POCKET HANDBOOK OF

SMALL ANIMAL MEDICINE

KIT STURGESS

MANSON PUBLISHING
This book is dedicated to... my wife, Avery, and my children William and Matthew for the family time they gave up to allow me to write this text... my parents’ memory for their endless encouragement and support... my veterinary colleagues without whom I would not have gained my knowledge and experience.
Preface

When I set out to write this handbook, it seemed a simple task: not too much detail and no references. It soon became clear that deciding what to include and – even more importantly – what to leave out, while still producing a useful and informative text, was a big challenge – so I hope that I have the balance right!

The Pocket Handbook of Small Animal Medicine has been designed as a ‘pick-up-and-get-started’ text for quick and easy reference. It aims to offer concise subject reviews and rapid interpretation and problem-solving of key clinical signs. Part 1 gives an overview of basic approaches and general health concerns, while Parts 2 and 5, ‘Clinical Presentations’ and ‘Critical Care’ present a series of algorithms intended to guide the reader from presentation to diagnosis, or to outline emergency management. Part 3 forms the core of the book, covering – in brief – the examination, diagnosis and treatment of the diseases and disorders of each body system, as well as multisystemic disorders, while Part 4 covers anaesthetics, analgesia, and surgery. For more in-depth discussion and treatments, a short Further Reading list is included.

ACKNOWLEDGEMENTS

I would like to thank Davina Anderson, Ewan Ferguson, Sue Fitzmaurice and Andy Moores for their contribution in writing or reviewing sections of this text, the Diagnostic Imaging section, University of Uppsala, for the GDV radiograph (page 80) and Dr Tim Nuttall of the University of Liverpool School of Veterinary Science for the dermatology photos (page 109). I would also like to thank the editorial team of Ayala Kingsley and Graham Topping for their unstinting efforts in producing this book.
# BASIC APPROACHES

1.1 General health  
1.2 History taking and physical examination  
1.3 Diagnostic techniques
1.1 General health

1.1.1 Dog breeds 1.1.2 Cat breeds 1.1.3 Congenital and hereditary disease 1.1.4 Genetic defects 1.1.5 Genetic screening 1.1.6 Vaccinology 1.1.7 Parasite control 1.1.8 Feeding healthy pets 1.1.9 Influence of life stage and lifestyle 1.1.10 Feeding the hospitalized patient; tube feeding techniques

INTRODUCTION

- While lifestyle and care clearly affect pet health, disease risk is also affected by genetic factors (1.1.3–1.1.5).
- The ultimate goal of medicine is to prevent patients becoming sick. Vaccination (1.1.6), worming (1.1.7) and appropriate nutrition (1.1.8) reduce this risk.

BREEDS, BREEDING AND DISEASE

1.1.1 Dog breeds

- Dogs have been bred with an incredible variety of body shapes and sizes, with over 150 breeds divided into 10 groups.
  - Group 1: sheepdogs and cattle dogs.
  - Group 2: pinschers and schnauzers, molossians, Swiss cattledogs.
  - Group 3: terriers.
  - Group 4: dachshunds.
  - Group 6: scent hounds, leash hounds, related breeds.
  - Group 7: continental pointers, Irish pointers and setters.
  - Group 8: flushing dogs, retrievers, water dogs.
  - Group 9: companion and toy dogs (12 sections, including poodles, bichon, Tibetan, Dalmatian, Japanese chin).
  - Group 10: sight hounds and related breeds.

- In the UK, Labrador retriever, Border collie, JRT, Yorkshire terrier and German shepherd account for 50% of the 5.5 million pedigree dogs and 7.3 million total pet dogs.

- See www.thekennelclub.org for further information.

1.1.2 Cat breeds

- There are around 50 registered breeds of domestic cat, grouped in the UK as follows by the GCCF.
  - Persian.
  - Semi-longhair: Birman, Maine coon, ragdoll, Somali, Norwegian forest cat.
  - British shorthair, including Manx and Selkirk rex.
  - Foreign: Abyssinian, Bengal, rex, Russian, korat, sphynx.
  - Burmese.
  - Oriental: including Havana.
  - Siamese, including Balinese.
1.1 General health

In the UK, British shorthair, Siamese, Bengal, Persian and Burmese account for 60% of the 0.6 million pedigrees, from a total of 7.2 million pet cats.

See www.gccfcats.org for further information.

Congenital and hereditary disease

Animals born with any defect have **congenital** disease, which does not have to be heritable.

**Heritable disease** may not be seen until later in life, e.g. mitral valve disease in Cavalier King Charles spaniels.

The same phenotype, e.g. cleft palate, can occur both as a heritable and non-heritable congenital defect in cats.

If a litter is born with or develops a defect, the hallmark of heritable disease is that only a proportion of the litter is affected by an identical defect.

If all or most of a litter is affected and the defects are different, this indicates an insult in utero, not genetic defects.

Genetic defects

Genetic defects can be:

- Breed related: where the fact of being of that breed predisposes the animal to a disease (e.g. brachycephalic airway syndrome in bulldogs) and breeding out a defect would change the breed beyond recognition.
- Breed defects: defect is widespread in multiple lines within the breed, e.g. flat chests in Burmese, hip dysplasia in retrievers.
- A defect reported in a breed: many defects that are reported as breed defects have only appeared in a few individuals or specific lines within the breed. However, the true prevalence of the genetic tendency within the breed may be masked by individuals’ lack of expression, e.g. craniofacial malformation (USA Burmese; not UK).

Genetic screening

Tests are available for an increasing number of diseases, such as polycystic kidney disease in Persians, or von Willebrand disease.

The limitations of the particular test should be recognized. Some genetic tests actually identify the defect (most reliable) whereas others identify genotypes that are associated with the disease and are less reliable.

Some tests identify genetic traits such as dog leukocyte antigen (DLA) Class II haplotypes, associated with canine diabetes.

Care should be taken when using a test to check that it has been appropriately validated for that breed.
PREVENTIVE MEDICINE

1.1.6 Vaccinology

■ **Principles**: vaccination primes the immune response. If wild-type infection is encountered, a rapid anamnestic response occurs: as a result, a protective immune response occurs before the infectious agent has multiplied to a level which would cause clinical signs.

■ Vaccines for allergic and neoplastic disease are not preventative, but are aimed at modifying the immune response.

■ Vaccination is not 100% safe or effective.

■ Severe vaccine reactions are rare, although long-term risks associated with vaccination are not fully understood but do exist, e.g. vaccine-associated sarcoma in cats.

■ Vaccination has also been loosely associated with immune-mediated diseases.

■ Vaccine failure can be due to:
  - Animal factors, e.g. already incubating disease or strong maternally-derived immunity.
  - Disease factors, e.g. new strains or weight of infection.
  - Vaccine factors, e.g. inappropriate strain inclusion, inappropriate storage or delivery.

■ **Vaccination schedules** vary with vaccine manufacturer.

■ The aim of puppy/kitten courses is to achieve early vaccination to allow socialization.

■ Primary course is generally 2 vaccines 3–4 weeks apart with the 2nd vaccination at 10–12 weeks old.
  - Modified live vaccination of very young puppies and kittens risks causing the primary disease.

■ Not all puppies and kittens respond to the primary course (e.g. 65% to distemper). A first booster at 15 months is vital to cover all individuals.
  - Note that duration of immunity for distemper/parvovirus vaccination depends on the manufacturer, but will be a minimum of 2–3 years.

■ **Vaccine types**: modified live virus (replication attenuated); killed adjuvanted; genetically-engineered, containing part of the agent (adjuvanted or inserted in a replication-deficient carrier).

■ Choice of vaccine type (see table, above right) depends on local disease prevalence and patient factors, e.g. FIV status.
Parasite control

- Virtually all puppies and kittens will be born with, or acquire during suckling, a parasite load. Low-level parasitism is also common in adult dogs and cats: though rarely of great clinical significance, it does pose a public-health risk.
- It is therefore important to develop a clear, practice-based worming policy for puppies and kittens, adult dogs and cats, and pregnant bitches and queens.
- The choice of product and frequency of working will depend on local prevalence of specific parasites, and on the lifestyle of the patient.
- The table on pages 10–11 shows available parasiticides in the UK, their formulation (combination), their route of administration, and their spectrum of activity.

**Parasites in dogs and cats**

- **Dogs**
  - *Distemper*
  - *Hepatitis (CAV-2)*
  - *Leptospira canicola*
  - *Leptospira icterohaemorrhagiae*
  - *Parvovirus*
  - *Rabies*
  - *Bordetella bronchiseptica*
  - *Coronavirus (not UK)*
  - *Parainfluenza*
  - Canine herpes virus
  - Canine influenza virus (not UK)

- **Cats**
  - *Feline calicivirus*
  - *Feline herpes virus 1*
  - *Parvovirus*
  - *Rabies*
  - *FeLV*
  - *FIP (not UK)*
  - *FIV (not UK)*

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**Lungworm larva** (*Angiostrongylus vasorum*) seen on bronchoalveolar lavage. There are several parasitic nematodes, including *Oslerus osleri* in dogs and *Aelurostrongylus abstrusus* and *Capillaria aerophila* in cats, that can cause pulmonary disease.
### BASIC APPROACHES

#### PARASITICIDES

<table>
<thead>
<tr>
<th>Active ingredient</th>
<th>Route</th>
<th>Fleas</th>
<th>Flies</th>
<th>Ticks</th>
<th>Demodex</th>
<th>Sarcoptes</th>
<th>Otodectes</th>
<th>Lice</th>
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<tbody>
<tr>
<td>1. Amitraz</td>
<td>Topical</td>
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<td>2. Deltamethrin</td>
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<td>3. Dichlorophen</td>
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<td>9. Garlic*1</td>
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<td>11. Ivermectin*2</td>
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<td>12. Lufenuron*3</td>
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<td>15. Methoprene*4</td>
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<td>17. Moxidectin</td>
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<td>20. Piperazine*5</td>
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<td>22. Pyrantel/Oxantel</td>
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<td>25. Tiabendazole</td>
<td>Ear drop</td>
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*1 Efficacy not established
*2 Toxicity in collie breeds
*3 Insect growth regulator
*4 Combined with permethrins as a household spray
*5 Toxic to large felids; overdose relatively common in kittens
* Limited efficacy
### Endoparasites

<table>
<thead>
<tr>
<th>Taenia</th>
<th>Diphylidium</th>
<th>Echinococcus</th>
<th>Giardia</th>
<th>Angiostrongylus</th>
<th>Toxocara</th>
<th>Toxocara larva</th>
<th>Uncinaria</th>
<th>Ancylostoma</th>
<th>Trichuris</th>
<th>Lungworm</th>
<th>Dirofilaria</th>
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<th>Host species</th>
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*Note:* The table represents the possible combinations of endoparasites and their hosts. The symbols (e.g., •) indicate the presence of that parasite species and its suitability for the indicated host.
NUTRITION

1.1.8 Feeding healthy pets

- Energy requirements depend on life stage, lifestyle, presence of chronic disease, neutering and reproductive status in females.
- Recently, breed-specific diets have been designed to meet:
  - Anatomic need, e.g. jaw shape in Persian cats.
  - General requirements of a breed group associated with their body size, body shape, appetite or temperament.
  - Theoretical nutritional strategies to reduce the incidence/severity of diseases common in that breed.

Target energy requirements

- Metabolizable energy (expressed per 100 g of dry matter for comparison) is used to digest, absorb and utilize food (heat increment). The remainder is available for growth, lactation, reproduction, physical performance (net energy for production) and repair, basic activity and thermoregulation (net energy for maintenance). Excess energy is then stored as fat.
- **Resting energy requirement** (RER) is the basic energy expended at rest:
  - RER (kcal/day) = 70 \times (\text{bwt in kg})^{0.75}
  - \approx 30 \times (\text{bwt in kg}) + 70 \text{ (for animals from 2–48 kg)}.
- **Maintenance energy requirement** (MER) is the amount of energy required by an active animal. A basic MER is expressed by the following equation, but it should be further modified according to life stage and lifestyle.
  - MER (kcal/day) = 1.8 \times \text{RER (dog)}
  - \approx 80 \times (\text{bwt in kg}) \text{ (cat)}.

Metabolizable energy.

Gross energy from food passes through two stages, with loss in various forms en route.
### MAINTENANCE ENERGY REQUIREMENTS

<table>
<thead>
<tr>
<th>Life stage/lifestyle</th>
<th>Dog (x MER)</th>
<th>Cat (kcal/kg bwt)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 3 months (growth)</td>
<td>2</td>
<td>250</td>
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<tr>
<td>3–6 months (growth)</td>
<td>1.5</td>
<td>130</td>
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<tr>
<td>6–12 months</td>
<td>1.2</td>
<td>80–100</td>
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<tr>
<td>Inactive adult</td>
<td>0.75</td>
<td>70</td>
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<tr>
<td>Active</td>
<td>1</td>
<td>80</td>
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<tr>
<td>Working 1 hour/day</td>
<td>1.1</td>
<td>Not applicable</td>
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<tr>
<td>Working full day (mild to moderate work)</td>
<td>1.5–2</td>
<td>Not applicable</td>
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<tr>
<td>Gestation (&lt;42 days)</td>
<td>1–1.1</td>
<td>88</td>
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<tr>
<td>Gestation (&gt;42 days)</td>
<td>1.1–1.3</td>
<td>88–104</td>
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<tr>
<td>Peak lactation (3–6 weeks)</td>
<td>1 + (0.25 x no. of puppies)</td>
<td>80 x (1 + [0.25 x no. of kittens])</td>
</tr>
<tr>
<td>Cold weather (pet outside)</td>
<td>1.25–1.75</td>
<td>Data not available</td>
</tr>
</tbody>
</table>

### Influence of life stage and lifestyle

- **Growth**
  - **Pre-weaning**: ideally dam’s milk, if not then fostering.
  - Hand rearing requires proprietary milk substitute.
  - **Post-weaning**: puppy 50%; kitten 75% of adult weight by 6 months.
  - Maximal growth does not mean optimal growth.

- **Puppies** require energy dense, highly digestible food.
  - The amount and balance of vitamins/minerals, particularly calcium and phosphorus, are important especially for giant breeds.

- **Kittens** are best fed *ad libitum*.
  - Energy requirements per kg peak at 10 weeks, but are lower than for puppies, as the percentage increases over birth weight are less.

- **Maintenance** has few requirements outside of a balanced diet.
  - Cats and small dogs reach adult requirements at 12 months; medium dogs 15–18 months and giant breeds 18–24 months.

- **Gestation** requires increased feeding as follows:
  - For dogs in 5th–9th weeks (when most foetal weight gain occurs), increase ration by 15% per week, with small meals of higher density foods.
  - For cats feed *ad lib*: if portion-fed, increase by 4–5% per week.
**Lactation** makes a huge nutritional demand, peaking at 4 weeks postpartum. Small meals of highly palatable, highly digestible food required for bitches, ad libitum feeding of queens is recommended.
- Additional vitamins/minerals are *not* necessary if diet is balanced.

**Activity:** a 5 km run increases a dog's requirements by only 10%! Generally, working dogs need a highly palatable, energy dense (fat), highly digestible and nutritionally-balanced diet.

**Old age** requirements are poorly defined.
- Intestinal function declines from 8 years of age.
- It is generally felt that lower nutritional density is required, as energy demands decrease due to reduced levels of activity and lower lean body mass.
- Vitamin and antioxidant requirement may be increased.
- Palatability is important as appetite can be reduced.
- Increase feeding frequency.

### 1.1.10 Feeding the hospitalized patient
- Nutrition needs to be addressed if the patient is severely inappetant/anorexic for >3 days.
- If food manipulation or hand-feeding fails, then consider appetite stimulants (see table) or tube feeding.

**Tube-feeding techniques**
- **Nasoesophageal tube:** use with conscious or lightly sedated patient. Inappropriate if vomiting, oesophageal disease, unconscious patient.
- Tube measured to 9th rib (cat); to 7th intercostal space (dog).
- Nostril desensitized and tube (6–16 Fr) advanced in a ventral and medial direction with the neck flexed to the pre-measured point.

<table>
<thead>
<tr>
<th>Appetite stimulant</th>
<th>Dose rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diazepam (cat/dog)</td>
<td>0.1–0.2 mg/kg po or i/v q12–24hr</td>
</tr>
<tr>
<td>Oxazepam (cat)</td>
<td>0.2–0.4 mg/kg po q24hr</td>
</tr>
<tr>
<td>Cyproheptadine (cat)</td>
<td>2 mg/cat po q24hr</td>
</tr>
<tr>
<td>Mirtazapine</td>
<td>3.75 mg/cat po q3 days</td>
</tr>
<tr>
<td></td>
<td>0.5 mg/kg po q24hr</td>
</tr>
</tbody>
</table>

*Note:* Corticosteroids, anabolic steroids and megoestrol acetate are not first-choice drugs due to their potential side-effects.
Tube position can be checked with sterile water/saline or by radiography.
- Secure tube by tapes.
- Good for short-term feeding (5–7 days).
- **Oesophagostomy tube**: a small incision is made in the oesophagus distal to pharynx under light general anaesthesia and 14–18 Fr feeding tube inserted
  - Position and contraindications as for naso-oesophageal tube.
  - Take care regarding wound infection: there is a low risk of oesophageal stricture.
  - Use for short- to medium-term feeding.
- **Gastrostomy tube**: per-endoscopic gastrostomy (PEG), blind or surgical, 16–20 Fr tube with a suitable mushroom tip. PEG is quicker and much less traumatic than surgery.
  - Risks:
    - Inappropriate if vomiting, unacceptable anaesthetic risk.
    - Risk of leakage around the tube and peritonitis, necrosis of the stomach wall (tube pulled too tight), splenic laceration, interference with gastric emptying.
    - Risks highest with blind and lowest with surgical placement.
  - For long-term feeding (months to years).
- **Other**: jejunostomy tube feeding and total parenteral nutrition are specialized techniques used in vomiting and unconscious patients.

