (retina, NII, optic tract, optic radiation to the visual cortex)
- CN VII - facial - for the muscles for the ability to blink
- Cerebellum - the menace reflex is co-ordinated by the cerebellum
- Cortex - to perceive the menace cognitive function is necessary – if "out to lunch" may not react to menace

- **Pupils**
  - ✓ Assess both pupils for equality → bright pinpoint light between eyes → illuminates both eyes
  - ✓ Turn off room light → look at pupillary response (Pupillary Light Reflex)
  - ✓ Beam the light into the eye until the max pupil constricts → quickly move to other eye to assess its constriction → repeat the procedure on the other eye.

  ✓ **Pupillary Response Tests:**
  - CN II - as far as the branch to III (parasympathetic nuclei).
  - CN III - for the parasympathetic

- **Physiological Nystagmus**
  - ✓ Lift the upper eyelids to see the whites of the eyes
  - ✓ Move head from side to side → watch eye movement for normal physiological nystagmus
    - i.e. quickly flicking left and right with the movement of the head
  - ✓ If small animal – pick up and facing you at eye level, hold head then spin the animal to get enough excursion to see eye movements

  ✓ **Physiological nystagmus Tests:**
  - Nerves III, IV and VI - to the eye muscles to move the eye
  - Medial Longitudinal Fasciculus (MLF) - carries signal from the vestibular system to nuclei of III, IV and VI
  - Cerebellum - co-ordinator of the vestibular system and nuclei of the nerves to the eye muscles.
  - Vestibular system - perceiving movement of the head

- **Test Nerve V**
  - ✓ Sensory branches of the trigeminal:
    - Medial canthus of the eye - ophthalmic branch
    - Lateral canthus of the eye - maxillary branch
    - Base of the ear - mandibular branch
    - Do the test quickly - look for the blink - need the element of surprise to get the reflex

  ✓ Motor component of the mandibular branch:
  - Palpate size and symmetry of the temporal muscles
  - Feel for prominence of the zygomatic arch and occipital crest

  ✓ **Tests:**
  - V - ability to feel sensation
  - VII - ability to blink

  ✓ **Nasal Septum Test:**
  - Cover both eyes → stroke gently the edge of the nostrils with a cotton bud
    - If no response, touch the septum inside the nostrils
  - Start gently and gradually increase the stimulus until a reaction

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- The dog tries to avoid and pulls its head away - look for symmetry of reaction
- The nasal septum response may be abolished in a frightened patient

✓ **Nasal Septum Tests:**
- Ophthalmic branch of CN V - ability to feel sensation.
- Cortex - sensory perception of cortex is required to get reaction (pulling away)
  - Tests the contralateral cortex

- **Test Nerve VII:**
  - ✓ Already partly tested by ability to blink in Menace and CN V tests
  - ✓ Look for facial symmetry
    - Drooping of ears, eyelids, commissures of the lips
      - Both hands hold the muzzle with thumbs between mandibular rami and touch the commissures of the lips with the fingers - are they equal/level both sides?
  - ✓ Test for full closure of the eyelids. Run a finger over the upper and lower eyelids at the same time and see if there is complete closure.

- **Test Nerve VIII:**
  - ✓ Look for a head tilt
  - ✓ Vestibular (Positional) strabismus
    - Lift the nose up so that it points upward, the eyes should stay in the centre of the orbit

- **Test Nerves I, IX and X:**
  - ✓ Function of these nerves is obtained from the history
  - ✓ Ask the owner does the animal sniff
  - ✓ Ask the owner about the ability of the animal to eat and drink normally, without choking, gagging, and if it is able to swallow normally.

- **Test Nerve XII:**
  - ✓ Look at the action of the tongue - e.g. does the dog lick its nose evenly?
  - ✓ Open the mouth and look at the tongue
    - Evenly placed in the mouth or is it pulled to one side?

4. PROPRIOCEPTION AND HOPPING

- **Proprioception**
  - ✓ **Knuckling:**
    - Stand animal square →legs in normal position.
    - Front legs - stand over animal, hand under chest, support weight→ then knuckle
    - Repeat on the other leg
    - Back legs - stand at back and hand between back legs, support weight→ then knuckle
    - Repeat for the other leg
  - ✓ **Paper slide:**
    - Place foot on a piece of paper and pull it laterally

  - ✓ **Lateral Placing Proprioceptive test:**
    - **Cat-** May not co-operate with knuckling → do instead
    - One hand under chest → hold 3 legs away from table against so only 1 leg is near table.
    - Blindfold cat (tucking head under arm) and hold it away from seeing the table.
    - Bring free limb up to the edge of the table→ barely touch table
    - Should lift it up onto table when just touches

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- ✓ **Hopping:**
  - ➢ Stand over dog to test the front legs
  - ➢ Stand at the side to test the back legs
  - ➢ Do not support weight → pick up leg and push dog to side and watch its ability to hop
  - ➢ Front leg - place hand at or just below the shoulder joint
  - ➢ Back leg - pick up the tibia and push dog towards the other side
  - ➢ Pick up one leg at a time and leave the dog standing on the other legs
  - ➢ Repeat front and back legs a number of times to gauge consistency of the reaction
  - ➢ With a cat or very small dog the test can be done by holding up the animal and 3 legs then makin the animal hop on the one leg that is on the ground

5. SPINAL REFLEXES

- **PATELLA → Tests Femoral Nerve L4-6**
  - ✓ Manipulate leg to ensure patella ligament is tight to get the reflex:
    - ➢ May need to flex up the toes with the holding hand to tighten the patella ligament.
  - ✓ Test each side by turning the animal over. Only test the upper leg

- **WITHDRAWAL/FLEXION → Tests Spinal Reflexes of the Limbs – Plexuses**
  - ✓ **Hind leg** - test lateral **(Sciatic n: L6 – S2)** and medial toes **(Femoral n: L4-L6)**
    - ➢ Lie the dog on its side
    - ➢ Hold toe, pinching it, and pull on the leg
    - ➢ Withdrawal is a difficult test to interpret clinically
  - ✓ **Front leg** - test pinch the middle toe (No differentiation between Radial (D) and Medial (V))
    - ➢ Small dogs and cats - can hold them in the arms with back against the body, this can be easier to test the reflexes

- **PERINEAL → Test Spinal Reflexes Sacrum: S1-S3**
  - ✓ Touch either side of the tail (perineal region), if the tail contracts down then don't need to do perineal reflex
  - ✓ For the perineal reflex can lift the tail at the base with one hand then touch the anus/perineal region with the other - can feel the tail contract down

6. CUTANEOUS TRUNCI REFLEX → Test Spinal Reflexes of the Trunk: T2 – L3

- ✓ Start testing at the level of the wing of the ilium:
  - ➢ Lightly pinch skin about 2.5 cm each side of the back bone (spine)
- ✓ Pinch from ilium forwards until the skin flicks:
  - ➢ Repeat on the other side
- ✓ Once the skin flick is elicited there is no need to pinch above this point