**History taking and physical examination**

1.2.1 Consultation as a problem-solving exercise
1.2.2 Consultation as a collaboration with the owner
1.2.3 Open and closed questions
1.2.4 General background questions
1.2.5 Presentation-related questions
1.2.6 Examination basics
1.2.7 Normal parameters

**GENERAL APPROACH**

**Consultation as a problem-solving exercise**

- History taking and physical examination are key to achieving a diagnosis and treating cases appropriately.
- A thorough knowledge of normal variation is important in the interpretation of findings.
- Consultations should be conducted using a problem-solving approach, as this:
  - Helps to apply vast amounts of factual information from a number of different sources to an individual case.
  - Ensures the approach is logical and thorough, allowing important facets of the case to be appreciated, and reducing the risk of missing crucial information.
Provides accurate and complete medical records for effective communication between colleagues, thus aiding case management, which is particularly important in a multi-person/multicentre practice.

- Provides accurate and complete medicolegal records.
- Identifies critical patient problems.
- Records less-critical problems which may be important later in patient management, or may be of greater significance than originally apparent.
- Encourages clear decision making.

1.2.2 Consultation as a collaboration with the owner

Planning and management

- Construct a differential list.
- Explain why each major differential is relevant; use visual aids if helpful.
- Relate differentials to owner concerns and check understanding:
  - Explain benefits/drawbacks for each management strategy, solicit owner's thoughts.
  - Offer choice of approach, encourage owner to express preference.
  - Negotiate an approach that is appropriate, practical, effective and which the owner understands.
  - Check agreement on the plan of action and expected outcomes.

Summary and closure

- Summarize and clarify plan of action.
- Provide a safety net: a plan B if the response is not good.
- Address outstanding concerns which the owner may have expressed.
- Agree date of follow-up appointment or further action.

CLINICAL HISTORY

1.2.3 Open and closed questions

- Owner information is often undervalued. Fitting a full clinical history into a time-restricted consultation is difficult, and is often overlooked in the desire to begin the physical examination.
- It is important to address the patient's problems not only as you identify them, but also as they are perceived by the owner.
- When asking an owner questions, where possible they should initially be phrased so as not to imply the answer that is expected.
- Closed questions invite a yes/no answer – quicker in obtaining a ‘history’ but may miss vital information.
Open questions allow a range of responses, as well as triggering other unasked but potentially important history.

Example 1:
- ‘Does Rover have diarrhoea?’ (closed) is answered ‘No’, but
- ‘What is Rover’s poo like?’ (open) receives the answer ‘covered in slime with a lot of straining’.
- ‘Is Henry vomiting?’ (closed) is answered ‘Yes’, not understanding the exact medical terminology, when Henry is in fact regurgitating.

Example 2:
- ‘Is Fluffy drinking more?’ tends to invite the answer ‘Yes’, partly because the question implies that the owner should know, and partly because it seems that the vet wants this answer to aid diagnosis.
- ‘Have you noticed a change in the amount Fluffy is drinking?’ allows the owner more easily to answer that they have not noticed any change – either because they have not looked, or there has been no change.

Closed questions are often more appropriate later on in the history when specific facts are being sought.

**General background questions**

Practice records should not be the sole source of the following information.
- Age, breed, sex.
- Time in owner’s possession.
- Past history.
- Worming, vaccination, flea control: this may not be on the medical record as products may have been obtained from other sources, vaccination clinics and so on.
- Environment: access to outside (cats), where and how much exercise (dogs).
- Other pets: are they well?
- Neutering status, or stage in reproductive cycle.

**Presentation-related questions**

- Why has the pet been presented (now)?
- General demeanour.
- Appetite and weight.
- Thirst/urination.
- Defecation, vomiting, diarrhoea.
- Coughing, sneezing, respiratory effort.
- Lameness.
- Lumps, bumps and skin changes.
- For additional history elements related to specific presentations see Part 2; and related to specific body systems, see Part 3.
1.2.6 Examination basics

- Time should be taken to complete the physical examination although this may not be possible in all patients, for a variety of reasons.
- If parts of the physical examination cannot be completed, e.g. auscultation of the thorax of a purring cat, this should be recorded and the owner should be informed.
- Maximum information will be gained if both the patient and the owner are calm and co-operative.
- Treat seriously any owner accounts of previous issues with physical examination.
- Ask the owner if their pet will react better if the owner holds it; or whether you should ask a nurse to help.
- Consider examining dogs on the floor: some dogs will be more relaxed there than on a table.
- If you think that you need to use a muzzle, ask the owner if this is acceptable, and about any previous response to muzzling.
- Elements of the physical examination related to specific body systems are presented in the relevant sections.

### HEART AND RESPIRATORY RATES

<table>
<thead>
<tr>
<th>(Consulting room)</th>
<th>Adult dog</th>
<th>Puppy</th>
<th>Adult cat</th>
<th>Kitten</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate (range/min)</td>
<td>70–160</td>
<td>70–220</td>
<td>120–200</td>
<td>140–240</td>
</tr>
<tr>
<td>Respiratory rate (range/min)</td>
<td>18–28</td>
<td>Tends to be higher in kittens, puppies and small breed dogs or if the animal is stressed. Most cats at home at rest have rates less than 24/minute</td>
<td>20–30</td>
<td></td>
</tr>
</tbody>
</table>

### NEONATAL PARAMETERS

<table>
<thead>
<tr>
<th>Age (days)</th>
<th>Rectal temperature (°C [°F])</th>
<th>Heart rate (bpm)</th>
<th>Respiratory rate (/min)</th>
<th>Recommended environmental temperature (°C [°F])</th>
</tr>
</thead>
<tbody>
<tr>
<td>0–7</td>
<td>35.5±0.8 [96±1.4]</td>
<td>200–500</td>
<td>15–35</td>
<td>29.5–32 [85–90]</td>
</tr>
<tr>
<td>8–14</td>
<td>37.8 [100]</td>
<td>70–220</td>
<td>15–35</td>
<td>26.5 [80]</td>
</tr>
<tr>
<td>15–28</td>
<td>No data</td>
<td>70–220</td>
<td>15–35</td>
<td>26.5 [80]</td>
</tr>
<tr>
<td>29–35</td>
<td>Adult</td>
<td>70–220</td>
<td>15–35</td>
<td>21–24 [70–75]</td>
</tr>
<tr>
<td>&gt;35</td>
<td>Adult</td>
<td>70–220</td>
<td>Adult</td>
<td>21 [70]</td>
</tr>
</tbody>
</table>
Normal parameters

Heart and respiratory rates
- Reference ranges for heart and respiratory rates are shown in the tables, below left. While absolute heart rate is important, it should also be appropriate for the situation.
- For example, normal dogs asleep can have heart rates less than 30/min and pauses of 4–5 seconds.
- A heart rate of 60/min in the consulting room in a highly stressed Yorkie is unexpectedly low, but may be expected in a laid-back labrador.
- The heart rhythm, character of the pulse and respiratory effort are also important parameters to assess and record.

Temperature
- Generally 38–39°C (100.5–102.5°F) is the normal range, but this can increase to 39.5°C in stressed patients and rise to 40.5°C after exercise.
- If there is concern that the patient’s pyrexia is physiologic, the owner should be encouraged to take the rectal temperature at home.
- Unfortunately, ear-based temperature measurement has not proven reliable; microchip-based temperature sensors tend to underestimate core temperature.

Diagnostic techniques

INTRODUCTION
- This section covers the most frequently employed diagnostic techniques used in veterinary practice. Further information on the interpretation of specific tests is given in Part 3. For additional reference material see page 190 and the BSAVA Guide to Procedures in Small Animal Practice (2010).
- It is important to develop a good relationship with any laboratory you use, and for both parties to be prepared to discuss the interpretation of results.

Good case information aids the laboratory in interpreting the results.
LABORATORY TESTS

1.3.1 Haematology

- Precise reference ranges will vary between laboratories and in-house machines; breed- and age-specific variations also occur.
- Moderate to marked changes are more likely to be clinically significant and relate to a primary disease process. Mild changes (especially in older animals) are common, may be difficult to explain, and may reflect disease elsewhere.
- Haematocrit (HCT) from most automated machines (derived parameter = RBCC × MCV) approximates to PCV. It can be significantly higher than PCV if the red-cell count is elevated, or in postal samples where red cell swelling raises MCV.

### HAEMATOLOGY: REFERENCE VALUES/INTERPRETATION

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Low MARKED</th>
<th>MODERATE</th>
<th>MILD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Packed cell volume (PCV) (%)</td>
<td>15</td>
<td>23</td>
<td>30</td>
</tr>
<tr>
<td></td>
<td>12</td>
<td>18</td>
<td>22</td>
</tr>
<tr>
<td>Red blood cell count (RBCC) (x10^{12}/l)</td>
<td>2.0</td>
<td>3.5</td>
<td>4.8</td>
</tr>
<tr>
<td></td>
<td>1.7</td>
<td>3.4</td>
<td>5.2</td>
</tr>
<tr>
<td>Haemoglobin (Hb) (g/dl)</td>
<td>3.3</td>
<td>7.5</td>
<td>10.0</td>
</tr>
<tr>
<td></td>
<td>3.0</td>
<td>5.5</td>
<td>7.0</td>
</tr>
<tr>
<td>Mean cell volume (MCV) (fl)</td>
<td>50</td>
<td>55</td>
<td>58</td>
</tr>
<tr>
<td></td>
<td>32</td>
<td>37</td>
<td>39</td>
</tr>
<tr>
<td>Mean cell haemoglobin (pg)</td>
<td>22</td>
<td>27</td>
<td>30</td>
</tr>
<tr>
<td>Platelets (x10^9/l)</td>
<td>10</td>
<td>50</td>
<td>120</td>
</tr>
<tr>
<td>WBCC (x10^9/l) (excludes leukaemia)</td>
<td>1.5</td>
<td>3.0</td>
<td>5.0</td>
</tr>
<tr>
<td></td>
<td>1.0</td>
<td>2.0</td>
<td>3.5</td>
</tr>
<tr>
<td>Neutrophil</td>
<td>1.0</td>
<td>2.0</td>
<td>2.8</td>
</tr>
<tr>
<td></td>
<td>1.0</td>
<td>1.8</td>
<td>2.3</td>
</tr>
<tr>
<td>Lymphocyte</td>
<td>0</td>
<td>0.5</td>
<td>0.9</td>
</tr>
<tr>
<td></td>
<td>0</td>
<td>0.7</td>
<td>1.3</td>
</tr>
<tr>
<td>Monocyte</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Eosinophil</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
</tbody>
</table>

### HAEMATOLOGY: NEONATES

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Kittens (mean or range)</th>
<th>0–3 DAYS</th>
<th>2 WKS</th>
<th>4 WKS</th>
<th>6 WKS</th>
</tr>
</thead>
<tbody>
<tr>
<td>PCV (%)</td>
<td>41.7</td>
<td>33.6–37.0</td>
<td>25.7–27.3</td>
<td>26.2–27.9</td>
<td></td>
</tr>
<tr>
<td>Hb (g/dl)</td>
<td>11.3</td>
<td>11.5–12.7</td>
<td>8.5–8.9</td>
<td>8.3–8.9</td>
<td></td>
</tr>
<tr>
<td>MCV (fl)</td>
<td>81.6</td>
<td>65.5–69.3</td>
<td>52.7–55.1</td>
<td>44.3–46.9</td>
<td></td>
</tr>
<tr>
<td>WBCC (x10^9/l)</td>
<td>7.55</td>
<td>9.1–10.2</td>
<td>14.1–16.5</td>
<td>16.1–18.8</td>
<td></td>
</tr>
</tbody>
</table>
Reticulocyte counts: used to evaluate an appropriate bone marrow response to the level of anaemia. Regenerative response in mild–moderate anaemia >100,000 (dog) 65,000 (cat)/μl; severe anaemia reticulocyte >250,000 (dog), 175,000 (cat)/μl.

Low reticulocyte count can occur due to extra marrow factors, e.g. iron deficiency or insufficient response time.

In leukaemia or leukaemic lymphoma, white cell count and the cell line affected can increase to >100,000/μl.

| Reference | High | Moderate | Marked |  |
|-----------|------|---------|--------|  |
| 37–55     | 60   | 65      | 75     | dog |
| 27–50     | 55   | 60      | 70     | cat |
| 5.0–8.5   | 9.0  | 10.0    | 11.0   | dog |
| 5.5–10.0  | 11.0 | 13.0    | 15.0   | cat |
| 12.0–18.0 | 20.0 | 22.0    | 25.0   | dog |
| 9.0–17.0  | 18.5 | 20.0    | 23.5   | cat |
| 60–80     | 81   | 83      | 87     | dog |
| 40–55     | 56   | 59      | 62     | cat |
| 31.5–37.0 | NA   | NA      | NA     | dog/cat |
| 160–600   | 650  | 800     | 1000   | dog |
|           | 700  | 1000    | 1500   | cat |
| 6.0–15.0  | 20.0 | 30.0    | 40.0   | dog |
| 4.0–15.0  | 20.0 | 30.0    | 40.0   | cat |
| 3.0–11.5  | 15.0 | 25.0    | 35.0   | dog |
| 2.5–12.5  | 15.0 | 25.0    | 35.0   | cat |
| 1.0–4.8   | 5.5  | 7.0     | 10.0   | dog |
| 1.5–7.0   | 7.5  | 9.0     | 12.0   | cat |
| 0–1.0     | 1.5  | 3.0     | 5.0    | dog/cat |
| 0–1.3     | 1.8  | 3.0     | 5.0    | dog/cat |

Puppies (mean ± standard deviation, or range)

<table>
<thead>
<tr>
<th>0–3 DAYS</th>
<th>0–2 WKS</th>
<th>2–4 WKS</th>
<th>6 WKS</th>
</tr>
</thead>
<tbody>
<tr>
<td>46.3±8.5</td>
<td>33–52.5</td>
<td>27–37</td>
<td>34</td>
</tr>
<tr>
<td>15.8±2.9</td>
<td>14–17.5</td>
<td>8.5–11.6</td>
<td>9.59</td>
</tr>
<tr>
<td>94.2±5.9</td>
<td>89–93</td>
<td>78–83</td>
<td>–</td>
</tr>
<tr>
<td>16.8±5.7</td>
<td>16.8–23</td>
<td>23–25.5</td>
<td>15.00</td>
</tr>
<tr>
<td>Parameter</td>
<td>Low MARKED</td>
<td>MODERATE</td>
<td>MILD</td>
</tr>
<tr>
<td>-----------------------------------</td>
<td>------------</td>
<td>----------</td>
<td>------</td>
</tr>
<tr>
<td>Total protein (g/l)</td>
<td>35</td>
<td>42</td>
<td>47</td>
</tr>
<tr>
<td>Albumin (g/l)</td>
<td>15</td>
<td>20</td>
<td>24</td>
</tr>
<tr>
<td>Globulin (g/l)</td>
<td>15</td>
<td>18</td>
<td>22</td>
</tr>
<tr>
<td>Sodium (mmol/l)</td>
<td>120</td>
<td>128</td>
<td>133</td>
</tr>
<tr>
<td>Potassium (mmol/l)</td>
<td>2.0</td>
<td>2.5</td>
<td>3.5</td>
</tr>
<tr>
<td>Chloride (mmol/l)</td>
<td>85</td>
<td>90</td>
<td>95</td>
</tr>
<tr>
<td>Total calcium (mmol/l)</td>
<td>1.0</td>
<td>1.5</td>
<td>1.8</td>
</tr>
<tr>
<td>Calcium (ionized) (mmol/l)</td>
<td>0.5</td>
<td>0.8</td>
<td>1.0</td>
</tr>
<tr>
<td>Phosphate (mmol/l)</td>
<td>0.5</td>
<td>0.6</td>
<td>0.7</td>
</tr>
<tr>
<td>Urea (mmol/l)</td>
<td>1.0</td>
<td>1.5</td>
<td>1.8</td>
</tr>
<tr>
<td>Creatinine (μmol/l)</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Alkaline phosphatase (ALKP) (iU/l)</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Alanine aminotransferase (ALT) (iU/l)</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Aspartate aminotransferase (AST) (iU/l)</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Gamma glutamyl transferase (γ-GT) (iU/l)</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Total bilirubin (μmol/l)</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Bile acids (BA) (μmol/l)</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>BA – post-feeding (2hr)</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Glucose (mmol/l)</td>
<td>1.0</td>
<td>1.5</td>
<td>2.0</td>
</tr>
<tr>
<td>Fructosamine (mmol/l)</td>
<td>Low values may indicate periods of hypoglycaemia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Creatine kinase (iU/l)</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Cholesterol (mmol/l)</td>
<td>1.5</td>
<td>2.5</td>
<td>3.0</td>
</tr>
<tr>
<td>Triglyceride (mmol/l)</td>
<td>Not been significantly associated with disease</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Amylase (iU/l)</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Lipase (iU/l)</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Magnesium (mmol/l)</td>
<td>0.4</td>
<td>0.5</td>
<td>0.55</td>
</tr>
</tbody>
</table>
### 1.3 Diagnostic techniques

<table>
<thead>
<tr>
<th>Reference</th>
<th>High</th>
<th>MILD</th>
<th>MODERATE</th>
<th>MARKED</th>
<th>dog</th>
<th>cat</th>
</tr>
</thead>
<tbody>
<tr>
<td>54–77</td>
<td>80</td>
<td>90</td>
<td>100</td>
<td>110</td>
<td></td>
<td></td>
</tr>
<tr>
<td>54–80</td>
<td>90</td>
<td>100</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>26–42</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>dog/</td>
</tr>
<tr>
<td>24–47</td>
<td>55</td>
<td>60</td>
<td>70</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>139–154</td>
<td>160</td>
<td>165</td>
<td>170</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>135–160</td>
<td>164</td>
<td>168</td>
<td>172</td>
<td></td>
<td>dog</td>
<td></td>
</tr>
<tr>
<td>3.5–5.5</td>
<td>6.0</td>
<td>7.0</td>
<td>8.0</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>99–125</td>
<td>130</td>
<td>135</td>
<td>140</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>110–130</td>
<td>135</td>
<td>140</td>
<td>145</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2–3</td>
<td>3.5</td>
<td>4.0</td>
<td>5.0</td>
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<td>25</td>
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<td>cat</td>
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<td>0.5–2.0</td>
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<td>1.5</td>
<td>2.0</td>
<td>3.0</td>
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</tbody>
</table>
1.3.2 Biochemistry

- Spun, separated serum is preferable to heparinized plasma for biochemical analysis (see also ‘Blood sample types/tests’ table).
- Bromide will cross-react with chloride measurement; if chloride is markedly high check if the patient is on potassium bromide for seizures.

1.3.3 Other tests

- In addition to biochemistry and haematology, other tests may be appropriate when presented with patients with signs suggesting:
  - GIT disease: full faecal analysis, vitamin B₁₂ and folate, trypsin-like immunoreactivity (TLI), pancreatic-specific lipase, ACTH stimulation test (to rule out atypical hypo-adrenocorticism as a cause of GI signs).
- **Renal disease**: urinalysis including UPC and culture, estimate GFR, erythropoietin level, acid/base status.
- **Liver disease**: dynamic bile acid testing, blood ammonia, bile cytology and culture, clotting profile.
- **Cardiac disease**: cardiac troponin I, NT-proBNP, taurine.
- **Anaemia**: reticulocyte count, erythropoietin level, iron and iron binding level.
- **Unexplained lymphocytosis**: flow cytometry.

### Hormonal tests
- Many hormones, e.g. parathyroid hormone (PTH), require careful handling, as they are easily destroyed during sampling or postage; check requirements.
- **ACTH stimulation test** for hyper- and hypoadreno-corticism: measure basal cortisol, inject tetracosactide (Synacthen) i/v or i/m and measure cortisol after 45–60 minutes.
  - For a dog weighing >10 kg use 0.25 mg (1 vial); weighing <10 kg, use a half-vial.
  - Cats should be sampled at 1 and 3 hours, use a half-vial tetracosactide.
- **Low-dose dexamethasone suppression test** (LDDST) for hyperadrenocorticism: give 0.01 mg/kg (dog) 0.1 mg/kg (cat) i/v dexamethasone sodium phosphate, and measure at 0, 3 and 8 hrs.

  *For accuracy, dilute dexamethasone to 0.2 mg/ml solution with water for injection and use an insulin syringe.*

- High-dose dexamethasone suppression test (HDDST) to distinguish pituitary vs. adrenal dependent disease: give 0.1 mg/kg (dog) 1.0 mg/kg (cat) dexamethasone i/v and measure as LDDST.
- **TSH stimulation test** for hypothyroidism: inject recombinant human TSH 50 μg i/v and measure thyroxin at 0 and 6 hrs.
- See endocrine system (3.11) for further information.

### Genetic tests
- Genetic testing is most valuable for breeding programmes and for individual patients with a specific diagnosis.
- Increasing numbers of tests are being offered, e.g. for polycystic kidney disease in Persian cats. The amount of validation that has been undertaken for a particular test is very variable.
- Counselling of owners before genetic testing is important. A positive result indicates the genetic potential to develop the disease. A negative result does not always exclude disease.
1.3.6 Urinalysis

- Definitions: hyposthenuric = SG < 1.008; isosthenuric = 1.007–1.015; hypersthenuric = >1.015.
- **Collection:** catheter samples are rarely justified.
  - For cats, free catch using polystyrene packing, plastic beads (e.g. Katkor) or washed fish-tank gravel.
  - For dogs mid-stream urine flow is preferred.
- **Cystocentesis** allows accurate full urinalysis – send in a plain tube.
- If there is gross haematuria on urination, compare a cystocentesis sample with a free catch, as this will help localize the area that is bleeding.
- **Dipsticks** are designed for humans: SG, leucocytes, protein nitrite and urobilinogen can be unreliable or of no value.
- SG should be measured with a refractometer.

### Urinalysis Reference Ranges

<table>
<thead>
<tr>
<th>Specific gravity (SG)</th>
<th>pH</th>
<th>Bilirubin</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cat</td>
<td>&gt;1.035</td>
<td>1.001–1.080</td>
</tr>
<tr>
<td>Dog</td>
<td>&gt;1.025</td>
<td>1.001–1.065</td>
</tr>
</tbody>
</table>

### Blood Sampling Tubes for Different Tests

<table>
<thead>
<tr>
<th>EDTA</th>
<th>Haematology, fibrinogen, Coombs’ testing, genetic and PCR testing (e.g. FeLV, FIV, <em>Mycoplasma haemofelis</em>)</th>
</tr>
</thead>
<tbody>
<tr>
<td>EDTA plasma</td>
<td>lipid electrophoresis*, plasma osmolality</td>
</tr>
<tr>
<td>Frozen EDTA plasma</td>
<td>Endogenous ACTH*, PTH, PTH-RP*, renin*, NT-proBNP</td>
</tr>
<tr>
<td>Protease inhibitor NT-proBNP</td>
<td></td>
</tr>
<tr>
<td>Heparin plasma</td>
<td>Carnitine (frozen), ionized calcium, in-house laboratory analysis, can be used for external samples, plasma amino acids (frozen)*</td>
</tr>
<tr>
<td>Serum</td>
<td>Most biochemistry, hormonal tests, antibody titres, drug levels, erythropoietin*, gastrin (frozen)*</td>
</tr>
<tr>
<td>Citrate</td>
<td>Clotting times, anti-thrombin 3, D-dimer, von Willebrand factor, lead</td>
</tr>
<tr>
<td>Oxalate/fluoride</td>
<td>Glucose</td>
</tr>
</tbody>
</table>

* sample greater than 0.5 ml required
- **Sediment**: should be inactive – few cells and no casts.
  - More than one or two casts per low power field usually indicates damage to renal tubules.
  - Presence of bacteria reported in sediment does not demonstrate urinary infection.
    - Infection must be confirmed by culture as mis-identification rate is high.
- **Culture**: positive on cystocentesis, or >10^5 cfu/ml on free-catch/catheter, would be significant results.
- **Urine protein:creatinine ratio** gives a better estimate of proteinuria:
  - For dogs, a ratio of <0.4 is normal, >0.6 would be abnormal.
  - For cats, a ratio of <0.2 is normal, >0.4 would be abnormal.
- **Urine cortisol:creatinine ratio** can be a useful negative rule out for hyperadrenocorticism.
  - For dogs the ratio should be <20 × 10^-6.
  - For cats the ratio should be <50 × 10^-6.

### Sample collection and preparation

- Correct sampling and handling are vital to obtain valid results.
- **Blood samples**: a 0.5 ml sample is usually sufficient; see table, below left, for those tests which need more.
- **Urinalysis**: requires a sample >5 ml. Only use boric acid tube if free-catch for culture.
- **Fine-needle aspiration biopsy (FNAB)**: use a 21g needle; take multiple samples using either a needle-insert technique without suction, or suction with a 5 ml syringe.
  - Remember to release the suction pressure before removing the needle, as this reduces the likelihood of sucking material into the syringe that cannot be retrieved.
  - Storage in the fridge or in the same package as formalin reduces quality of FNAB slides.
- **Biopsy**: try to gain material from advancing edge of a lesion or mass.
  - Ensure adequate formalin is used (10 × volume of biopsy).
  - If the biopsy is large, section into 10 mm strips.
ECG BASICS

1.3.8 ECG recording
- A calm, quiet room is required.
- Patient should preferably be in right lateral recumbency, but for rhythm it is better to place the patient in a position in which they are comfortable.
- Chest leads are rarely required or of value. Electrode placement:
  - Red – right fore.
  - Yellow – left fore.
  - Green – left hind.
  - Black – right hind.
- Maximize complex deviation by turning up gain.
- If baseline artefacts are present, turn off electrical equipment in the room, and use the filter options.
- Record leads I, II and III at 25 and 50 mm/sec; aVR, aVL and aVF at 25 mm/sec.

1.3.9 ECG interpretation
- Diagnostic quality: can complexes be clearly identified?
- Rate: consider overall rate as well as beat–beat variation.
- Rhythm: is it sinus (P for every QRS and QRS for every P)?
- Every QRS must be followed by a T wave.
- Mean electrical axis: angle of lead at 90° to the isoelectric lead (sum of positive and negative deviation = 0°).
- If abnormal beats or artefact(s) are present: how many? Are they all the same?
  - Supraventricular beats tend to be narrow and similar to normal QRS complexes; ventricular beats tend to be wide and bizarre.
- Measure complex deviation and time intervals.
- What might other deviations from baseline be?
RADIOGRAPHY

Radiographic tips

- Use positioning aids and sedation/anaesthesia as appropriate.
- In lateral view, use a wedge to raise the sternum parallel to the spine.
- Inflated anaesthetized films give most detail of the lungs, but don’t overinflated.
- For lung views, maximize kV and minimize mAS to reduce movement blur.
- Deep-chested dogs may need separate cranial and caudal abdominal views.
- Obtain at least two views (left and right lateral, or right lateral and DV/VD of the chest; right lateral and VD of the abdomen).
- VD views in dyspnoeic patients can be dangerous.
- Use a bright light to examine lung detail on the radiograph.
- For orthopaedic views: remember that soft tissue is important. Flexed and extended views may be needed.
- For spinal films: try to keep the spine parallel to the plate.

Thoracic radiography

- The diagrams below illustrate normal thoracic radiographic anatomy.

Thorax: lateral view.

1. Aorta
2. Cranial mediastinum
3. Trachea
4. Pulmonary vessels
5. Cardiac silhouette
6. Fundus of the stomach
7. Lung fields
8. Carina
9. Caudal vena cava
10. Diaphragm
11. Caudal mediastinum

Thorax: dorsoventral view.
1.3.12 Abdominal radiography

- At least two views of the abdomen are essential in order to locate the position of any abnormality. Usually these are a right lateral and VD, as in the diagrams above.

- For complete evaluation of the stomach, four views are required; in normal cats and dogs gastric gas is located as follows:
  - VD view – fundus.
  - DV view – body.
  - Left lateral view – antrum.
  - Right lateral view – fundus and body.

Note species difference: pylorus is more midline in cats than dogs.
ULTRASOUND BASICS

- Full ultrasound of the abdomen, thorax and other structures is a specialist technique that requires a lot of knowledge, understanding and experience of normal variation and the interpretation of abnormalities, as well as appropriate equipment.
- However, the necessary skills can be acquired relatively easily to answer key questions that help with problem solving a case, e.g. the presence of free abdominal fluid or evidence of left atrial enlargement.
- When performing an ultrasound examination it is important that the areas examined, limitations and findings of the examination are recorded.

Abdominal ultrasound

- Good patient preparation is important. Sedation may be necessary in some cases but can be minimized by a suitably comfortable table and quiet surroundings.
- A systematic approach should be adopted and abnormal findings described and then interpreted.
- A suitable reference text will be helpful in illustrating probe positioning and normal variation. Correct machine settings can also make a significant difference.

Echocardiography

- Imaging the patient in lateral recumbency from underneath, via a cutout in the table top, produces the best images.
- Phased array probes are preferred as it is easier to get a good image window between the ribs.
- Full cardiac ultrasound is complex, time consuming and requires significant experience. However, a right parasternal approach to produce 2D short- (SA) and long-axis (LoA) views can generate useful decision making information (see images, next page).
  - **Long axis (LoA)**: ultrasound beam perpendicular to the long axis of the body and parallel to the left atrium of the heart showing the four chambers. A left ventricular outflow view showing the aortic valve is created by cranial angulation or clockwise rotation of the probe.
  - **Short axis (SA)**: rotate transducer by 90° clockwise trying to produce a left ventricle with circular symmetry and an internal mushroom shape (created by the papillary muscles). Progressive dorsal angulation and slight clockwise rotation will bring the aorta into view.

*If it’s round and central, it’s probably the aorta.*
Key questions

- Is there a pericardial effusion – a separation of the myocardium from the pericardium by hypo/anechoic fluid?
- Is there volume overload of the left heart – an enlargement of the left atrium (most easily assessed in short-axis view)
- What does the myocardium look like and how well is it moving?

Measuring the left atrial size

- The ratio between the left atrium and aorta in diastole just before the aortic valves open is relatively unaffected by patient size.
  - Ratio of less than 1.4 is normal and over 1.6 abnormal.
- The aortic measurement is taken when the valve is closed along the closure line between the non-coronary and right cusps. The left atrium is measured parallel from the line between the non-coronary and left cusp (red arrows, above).

Echocardiography. Standard right parasternal long and short axis views of the heart of a Cavalier King Charles Spaniel with mitral valve disease.

RV = right ventricle; LV = left ventricle; RA = right atrium
LA = left atrium; RPA = right pulmonary artery; Ao = aorta; MV = mitral valve
2

CLINICAL PRESENTATION

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2.12 Pyrexia 52
2.13 Anaemia 54
2.14 Jaundice 56
2.1 Sneezing and nasal discharge

**Key aspects of history and physical examination**
- Duration and progression of disease.
- Likelihood of infectious disease.
- Character of discharge: mucoid, serous, bloody (alone or mixed with secretions).
- Halitosis.
- Facial pain, asymmetry, or ocular signs.
- Physical examination:
  - Assess for regional lymphadenopathy.
  - Percuss sinuses.
  - Tracheal pinch test.
  - Oral and aural examination.
  - Assess nasal airflow.
2.1 Sneezing and nasal discharge

- **Infectious (viral, fungal), cleft palate, allergic, \*dental disease**
- **Foreign body, fistula, dental disease, neoplasia, fungal, polyp (cat)**

**Yes**

- **Nasopharyngeal or sinus disease**

**No**

- **Infectious (viral, fungal, \$bacterial, parasitic), dental disease, neoplasia**

**Antegrade:** infectious, neoplastic, allergic, foreign body, granuloma, dental disease, squamous metaplasia

- **Nasopharynx (retrograde):** pharyngitis, foreign body, neoplasia, parasites, polyps

- **Turbinate destruction:** aspergillosis, osteomyelitis (dental), neoplasia

- **Increased density suggests infection, neoplasia, granuloma, foreign body, blood**

**Notes**

- \*Reverse sneezing = violent paroxysmal inspiratory effort, often confused by owners as retching, gagging or coughing.
- \*Occasionally non-nasopharyngeal disease can cause nasal discharge, e.g. pneumonia and oesophageal reflux.
- \$Bacterial culture of nasal discharge is usually positive, but primary bacterial infections are very rare.
2.2 **Coughing**

**Approach to coughing**
- Coughing should be differentiated from reverse sneezing, gagging and retching.
- Consider recent exposure to allergens or irritants; seasonality; progression; single or paroxysmal coughs.
Notes

- *Many animals do not expectorate even when their cough is productive. Check whether there is a terminal retch/swallowing, which indicates that the cough is productive.
- Cardiac disease rarely causes coughing in cats.
2.3 Dyspnoea

**Approach to dyspnoea**

**Key decisions**
- Dyspnoea is caused by a failure of oxygenation of tissues (and removal of carbon dioxide) due to:
  - Obstruction to airflow, e.g. tracheal collapse, feline asthma.
  - Failure of diffusion across the alveolar membrane, e.g. pneumonia, pulmonary oedema.
  - Failure of lung circulation, e.g. cardiac disease, pulmonary hypertension, pulmonary thromboembolism, anaemia.
- Deciding which of these processes is responsible for the dyspnoea significantly aids disease localization.
2.3 Dyspnoea

- Alveolar or interstitial infiltrates or masses including pulmonary oedema
- Pleural effusion, mediastinal mass, diaphragmatic rupture
- Pneumothorax
- Is abdominal enlargement or muscle weakness causing restrictive pattern?
  - Yes: Nasal/nasopharyngeal disease
  - No: inspiratory effort
    - No: expiratory effort
      - No: Lower airway disease
    - Yes: Pharyngeal/laryngeal or tracheal disease

**Investigations**

- **Alveolar/interstitial disease**: CBC, bronchoalveolar lavage with cytology, serology (fungal, heartworm [region dependent]), faecal for lungworm, cardiac evaluation, bronchoscopy, guided biopsy, thoracotomy.
- **Pleural disease**: CBC, biochemistry, FeLV, FIV, FIP, cytology and culture on sample, drainage and repeat imaging, exploratory thoracotomy.
### 2.4 Inappetence and anorexia

**History**
Watch the animal eating or get the owner to video eating

- **No apparent environmental stress or dietary change**
  - Is there interest in food?
    - **No**
      - Oral, cranial and neurologic examination normal
        - Rule out systemic disease, particularly disease of the pancreas and GI tract
    - **Yes**
      - Neurologic examination of cranial nerves for abnormalities
        - **No**
          - Examination, imaging and endoscopy under GA. Nasal, oral or facial mass/fracture; dental disease; oral/pharyngeal foreign body or inflammation; retrobulbar abscess; TMJ disease
        - **Yes**
          - Trigeminal neuritis; masticatory muscle myositis
2.4 Inappetence and anorexia

Approach to inappetence/anorexia

- Inappetence/anorexia is a very non-specific sign, particularly in some cats and small-breed dogs that normally have poor appetites, even when well.
- Inappetence/anorexia will usually resolve once the primary condition has been treated; it is usually more productive to focus on the other clinical signs with which the patient presents.
- Rarely, significant food aversion will have developed as a result of an underlying disease process; e.g. severe oral pain may result in persistent inappetence, so that the patient may potentially require long-term tube-feeding.