7. TESTING FOR BACK AND NECK PAIN

- **Back Pain:**
  - ✓ Stand the dog up squarely on its 4 legs, if possible
  - ✓ Stand behind the dog and support with a hand underneath between the back legs NOT under the abdomen:
    - ➢ Press with the ball of the fingers, not just the tips, avoid using fingers as needles!
    - ➢ Press about a knuckles width, evenly, either side of the spine starting at the scapulae (T1)

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- **Neck Pain:**
  - ✓ Press down at each vertebra
  - ✓ Go down the spine first lightly so as to gauge the reaction of the animal and judge its tolerance to pain, then go down again firmly

    - ➢ ***Neck pain testing***- use food to get dog to move neck to test the range of motion, rather than physically manipulating the neck

    - ➢ The dog should be able to move laterally to touch nose to shoulders each side, move ventrally sufficiently to touch nose to chest and move the head back dorsally freely

- **Severity of spinal Lesion and likelihood of regeneration:**

Loss of:	Depth of damage:	Prognosis:
Proprioception	• Superficial	• Good
Voluntary motor	• Superficial	• Fair
Above and **cutaneous pain**	• Middle	• Moderate
Above and **deep pain**	• Deep	• Poor

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Sample Collection and Storage:

Tube Type:	Colour:	Type of sample:	Applications:
EDTA	**Purple**	Whole blood	• **Haematology** • Not for smear (cells may be lysed) • Electrolytes (not K) • **Toxins**
Serum	**Red**	Clotted blood / serum	• **Biochemistry (serum)** • Serology • Not blood smear (clotted)
Lithium heparin	**Green**	Whole blood and plasma	• **Blood smear (best one)** • Biochemistry (plasma – not as good as serum) • **Electrolytes** • **Lead toxicity** (not other toxins)
Sodium heparin	**Green**	Whole blood and plasma	• **Blood smear (best one)** • Biochemistry (plasma not as **good** as serum) • Not electrolytes (due to Na)
Oxalate fluoride	**Grey**	Whole blood	• **Glucose**
Sodium citrate	**Light blue**	Whole blood and plasma	• **Coagulation**

- **Handling:**
  - ✓ Use sterile collection method
  - ✓ Mix tubes well by inversion
  - ✓ Remove stopper from tube to fill; do not inject through lid as will cause haemolysis
  - ✓ Chill (2-8°C) but not freeze for storage
  - ✓ Separate plasma/serum from red cells if not going to lab within 4 hours – will last up to 24 hours
  - ✓ Store blood smears at room temperature

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Common Drugs Summary

Anaesthetic – Sedatives / Premedicants

- **See "Anaesthesia and Analgesia" for combinations of drugs**

Drug (*Trade Name*):	Action/Effect:	Indications:	Dosage:	Side Effects/Comments:
Acepromazine *(ACP)* *2mg/ml*	Tranquilliser (phenothiazine) Dopamine antagonist (α1) → ↓ sympathetic Anti-emetic	Sedation (calming) Premedicant Care in Boxers NOT OP poisoning	0.01-0.05mg/kg IV (slow)/SC 1/2 dose if cardiomyopathy	Hypotension (peripheral vasodilator) / ↓ HR – don't use in shock/cardiac patients Anti-arrhythmic ↓ Ictal threshold Antihistamine Ceiling effect → ↑ side effects not sedation
Atropine *(Atrosite)* *0.6mg/ml*	Anticholinergic Antispasmodic Mydriatic Inhibit vagal tone → ↑ HR	Bradycardia; asystole OP toxicity ↓ secretions (oral/airway) Relax smooth muscle Cane toad toxicity?	0.02-0.04mg/kg IV IM SC E.g. 0.02mg/kg dog pre med	Can cause tachycardia (and ↑ BP), dry mouth Do not administer if high HR Caution if heart disease Is also bronchodilator (but ↓ secretions) Use glycopyrrolate instead (no CNS effects)
Diazepam *('Valium'; Pamlin)* *5mg/ml*	**Benzodiazepines** BZ → ↑ GABA → ↑ Cl → hyperpolar → refractory Anti-convulsant Anti-anxiety	Control seizures/tremors Tranquilizer (not potent) Sedative (not sole) Muscle relaxant	Seizures: 0.2-1.0mg/kg IV Sedation: 0.2-0.5mg/kg IV or IM	Give slow IV (fast can cause bradycardia/hypotension and excitement) Poor absorption SC; stings IM Can get ataxia (→ ↑ anxiety); little sedation Use for appetite ↑ in cats Respiratory depression Can give intra-nasal or intra-anal Possible idiosyncratic hepatoxicity in cats
Midazolam	Faster onset Shorter duration	Sedative (not sole) Muscle relaxant	0.1-0.5mg/kg IV IM SC	Less CNS depression CRI better
Xylazine *(Xylazil)* *20mg/ml*	**α2 agonists** Stimulate inhibitory (α2) → ↓ NAd → ↓ CNS sympathetic Duration: 20-40mins	Sedative (calming) Analgesia (profound) Muscle relaxation Epidural	0.25-0.5mg/kg *IV* *1.0-2.2mg/kg IM SC* *0.1-0.2ml/10kg pre-an; 0.3-0.8 for anaesthesia*	Poor CV stability (↓ HR; initial ↑ BP then hypotension); ↓ cardiac contractility Side effects (α1) = ↓ gut motility/secretion; ↑ saliva; ↓ insulin; ↑ urine (↓ renin/ADH) Nausea/V+ (especially cats – 5mins after injection)
Yohimbine *(Reverzine)10mg/ml* *(Reverzine SA)1.25mg/ml*	α2 antagonist	Reverse α2 agonist (xylazine)	Cow: 0.125mg/kg (1ml/80kg) IV Dog: 1ml/10kg IV Cat: 0.25ml/10kg IV	Used to reverse Xylazine (and xylazine/ketamine) in cattle/deer/dogs/cats