Note

- *Manufacturers will sometimes change dietary formulation and this can cause problems for some pets. Was the inappetence associated with a new bag/batch of food, or large bags of dry food that has spoiled?
2.5 **Vomiting and regurgitation**

- **VOMITING or REGURGITATION?**
  - Vomiting: acute or chronic
  - Regurgitation

**Patient well**
- Responds to conservative treatment?
  - No
    - CBC and biochemistry, urine and faecal exam, imaging
      - Specific therapy if available, otherwise symptomatic and supportive
  - Yes
    - Gastrointestinal or abdominal disease
      - Non-surgical investigation
      - Systemic or metabolic disease
      - CNS or vestibular disease
      - Surgical investigation

**Patient unwell**
- Fails to respond
Approach to vomiting and regurgitation

- Some animals will present with signs consistent with both vomiting and regurgitation.
- The easiest way to decide whether material has been vomited (acidic) or regurgitated (alkaline) is to test the pH of the material using litmus paper.
- Chronic vomiting can lead to oesophagitis and secondary regurgitation associated with oesophageal dysfunction.
2.6 **Diarrhoea**

- **Acute, severe**
  - CBC and biochemistry; urine and faecal exam; imaging
  - Inconclusive results
  - Hypoproteinaemic: albumin $<22\text{g/l (dog)}$
    - $<18\text{ g/l (cat)}$
  - Treat cause if evident, if not symptomatic; supportive care

- **Chronic but significant**
  - Inconclusive results
  - Hypoproteinaemic: albumin $<22\text{g/l (dog)}$
    - $<18\text{ g/l (cat)}$
  - Treat cause if evident, if not symptomatic; supportive care

- **Mild, non-debilitating**
  - Inconclusive results
  - Hypoproteinaemic: albumin $<22\text{g/l (dog)}$
    - $<18\text{ g/l (cat)}$
  - Treat cause if evident, if not symptomatic; supportive care

2.7 **Weight loss**

- **Assess appetite**
  - **Inappetence/anorexia**
    - **Is weight loss $<2-3%/week$?**
      - **Yes**
        - Investigate causes of inappetence
      - **No**
        - **Excessive calorie loss?**
          - **Yes**
            - Reduce calorie loss and increase intake; dietary modification may be appropriate
          - **No**
            - **Adequate calorie intake?**
              - **Yes**
                - Continue with current diet
              - **No**
                - Investigate causes of inappetence
2.6 Diarrhoea; 2.7 Weight loss

Chronic

- Faecal examination and tests: selective culture, ZnSO₄ flotation, *Trichomonas foetus* (cat); anthelmintics; dietary trial

Acute

- Symptomatic treatment and supportive care

Further diagnostics: T₄, cTSH; ACTH stimulation test; FeLV/FIV; TLI; folate; B₁₂; pancreatic specific lipase

Inconclusive results

- Intestinal biopsies: endoscopic or surgical

Empiric therapy (often unrewarding)

- Exclude causes of hypoproteinaemia: UPC and bile acids

Empiric therapy (often unrewarding)

- Treat diagnosed cause

Empiric therapy (often unrewarding)

- Stop catabolic drugs especially corticosteroids if possible

Check diet is appropriate for life stage/lifestyle

- Correct inadequate calorie intake

Evidence of GIT disease?

- Catabolic states: increased metabolic rate due to endocrine (e.g. hyperthyroidism), neoplastic, congestive heart failure, inflammatory or infectious disease

Evidence of GIT disease?

- Investigation of GIT disease (see 3.4)

Yes

No

No

Evidence of GIT disease?

- Correct inadequate calorie intake

No

Check diet is appropriate for life stage/lifestyle

- Correct inadequate calorie intake

No

Evidence of GIT disease?

- Catabolic states: increased metabolic rate due to endocrine (e.g. hyperthyroidism), neoplastic, congestive heart failure, inflammatory or infectious disease

Evidence of GIT disease?

- Investigation of GIT disease (see 3.4)

Yes

Evidence of GIT disease?

- Correct inadequate calorie intake

No

Evidence of GIT disease?

- Catabolic states: increased metabolic rate due to endocrine (e.g. hyperthyroidism), neoplastic, congestive heart failure, inflammatory or infectious disease

Evidence of GIT disease?

- Investigation of GIT disease (see 3.4)

Yes

Evidence of GIT disease?

- Correct inadequate calorie intake

No

Evidence of GIT disease?

Note

- Look for calorie loss through urine (protein/glucose) or due to severe skin disease.
2.8 Polyuria/polydipsia

POLYURIA/ POLYDIPSIA

Does fluid intake exceed 80 ml/kg/day?§

No

PU and high urine SG

Yes

Check for use of diuretics, corticosteroids or recent change to dry diet

No

Evidence of pain, neurologic disease

No

Just PU

CBC; biochemistry (inc. bile acids) urinalysis; urine culture; $T_4$ (cat)

$T_4$/cTSH (dog); TLI

Test for HAC (dog); Urine Co:Cr ratio; ACTH stimulation test; LDDST; imaging

GFR

CT/MRI pituitary

Culture from renal pelvis

Modified water deprivation test†

• Nephrogenic DI
• Central DI
• Behavioural
• Neurologic/liver

ADH response test

Central diabetes insipidus (DI), but test can give false positive and negative results
2.8 Polyuria/polydipsia

Look for causes of incontinence, cystitis, or behavioural change

Look for other causes of fluid loss, e.g. fever, vomiting, diarrhoea, excessive panting, recent dehydration

Rule out:
- Diabetes mellitus
- Glycosuria
- Post-obstructive diuresis
- Infection (pyometra)*
- Renal disease*
- Hypokalaemia
- Hypercalcaemia
- Hyperthyroidism
- Pyelonephritis*
- Polycythaemia
- Liver disease

Rule out:
- Hypothyroidism
- Pancreatic insufficiency

Rule out:
- Hyperadrenocorticism (HAC)

Rule out:
- Compensated renal disease
- Pyelonephritis
- Pituitary disease*

Notes

- §Don’t forget to add water in wet food (75% of weight). Investigation is also justified if the owner has noticed a clear change in consumption.
- *Rule-out of these diseases is partial; further investigation is warranted if clinical suspicion remains.
- †Water deprivation tests are potentially dangerous and difficult to do effectively without considerable time input and nursing care.
2.9 Feline lower urinary tract disease

Approach to feline lower urinary tract disease (FLUTD)
- The majority (55–65%) of cases of FLUTD are idiopathic.
- These cases are best managed using a multimodal approach, involving changes to home environment, increasing water turnover through diet, and medication.
2.10 Urinary Incontinence

Note
- Be sure that is true incontinence; differentiate from inappropriate urination associated with behaviour, pollakiuria or PU/PD.
### CLINICAL PRESENTATION

#### 2.11 Renal disease

**Approach to renal disease**
- Differentiating pre-renal vs. acute failure vs. chronic renal disease can be difficult. Acute and chronic changes may co-exist, as animals with pre-existing renal disease are more at risk of developing acute failure as well.
- The signs or changes in laboratory parameters listed are generally the case, but exceptions do occur.
2.11 Renal disease

Acute on chronic renal failure

Acute renal failure (ARF)

Sudden increase in urea/creatinine in previously azotaemic case; rapid progression of signs; abnormal palpation/ultrasound; acute failure on CRD

Note
- *Suggests disease which interferes with ADH function, e.g. hypoadrenocorticism, hypercalcaemia, sepsis.
2.12 Pyrexia

**Approach to pyrexia of unknown origin**

- There is always pressure to treat a pyrexic case on presentation. However, watchful waiting and/or targeted investigation is frequently a better option.
- If the fever is high enough to require immediate treatment, use antipyretics rather than other treatments such as antimicrobials, which may interfere with subsequent investigation.
- Non-specific treatment with aspirin 10–25 mg/kg po q8–24hr (dog); 10 mg/kg po q48–72hr (cat); or paracetamol 10 mg/kg po q12hr (dog). Other NSAIDs have variable antipyretic effects.
- Ranking of causes of pyrexia in referred cases: immune mediated disease > localized/systemic infection > neoplasia.
- Approximately 15% of referred cases are idiopathic.

---

**Diagram Description**

- **<40°C; patient well**
  - Record temperature over 48 hr, observe

- **40–41°C; patient well**
  - Admit to hospital; observe over 24 hr, give antipyretics ± gastric protectant drugs

- **Patient unwell and/or >41°C**
  - Admit to hospital, obtain minimum database* and observe over 24 hr; give antipyretics

- **Recurrent pyrexia**
  - Database unremarkable, pyrexia resolving
  - Continue antipyretics and recheck in 48 hr
Pyrexia continues

- Treat cause
  - Idiopathic: empirical treatment

- Full investigation: serology, echocardiography, joint and bone marrow aspirates, CSF, advanced imaging, exploratory laparotomy, nuclear medicine

Pyrexia resolves

- No further action necessary

Pyrexia continues

- Antipyretic ± gastric protectant drugs; monitor for 48 hr

Further investigation:
- Fundic and neurologic examination, blood culture, thoracic and abdominal imaging, SPE and PLI, serology

Pyrexia still continuing

Notes
- *Full haematology, biochemistry, urine and faecal culture, FeLV/FIV.
- §Assuming no localizing signs have developed, in which case investigation should be focused on these.
- †For tick-borne disease, coronavirus, Toxoplasma, fungal disease (depending on location).
- ‡Informed owner consent is needed for trial with antimicrobials, corticosteroids, etc.
2.13 Anaemia

Approach to anaemia/pallor

- Differential diagnosis of pallor:
  - Hypovolaemia.
  - Shock.
  - Poor cardiac output.
  - High sympathetic tone.
  - Anaemia.

- Measure packed cell volume (PCV) to determine if anaemia is present and at a level to cause clinical signs: PCV <23% (dog); <18% (cat).
2.13 Anaemia

Notes
- Anaemia can have multiple causes in a particular disease.
- Features of regeneration include:
  - Increased reticulocyte count.
  - High MCV (especially dogs).
  - Anisocytosis and polychromasia.
2.14 Jaundice

Jaundice

PCV normal or near normal

Yes

Hepatobiliary disease

Laboratory results

Full haematology and biochemistry; FeLV; FIV; FIP; canine adenovirus; cPLI; abdominal imaging; hepatic biopsy; exploratory surgery

History and clinical signs

Hepatocellular Systemic disease precedes onset of jaundice; PU/PD; abdominal distension; encephalopathy; bleeding tendency

Obstructive Vomiting; diarrhoea; abdominal pain; pale, fatty stool

Hepatocellular (intrahepatic) cholestasis Treat primary cause of hepatic disease if possible; otherwise symptomatic and supportive

Obstructive (extra-hepatic) cholestasis May require surgery. Bile sludge; cholelithiasis; pancreatitis; mural duodenal disease; biliary tumour

Haemolysis (see 2.13)

No

Notes

- Abdominal imaging should include detailed ultrasound of the liver, gall bladder, extrahepatic bile duct, duodenal papilla and pancreas.
- Bile acids are unreliable in jaundiced patients.
- Cats with extrahepatic biliary obstruction do not always have dilation of the gall bladder.
3

BODY SYSTEMS AND MULTISYSTEMIC DISEASE

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3.1 **Eyes**

3.1.1 *Approach to diseases of the eye*

3.1.2 *Ophthalmic examination*

3.1.3 *Diagnostic tests and objectives*

3.1.4 *Topical ocular products*

3.1.5 *Role of nutrition*

3.1.6 *Diseases by anatomic area*

3.1.7 *Glaucoma*

**INTRODUCTION**

3.1.1 *Approach to diseases of the eye*

- Diseases of the eye are best evaluated by deciding which anatomic structures within the eye are affected.
- Many systemic, especially infectious diseases can present with ocular signs, e.g. uveitis as a result of FIP; or retinal haemorrhage associated with hypertension.

**HISTORY AND PHYSICAL EXAMINATION**

3.1.2 *Ophthalmic examination*

- **Technique**
  - Use a quiet, well-illuminated room which can be darkened.
  - Use bright focal light to examine the adnexa, anterior chamber and PLR. Magnification is preferable.
  - Dilate the pupil (mydriasis – use 1% tropicamide and allow 20 minutes) to allow fundic examination.

- **Direct ophthalmoscopy** (below left)
  - Distant direct – set at 0 or +1 looking for black opacities on the reflected path from the tapetum.
  - Close direct – set at -2 to +2; find and evaluate the optic disc and then the rest of the fundus. Then focus back through the anterior segment (lens about +10).

- **Indirect ophthalmoscopy**, in its simplest form, requires a light + hand lens (below right). Start with the lens close to the eye and withdraw until the image fills the field of view. Keep the lens at 90° to the light beam.

![Direct ophthalmoscopy](image1.png) **Direct ophthalmoscopy.** The ophthalmoscope is held close to the cornea and the angle adjusted to fully examine the retina.

![Indirect ophthalmoscopy](image2.png) **Indirect ophthalmoscopy.** The upper lid is held open, the lens is aligned and the hand and lens move with the head.
3.1 Eyes

DIAGNOSIS

Diagnostic tests and objectives

- **Schirmer tear testing**: place the short end of the tip in lateral half of lower conjunctival sac holding the eyelid closed. Measure over 1 minute. Cats and dogs should produce 12–27 mm, but breed variations do occur.
- **Fluorescein** demonstrates ulceration and nasolacrimal patency. Note that deep ulcers do not stain centrally; mucus on the corneal surface can give a ‘false positive’.
- **Blood tests** primarily serve to look for systemic conditions causing ocular signs.
- **Cytology and histology** can provide valuable information in diseases affecting the eyelid, conjunctiva and cornea.
- **Culture**: aerobic bacterial culture is appropriate for the majority of suspected infections. Special transport media are required for *Chlamydia phila* and *Mycoplasma*.
  - Fungal disease should be considered in animals that have travelled abroad.
- **Imaging**: ultrasound, CT and MRI are valuable for investigating disease of the deeper structures of the eye, retrobulbar area and optic nerve.
  - Radiographs have limited value, except to detect radiopaque foreign bodies and diseases affecting the orbital bone.
- **Electroretinography** evaluates retinal function but requires general anaesthesia.

TREATMENT

Topical ocular products

- Provided the patient is amenable, topical therapy (see table, next page) is the route of choice for diseases of the anterior segment of the eye, as it maximizes exposure to the drug while minimizing systemic side-effects.

Lacrimal flushing

- In many cases, lacrimal flushing via the upper (dorsal) punctum can be undertaken with the patient conscious, or sedated rather than anaesthetized. Fine nasal cannulae (23–24 g) are needed.
3.1.5 Role of nutrition

- Nutrition does not play a crucial role in the genesis or management of ocular disease. Notable exceptions would include taurine deficiency, vitamin E deficient retinopathy and lipaemia retinalis.
- Lysine has been recommended as adjunctive treatment for chronic FHV-1; efficacy has not been shown.

3.1.6 Diseases by anatomic area

- See table, right and page 62. Note that injury/trauma, foreign bodies and neoplasia can affect all areas.

3.1.7 Glaucoma

- **Signs** include:
  - Acute: pain, corneal oedema or vascularization, unresponsive pupil, episcleral congestion, vision loss.
  - Chronic: globe enlargement, corneal ulceration and neovascularization, scleral thinning, iris atrophy, lens luxation, cataract, haemorrhage, blindness.
Glaucoma is classified as primary in a number of dog breeds, or secondary to other ocular pathology, such as lens luxation, uveitis, intraocular haemorrhage, ocular melanosis, or swollen lens, e.g. in diabetes.

**Therapy**: lower the intraocular pressure to <20 mmHg using surgical or medical therapy, e.g. osmotic diuretics, carbonic anhydrase inhibitors, miotics, β-adrenergic blockers.

---

### OCULAR DISEASES BY ANATOMIC AREA

<table>
<thead>
<tr>
<th>Area</th>
<th>Defect</th>
<th>Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Eyelid and nictitating membrane</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Congenital</td>
<td>Coloboma, epibulbar dermoid</td>
<td></td>
</tr>
<tr>
<td>Eyelid position</td>
<td>Entropion (can be breed-related), ectropion, e.g. diamond eye in Clumber spaniels</td>
<td></td>
</tr>
<tr>
<td>Ptosis</td>
<td>Defect CN III or Horner’s syndrome</td>
<td></td>
</tr>
<tr>
<td>Blepharitis</td>
<td>Infection/inflammation of eyelid</td>
<td></td>
</tr>
<tr>
<td>Distichiasis</td>
<td>Ectopic cilia, trichiasis (facial hair on ocular surface) e.g. nasal fold in brachycephalic dogs</td>
<td></td>
</tr>
<tr>
<td>Eosinophilic plaque (cat)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3rd eyelid</td>
<td>Scrolling, prolapse, inflammation, protrusion (suggests underlying disease), neoplasia, diarrhoea-associated (cats)</td>
<td></td>
</tr>
<tr>
<td><strong>Conjunctiva</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Primary</td>
<td>Viral, irritant, allergic, immune-mediated FHV-1, FCV (cat) Chlamyphila felis</td>
<td></td>
</tr>
<tr>
<td>Secondary</td>
<td>Bacterial, mechanical</td>
<td></td>
</tr>
<tr>
<td>Symblepharon – adhesion to itself/cornea, common in cats</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Cornea</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Congenital</td>
<td>Dermoid, opacity, microcornea</td>
<td></td>
</tr>
<tr>
<td>Dystrophy – thinning of the cornea</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Keratitis</td>
<td>Often ulcerative associated with eyelid abnormalities, immune-mediated, tear film, infection</td>
<td></td>
</tr>
<tr>
<td>Pannus (chronic superficial keratoconjunctivitis)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Herpetic keratitis (cat)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
## OCULAR DISEASES BY ANATOMIC AREA (continued)

<table>
<thead>
<tr>
<th>Area</th>
<th>Defect</th>
<th>Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cornea</td>
<td>Exposure – loss or incomplete blink</td>
<td>CNV, anaesthesia</td>
</tr>
<tr>
<td></td>
<td>Corneal sequestrum</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Eosinophilic (proliferative) keratoconjunctivitis</td>
<td></td>
</tr>
<tr>
<td>Uveal tract</td>
<td>Developmental</td>
<td>Deficiency in pigmentation, coloboma and uveal cysts, persistence of embryonic pupillary membrane</td>
</tr>
<tr>
<td></td>
<td>Benign melanosis (dog) – foci of pigment</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Synechiae: adhesions iris to cornea or lens</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Uveitis: pain, photophobia, lacrimation</td>
<td>Infectious, e.g. FIP, parasitic, traumatic, immune mediated, lens-induced, intraocular foreign body</td>
</tr>
<tr>
<td></td>
<td>Neoplasia</td>
<td>Melanoma, lymphoma</td>
</tr>
<tr>
<td></td>
<td>Lipaemic aqueous (check serum triglycerides)</td>
<td></td>
</tr>
<tr>
<td>Lens</td>
<td>Congenital</td>
<td>Missing/small, coloboma, embryonic remnants</td>
</tr>
<tr>
<td></td>
<td>Nuclear sclerosis (age-related)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Cataract</td>
<td>Secondary to other ocular disease, trauma, diabetes, hypocalcaemia, toxic, senile</td>
</tr>
<tr>
<td></td>
<td>Luxation</td>
<td>Inherited (terriers), glaucoma, cataract, uveitis</td>
</tr>
<tr>
<td>Retina*</td>
<td>Normal</td>
<td>Significant variation</td>
</tr>
<tr>
<td></td>
<td>Heritable disease</td>
<td>Collie eye anomaly, retinal dysplasia</td>
</tr>
<tr>
<td></td>
<td>Acquired disease</td>
<td>Generalized progressive retinal atrophy, sudden acquired retinal degeneration, retinal detachment, lipaemia, taurine deficiency (cat) chorioretinitis (often indicates systemic disease, e.g. FIP, Toxoplasma, Ehrlichia, Aspergillus)</td>
</tr>
</tbody>
</table>

*Assess tapetal reflectivity, changes in pigmentation, optic disc, vasculature and haemorrhage
INTRODUCTION

- Dental disease is the most common abnormality found on physical examination of cats and dogs.
- Association has been made between the level of periodontal disease and both chronic renal disease and heart disease. This is presumed to be due to chronic bacteraemia and inflammation.