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Medetomidine *(Domitor)* *1mg/ml*	Stimulate inhibitory (α2) → ↓ NAd → ↓ CNS sympathetic Dose-dependent effects Synergistic with opioids or ketamine or isoflurane → deep anaesthesia	Pre-med Sedation Analgesia Anaesthesia Vasoconstriction (↑ BP initially)	Cat: 50-150µg/kg IM; 50-80µg/kg (with butorphanol) Dog: 10-80µg/kg IM IV Premed 10-20µg / kg IV; 20-40 µg / kg SC 10-25µg/kg (with butorphanol)	↓ Side effects compared to xylazine Sedation: See **"Anaesthesia and Analgesia"** for combinations of drugs
Atipamezole *(Antisedan)* *5mg/ml*	Displaces medetomidine from α2 receptors → ↑ NAd secretion	Reversal of medetomidine (sedation not side effects)	Cat: 125-200 µg / kg IM SC (2.5x = 1/2 Domitor vol) Dog: 125µg/kg IM IV (5x Domitor = same vol)	Cat: reverse 50µg/kg Medetomidine (at 125µg/kg) or 80µg/kg Medet and ketamine (at200µg/kg) IM or 40µg/kg Medet/ketamine (at100µg/kg) IV Dog: reverse 25µg/kg Medetomidine (at 125µg/kg)
Detomidine *(Dormosedan)* *10mg/ml*	Stimulate inhibitory (α2) → ↓ NAd → ↓ CNS sympathetic Duration: 90-120min	Analgesia X rays; transport; colic	Horse: 10-80µg/kg (0.1-0.8ml/100kg) 0.6ml plus 0.6ml Torbugesic IV IM	Inhibits CNS mediated transmission of pain impulses ↑ BP; ↓ HR; ataxia; head droop
Romifidine *(Sedivet)* *10mg/ml*	Stimulate inhibitory (α2) → ↓ NAd → ↓ CNS sympathetic Longer acting	Analgesia X rays; transport; rectal palpation Premedicant	0.4-1.2ml/100kg IV	↑ BP; ↓ HR; second degree AV block; head droop

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Anaesthetic – Induction and Maintenance

Drug (*Trade Name*):	Action/Effect:	Indications:	Dosage:	Side Effects/Comments:
Alfaxan *10mg/ml*	Steroid anaesthetic (near BZ site) → ↑ GABA Dampens CNS	Induction Maintenance NO analgesia Muscle relaxation	*Dog: 1-2mg/kg* *= 0.1-0.2ml/kg* 1-2ml/10kg IV or IM *Cat: 2-5mg/kg* 2ml/5kg	Non-cumulative; high therapeutic index Little CV/respiratory depression (no arrhythmia/vasodilation) Rapid recovery (short duration); may shiver/get excitement (if no pre-med) Recovery by metabolism (mostly); redistribution; eliminate
Ketamine *100mg/ml*	Dissociative Acts on GABA (inhibits → overexcite); antagonize NAd / serotonin; NMDA antagonist (analgesia)	Induction Maintenance Analgesia (intense but short) Not for eye/brain surgery (↑ pressure)	5mg/kg ketamine and 0.25mg/kg diazepam 1-3mg/kg cat SC (with ACE/meth) 0.1-0.6mg/kg/hr infusion	Maintain BP/CO and reflexes Causes hypertonicity/convulsions → use with midazolam/ diazepam / xylazine Recovery = redistribution; liver metabolism; renal excretion
Propofol *(Rapinovet)* *10mg/ml*	↑ GABA → open Cl channel → hyperpolarise → less excitable → CNS depression/inhibition	Induction (rapid) Maintenance (duration 20-30mins; infusion) Anti-convulsant NO analgesia	*Dog: 4-6.5mg/kg* 4ml/10kg (pre-med); 6.5 if none *Cat: 6-8mg/kg* 3-4ml/5kg	↓ Respiration/BP (vasodilate; ↓ cardiac contract) = worst Non-cumulative; fast recovery (some twitches) 15% get pawing; sneezing; vomiting (if no pre-med) Slightly irritant Recovery = redistribution to muscles; liver/lung metabolism
Thiopentone 2.5% *25mg/ml*	Barbiturate (↓ GABA dissociation → Cl channels open) Depress cortex/reticular before medulla (CV/respiratory centres)	Induction (onset 20-30 secs; duration 15-20 mins) Anti-convulsant NO analgesia	15-20mg/kg IV	Very irritant (high pH) Cumulative ↓ respiration (↓ sensitivity to CO2) ↓ cardiac contractility; slight ↓ BP/HR; arrhythmias Not for neonates or sight hounds Recovery = redistribution to muscles; liver metabolism
Zoletil = Tiletamine plus Zolazepam *50mg/ml*	Dissociative	Induction Maintenance Analgesia (short) Muscle relaxation	Dog: 7-25mg/kg IM 5-10mg/kg IV Cat: 10-15mg/kg IM 5-7.5mg/kg IV 0.1-0.2ml cat IM/SC	Wide safety margin Maintain reflexes Long recovery (2-6hrs) – use 0.5mg/kg midazolam and 3-5mg/kg ketamine instead Non-cumulative Do not use ACE as premedication (use atropine)

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Anaesthetics	Blood/gas Solubility *Low = fast wakeup / induction*	Oil/gas Solubility *High = enter brain rapidly; ~ potency*	MAC % *Low = more potent*	SVP mmHg *High = high % vapour achievable*	Metabolism %	Toxicity	Comments
Methoxyflurane	13	635	0.23	22.5 = 3% 13 x MAC	50%	Renal	Analgesia (sub-anaesthetic doses) Low SVP and high solubility → hard to achieve MAC (slow induction) Halogenated ethyl methyl ether
Halothane	2.3	225	0.87	243 = 32% 37 x MAC	10-25%	Hepatic	Dose related CV depression → hypotension (↓ CO/SV/cardiac contractility); vasodilation; bradycardia Least respiratory depression (↓ TV → ↓ AV) Halogenated hydrocarbon; no analgesia Preservative = thymol
Isoflurane	1.4	91	1.28	251 = 33% 26 x MAC	0.17%		Less potent (cf. halo) but faster inductions Better CV stability (some vasodilation and hypotension) – low solubility and metabolism More respiratory depressant (cf. halo) No analgesia Halogenated ethyl methyl ether
Sevoflurane	0.6	42	2.25	160 = 21% 9 x MAC	~3%	Renal	Fast induction/wakeup (> iso) Best CV stability > iso/des > halo Worst respiratory depress > iso/des > halo Fluorinated methyl-isopropyl ether (soda lime)
Desflurane	0.42	18.7	7.2	700 = 92% 13 x MAC	0.02%	Liver?	Very fast inductions/wakeups > sevo > iso CV stability < sevo but = iso (vasodilate) Respiratory depress = iso
Nitrous Oxide	0.47	1.4	200	39,000 26 x MAC	0%		Analgesia; weak anaesthetic Deliver 30+% O2 Little CNS/CV/respire/GIT effects Bone marrow depression (prolonged) Diffuses into gas filled body cavities (N2O in > N2 out); hypoxia (N2O from blood to alveoli → ↓ O2); 2nd gas effect (N2O from alveoli to blood → ↓ alveoli vol → ↑ induction speed)