HISTORY AND PHYSICAL EXAMINATION

Dental examination

- The following areas should all be examined:
  - Oropharynx: palate, tonsils, fauces.
  - Lips and cheeks: mucocutaneous junction, philtrum, frenula, salivary papilla.
  - Oral mucosa: buccal, lingual, gingival surfaces.
  - Hard palate.
  - Floor of mouth and tongue, lingual frenulum.
  - Teeth: life stage, missing/extra teeth, abnormalities of individual teeth, wear patterns, pathological changes, e.g. caries, fracture.

- Full oral examination requires the following:
  - Time to record findings using a standard dental chart, so that severity and change over time may be assessed.
  - General anaesthesia, in most patients.
  - Screening blood tests are often recommended prior to general anaesthesia, as many patients will be elderly.

It is important that if screening blood tests are undertaken a clear action plan is available in response to any abnormalities found.
DIAGNOSIS

3.2.3 Diagnostic tests
- Thorough visual examination and probing in good lighting, together with dental radiographs, are the key diagnostic tools.
- Although general skull radiographs can be used to examine the teeth, it is difficult to separate the arcades adequately. Intraoral dental films give much greater detail.
- Viral screening (FIV, FCV) should be considered in cats.

3.2.4 Dental score charts
- On a dental record card, record evidence of:
  - Gingivitis.
  - Gingival index.
  - Gingival recession.
  - Loss of tissue between multi-rooted teeth (furcation).
  - Tooth mobility.
  - Degree of tooth attachment.

3.2.5 Staging periodontal disease
- Periodontal disease results from plaque-induced inflammation. The vast majority of cats and dogs over 3 years of age have a degree of disease which warrants intervention.
- The earliest stage of periodontal disease, gingivitis, is reversible. Once there is bone loss, changes become irreversible. The stages are defined as:
  - Gingivitis: inflammation limited to gingiva.
  - Gingival hyperplasia: increased periodontal probe depth but no loss of periodontal support.
  - Periodontitis with vertical bone loss: early loss of periodontal support and destruction of alveolar bone, root not yet exposed.
  - Periodontitis with horizontal bone loss: loss of periodontal ligament and exposure of the root.

TREATMENT

3.2.6 Management of dental disease
- The most important element is the maintenance of oral hygiene – tooth brushing (q24hr with toothpaste), dental diets and dental chews and oral antiseptics (topical chlorhexidine).
- Cats, unlike dogs, can develop severe gingivostomatitis that is intractable to medical and periodontal therapy, leading to extraction of all premolars and molars, and ultimately the canines too.
Therapy for periodontal disease includes:
- Supragingival scaling and polishing.
- Subgingival scaling, root planing and polishing.
- Sulcular lavage.
- Periodontal surgery: gingivoplasty, tooth removal.

Tooth extraction
- Indications: advanced periodontitis, advanced destruction of dental hard tissue including feline odontoclastic resorptive lesions (FORL), persistent primary teeth, malocclusion, traumatic tooth injury, overcrowding.
- **Techniques**: be sure to use sharp, well-maintained instruments of the correct size.
  - **Closed**: luxation and elevation, socket heal by granulation.
  - **Open**: mucoperiosteal flap to expose buccal surface of tooth. Indicated if bizarre root morphology, extensive root resorption or ankylosis, sound upper/lower canines or multi-rooted teeth, retained root remnants.
    - Cats’ teeth (especially with FORL) fracture easily, so open extraction is preferable unless there is severe periodontitis.
    - Multi-rooted teeth should be sectioned into single roots. There are three roots in the maxillary 4th premolar (cat/dog), and 1st and 2nd molar (dog).
  - Always cut gingival attachment first, by scalpel or sharp luxator.
  - Luxate the tooth: the concave surface should approximate the curve of the root; advance into sulcus and work around the tooth, applying gentle apical pressure.
  - Once there is sufficient space, work an elevator around in a similar fashion with gentle rotational pressure.
  - Remove the tooth with fingers or forceps.
  - Retained roots: radiograph to assess the amount of root left and then decide on whether open extraction is required. Small retained tips can be atomized. Clients should be informed if roots cannot be removed.

**NUTRITION**

**Nutrition and dental disease**
- The relationship between processed (especially wet) pet foods and dental disease is the subject of much debate but few good studies.
- When choosing a diet, all the health needs of the patient should be considered, together with owner factors such as their ability to perform routine oral health care.
- Some products are marketed as main diets and treats aimed at reducing tartar build-up.
3.2.8 Equipment

- Good dental care does not necessarily require expensive equipment, but an appropriately-sized selection of well-maintained, sharp hand tools is essential (see above).

3.3 Heart and lungs

3.3.1 Prevalence and causes of cardiorespiratory disease
3.3.2 Checklists  
3.3.3 Diagnostic tests and their objectives
3.3.4 Thoracic radiography: lungs  
3.3.5 Thoracic radiography: heart
3.3.6 Drug therapy  
3.3.7 Nutritional management in cardiac disease
3.3.8 Blood pressure

INTRODUCTION

3.3.1 Prevalence and causes of cardiorespiratory disease

- Between 15% and 20% of sick cats and dogs presenting to veterinary surgeons have cardiorespiratory disease.
- Congenital cardiac disease in dogs is relatively common, affecting between 0.5% and 1% of puppies; congenital respiratory disease is rare.
- Primary bacterial causes of respiratory disease are uncommon and the presence of bacteria suggests another underlying condition that has damaged the defence system.
- For approaches to common presenting signs of cardiorespiratory disease, see 2.1 Sneezing and nasal discharge; 2.2 Coughing; 2.3 Dyspnoea.

HISTORY AND PHYSICAL EXAMINATION

3.3.2 Checklists

- On history and physical examination, there may not be a clear separation between cardiac and respiratory disease, especially in cats.
- In such cases, the history and physical examination pointers for both body systems should be used.
**History: cardiac disease**
- Recent changes in attitude, activity or exercise tolerance.
- Changes in respiratory rate/effort noticed at rest.
- Excessive panting at exercise: is there an association with degree of exercise and weather?
- Falling over or weakness: what had the patient just done, or was doing at the time?
- Whether the patient is coughing: type, timing, progression, description.
- Check colour of mucous membranes, particularly at exercise.

**History: respiratory disease**
- History should be focused on differentiating between upper and lower respiratory tract disease – see 2.1, 2.2 and 2.3.

**Physical examination: cardiac disease**
Abnormalities in the following would tend to indicate the presence of cardiovascular disease. You should record:
- Regularity of rate and rhythm.
- Is heart beat *regularly or irregularly* irregular?
- Synchrony of heart beat with pulse.
- Pulse quality.
- Mucous membrane colour (if pale, see 2.14).
- Presence of heart murmur; focus on:
  - Systolic or diastolic – all, or part of phase.
  - Point of maximum intensity.
  - Radiation.
  - Loudness (see table, below).
  - Shape, quality and pitch.

**HEART MURMUR: GRADES OF LOUDNESS**

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grade I</td>
<td>Quiet: need good listening conditions to hear</td>
</tr>
<tr>
<td>Grade II</td>
<td>Soft but easily heard</td>
</tr>
<tr>
<td>Grade III</td>
<td>Moderate: similar to intensity of normal heart sounds</td>
</tr>
<tr>
<td>Grade IV</td>
<td>Loud: louder than intensity of normal heart sounds</td>
</tr>
<tr>
<td>Grade V</td>
<td>As IV but with precordial thrill</td>
</tr>
<tr>
<td>Grade VI</td>
<td>Very loud, precordial thrill, heard away from body wall</td>
</tr>
</tbody>
</table>
Physical examination: respiratory disease

- Observe rate, pattern and character of breathing.
- Examine nasal area for symmetry, hair discoloration and nasal discharge.
- Check air flow from both nares.
- Palpate the upper respiratory tract and thorax (for integrity, compressibility and position of the apex beat).
- Gently squeeze the larynx to assess cough response.
- Auscultate the thorax and trachea for the location, intensity and normality of breathing and cardiac sounds.
- Percuss the thorax for increased (air) and decreased (fluid, soft tissue) resonance.

DIAGNOSIS

3.3.3 Diagnostic tests and their objectives

- Routine haematology and biochemistry are often of limited value.
- Infectious disease testing should be considered: FeLV, FIV, FHV-1, FCV.
- Cardiac troponin I: looking for ongoing myocardial damage.
- NT-proBNP: looking for left ventricular stretch and overload; differentiating heart failure from respiratory disease as a cause of dyspnoea (see table below).
  - Check appropriate sample type and handling before sending.
- Electrocardiography: ECG is best at assessing rhythm and type of rhythm disturbance; see 1.3.8, 1.3.9.
- Blood pressure: see 3.3.8.
- Radiography: best used for nasal, lung and pleural disease; heart size; see 1.3.10.
- CT is better than radiography for nasal and lung disease.
- Echocardiography for myocardial and valvular function, congenital heart disease, pleural fluid and mediastinal disease; see 1.3.14.
- MRI is of limited value in cardiac and lower respiratory tract disease.

<table>
<thead>
<tr>
<th>NT-proBNP RESULTS: INTERPRETATION</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Interpretation</strong></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>------------------------------</td>
</tr>
<tr>
<td>Normal – heart disease unlikely</td>
</tr>
<tr>
<td>Elevated – heart disease possible</td>
</tr>
<tr>
<td>Heart volume overload likely</td>
</tr>
<tr>
<td>Congestive heart failure</td>
</tr>
</tbody>
</table>
Common lung patterns

- There are 3 common lung patterns. Although often mixed, there is usually one pattern that is dominant and can be used to construct a differential diagnosis list.
- **Alveolar pattern** (above left) is caused by filling of the alveoli with fluid or cells, resulting in a ‘fluffy’ appearance that is more dense towards the centre of the area, with an ill-defined edge as the affected lung merges into normal lung. In extreme cases, where all the alveoli are filled, an air bronchogram is created.
- **Bronchial pattern** (above right) occurs when the bronchial wall is prominent (inflammation, fibrosis, calcification) or there is peribronchial infiltration.
  - End on, bronchi appear as ‘doughnuts’ or ‘signet rings’; longitudinal airways are described as ‘tramlines’.
  - In acute bronchial disease the edge of the wall tends to be thicker, less radiodense and less well defined.
  - In chronic disease the wall tends to be brighter and more sharply defined.
- **Interstitial patterns** are caused by diseases that affect the support tissue of the lungs. This is seen either as a diffuse ‘chicken-wire’ appearance, where the general lung density is increased and structures such as the blood vessels become less distinct, or as a nodular pattern such as occurs with metastatic disease.

Radiographic signs of pleural disease

- Free pleural fluid usually causes scalloping of the ventral lung fields on a lateral view (LV) and separation of the lung from the body wall by a soft-tissue density on a dorsal view (DV).
- Pneumothorax causes the lungs to collapse centrally, so dense soft tissue is surrounded by black air that, even on bright light, has no lung structures within.
- Finding abdominal contents in the pleural space indicates ruptured diaphragm.
3.3.5 Thoracic radiography: heart

**Common heart changes**

- **Heart size** can be assessed in a variety of ways:
  - 2.5–3.5 rib spaces and two-thirds thoracic height on lateral.
  - Two-thirds thoracic width at the 5th intercostal space on the DV.
  - Vertebral heart score. Long- and short-axis heart dimensions are transposed onto the vertebral column and recorded as the number of vertebrae, beginning with the cranial edge of T4; the vertebral heart score is the sum of these values (see radiograph, below) and is normally 8.5–10.5 (dog); 6.7–8.5 (cat).
  - These criteria are not perfect: use judgement.
  - Note that heart shape varies with breed and a quick glance can be misleading, e.g. the heart always looks big in small terrier breeds; Labradors often have a rounded heart shape.
  - Small heart: consider dehydration, over-inflation of lungs, shock, pneumothorax, obstruction to venous flow.
  - Normal heart: consider atrial or small ventricular septal defect, mild valvular or myocardial disease, early bacterial endocarditis, constrictive pericarditis, neoplasia, congestive heart failure on diuretics.
  - Large heart: consider volume overload associated with severe valvular disease or myocardial failure, marked HCM, pericardial effusion.
  - **Increased chamber size**
    - Left atrial enlargement is usually seen as a straight caudal border to the heart (dog) and increased density at the carina due to volume overload and is often associated with pulmonary oedema.
    - Right side enlargement causes increased sternal contact, secondary to tricuspid/pulmonic valvular disease, pulmonary hypertension.

![Vertebral heart score](image)
**Pulmonary vasculature**

- Cardiovascular disease may lead to changes in pulmonary vasculature. Assessment criteria are:
  - In a normal individual arteries and veins are of a similar diameter.
  - Lateral view: arteries lie dorsally, and veins lie ventral to the bronchus. Width of cranial pulmonary vessels normally less than width of the proximal third of rib 4.
  - Dorsoventral view: the arteries lie lateral to the vein. Normally less than the diameter of rib 9 as the vessel crosses the ribs.

**TREATMENT**

**Drug therapy**

- **Cardiac disease**: see table, page 72. Therapy for cardiac disease should be directed as far as possible at the cause of reduced cardiac output.
- Many drugs can be hypotensive, so take care when starting multiple therapies at the same time.
- **Coughing and dyspnoea**: see table, page 74. Successful treatment is very dependent on an appropriate diagnosis. Therapy based on clinical signs will often fail to produce sustained improvement in chronic disease.

**NUTRITION**

**Nutritional management in cardiac disease**

- See table below for general recommendations. The true value of dietary intervention has been difficult to document.

### NUTRITIONAL MANAGEMENT IN CARDIAC DISEASE

<table>
<thead>
<tr>
<th>Asymptomatic disease</th>
<th>Mild–moderate disease</th>
<th>Severe disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild sodium restriction</td>
<td>Moderate sodium restriction</td>
<td>Increased sodium restriction</td>
</tr>
<tr>
<td>Maintain optimal BCS</td>
<td>Maintain optimal BCS</td>
<td>Enhance palatability to maintain BCS</td>
</tr>
<tr>
<td>n-3 PUFA</td>
<td>n-3 PUFA Maintain serum magnesium and potassium levels in reference range</td>
<td>n-3 PUFA Benefits of taurine, carnitine, co-enzyme Q, B-vitamins and L-arginine supplementation, where specific deficiency has not been documented, are unknown</td>
</tr>
</tbody>
</table>

BCS: body condition score
PUFA: polyunsaturated fatty acid
<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose rate (dog)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Furosemide</strong>§ (diuretic)</td>
<td>Acute pulmonary oedema 1–2 mg/kg q2–6 hrs i/v or i/m until stable. Can be given s/c. Oral maintenance therapy 1–4 mg/kg q12–24hr</td>
</tr>
<tr>
<td>50 mg/ml inj; 20/40 mg T; 4–10 mg/ml S (NL)</td>
<td></td>
</tr>
<tr>
<td><strong>Spironolactone</strong>* (diuretic)</td>
<td>2–4 mg/kg orally once daily</td>
</tr>
<tr>
<td>10, 40, 80 mg T; 20 mg/ml S (NL)</td>
<td></td>
</tr>
<tr>
<td><strong>ACE inhibitors</strong></td>
<td></td>
</tr>
<tr>
<td>Benazepril* 5, 20 mg T</td>
<td>0.25–0.5 mg/kg q24hr</td>
</tr>
<tr>
<td><strong>Enalapril</strong>§</td>
<td>0.25–1 mg/kg q12–24hr</td>
</tr>
<tr>
<td>1, 2.5, 5, 10, 20 mg T</td>
<td></td>
</tr>
<tr>
<td><strong>Imidapril</strong>§</td>
<td>0.25 mg/kg q24hr</td>
</tr>
<tr>
<td>75, 150, 300 mg powder</td>
<td></td>
</tr>
<tr>
<td><strong>Ramipril</strong>* 1.25, 2.5, 5 mg T</td>
<td>0.125 mg/kg q24hr</td>
</tr>
<tr>
<td><strong>Pimobendan</strong>* (inodilator)</td>
<td>0.1–0.3 mg/kg q12hr one hour before food</td>
</tr>
<tr>
<td>1.25, 2.5, 5 mg T</td>
<td></td>
</tr>
<tr>
<td><strong>Diltiazem</strong>§</td>
<td>0.5–2.0 mg/kg po q8hr</td>
</tr>
<tr>
<td>(calcium channel blocker)</td>
<td></td>
</tr>
<tr>
<td>10 mg T (60 mg T [NL])</td>
<td></td>
</tr>
<tr>
<td><strong>Digoxin</strong></td>
<td>0.22 mg/m² po q12hr</td>
</tr>
<tr>
<td>0.0625, 0.125, 0.25 mg T; 50 μg/ml S</td>
<td></td>
</tr>
<tr>
<td><strong>Beta adrenergic blockers</strong></td>
<td>0.5–2 mg/kg po q12hr</td>
</tr>
<tr>
<td>Atenolol</td>
<td></td>
</tr>
<tr>
<td>25, 50, 100 mg T; 5 mg/ml S</td>
<td></td>
</tr>
<tr>
<td><strong>Propranolol</strong></td>
<td>0.02–0.08 mg/kg slow i/v over 5 min q8hr,</td>
</tr>
<tr>
<td>1 mg/ml inj; 10, 40, 80, 160 mg T</td>
<td>0.25–1.5 mg/kg po q8hr</td>
</tr>
<tr>
<td>(Sotalol, metoprolol,</td>
<td></td>
</tr>
<tr>
<td>carvedilol)</td>
<td>See formulary for dose rates</td>
</tr>
</tbody>
</table>

T = tablet; S = syrup; inj = injectable
* Licensed for use in dogs; § licensed for use in cats (UK); other drugs are used off-licence; NL = non-licensed
### 3.3 Heart and lungs

<table>
<thead>
<tr>
<th>Dose rate (cat)</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute pulmonary oedema 1–2 mg/kg q4–8 hrs i/v or i/m until stable. Can be given s/c. Oral maintenance therapy 0.5–2 mg/kg q8–24hr</td>
<td>Use lowest dose possible. If high doses (&gt;2–4 mg/kg/day) consider additional diuretic. Continuous rate infusion (0.1–0.5 mg/kg/hr) in acute cases may be better than pulse-dosing</td>
</tr>
<tr>
<td>2–4 mg/kg orally once daily</td>
<td>Potassium-sparing diuretic, synergistic with furosemide; rarely sufficient alone</td>
</tr>
<tr>
<td>0.25–0.5 mg/kg q24hr</td>
<td>All have similar action and act as a balanced vasodilator, reduce renin–angiotensin–aldosterone system activation and improve vascular remodelling. Liver/kidney excretion varies between agents. Tend to reduce blood pressure: use with care in hypovolaemic or hypotensive cases</td>
</tr>
<tr>
<td>0.25 mg/kg q24hr</td>
<td></td>
</tr>
<tr>
<td>0.125 mg/kg q24hr</td>
<td></td>
</tr>
<tr>
<td>1.25 mg/cat q12hr one hour before food</td>
<td>Positive inotrope sensitizing the myocardium to calcium, without significant increase in oxygen demand. Vasodilation mediated by phosphodiesterase III activity</td>
</tr>
<tr>
<td>0.5–2.5 mg/kg po q8hr</td>
<td>Slow-release tablets have been used, but pharmokinetics are not defined and may have hepatotoxicity in cats. Acts to interfere with calcium movement and has a negative inotropic activity; retards atrio-ventricular conduction and dilation of the large arteries</td>
</tr>
<tr>
<td>10 μg/kg po q24–48hr [rarely used in cats]</td>
<td>Unlikely to have significant positive inotropic activity. Most useful in slowing heart rate in atrial fibrillation. Narrow therapeutic index; serum monitoring advised after 5–7 days use</td>
</tr>
<tr>
<td>6.25–12.5 mg/cat po q24hr</td>
<td>Negative chronotropic and inotropic activity in cardiac arrhythmia (both supraventricular and ventricular). Use in congestive heart failure controversial; requires very careful patient selection and introduction</td>
</tr>
<tr>
<td>0.04–0.06 mg/kg slow i/v over 5 min q8hr, 2.5–5 mg/kg po q8hr</td>
<td></td>
</tr>
<tr>
<td>See formulary for dose rates</td>
<td>See formulary for contraindications and side-effects</td>
</tr>
<tr>
<td>Drug</td>
<td>Dose rate (dog)</td>
</tr>
<tr>
<td>---------------------------</td>
<td>--------------------------------------------------------------------------------</td>
</tr>
<tr>
<td><strong>Mucolytic</strong></td>
<td></td>
</tr>
<tr>
<td>Bromhexine*§ (1% powder)</td>
<td>2–2.5 mg/kg po q12hr</td>
</tr>
<tr>
<td><strong>Cough suppressant opiates</strong></td>
<td></td>
</tr>
<tr>
<td>Morphine (wide variety of formulations and strengths)</td>
<td>Injectable: 0.5 mg/kg s/c q6–8hr</td>
</tr>
<tr>
<td>Codeine</td>
<td>3 mg/ml S; 15, 30, 60 mg T</td>
</tr>
<tr>
<td>Butorphanol*</td>
<td>10 mg/ml inj; 5, 10 mg T</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Bronchodilators</strong></td>
<td></td>
</tr>
<tr>
<td>Etamiphylline*§ 140 mg/ml inj; 100, 200, 300 mg T</td>
<td>15 mg/kg s/c, i/m q8hr; 10–20 mg/kg po q8hr</td>
</tr>
<tr>
<td>Theophylline*</td>
<td>100, 200, 500 mg SR capsule</td>
</tr>
<tr>
<td>Terbutaline</td>
<td>0.3 mg/ml S; 5 mg T</td>
</tr>
<tr>
<td><strong>Antihistamines</strong></td>
<td></td>
</tr>
<tr>
<td>Many types, e.g. chlorphenamine</td>
<td>4–8 mg/dog po q8–12hr</td>
</tr>
<tr>
<td>10 mg/ml inj; 4 mg T; 0.4 mg/ml S</td>
<td></td>
</tr>
<tr>
<td>Clemastine</td>
<td>1 mg T</td>
</tr>
<tr>
<td><strong>Anti-inflammatories</strong></td>
<td></td>
</tr>
<tr>
<td>Prednisolone</td>
<td>0.5 mg/kg po – anti-inflammatory 1–2 mg/kg po – immunosuppressive</td>
</tr>
<tr>
<td>NSAIDs</td>
<td>see 4.2.4 for options and dose rates</td>
</tr>
<tr>
<td><strong>Antimicrobials</strong></td>
<td></td>
</tr>
<tr>
<td>see 3.13.4 for appropriate antimicrobial choice</td>
<td></td>
</tr>
<tr>
<td><strong>Inhaled therapy</strong></td>
<td></td>
</tr>
<tr>
<td>Other corticosteroid-based inhalers available</td>
<td>50–250 μg q12–24hr</td>
</tr>
<tr>
<td>Fluticasone propionate (Flixotide)</td>
<td></td>
</tr>
<tr>
<td>Salbutamol (Ventolin)</td>
<td>100 μg q4–6hr</td>
</tr>
</tbody>
</table>

T = tablet; S = syrup; SR = slow release; inj = injectable
s/c = subcutaneous, i/m = intramuscular; i/v = intravenous
* Licensed for use in dogs; § licensed for use in cats
## 3.3 Heart and lungs

<table>
<thead>
<tr>
<th>Dose rate (cat)</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 mg/kg po q24hr</td>
<td>Bronchial secretolytic making expectoration easier; efficacy difficult to assess</td>
</tr>
<tr>
<td>Injectable: 0.5 mg/kg sc q6–8h</td>
<td>Can cause sedation, respiratory depression and constipation; contra-indicated if coughing productive</td>
</tr>
<tr>
<td>0.3–0.5 mg/kg po q8–12hr</td>
<td></td>
</tr>
<tr>
<td>0.05–0.1 mg/kg s/c, i/m, i/v q6–12hr; 0.5–1 mg/kg po q6–8hr</td>
<td></td>
</tr>
<tr>
<td>15–25 mg/kg s/c, i/m q8hr; 100 mg/cat po q8hr</td>
<td>CNS and cardiovascular stimulation, mild diuretic action</td>
</tr>
<tr>
<td>20–25 mg/kg po q24hr</td>
<td></td>
</tr>
<tr>
<td>0.3–1.25 mg/kg po q8–12hr</td>
<td></td>
</tr>
<tr>
<td>2–4 mg/cat po q8–12hr</td>
<td>Side-effects depend on drug used; can cause sedation and other anticholinergic activity; efficacy in allergic respiratory disease often poor. Second-generation antihistamines have fewer side-effects but have been less used in dogs and cats</td>
</tr>
<tr>
<td>0.05–0.1 mg/kg po q12hr</td>
<td></td>
</tr>
<tr>
<td>Prednisolone (or other corticosteroids) useful in allergic and inflammatory airway disease, but can cause muscle wasting and tachypnoea; <strong>contraindicated</strong> in bacterial pneumonia and viral respiratory disease</td>
<td></td>
</tr>
<tr>
<td>NSAIDs useful in some nasal disease, viral upper respiratory tract disease; <strong>contraindicated</strong> in bronchoconstriction</td>
<td></td>
</tr>
<tr>
<td><em>Bordetella, Pseudomonas</em> and <em>Streptococcus zooepidemicus</em> are the primary bacterial invaders of the respiratory tract</td>
<td></td>
</tr>
<tr>
<td>50–250 μg q12–24hr</td>
<td>Spacing device required – if multiple actuations required, should be given as one dose multiple times. More expensive than oral therapy, but fewer side-effects in long-term use. Salbutamol can cause tachycardia, tremor and CNS stimulation</td>
</tr>
<tr>
<td>100 μg q4–6hr</td>
<td></td>
</tr>
</tbody>
</table>
### SELECTED TOPIC

#### 3.3.8 Blood pressure

- Both hypertension and hypotension can cause life-threatening disease.
- **Measurement**: normal systolic BP measured by Doppler is 120–160 mmHg. Oscillometric methods are less reliable in conscious patients, and tend to underestimate BP, if a reading is possible at all.
- Note that BP measurements in conscious animals are inherently unreliable: the ‘white coat effect’ can be as much as 50 mmHg, especially in cats.
- In anaesthetized/recumbent animals, BP trends are more important than absolute values (unless extreme).
- **Diagnosis** of hypertension should therefore only be made after multiple measurements, on more than one occasion, show high BP.
- If hypertension is noted, the patient should also be examined for evidence of clinical effects such as:
  - Retinopathy or intraocular haemorrhage.
  - Chronic or acute renal failure.
  - Hyperadrenocorticism, diabetes mellitus, or hyperthyroidism.
  - Unexplained neurologic signs.
  - Left ventricular hypertrophy.
**Approach** to hypertension: see the algorithm below. This is based on systolic BP measured by the Doppler method. Ideally, diagnosis is based on multiple measurements obtained at more than one visit.

**Management:** hypertension can be managed by oral therapy as follows:

- **Cat:**
  - Amlodipine 0.625–1.25 mg/cat q24hr–q12hr; or
  - Hydralazine 2.5–5 mg/cat q24hr–q12hr.

- **Dog:**
  - Amlodipine 0.05–0.1 mg/kg q24hr–q12hr;
  - ACE inhibitors; or
  - Atenolol 0.5–2 mg/kg q12hr.

---

### BLOOD PRESSURE REFERENCE RANGES

<table>
<thead>
<tr>
<th>mmHg</th>
<th>Systolic</th>
<th>Diastolic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Doppler</td>
<td>(Dog and cat)</td>
<td>(Dog and cat)</td>
</tr>
<tr>
<td></td>
<td>120–160</td>
<td>80–120</td>
</tr>
<tr>
<td>(Oscillometric)</td>
<td>Cat: 124 av.</td>
<td>Cat: 84 av.</td>
</tr>
</tbody>
</table>

---

- Begin antihypertensive therapy immediately; treat underlying disease
- Treat underlying disease if present, otherwise monitor BP
- Repeat testing
- BP consistently >180/120: trial antihypertensive therapy
- Evaluate BP every 3–6 months
- No further testing indicated

---
3.4 Digestive tract

3.4.1 Acute/chronic gastrointestinal disease
3.4.2 Key questions and examination tips
3.4.3 Preliminary diagnostic tests
3.4.4 Abdominal imaging
3.4.5 Drug treatment
3.4.6 Dietary approach to intestinal disease
3.4.7 Pancreatitis

INTRODUCTION

3.4.1 Acute/chronic gastrointestinal disease

- GIT disease presents in both acute and chronic forms; for approaches see 2.4, 2.5, 2.6 and 2.12.
- With acute disease, the key question is whether the problem is self-limiting. For such cases there will be a short history and a known exciting cause; the patient will be systemically well with good appetite, and there will be negative physical findings. However, if the problem is severe, the patient is usually unwell with positive physical findings indicative of GIT disease; such cases require immediate investigation.
- For chronic or recurrent disease the key is to follow a logical path of investigation and treatment (preferably monotherapy) so that effective management is eventually achieved.

HISTORY AND PHYSICAL EXAMINATION

3.4.2 Key questions and examination tips

- History is crucial, especially in chronic disease where there may be minimal physical findings. Description of the signs when the disease first began is often the most helpful.
- In patients that are ‘vomiting’, it is essential to be certain the owner is not describing regurgitation, gagging, dysphagia, expectoration or retching. On rare occasions, patients will show a combination of signs.
- Regurgitated material (alkaline) can be distinguished from vomitus (acidic) by measuring the pH, using litmus paper.
- In vomiting cases, the relationship to feeding and the nature of the vomitus are important.
- Physical examination: improved access to the abdomen can sometimes be achieved by raising the front legs.
- Limited information can be gained from large breed and fat dogs.

The intestine of very thin animals can often feel abnormally prominent, but in many cases this is a normal finding due to the lack of abdominal fat.
**Faecal score chart**: Can be useful for in-hospital description by nurses, and for home use by owner.

**Small- vs large-bowel diarrhoea**: Small-intestinal bacterial overgrowth can cause large-intestinal signs. Localization of disease according to faecal characteristics is less reliable in cats.

- Small intestinal diarrhoea is characterized by increased fatty stools, melaena and undigested food. Large bowel diarrhoea is characterized by increased frequency, urgency, tenesmus, mucus and fresh blood.

### DIAGNOSIS

#### Preliminary diagnostic tests

- Full faecal examination is essential in cases of chronic diarrhoea.
- Routine haematology and biochemistry are of limited value in chronic disease, but important in acute disease where the patient is systemically unwell. In such cases it will help to exclude non-GIT disease as the underlying cause, as well as documenting any metabolic consequences of the disease process.
- GIT-focused blood tests include:
  - TLI, PLI, amylase and lipase – see pancreatitis (3.4.7).
  - Folate and cobalamin – changes outside of the reference range can be supportive of GIT disease.
  - Hypocobalamininaemia requires parenteral treatment.
  - An ACTH stimulation test looking for atypical Addison's disease is appropriate in some dogs.
  - A thyroid hormone level test is appropriate in older cats.
3.4.4 Abdominal imaging

Radiographic changes in common GIT diseases

- For basic abdominal radiographic anatomy see 1.3.12.

**Megaoesophagus**
- Wall is highlighted by intraluminal gas (see radiograph, below left).
- Heart/trachea is displaced ventrally.
- Check for aspiration pneumonia (dependent parts of lobes, especially right middle and left cranial) and mediastinal mass.

**Gastric dilation/torsion**
- Torsion shows as a gas-filled stomach, usually with linear soft-tissue density crossing the stomach on a lateral view.
- Gas may be present in the intestines. The pylorus is displaced dorsal, cranial and to the left (see radiograph, below right).

**Small intestinal obstruction**
- Small intestinal loops are dilated proximal to obstruction (greater than diameter of large bowel).
- Colon may be empty.
- Gravel signs may be present.

**Peritonitis**
- Septic vs non-septic:
  - Check with ultrasound; septic fluid tends to be granular.
  - Microscopic examination of fluid: bacteria = septic.
- Regionalized vs generalized.
- Loss of sharp outline of abdominal organs.
- Serosal surfaces not clearly seen.

**Ultrasound:** complete ultrasound of the GIT is challenging and requires an experienced ultrasonographer. However, useful information can still be gained by looking for evidence of free fluid, dilation of the stomach or bowel and gross changes in wall thickness or layering.
TREATMENT

3.4 Digestive tract

Drug treatment

- Drugs commonly used in the management of GI disease are listed in the table below.

### DRUGS FOR MANAGEMENT OF GI DISEASE

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose (mg/kg)</th>
<th>Side-effects</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>H₂ blockers</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Cimetidine</em> (T, S, inj, s/c, i/m, i/v)</td>
<td>5–10 q8hr (dog po, inj) 2.5–5 q12hr (cat po, inj)</td>
<td>Rare: hepatic, renal and thrombocytopenia</td>
</tr>
<tr>
<td>Ranitidine (T, S, inj, s/c, i/m, i/v)</td>
<td>2 q8–12hr (dog po, inj) 2.5 (inj) 3.5 (po) (cat)</td>
<td></td>
</tr>
<tr>
<td>Famotidine (T)</td>
<td>0.5–1 q12–24hr (po)</td>
<td></td>
</tr>
<tr>
<td><strong>Proton pump inhibitors</strong></td>
<td>0.5–1.5 q24hr (dog po), 0.75–1 q24hr (cat po)</td>
<td>Nausea; diarrhoea. Use for no more than 8 weeks</td>
</tr>
<tr>
<td>Omeprazole (T, capsule, inj, i/m, i/v)</td>
<td>1 q24hr (dog/cat inj)</td>
<td></td>
</tr>
<tr>
<td><strong>Protectants</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sucralfate (T, L)</td>
<td>25–50 q6–8hr (dog po) 250/cat q8–12hr (po)</td>
<td>Constipation</td>
</tr>
<tr>
<td>†Kaolin-based pastes</td>
<td>See product</td>
<td></td>
</tr>
<tr>
<td><strong>Antiemetics</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Maropitant</em> (T, inj, s/c)</td>
<td>2 q24hr (dog po) 1 q24hr (dog inj)</td>
<td>Injection can be painful</td>
</tr>
<tr>
<td>†Metoclopramide (T, L, inj, s/c, i/m, i/v)</td>
<td>0.2–0.5 q6–8hr (po) 0.2–0.5 q6–8hr (inj)</td>
<td>Neurologic: can be severe</td>
</tr>
<tr>
<td>Prochlorperazine</td>
<td>0.5–1 q8–12hr (po) 0.1–0.5 q6–8hr (inj)</td>
<td>Sedation, hypotension, neurologic</td>
</tr>
<tr>
<td><strong>Prokinetics</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ranitidine (T, S, inj, s/c, i/m, i/v)</td>
<td>As above</td>
<td>As above</td>
</tr>
<tr>
<td>†Metoclopramide (T, L, inj, s/c, i/m, i/v)</td>
<td>As above, also 1–2 q24hr CRI</td>
<td>As above</td>
</tr>
<tr>
<td>Erythromycin (T, L)</td>
<td>0.5–1 q8hr</td>
<td>Rare at this dose</td>
</tr>
<tr>
<td><strong>Motility modifiers</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diphenoxylate (T)</td>
<td>0.05–0.1 q6–8hr (dog po)</td>
<td>Sedation, constipation, ileus</td>
</tr>
<tr>
<td>Loperamide</td>
<td>0.04–0.2 q8–12hr (po)</td>
<td>Constipation; Toxicity in collie types</td>
</tr>
</tbody>
</table>

* Licensed for dogs; † licensed for cats and dogs;

T = tablet; S = syrup; L = liquid; inj = injectable;
s/c = subcutaneous; i/m = intramuscular; i/v = intravenous
NUTRITION

3.4.6 Dietary approach to intestinal disease

- **Adverse reactions to food** can be immunologically-based or non-immunologic (pharmacologically active micronutrients such as caffeine or theophylline, histamine in cheap fish as well as preservatives, additives and dyes and toxic contamination from bacteria or chemicals).