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Analgesia – Opioids

Drug (*Trade Name*):	Action/Effect:	Indications:	Dosage:	Side Effects/Comments:
Buprenorphine *(Temgesic)*	Partial μ agonist (ceiling effect) ↓ effect; longer lasting (6hrs)	Analgesia	0.006-0.01mg/kg IM IV; 0.02mg/kg SC	Reduced respiratory depression. 3-5x morphine potency Better analgesia than methadone in cats? *Do not combine partial agonist/antagonist with agonist (morphine; methadone) as competitive*
Butorphanol *(Torbugesic)* *10mg/ml*	Agonist (κ)/μ antagonist (ceiling effect) Lasts for 2 hours	Analgesia Pre-medication (same dose for cats) Antitussive	*Dog: 0.2-0.3 mg / kg IM SC IV* *0.2-0.3ml/10kg* *0.1-0.2mg/kg pre-anaesthetic* Cat: 0.2-0.5mg/kg IM *SC; 0.1mg/kg IV*	Rapid onset of analgesia Less respiratory depression *Do not combine partial agonist/antagonist with agonist (morphine; methadone) as competitive*
Fentanyl	μ agonist 20min duration Very potent (100x morphine)	Analgesia	2-5μg/kg IV 2μg/kg/hr (dog) 10μg/kg/hr (cat)	↓ Cardiovascular system effects; still get respiratory depression ↑ Vagal tone → bradycardia Patches take 12-24 hours till effect (6-12 in cats); lasts 72 hours
Hydromorphone	μ agonist 4-6 hours	Analgesia Pre-medication	*Dogs:* 0.1-0.4mg/kg SC/IM/IV *Cats:* 0.05-0.2mg/kg SC/IM/IV	Respiratory depression and bradycardia Caution patients with head trauma or increased intracranial pressure, adrenocortical insufficiency or severely debilitated patients High doses in cats can cause ataxia and hyperaesthesia Avoid in patients taking MAOI's
Methadone *(Methone)* *10mg/ml*	Opioid (μ agonist) Maximum stimulation; dose dependent effect 4 hours duration	Analgesia Pre-medication	0.25-0.5mg/kg SC premedication 0.25mg/kg SC q4h analgesia Mild: 0.1-0.2mg/kg IV Moderate/severe: 0.2mg/kg IM SC (cat) 0.2-0.3mg/kg IV (dog)	Respiratory depression (lose sensitivity to CO_2) Good cardiovascular stability Vasodilation ↓ gut motility (constipation) Nausea/V+ (↑ with morphine if pain free) Urine retention ↑ vagal tone

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Morphine	μ agonist 4-6hr duration	Analgesia Bolus/titrate/infusion	Cat: 0.05-0.1mg/kg IM SC Dog: 0.5-1.0mg/kg IM SC; 0.2mg/kg IV	Epidural (preservative free): 0.05-0.2mg/kg (20-60 min onset; 16-24hr duration) with bupivacaine or lignocaine (make up to 0.2ml/kg) CRI: 0.1-0.2mg/kg/hr Histamine release
Oxymorphone	μ agonist 3-4 hours	Analgesia Pre-medication	*Dogs:* 0.1-0.4mg/kg SC/IM/IV *Cats:* 0.05-0.2mg/kg SC/IM/IV	Respiratory depression and bradycardia Caution patients with head trauma or increased intracranial pressure, adrenocortical insufficiency or severely debilitated patients High doses in cats can cause ataxia and hyperaesthesia Avoid in patients taking MAOI's
Pethidine	μ agonist 1.5-2hr duration		1-4mg/kg SC (not IV)	Histamine release → vasodilation and ↓ BP (not use if history of mast cell tumours) 0.5x potency of morphine
Tramadol *(Tramadol)*	μ agonist Serotonin reuptake inhibitor	Analgesia Antitussive	1-5mg/kg PO BID-TID (dogs) 1-2mg/kg PO BID (cats)	CNS effects (including seizures) or GIT Do not use with Amitriptyline
Naloxone Naltrexone	Antagonist Short duration (45-60min)	Overdose	0.04mg/kg IV IM SC	Titrate to reduce side effects and maintain analgesia

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Analgesia – Local Anaesthetics

Drug (*Trade Name*):	Action/Effect:	Indications:	Dosage:	Side Effects/Comments:
Bupivacaine 0.5% *100mg/20ml*	Slower effect cf. lignocaine but longer duration (3-10hrs)	Analgesia (intra and post op)	NOT IV 2mg/kg/4hr MAX	Epidural (with morphine) – preservative free; lasts 4-6 hours **Cardiotoxic** Used for thoracotomy (intercostal nerves)
Lignocaine 20 = 2% *20mg/ml*	Anti-arrhythmic Blocks Na channels → slows AP/depol → stabilises membranes	Arrhythmias; tachycardia Local anaesthesia Analgesia (IV)	Nerve block (0.5ml) Epidural 4.5mg/kg MAX	Epidural (with morphine) 3-5mg/kg – preservative free; lasts 1-2 hours (90-200min) Rapid onset (5-10min) Can do CRI

Analgesia – Non-steroidal anti-inflammatory Drugs

Drug (*Trade Name*):	Action/Effect:	Indications:	Dosage:	Side Effects/Comments:
Meloxicam (*Metacam*) *5mg/ml*	COX II selective Inhibits PGs Antipyretic	Low grade pain relief; mild injuries (musculo-skeletal); post-op (ortho / soft tissue)	0.2mg/kg IV/SC/PO initial then 0.1mg/kg SID	Renal failure and gastric ulcers, particularly if dehydrated/low BP/shock/cats → anaemia / hypoproteinaemia May predispose to bleeding (↓ platelet function) Oral doses with food
Firocoxib *(Previcox)*	COX II selective	Osteoarthritis; musculo-skeletal; post-op	5mg/kg SID	
Ketoprofen *(Ketofen)* *10mg/ml*	COX I selective	Osteoarthritis; musculo-skeletal; post-op	2mg/kg IM IV SC (D and C) 0.5-1mg/kg/day (chronic)	One dose only in cats
Carprofen *(Rimadyl; Norocarp)* *50mg/ml*	Inhibits COX I/II and phospholipase A2	Mild-moderate analgesia 24 hour duration Orthopaedic/soft tissue surgery; DJD	4mg/kg SC IV Post-op 2mg/kg BID PO (dogs)	Less ulcerogenic (COX II selective) One dose only in cats
Aspirin	Platelet inhibition	Used in thrombogenic diseases	10-20mg/kg PO BID-TID (dog) 10mg/kg PO q48-72hrs (cat)	One dose (10mg/kg) only in cats

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/pneumonia **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)	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)

Drug (*Trade Name*):	Action/Effect:	Indications:	Dosage:	Side Effects/Comments:
Enrofloxacin *(Baytril)*	Fluoroquinolone Bactericidal Gram +ve (most) **Gram –ve** (many) Not anaerobes Pseudomonas	Dental infections Abscesses Severe infections **Urogenital tract** (incl. prostate) Otitis externa **CNS** **Respiratory tract** Bone, skin, CSF	Dogs: 5 – 20 mg/kg IV/ PO SID or 2.5 – 10 mg/kg IV/ IM/ PO BID Cats: 5mg/kg PO SID or 2.5mg/kg PO / IM BID	Avoid in young growing animals from 2 months to 8 months of age (cartilage defects; OK till 4wks old) May cause nausea/vomiting – administer on empty stomach May cause blindness in cats Slow IV (anaphylaxis)
Erythromycin	Macrolide Bactericidal Gram +ve Anaerobes	Respiratory Skin	10-20mg/kg PO BID-TID	Hepatotoxic NOT – CSF
Gentamicin *(Otomax)*	Aminoglycoside Bactericidal **Gram –ve**	ECF; placenta; milk		**Kidney damage** (not use in cats) **Ototoxic** (topical only) NOT – urine; pus; necrotic tissue; abscess; CNS
Metronidazole *(Metrogyl)*	Nitroimidazole Bactericidal **Anaerobes** *Giardia*	**GI disease** (HGE) **CNS** Severe injuries with ↓ blood flow Pancreatitis; PSS	Dogs:10mg/kg IV or PO BID Cats:10mg/kg IV/ PO BID	Give IV dose slowly → neurological signs if fast Also anorexia, nausea, vomiting, haematuria, ↓ WCC, lethargy Light sensitive so cover bag/syringe
Neomycin *(Tricin)*	Aminoglycoside Bactericidal Gram –ve aerobes	Otitis externa	Topical	More nephrotoxic than gentamycin
Ticarcillin-clavulanic acid *(Timentin)*	Cephalosporin Gram +ve **Gram –ve** (better) Pseudomonas **Anaerobes**	Severe infections Compromised patients	40-50mg/kg IV TID-QID	Give slow IV → allergic reactions if fast Make up from powder form; store in fridge for 3d or freezer for 30d Add 30ml into a vial for 100mg/ml
Trimethoprim-sulpha *(Tribrissen)*	Bactericidal Gram +ve Gram –ve	**Urogenital tract** (incl. prostate) **Pyometra** **URT** (rhinitis)/pneumonia **CNS**	30mg/kg SID PO or 15mg/kg BID	Also *Toxoplasma; Neospora; Coccidia* Risk of KCS NOT – pus/blood/necrotic tissue Low safety margin in cats NOT to Doberman, min Schnauzer, Samoyed
Tylosin	Macrolide	**Chronic diarrhoea**	10mg/kg PO SID-BID	Gastrointestinal upset

Antifungals

Drug (*Trade Name*):	Action/Effect:	Indications:	Dosage:	Side Effects/Comments:
Griseofulvin ▪ Griseofulvin	Fungistatic Dermatopytosis *Malassezia*	▪ Dermatophytosis	*Micronized:* ▪ 30-40mg/kg/day PO SID or divided BID *Ultramicronized:* ▪ 5-10mg/kg/day PO SID or divided BID	▪ Not in pregnant animals, teratogenicity ▪ Not if FIV positive ▪ Haematological abnormalities ▪ Gastrointestinal upset
Imidazole ▪ Enilconazole ▪ Clotrimazole ▪ Fluconazole ▪ Itraconazole ▪ Ketoconazole ▪ Miconazole	Depends on dose: Fungicidal/static Aspergillosis Cryptococcus Histoplasmosis Pythiosis *Malassezia* Dermatopytosis	*Systemic infections:* *(skin, nasal, CNS, spinal, respiratory, GIT)* ▪ Fluconazole ▪ Itraconazole ▪ Ketoconazole *Nasal infections:* ▪ Enilconazole ▪ Clotrimazole *Otitis externa:* ▪ Miconazole *Skin infections:* *(Malassezia)* ▪ Ketoconazole	▪ Fluconazole: 5-10mg/kg PO SID-BID ▪ Itraconazole: 2.5mg/kg PO BID ▪ Ketoconazole: 10-20mg/kg PO BID *Nasal infusions:* ▪ Enilconazole 1-2% ▪ Clotrimazole 1% *Topical preparations:* ▪ BID for 7 days after negative cytology ▪ Ketoconazole: Dog: 5-10mg/kg PO BID, Cat: 40mg/cat PO BID	▪ Not in pregnant animals ▪ Gastrointestinal upset ▪ Hepatopathy
Macrolide Antibiotics ▪ Amphotericin B ▪ Nystatin (topical)	Fungicidal Cryptococcus Dermatophytosis Histoplasmosis Candidiasis	*Systemic infections:* ▪ Amphotericin B *Otitis externa:* ▪ Nystatin (topical)	0.5mg/kg slow IV EOD, total dose of 5-9mg/kg *Topical preparations:* BID for 7 days after negative cytology	▪ Nephrotoxic

Behaviour Drugs

Start with low dose

Drug (*Trade Name*):	Action/Effect:	Indications:	Dosage:	Side Effects/Comments:
Fluoxetine *(Lovan; Zactin; Prozac)*	Specific serotonin reuptake inhibitor	FAVOURITE DRUG Lick granulomas Separation anxiety	Dogs: once a day 1mg/kg Cats: liquid 10mg/ml – Start at 0.25ml/cat SID then increase to 0.5ml/cat	Side effects: ↓ appetite (nausea) in 5% dogs; lethargy; nightmares; ataxia Get liquid for cats – 10mg/ml
Diazepam *(Valium; Xanax)*	Anti-anxiety	When can predict problem – noise / travel / separation	Dogs: 0.5-2mg/kg PO PRN Cats: 0.2-0.5mg/kg PO BID	Use like ACE for behaviour issues (when you need it) Short half-life in dogs
Alprazolam *(Kalma)*	Use for early treatment (e.g. separation anxiety) before TCAs etc. kick in	As needed; give 2 hours before effect needed	Vague dose rate (0.5mg/10kg) – start at 0.25mg and titrate up	Longer half-life than diazepam Side effects: ↑ appetite/olfaction then serene/peaceful/dopey (<ACE)
Amitriptyline *(Endep)*	Tricyclic antidepressant – regulates NAd/dopamine (blocks reuptake)	Stereotypies – barking; self-mutilation Anxiolytic Urinary incontinence	1-2 mg/kg PO BID	Cheapest BUT side effects = dries mucous membranes; constipation; ↓ urine flow (tightens sphincter); arrhythmias; lethargy; personality change Then muscle weakness; ataxia (drunk) Can get hyperactivity/rebound anxiety when stop using it DO NOT USE WITH TRAMADOL
Clomipramine *(Clomicalm)*	Tricyclic antidepressant	Only one registered in animals – compulsive behaviours/anxiety	Dogs: 1-3mg/kg PO BID Cats: 0.5mg/kg PO SID	High cost; side effects similar to Amitriptyline
Pheromones				DAP – intramammary cleft for puppies - successful in calming noise/separation anxiety Feliway – spraying/urine marking/scratching