- **Dietary hypersensitivity**: typical signs are weight loss, vomiting and diarrhoea. 10% of cases have pruritic skin disease. Investigate by elimination diet as follows.
  - Diet should contain a novel protein and carbohydrate source. It can be commercial (single source protein or hydrolyzed) or home-cooked (e.g. boiled white fish and sweet potato).
  - Strict adherence is essential. Most cases likely to respond will start to do so in 1–2 weeks.
  - Responders should ideally be confirmed by rechallenge.

- **Acute vomiting and diarrhoea**: prescribe GI rest for 24 hours in patients that are systemically well, then introduce a ‘bland’ diet – palatable, low fat, highly digestible, low residue. For home-cooked food, protein:carbohydrate ratio = 1:1 (cat); 1:2 (dog). Malnutrition can occur with frequent starvation; feeding through episodes is advised.

- **Protein losing enteropathy (PLE)**: give increased levels of high biological value protein, increased carbohydrates as an energy source and reduced fat (<4% DM). Give increased fat-soluble (A, D and E) vitamins.

- **Colitis**: similar diets as for dietary hypersensitivity; for chronic cases, increasing fibre may help.

- **Faecal incontinence**: manage with low residue diets.

- **Constipation** cannot be treated by diet. However, the likelihood of recurrence can be reduced by using insoluble fibre to increase faecal bulk and stimulate defecation, while producing a softer stool.
  - 4 tsp of a high-fibre breakfast cereal per 100 g of food increases the fibre content of a diet by ~10%.

- Probiotics (cultured bacteria) and prebiotics (soluble fibre, e.g. fructo-oligosaccharide) have a role in the management of chronic GIT disease. Their precise mechanism(s) of action are unclear. Avoid natural yoghurts, because of the level of lactose.

SELECTED TOPIC

3.4.7 Pancreatitis

- Pancreatitis can occur as a primary problem, or secondary to another disease.

- Sick hospitalized patients are at risk of developing pancreatitis, even if it is not present on admission.
Mortality in acute pancreatitis cases is likely to be 25–50%.

**Presenting signs** might include:
- Acute, dogs: anorexia, vomiting, diarrhoea, abdominal pain, fever.
- Acute, cats: lethargy, anorexia, jaundice, fever and less commonly vomiting and diarrhoea.
- Chronic, dogs: anorexia, vomiting, weight loss, jaundice.
- Chronic, cats: anorexia, weight loss, vomiting, jaundice.
  - Chronic pancreatitis in cats may eventually lead to diabetes mellitus and/or exocrine pancreatic insufficiency.

**Absence of abdominal pain does not reliably rule out pancreatitis.**

**Laboratory changes**
- TLI, amylase and lipase are insensitive.
- PLI is relatively much more sensitive and specific, but note that false positive and false negative results do occur.
- Acute cases need full haematology and biochemistry.

**Imaging**
- **Acute:** radiographic changes are those associated with focal or generalized peritonitis and/or cranial abdominal mass. Ultrasound is more specific and aids aspiration of fluid from the pancreas and small pockets of fluid around it, as well as assessing the level of biliary obstruction and other disease.
- **Chronic:** requires ultrasound by an experienced ultrasonographer.

**Treatment**
- Starvation of acute pancreatitis cases is no longer recommended.
- **Acute:** treatment should be individualized according to patient’s problem list, but may include the following:
  - Pain relief (opioids not NSAIDs) and fluid therapy (Hartmann’s).
  - Plasma and/or albumin.
  - Gastric protection, antiemetics and prokinetics – omeprazole, ranitidine, CRI metoclopramide.
  - Antimicrobial (i/v – metronidazole, amoxicillin clavulanate).
  - Vitamins B₁₂ and K.
  - Encouragement to eat low-fat food, e.g. baby rice and Bovril.
  - Regular monitoring is required of proteins, haematocrit, electrolytes, liver.
- **Chronic:** low-fat (but not high-fibre) diets are beneficial in dogs but have not been demonstrated to be useful in cats.
3.5 Liver and biliary tract

3.5.1 Presentation
- Raised liver enzymes are commonly found on routine blood screening. Appropriate investigation and management is important, as many patients do not have clear signs associated with hepatobiliary pathology.
- Jaundice (see 2.14) is a worrying presenting complaint; accurate diagnosis is essential to allow prompt treatment.

3.5.2 Principal functions of the liver
- Besides metabolism of food, hormones, drugs and toxins, the liver also has haemopoietic and immune functions, as well as producing enzymes and clotting factors and storing iron, vitamins, glycogen and so on.

3.5.3 Major signs of liver dysfunction
- Jaundice (2.14), hypoglycaemia, hypoalbuminaemia, ascites, hepatic encephalopathy (episodes of stupor/disorientation with or without apparent blindness, especially post-prandially), bleeding (5.2), PU/PD (2.8), lethargy, inappetance (2.4), weight loss/poor growth (2.12).

3.5.4 Signs on physical examination
- Unless the liver is significantly enlarged, it is rarely palpable.
- Gross ascites can be diagnosed on ballottement; mild to moderate ascites and focal fluid may not be easily discernible on physical examination.
- Biliary obstruction associated with pancreatic disease may cause cranial right quadrant pain and will often be associated with jaundice.

3.5.5 Interpretation of blood tests
- Biochemistry is an important initial investigation for liver disease. Tests that indicate liver damage (ALT, ALKP) should be distinguished from tests of liver function (bile acids, ammonia). See table, page 86.
Approach to raised liver enzymes

- For an approach to raised ALKP and ALT in dogs, see page 87.
- Mild to moderate increases in liver enzymes are of greater clinical significance in cats and generally warrant further investigation. This is because in cats:
  - Cellular levels of liver enzymes are lower.
  - There is no steroid-induced isoenzyme of ALKP.

Further diagnostics

- **Radiography** is generally limited to assessing liver size.
- For normal liver size:
  - The gastric angle is between vertical and parallel to ribs.
  - The left lateral lobe of the liver has a sharp angle and is just within/beyond the costal arch.
- Lobar masses may be visible as they will displace other abdominal organs caudally.
- **Ultrasound** is usually more sensitive (though not more specific) than radiography and allows assessment of the biliary tract. Diffuse disease can be difficult to assess. In older dogs benign nodular hyperplasia can mimic neoplasia.
- **Aspiration/biopsy** – clotting times should be checked before biopsy. Biopsies obtained at laparoscopy/laparotomy preferred in diffuse disease; ultrasound-guided more valuable if focal or deep lesion present. Aspirates are useful in diffuse disease such as hepatic lipidosis, lymphoma and hepatitis. Take bile aspiration for culture if bacterial cholangiohepatitis suspected.
### BODY SYSTEMS AND MULTISYSTEMIC DISEASE

#### INTERPRETATION OF BLOOD TESTS

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Description</th>
<th>Non-hepatic causes of change</th>
</tr>
</thead>
<tbody>
<tr>
<td>ALKP increase</td>
<td>Inducible group of isoenzymes primarily associated with biliary disease. T&lt;sub&gt;1/2&lt;/sub&gt; 70 hrs (dog) 6 hrs (cat). Smaller increase seen in cats with hepatobiliary disease.</td>
<td>Growing animals, enteritis, pancreatitis, bone disease, endocrine disease, drugs (esp. corticosteroids [dog])</td>
</tr>
<tr>
<td>ALT increase</td>
<td>Cytosolic enzyme released on damage to hepatocytes T&lt;sub&gt;1/2&lt;/sub&gt; 60 hrs (dog) &lt;24 hrs (cat). Will show chronic increases as liver regenerates.</td>
<td>Endocrine disease, hypoxia, enteritis, pancreatitis, peritonitis, toxins, trauma, drugs</td>
</tr>
<tr>
<td>AST increase</td>
<td>Cytosolic enzyme released on damage to hepatocytes T&lt;sub&gt;1/2&lt;/sub&gt; 5 hrs (dog) 80 min (cat).</td>
<td>Muscle damage/inflammation, red cell damage</td>
</tr>
<tr>
<td>γ-GT increase</td>
<td>Bile ducts and perilobular tissue (dog). Increase due to de novo synthesis, regurgitation and from cell membrane. Increase more in biliary vs. parenchymal disease.</td>
<td>Corticosteroids (dog) and anticonvulsants, pancreatitis, enteritis</td>
</tr>
<tr>
<td>GLDH</td>
<td>Mitochondrial enzyme increases in hepatic necrosis (dog).</td>
<td></td>
</tr>
<tr>
<td>Bilirubin increase</td>
<td>Bilirubin can be elevated due to excess production (pre-hepatic), an inability of the liver to process bile (hepatic) or obstruction to bile flow (post-hepatic).</td>
<td></td>
</tr>
<tr>
<td>Bile acids increase</td>
<td>Synthesized by hepatocytes and secreted in bile. 95% enterohepatic circulation and should fall to baseline within 2 hours.</td>
<td>Jaundice, elevated glucocorticoids, pancreatic disease</td>
</tr>
<tr>
<td>Albumin</td>
<td>Severe impairment of functional liver mass.</td>
<td>Urinary, intestinal or 3rd space loss</td>
</tr>
<tr>
<td>Ammonia increase; urea decrease</td>
<td>Produced from nitrogen metabolism and intestinal bacteria; conjugated in liver to form urea. Increases indicate severe hepatic impairment. Volatile, so rapid measurement essential; false positives if ammoniacal cleaners recently used.</td>
<td></td>
</tr>
<tr>
<td>Cholesterol change</td>
<td>Tends to be increased with severe intrahepatic cholestasis, and decreased in chronic severe hepatocellular disease.</td>
<td></td>
</tr>
</tbody>
</table>

ALKP = Alkaline phosphatase; ALT = alanine aminotransferase; AST = aspartate aminotransferase; γ-GT = gamma glutamyltransferase; GLDH = glutamyl dehydrogenase
3.5 Liver and biliary tract

**Raised ALKP and/or ALT**

- **>2× upper limit of reference range**
  - Extend liver screening: gamma-GT, AST, CPK, bilirubin, dynamic bile acid
  - Ultrasound scan of liver
    - Abnormal
      - Biopsy liver and gall bladder aspirate
    - Normal
  - Case unwell: look for primary disease elsewhere
    - Case clinically well: re-check in 3–6 months
    - Results still abnormal
      - Discuss with client: chronic monitoring or further investigation*

- **<2× upper limit of reference range**
  - Case clinically well: re-check in 6–8 weeks
  - Case clinically well: re-check in 6–8 weeks

---

* Further investigation is indicated in breeds predisposed to chronic hepatopathy, e.g. Doberman. Monitoring is advised in breeds known to have chronically high enzymes without clear pathology, e.g. Scottish terrier.

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**TREATMENT**

**Choice of therapies**

- Acute liver disease is managed symptomatically and supportively. Coagulopathies and embolism are more likely than in chronic disease.
- Chronic liver disease in dogs and cats has relatively few specific treatments. Doses of potentially toxic drugs should be reduced as much as possible, and alternative therapies sought.
- **Antimicrobials** are indicated if proven infection or in severe dysfunction/portosystemic shunts: give ampicillin 10–20 mg/kg po q8hr.
- **Vitamin K₁** should be given to patients at risk of bleeding: 0.5–1 mg/kg s/c or po q12hr.
Diet and nutraceuticals

- Diet is indicated if there is hepatic dysfunction (3.5.9).
- S-adenosyl methionine replenishes antioxidant capacity and aids metabolite detoxification. Clinical benefit has been difficult to prove. 20 mg/kg po q24hr. Must be enteric-coated, bioequivalence varies between products.
- Vitamin E (α-tocopherol) is an antioxidant; give 4 mg (6 iU)/kg po SID.
- Silybin phosphatidylcholine (silymarin; milk thistle) is an antioxidant, benefit unproven; dose rate 9–70 mg po q24hr.
- Corticosteroids are indicated in inflammatory liver disease; additional immunosuppressive agents may be necessary.
- Colchicine is antifibrotic; its efficacy is unproven; it can have side-effects.
- Lactulose is used in hepatic encephalopathy as it reduces colonic bacterial numbers and converts NH₃ to NH₄⁺ in the colon that is not absorbed. 0.5–1 ml/kg po q8hr to give soft stool consistency. Use as an enema in acute encephalopathic crisis, 20 ml/kg (30% solution).
- Ursodeoxycholic acid is choleretic and anti-inflammatory.
- Diuretics may help in ascitic cases: give spironolactone (2–4 mg q24hr).
- Zinc is antifibrotic and decoppering: give 1–2 mg elemental zinc po q24hr.
- D-penicillamine and trientine are decoppering agents (consult appropriate text for use, adverse reactions and drug interactions).

Nutrition

3.5.9 Dietary approach to reduced liver function

- Aim to meet necessary nutritional requirements during times of hepatic insufficiency.
- Malnutrition is common in chronic hepatic disease and adversely affects cellular function, worsening the disease process.
- It is associated with anorexia, impaired nutrient digestion and absorption, increased energy requirements and accelerated protein breakdown in conjunction with reduced synthesis.
- Significantly less is known about the nutritional requirements of cats with liver disease than dogs.
- Control of ammonia production:
  - Ammonia is produced by protein catabolism and bacterial fermentation.
50% of whole body ammonia uptake occurs in the muscle.

Protein restriction is only appropriate when protein intolerance has developed, but subtle encephalopathy is difficult to diagnose.

Dietary proteins should be moderately restricted and of high biological value, limiting nitrogenous waste while maintaining positive nitrogen balance to prevent muscle breakdown.

Frequent small meals are beneficial.

Protein restriction in puppies/kittens can cause critically low blood albumin; standard hepatic diets are inappropriate and if used should be supplemented: milk or soya-based protein, e.g. cottage cheese. Increase protein to ~20% calories as protein, and monitor clinical signs and albumin.

**Fat**: should be restricted to 25–30% of calories.

**Carbohydrates**: complex carbohydrates as major energy source.

**Fibre** decreases ammonia production and colonic transit time.

**Vitamins**: requirement is increased, as liver is a major site of synthesis, storage and conversion. Vitamin E scavenges free radicals.

**Copper**: should be restricted, as it accumulates due to cholestasis or copper storage diseases. Consider copper content in water: this can be high.

**Zinc**: supplement as absorption is decreased and loss increased. Zinc acts to decrease copper absorption and aid ammonia detoxification in the urea cycle. May have antifibrotic activities.

**Amino acids**: L-carnitine, taurine, glutamine, arginine.

**Nutritional supplements**: S-adenosyl methionine, silybin.

**SELECTED TOPIC**

**Portosystemic shunts** 3.5.10

A vascular anomaly resulting in blood bypassing the liver.

They can be intrahepatic (large-breed dogs) or extrahepatic (small dogs and cats). Extrahepatic shunts are most commonly from portal or mesenteric vein to vena cava or azygous vein.

**Presentation** is usually poor growth and appetite, with signs of severe liver dysfunction (see 3.5.4). Kittens often have copper-coloured irises.

**Diagnosis** requires skilled ultrasonography or portovenography for confirmation.

**Management** (surgical vs. medical) remains controversial.

Acquired shunting will occur if there is portal hypertension.
3.6 Kidneys

3.6.1 Role of the kidney

The main functions of the kidney are:

- Excretion.
- Water balance.
- Blood pressure regulation.
- Electrolyte balance.
- Acid/base status.
- Hormones:
  - Excretion, e.g. gastrin.
  - Production of erythropoietin, calcitriol and renin.
  - Conversion of vitamin D.

The basic functional unit of the kidney is the nephron; see table and illustration, right.

Chronic disease leads to progressive loss of nephrons, with hyperfiltration occurring in the remainder to compensate. Eventually a tipping point occurs, where the level of hyperfiltration causes further nephron loss regardless of initiating cause, resulting in disease progression.

3.6.2 Risk factors and signs of acute renal disease

- Risk factors are additive and include pre-existing renal disease, sepsis, muscular damage, diabetes, hypovolaemia, electrolyte and acid/base imbalance, nephrotoxic drugs, and low-protein diets.
- Clinically these cases present with non-specific signs of lethargy, depression, anorexia (2.4) and collapse (5.6).
  - Owners may not have noticed changes in urine production.
- Cases will occur in hospitalized patients. It is therefore essential to flag a lack of urine production as soon as possible, to allow aggressive early intervention.