Cardiopulmonary Drugs

Drug (*Trade Name*):	Action/Effect:	Indications:	Dosage:	Side Effects/Comments:
Aminophylline *(Aminyllin)*	Bronchodilator Diuretic +ve inotrope	Heart failure Pulmonary oedema Chronic lung disease	100mg/10kg BID-QID 6-10mg/kg IV TID (dog) 4-6mg/kg IV BID (cat)	CNS excitement; arrhythmia; tachypnoea; insomnia; vomiting; anorexia
Benazepril; Enalopril (*Fortekor*)	ACE inhibitors = peripheral vasodilator; ↓ GFR; ↓ blood pressure	Heart failure (mitral valve regurgitation; DCM – dogs; HCM – cats) Renal insufficiency (cats)	Dogs: 0.25-0.5mg/kg PO BID Cats: 0.5mg/kg PO EOD	Retain sympathetic tone
Clenbuterol	β2 agonist Bronchodilator	Asthma Emphysema Bronchopneumonia COPD		Nebulizers; puffers
Digitalis *(Digoxin)*	+ve inotrope -ve chronotrope ↑ CO → ↑ GFR → U urine → ↓ preload / afterload	Long term management of heart failure (DCM) Arrhythmia (atrial fib) Hypertension	*Dog for CHF:* <20kg: 0.01mg/kg BID PO >20kg: 0.22mg/m^2 BID PO *Rapid digitalization:* 0.011mg/kg q1hr IV to effect	Heart beats more slowly and strongly Narrow therapeutic index
Diltiazem	Ca+ channel blockers = -ve inotrope (↓ contraction) and –ve chronotrope (↓ HR/heart block); vasodilator (antihypertensive)	Arrhythmia Hypertension HCM	Dog: 0.5 – 1.5mg/kg PO TID Cat: 1.75-2mg/kg TID PO (for Dilacor XR) OR 10mg/kg SID PO (Cardizem CD)	
Dopamine (200mg vial in 1L 5% dextrose or 0.9% NaCl = 200µg/ml)	+ve inotrope ↑ cardiac contractility	Acute HF Oliguric RF (dogs)	6-10 µg/kg/min IV (dog) 2-5 µg/kg/min IV (cat) 2-11µg/kg/min IV 0.5-3µg/kg/min (+ furosemide)	
Furosemide *(Frudix)*	Diuretic – ↓ blood volume and fluid in lungs (stops Na/K exchange)	Heart failure Fluid overload	Dog: 2-4mg/kg IV/SC, 1-4mg/kg PO TID Cat: 0.5-1.5mg/kg IV Frequency to effect	Caution in dehydrated or renal patients **RESCUE DOSES:** 4mg/kg every 1-2hrs until dyspnoea reduces
Lignocaine 20 = 2% *20mg/ml*	Anti-arrhythmic Blocks Na channels	Arrhythmias Tachycardia Local anaesthesia	1-2mg/kg IV bolus dog, repeated q5 mins max 8mg/kg, then 20-80µg/kg/min	Can cause seizures

Linctol Dextromethorphan hydrobromide 2 mg/mL, chlorpheniramine maleate 0.4 mg/mL, ephedrine hydrochloride 1 mg/mL	Cough suppressant		2.5ml BID-TID (cat; small dog) 5-10ml BID-TID (large dog)	Can cause tachycardia
Pimobendan *(Vetmedin)*	↑ Ca sensitivity Inhibits phosphodiesterase Vasodilation; +ve inotrope	Congestive heart failure (DCM; mitral valve)	0.1-0.3mg/kg PO BID	May be combined with diuretic Tx
Propranolol	β blockers = ↓ sympathetic so ↓ chrono and inotrope → ↓ CO = antihypertensive	Hypertension Arrhythmias HCM – cats Anxiety	0.05-0.1mg/kg IV	Will block sympathetic tone that may be keeping patient alive (by ↑ HR, vasoconstriction and bronchodilation) → start at low dose
Rikodeine	Cough suppressant		1.5ml/10kg BID/TID	
Spironolactone	Aldosterone antagonist Weak diuretic	Refractory oedema Hypertension		Potassium sparing
Terbutaline *(Bricanyl)*	Bronchodilator	Asthma Chronic respiratory dis Bronchospasm	1.5ml/10kg BID-TID	

Corticosteroids, Anti-histamines, Immune suppressants

Drug (*Trade Name*):	Action/Effect:	Indications:	Dosage:	Side Effects/Comments:
Dexamethasone sodium (*Dexapent; Dexadreson; Dexaphos*) *2mg/ml*	Anti-inflammatory Immunosuppressive	Immune mediated dis. Used for milder allergic reactions (e.g. vaccinations – or could use SDC)	Anti-inflammatory: 0.2mg/kg IV or IM Immunosuppression: 0.8mg/kg IV or IM	Give slow IV as can cause hypotension Rapid onset – 20 mins; peak levels by 2 hours Lasts 24-48 hours Many therapeutic uses where rapidity of onset is not an issue and a duration of activity of 1-2 days is desirable
Dexamethasone (*Dexafort*) *2mg/ml* phenylpropionate *1mg/ml* sodium phosphate	Slower onset and longer lasting – lasts 5-7 days Rapid onset and short acting – lasts 24-48 hours	Itchy skin Tracheitis Used where chronic problems need longer effect	Anti-inflammatory: 0.2mg/kg IV or IM Immunosuppression: 0.8mg/kg IV or IM	Take care if aetiology is infectious
Methylprednisolone acetate *(Depo Medrol)* *20mg/ml*	Very long acting (4-6 weeks)	Used for very chronic problems (arthritis?)	Dose: 1mg/kg or (10-40mg/dog) Cats 10-20mg IM (4mg/kg) (NOT IV)	**Care with use re adrenal axis suppression – can cause diabetes mellitus to become clinical in cats**
Prednisolone *(Macrolone: 20mg)* *(Delta cortef*: 5mg)	Short acting	Ongoing therapy	***Immunosuppressive:*** Dogs: 1-2mg/kg PO Cats: 2-4mg/kg PO ***Anti-inflammatory:*** Dogs: 0.2-0.5mg/kg PO Cats: 0.4-1.0mg/kg PO ***Anti-pruritic:*** Dogs: 0.2-0.5mg/kg PO Cats: 1-2mg/kg PO	Taper dose before stopping (BID 7d; SID 7d; EOD) If a prolonged immunosuppressive course, then taper slowly e.g. 20% reduction every 2 weeks while monitoring for signs of reoccurrence of disease
Prednisolone sodium succinate (Solu Delta Cortef) 10mg/ml	Rapid and intense action – short acting Decreases vascular permeability; diminishes exudation; impairs migration of inflammatory cells	Used for anaphylaxis; allergic reactions with urticaria/ wheals	Anti-inflammatory: 2mg/kg IV Anaphylaxis: 5-10mg/kg IV	Can be used for vaccine reactions as fast acting and wears off quickly so less interference with vaccine

Chlorpheniramine	Anti-histamine	Allergic reaction; pruritus Sedative (mild)	0.5mg/kg BID (4-8mg) dog PO/IM 2-4mg/cat BID PO	Sedation
Dexchlorpheniramine	Anti-histamine	Allergic reaction; pruritus Sedative (mild)	<20kg ½ a 6.5mg tablet SID >20kg 1x 6.5mg tablet SID	Sedation
Azathioprine (Imuran)	Immune suppressant	Immune mediated disease	2mg/kg PO SID until remission, then 0.5-2mg/kg EOD	Bone marrow suppression, monitor every month for haematological abnormalities Avoid if hepatic disease Not for cats
Cyclosporine (Atopica)	Immune suppressant Anti-inflammatory	Atopy Immune mediated disease	5-10mg/kg/day divided then EOD Cats: 5mg/kg SID	Gastrointestinal signs Hepatotoxicity and nephrotoxicity (rare)

Emergency Drugs

Drug (*Trade Name*):	Action/Effect:	Indications:	Dosage:	Side Effects/Comments:
Adrenaline 1 in 1000 *1mg/ml*	Cardiac stimulant α and β agonist Bronchodilator ↑ BP (central vasoconstriction) Peripheral vasodilation	Cardiac arrest: Anaphylaxis Pre-med to ↓ allergic reactions (e.g. TAS)	0.1mg/kg IV/ET 0.005-0.01mg/kg IV Premed = 1ml IM	Overstimulate heart + stress
Atropine *(Atrosite)* *0.6mg/ml*	Anticholinergic Antispasmodic Mydriatic Inhibit vagal tone → ↑ HR	Bradycardia: asystole OP toxicity ↓ Secretions (oral/airway) Relax smooth muscle	0.02-0.04mg/kg (= 0.33ml/10kg), IV,IM, SC, IT 0.2-2.0mg/kg IV for OP toxic	Can cause tachycardia, dry mouth Do not administer if high HR Caution if heart disease Is also bronchodilator (but ↓ secretions)
Bicarbonate 8.4% (1mEq/ml)		Acidosis	1mmol/kg IV (1mEq/kg)	Can cause hypernatraemia, paradoxical cerebral acidosis (leading to neurological signs), hypocalcaemia
Calcium gluconate 10% solution	Electrolyte	Hypocalcaemia Hyperkalaemia (to ↓ cardiac effects)	0.5-1.0ml/kg slow IV (to effect)	Do not give SC or IM (irritant) Give slow IV (over 10 mins) and monitor HR
Diazepam *(Valium; Pamlin)* *5mg/ml*	Benzodiazepine BZ → ↑ GABA → ↑ Cl + hyperpolar Sedative (not sole) Muscle relaxant	Control seizures/tremors Tranquilizer (not potent)	Seizures: 0.2-1.0mg/kg IV Sedation: 0.2-0.5mg/kg IV or IM	Give slow IV (fast can cause hypotension and excitement) Poor absorption SC Short half-life in dogs
Dopamine	+ve inotrope ↑ cardiac contractility	Low blood pressure, Bradycardia	2-10g/kg/min IV	Dilute 200mg vial in 1L 0.9% NaCl = 200µg/ml
Doxapram *(Dopram)* *20mg/ml*	Respiratory stimulant	Apnoea Bradypnoea Caesareans - pups	1-5mg/kg IV 5.5-11mg/kg IV 1-2 drops under tongue (pups)	Can cause seizures, hypertension, cardiac dysrhythmias Repeat every 15 mins
Pred sodium succinate (solu delta cortef)		Anaphylaxis	5-10mg/kg IV	
Terbutaline (Bricanyl)	Bronchodilator	Asthma Chronic respiratory dis Bronchospasm	1.5ml/10kg BID-TID	

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

Maropitant citrate *(Cerenia)*	Against Central and peripheral emetic centre	Acute vomiting Motion sickness	**Dog**: 1mg/kg (=1ml/10kg) SC SID or 2mg/kg PO SID **Cat**: 1mg/kg SC, PO SID 8mg/kg PO SID	For up to 5 consecutive days Less than 2 consecutive days
Prochlorperazine *(Stemetil)*	Anti-emetic	Vestibular dogs	0.1-0.5mg/kg SC IM TID	May cause sedation and hypotension Avoid if seizure history (ACE family)
Ranitidine *(Zantac)*	H_2 receptor antagonist (↓ acid release)	Gastric ulcers Oesophagitis	Dog and cats: 2mg/kg IV TD	Give slow IV → can cause arrhythmias if fast Pain if administered IM

Urinary System Drugs

Drug (*Trade Name*):	Action/Effect:	Indications:	Dosage:	Side Effects/Comments:
Bethanechol chloride (*Urocarb*)	Cholinergic and muscarinic agonist Parasympathomimetic agent	Increase detrusor contractility Poor bladder contractility	Dogs: 5-20mg/dog PO TID Cats: 2-4mg/cat PO TID	MUST ensure urethral patency before giving and use in conjunction with phenoxybenzamine
Dantrolene Sodium (*Dantrium*)	Muscle relaxant	Relax external (skeletal muscle) urethral sphincter	Dogs: 2 mg/kg PO TID Cats: 1-2 mg/kg PO TID	
Diazepam (*Valium*)	Muscle relaxant	Relax external (skeletal muscle) urethral sphincter	Dogs: 0.5mg/kg PO TID Cats: 0.3mg/kg PO TID	Beware cats with hepatic disease
Diethylstilboestrol Oestrogen	Synthetic oestrogen	Urinary incontinence due to lack of oestrogen	Dogs: 0.2-1mg/dog PO SID Cats: 0.1mg/cat PO SID After 1 week start to reduce dose and frequency to least possible	Avoid cumulative doses of >1mg/week Bone marrow suppression Dermatological changes
Phenoxybenzamine hydrochloride (*Dibenzyline*)	α-adrenergic antagonist Muscle relaxant	Relax internal (smooth muscle) urethral sphincter	Dogs: 0.2mg/kg PO BID-TID Cats: 2.5mg/cat PO BID-TID	Hypotension, miosis, tachycardia
Phenylpropanolamine (*Propalin*)	α-adrenergic agonist Sympathomimetic	Urinary incontinence Increase internal sphincter tone	1mg/kg PO BID-TID	Tachycardia and hypertension Excitement and restlessness Anorexia
Prazosin *(Minipress)*	Alpha-1 antagonist	Functional urethral obstruction	Dogs: 0.5-1mg/kg PO TID Cats: 0.25-1mg/cat PO TID-BID	Hypotension Syncope Weakness Gastrointestinal upsets
Propantheline *(Propan B)*	Anticholinergic and muscarinic antagonist Parasympatholytic	Reduce urge incontinence due to detrusor spasm Smooth muscle motility (block detrusor spasm)	0.25-0.4mg/kg PO BID-TID	Can decrease GI motility

Seizure Disorders

Drug (*Trade Name*):	Action/Effect:	Indications:	Dosage:	Side Effects/Comments:
Clonazepam *(Paxam)*	Anticonvulsant Anxiolytic/sedative Muscle relaxant	Status epilepticus (adjunct)	0.05-0.2mg/kg IV 0.5mg/kg PO BID-TID	Used after diazepam CRI Will develop tolerance with extended use
Diazepam *(Valium; Pamlin)* *5mg/ml*	Benzodiazepines (BZ → ↑ GABA → ↑ Cl → hyperpolar → refractory) Anti-convulsant Anti-anxiety	Control seizures/tremors Tranquilizer (not potent) Sedative (not sole) Muscle relaxant	Seizures: 0.2-1.0mg/kg IV repeated 3 times, q5 mins. CRI: 0.5-2mg/kg/hr 2mg/kg per rectum 0.5mg/kg intranasal	Give slow IV (fast can cause bradycardia/hypotension and excitement) Poor absorption SC; stings IM Can get ataxia (→ ↑ anxiety); little sedation Use for appetite ↑ in cats Respiratory depression Can give intra-nasal or per-rectum Possible idiosyncratic hepatoxicity in cats
Gabapentin	Analgesia Anticonvulsant	Refractory seizures Chronic pain	10-20mg/kg PO TID (seizures) 3mg/kg PO SID (pain) 1.25-5mg/kg PO BID	Wean off drug if used for seizure treatment (decrease dose slowly)
Levetiracetam *(Keppra)*	Anticonvulsant (unknown mode of action)	Status epilepticus (adjunct)	Maintenance therapy: 20mg/kg TID Seizures: Up to 60mg/kg IV	Used with phenobarbitone and bromide Can ↓ phenobarbitone dose required Can be used in status epilepticus Not hepatotoxic
Midazolam	Benzodiazepines (BZ → ↑ GABA → ↑ Cl → hyperpolar → refractory) Anti-convulsant	Control seizures/tremors Sedative Muscle relaxant	Seizures: 0.2mg/kg IV q5 mins CRI: 0.1-0.3mg/kg/hr	Possible respiratory depression
Phenobarbitone *(various)*	Anti-convulsant Barbiturate → ↑ GABA sensitivity → ↑ Cl → inhibitory	Ongoing control of seizures	Maintenance therapy: 4mg/kg/day PO divided Seizures: 2-4mg/kg IV repeated to a max of 18mg/kg/day Therapeutic range: 100-130µmol/l	Dilute in saline to inject Slow onset (20+ mins); administer slow IV May cause sedation for 48hrs (monitor airway) Will develop tolerance; 2-3 weeks till steady state Caution if liver disease/toxicity (>150µmol/l) Low fat diet Wean off drug if used for seizure treatment (decrease dose slowly)

Potassium bromide	Anti-convulsant	Idiopathic epilepsy (long term control) Patients with liver damage	Sole agent: 30mg/kg/day PO SID or divided Combined with phenobarbitone: 20mg/kg/day PO SID or divided Loading dose (GIT side effects and sedation): 400-600mg/kg PO divided Half dose BID Therapeutic range: 15-20µmol/l 20-30µmol/l (sole agent)	DO NOT use in cats Used with phenobarbitone if poor control Use instead of phenobarbitone if liver disease Takes 2-3 months for steady state Increased risk of pancreatitis

Other Drugs

Drug (*Trade Name*):	Action/Effect:	Indications:	Dosage:	Side Effects/Comments:
Dalteparin sodium *(Fragmin)*	Anticoagulant (> heparin)	Thromboembolism	100IU/kg SC BID	Haemorrhaging
Insulin *(Actrapid)*	Push potassium, glucose, phosphorus and magnesium into cells	Ketoacidosis Acute diabetic crisis Hyperkalaemia	CRI; dose varies with patient	Excess causes hypoglycaemia → depression, seizures, coma 0.5-1 IU/kg IV + 1g dextrose/IU + 5% D5W
Mannitol *(Osmitrol)*	Osmotic diuretic Stimulate urine production (kidneys) ↓ fluid pressure	Head trauma Seizures Anuria Pigmenturia	Increased ICP: 0.5g/kg as CRI over 20-30mins TID Anuria: 0.25g/kg as CRI over 20-30mins	Monitor urine output to ensure kidneys coping (preferably with urinary catheter) Not for cardiac patients 20% = 1gm per 5mls
Potassium chloride	Supplement	Hypokalaemia Anorexia	Depends on serum level – add to IV fluids	Mix KCl thoroughly in bag Overdose → dysrhythmias and death
Vitamin K1 *(Koagulon; K-Mav)*	Coagulant	Rodenticide Severe liver disease	5mg/kg IM then 2.5mg SC or PO BID NEVER IV	Dilute with 5ml saline (SC stings) Use small gauge needle (↓ haematoma) IV likely to cause anaphylaxis
Clopidogrel *(Plavix)*	Inhibition of platelet aggregation	Prevention of thromboembolism	Dogs: 2-4mg/kg PO SID Cats: 20mg/cat PO SID	Increased risk of bleeding

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(Hons class 1) MVS (Small Animal Practice) MANZCVSc (Emergency and Critical Care)

Gerardo completed his Bachelor of Veterinary Science at the University of Queensland, Australia in 2008. He graduated with first class honors, awarded the Australian Veterinary Association Student Award, he was also selected as valedictorian of his year. After graduation he spent almost 3 years in a busy small animal general practice. In 2010 he changed direction and started working in the field of emergency and critical care with Animal Emergency Service in Underwood, Brisbane where he is now a managing veterinarian.

He achieved Membership with the Australian and New Zealand College of Veterinary Scientists in the field of Emergency and Critical Care in 2012 and is currently the head examiner for future Membership candidates. In 2014 he completed his Masters of Veterinary Studies in Small Animal Practice through Murdoch University which focuses on the more advanced aspects of small animal medicine.

Gerardo has a strong interest in the stabilisation and management of critically ill patients, small animal ultrasound and radiology and emergency surgery. He is currently the coordinator of the internship program and the continuing education program for the emergency clinicians at three Animal Emergency Service practices.

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