3.6.3 Clinical signs of chronic renal disease

- Commonly: polyuria/polydypsia (2.8), weight loss (2.7), lethargy, poor appetite (2.4), depression, poor hair-coat.
- Rarely: vomiting (2.5), diarrhoea (2.6), blindness.
**Anatomy of the kidney and nephron.**

- **Cortex**
- **Medulla**
- **Renal vein**
- **Renal artery**
- **Ureter**
- **Proximal tubule**
- **Glomerulus**
- **Collecting duct**
- **Distal tubule**
- **Loop of Henlé**

---

**NEPHRON FUNCTIONS**

<table>
<thead>
<tr>
<th>Nephron region</th>
<th>Function</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glomerulus</td>
<td>Filtration, renin production, plasma volume regulation</td>
</tr>
<tr>
<td>Proximal tubule</td>
<td>Resorption of glucose, proteins, amino acids, vitamins, electrolytes; secretion of H⁺</td>
</tr>
<tr>
<td>Loop of Henlé</td>
<td>Counter-current concentration of Na⁺ and urea in the medulla</td>
</tr>
<tr>
<td>Distal tubule</td>
<td>Secretion of H⁺ and K⁺ ions</td>
</tr>
<tr>
<td>Collecting duct</td>
<td>Selective permeability to water influenced by antidiuretic hormone</td>
</tr>
</tbody>
</table>

---

**DIAGNOSIS**

**International Renal Interest Society (IRIS) classification**

- Chronic renal disease should be fully evaluated, as this will be part of the patient’s physiology for the rest of their life.
- IRIS publishes guidelines, including disease-stage classification (see table, next page). Staging facilitates appropriate treatment and monitoring (3.6.6).
- Note that creatinine value is irrespective of laboratory reference range, as animals in the upper reference range often have excretory failure and this extends into the mid-range in very thin patients.
- IRIS staging is subdivided according to:
  - Proteinuria, classified as non-proteinuric (NP), borderline (BP) and proteinuric (P).
  - Persistent hypertension (see table, next page).
  - Evidence of end-organ damage resulting from hypertension:
    - nc: no complications.
    - c: complications (end-organ damage).
    - rnd: risk not determined.
So a dog with a creatinine of 160 μmol/l, a UPC of 0.4 and a systolic BP of 155 mmHg, with no evidence of end-organ damage, would be classified as IRIS stage 2–BP–L–nc.

Beyond measuring creatinine, urea and blood pressure (3.3.8), management can be improved with knowledge of red cell count, electrolytes, calcium, phosphorus, albumin and full urinalysis (1.3.6).

### 3.6.5 Renal imaging

- Radiographs can be used to determine renal size and shape and the presence of pelvic mineralization/urooliths, as well as ureteroliths.
- Normal renal size can be compared with the size of lumbar vertebra L2 and the aorta, as in the table above right.
- Ultrasound is more sensitive for renal shape and architecture, including pelvic dilation, as well as variation within and differentiation between the medulla and cortex, e.g. cortical cysts. With skill, ureteric dilation can also be evaluated.
3.6 Kidneys

<table>
<thead>
<tr>
<th>Imaging mode</th>
<th>Normal renal size DOG</th>
<th>CAT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Radiography</td>
<td>2.5–3.5 x length L2</td>
<td>2.4–3.0 x length L2</td>
</tr>
<tr>
<td>Ultrasound</td>
<td>Ratio of kidney length to aorta diameter: 5.5–9.1</td>
<td>Length: 27–47 mm (35 av.). Width: 17–28 mm (21 av.)</td>
</tr>
</tbody>
</table>

L2 = second lumbar vertebra

### Therapy for chronic renal disease

Therapy is based on the IRIS staging system as follows:

- **Stage 1**
  - Discontinue any nephrotoxic drugs and rule out treatable underlying disease, e.g., pyelonephritis.
  - Correct dehydration and other pre- and post-renal causes.
  - Treat hypertension if end-organ damage, otherwise monitor.
  - Investigate proteinuria, if present; consider ACEi and aspirin if UPC > 2.0.

- **Stage 2**: As stage 1, plus
  - Treat if UPC >0.5 (dog); >0.4 (cat).
  - Manage phosphate levels to between 0.9 and 1.45 mmol/l, by diet, phosphate binders and ultra-low-dose calcitriol (dog 1.2 ng/kg q24).
  - Treat acidosis if present (sodium bicarbonate/potassium citrate).
  - Potassium gluconate if hypokalaemic – usually cats.

- **Stage 3**: As stage 2, plus
  - Target phosphate below 1.6 mmol/l.
  - Treat anaemia if affecting quality of life (erythropoietin, transfusion).
  - Manage nausea/vomiting (antacids, mucosal protectants, antiemetics).
  - Maintain hydration with parenteral fluids if necessary.

- **Stage 4**: As stage 3, plus
  - Target phosphate below 1.9 mmol/l.
  - Try to maintain body weight, nitrogen balance and hydration. If necessary consider fitting a PEG tube.

- Antimicrobials should be given if there is a positive urine culture, or evidence to support pyelonephritis (3.13.5).

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*Take care to ensure patient is normally-hydrated when introducing antihypertensive or ACEi therapy, especially in stage 4 disease.*
3.6.7 Dietary management of chronic renal disease

- Diet is an essential part of the management of CRD, more than doubling the median life expectancy in cats and dogs.
- Not all cats and dogs will tolerate the ideal dietary modification so compromises may need to be made. It is vital that the patient eats something, as body protein catabolism will have more serious adverse effects than almost any diet.

**Aims**

- Reduction in hyperphosphataemia and azotaemia.
- Reduction in associated nausea, vomiting and anaemia.
- Improvement in body condition and water-soluble vitamin status.
- Correction of hydration, acid/base balance and electrolytes.
- Reduction in blood pressure via omega-3 fatty acids and sodium balance.

- When to start: the ideal time is not known, but definitely when hyperphosphataemia develops and possibly when there is significant azotaemia (urea >12–13 mmol/l, creatinine >180 μmol/l (dog) 250 μmol/l (cat) even without elevated phosphate).

- Diet choice: highly palatable, wet food is preferable.

**Reduction in hyperphosphataemia and azotaemia.**

- Protein restricted: 14–15% (dog); 28–30% (cat) protein on a dry matter (DM) basis.
- Phosphate restricted: 0.15–0.3% (dog); 0.4–0.6% (cat) on a DM basis.
- Potassium and water-soluble vitamin supplemented.
- Soluble fibre up to 10% DM helps to trap nitrogen.
- Increased omega-3 polyunsaturated fatty acids may help.
- Non-acidifying with reduced salt (NaCl) levels.
- Energy from fat as it increases density and palatability.

**Fluid balance:** a crucial element of therapy is aimed at maximizing oral fluid intake. If this is inadequate, then subcutaneous fluid can be considered.

Always check whether a patient is eating normally before interpreting serial changes in urea and creatinine. Starvation will result in a fall in urea and creatinine levels.
SELECTED TOPICS

Diagnosis of acute renal disease

- This is signalled by a sudden rise in urea and/or creatinine by >50%. Prompt intervention is crucial to interrupt the induction phase and limit nephron loss.
- Diagnosis is via physical examination, risk factors and speed of onset of signs.
  - Note: kidneys often look normal on ultrasound.
  - Magnitude of azotaemia may not be especially extreme for the level of clinical signs, and often worsens despite treatment.
  - Patients are usually proteinuric, with alkaline urine. They can be anuric to polyuric.
  - Renal biopsy may help, but it is technically complicated.

Management of acute renal disease

Treatment goals

- Prevent further renal damage: induce emesis, use gastrointestinal adsorbents and diuresis, and stop nephrotoxic drugs.
- Maintain renal perfusion.
- Maintain urine flow and limit rise in uraemic toxins by diuresis, using fluid therapy, furosemide, mannitol.
  - Note: increased urine flow alone does not mean improved GFR (glomerular filtration rate).
- Correct acid/base and electrolyte abnormalities.
  - Note: electrolyte abnormalities are often associated with cellular shifts rather than absolute change, so they can alter very rapidly.
  - Primary correction of acidosis is rarely required.
- Control uraemic damage to GIT: use antacids and sucralfate.
- Control vomiting: use metoclopramide (not with dopamine CRI), maropitant, prochlorperazine, dolasetron, ondansetron.
- Provide antibacterial cover, as uraemic patients are immunocompromised.
- Peritoneal dialysis is possible, but technically challenging. Haemodialysis is available in some countries.

Treatment tips

- Always place a urinary catheter and measure output 1–2 hourly.
- Fluids and diuretics are the cornerstone of treatment.
- If there is oliguria or anuria, avoid over-hydration, particularly in cats.
- Safe volume expansion is 5% once hydration status is normal.
- An ‘in-and-out’ approach can be used: measure urine output/hr, add 1 ml/kg to that figure, and replace that amount in the following hour.
- Failure to re-establish urine output within 6–12 hours carries a guarded prognosis.
3.7 Lower urinary tract

3.7.1 Disease types, and bladder innervation

3.7.2 Differentiating disease types

3.7.3 Diagnostic tests

3.7.4 Drug therapy

3.7.5 Dietary management of LUT disease

3.7.6 Urinary catheterization

INTRODUCTION

3.7.1 Disease types and bladder innervation

Lower urinary tract (LUT) diseases fall into 3 main groups: cystitis (pollakiuria) (2.8), incontinence (2.10) and obstruction (5.7).

Neurologic control of bladder function is mediated by the hypogastric, pudendal and pelvic nerves (see diagram below). Spinal cord damage may affect bladder function, depending on its location. Damage above L4–L5 tends to cause a spastic bladder and inability to urinate, while damage to the spine below L4–L5 causes a flaccid overflow bladder.

HISTORY AND PHYSICAL EXAMINATION

3.7.2 Differentiating disease types

In the case of frequent attempts to urinate, initial examination should aim to differentiate cystitis from obstruction.

Cystitis patients tend to be restless, with frequent attempts to urinate – squatting and straining, often with haematuria but are rarely systemically unwell.

Obstructed patients tend initially to make frequent urination attempts and then will often appear to settle down, but they become quieter and more withdrawn as the metabolic changes associated with obstruction take effect.

Incontinence should be distinguished from increased urination associated with PU.

In chronic feline idiopathic lower urinary tract disease (FLUTD), an in-depth history covering the cat’s home environment is essential.
3.7 Lower urinary tract

**DIAGNOSIS**

**Diagnostic tests**

- In the absence of obstruction or signs of PU/PD, biochemistry and haematology are rarely of great diagnostic value.
- Full urinalysis (including culture) is an essential early diagnostic test.
- Crystals in urine sediment do not necessarily indicate:
  - That crystals are present in the bladder, unless it is a fresh sample placed on a warmed slide.
  - That any uroliths present are of the same composition.

**Imaging**

- Radiographic evaluation of the lower urinary tract can be achieved using:
  - Plain radiographs, with double contrast cystography and retrograde urethrography, to look at bladder size and position, wall thickness and regularity, filling defects, presence of uroliths and urethral stricture.
  - Intravenous urography to determine ureteral positioning.
  - Ultrasound can be used to evaluate all of these areas, apart from normal ureters, and much of the male and the distal female urethra. It has the advantages of being quicker, safer, less expensive and does not require anaesthesia.

**Radiodensity and appearance of uroliths**

- Uroliths vary in radiodensity. Diagnosis requires both plain and contrast radiographs.
- Air bubbles introduced with the contrast medium can mimic uroliths. To distinguish the two: bubbles rise, uroliths sink under gravity.

**TREATMENT**

**Drug therapy**

- Appropriate therapy for lower urinary tract disease is dependent on accurate diagnosis. See table on page 99 and algorithm 2.9.

**NUTRITION**

**Dietary management of LUT disease**

- For idiopathic cystitis or urolithiasis, diet should be the first-choice therapy, as it is safer and may be more effective than drugs or surgery.
  - **Crystalluria**: if asymptomatic, dietary intervention is not indicated.
  - **Idiopathic cystitis**: diets which increase urine output.
  - **Urolithiasis**: see table on next page.
Urinary catheterization tends to be underused; in addition to cases of obstruction, catheter placement should also be considered for recumbent or poorly-mobile patients.

There are several key elements for easy urinary catheterization.

- Choose the right catheter.
  - Polyamide catheters can be used but are relatively inflexible and traumatic especially for repeated use.
  - Silicone Foley catheters are best for indwelling catheters in dogs and female cats.
  - 11 cm catheters can be too short for some male cats (see table, below right).
- Restraint is important; this may require sedation in bitches and queens and general anaesthesia in tom cats. In cases that are systemically unwell, would cystocentesis drainage and stabilization be a safer first option?
- Use plenty of lubricant and keep everything as sterile as possible.
- Sometimes gently rotating the catheter, or flushing saline through the catheter as you insert, can help in males.
- A direct view with a speculum of the urethral orifice in bitches can make things a lot easier. Otherwise use gentle digital palpation in the ventral midline to find the orifice and then slide the catheter under your finger.
- If the catheter gets stuck, be patient and don’t push too hard, as this will traumatize and potentially rupture the urethra.
- Some patients are masters at removing catheters even when stitched in, so they may need an Elizabethan collar.
- Closed urine collection systems are preferred.
- Antimicrobial cover is NOT recommended.

### SELECTED TOPIC

#### 3.7.6 Urinary catheterization

- Struvite
  - Royal Canin Urinary S/O (P and D)
  - Purina UR (P and D)
  - Hill’s Prescription Diet: c/d (P), s/d (P), x/d (P and D), w/d (P, obesity)

- Calcium oxalate
  - Royal Canin Urinary S/O (P)
  - Purina NF (P)
  - Hill’s Prescription Diet: cd (P), u/d (P), x/d (P)

- Urate and cystine
  - Royal Canin Urinary U/C (P)
  - Hill’s Prescription Diet u/d (P and D)

D = dissolution; P = prevention

c/d, s/d, u/d, k/d and x/d are brand names

### MANUFACTURERS’ RECOMMENDED DIETS

<table>
<thead>
<tr>
<th>Stone Type</th>
<th>Diet 1</th>
<th>Diet 2</th>
<th>Diet 3</th>
<th>Diet 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Struvite</td>
<td>Royal Canin Urinary S/O (P and D)</td>
<td>Purina UR (P and D)</td>
<td>Hill’s Prescription Diet: c/d (P), s/d (P), x/d (P and D), w/d (P, obesity)</td>
<td></td>
</tr>
<tr>
<td>Calcium oxalate</td>
<td>Royal Canin Urinary S/O (P)</td>
<td>Purina NF (P)</td>
<td>Hill’s Prescription Diet: cd (P), u/d (P), x/d (P)</td>
<td></td>
</tr>
<tr>
<td>Urate and cystine</td>
<td>Royal Canin Urinary U/C (P)</td>
<td>Hill’s Prescription Diet u/d (P and D)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
### COMMON DRUG TREATMENT OF LUT DISEASE

<table>
<thead>
<tr>
<th>Problem</th>
<th>Generic drug</th>
<th>Oral dose (mg)</th>
<th>Side-effects</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Incontinence</strong></td>
<td><em>Phenylpropanolamine (S)</em></td>
<td>1.0/kg q8hr or 1.5/kg q12hr</td>
<td>Hypertension, restlessness, aggression</td>
</tr>
<tr>
<td></td>
<td>*Oestriol (T)</td>
<td>1/dog q24hr</td>
<td>Oestrogenic signs</td>
</tr>
<tr>
<td><strong>Reflex-dyssynergia or urethrospasm post obstruction</strong></td>
<td>Diazepam (T)</td>
<td>2–10/dog q8hr 1.25–5/cat q8hr</td>
<td>Sedation, ataxia, muscle weakness, hepatic necrosis (cat)</td>
</tr>
<tr>
<td></td>
<td>Phenoxybenzamine (C)</td>
<td>0.25–1/kg (dog) q8–24hr; 0.5–1/kg (cat) q12hr</td>
<td>Hypotension, tachycardia</td>
</tr>
<tr>
<td></td>
<td>Prazosin (T)</td>
<td>1/15 kg (dog) q8–24hr; 0.25–0.5/cat q12–24hr</td>
<td>Hypotension, sedation, heart failure in occult disease</td>
</tr>
<tr>
<td></td>
<td>Dantrolene</td>
<td>0.5–2/kg q12hr</td>
<td>Muscle weakness, hepatitis, pleural effusion</td>
</tr>
<tr>
<td><strong>Cystitis</strong></td>
<td>Consider glycosaminoglycan supplements, increasing water turnover (wet diet, frequency of urination), reducing environmental stressors (cats), amitriptylline</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Urolithiasis</strong></td>
<td>In conjunction with diet for certain uroliths, e.g. allopurinol (urate), D-penicillamine (cystine)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Urinary tract infection</strong></td>
<td>Should ideally be based on culture and sensitivity; re-culture after 7–10 days of therapy and 1–2 weeks thereafter. Empiric therapy – majority of UTI susceptible to amoxicillin/clavulanate, fluoroquinolone or potentiated sulphonamide; treat for 7–10 days</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Bladder neoplasia</strong></td>
<td>Transitional cell carcinomas most common tumour. Some respond well to COX-2 inhibitors (piroxicam, meloxicam). Variable response to chemotherapy (carboplatin, mitoxantrone)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Licensed for dogs; T = tablet; S = syrup; C = capsule; † two drugs usually combined to maximize muscle relaxation

### CATHETER SIZES

<table>
<thead>
<tr>
<th></th>
<th>Dog</th>
<th>Bitch</th>
<th>Tom cat</th>
<th>Queen</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diameter (Fr)</td>
<td>5–10</td>
<td>5–16</td>
<td>3–4</td>
<td>4–5</td>
</tr>
<tr>
<td>Length (cm)</td>
<td>30–60</td>
<td>30–40</td>
<td>11–14</td>
<td>11–30</td>
</tr>
</tbody>
</table>
3.8 Reproduction

3.8.1 Presentation
- The majority of cats and dogs present for routine neutering to control fertility.
- Presentation for reproductive problems is rare in cats other than for medication to postpone oestrus.
- Common presentations in dogs would include pseudopregnancy, pyometra and juvenile vaginitis.
- Apart from infertility and closed pyometra, the starting point for most reproductive problems is self-evident on history or physical presentation.
- Infertility frequently presents without any outward signs of ill health and can be daunting when dealing with valuable patients and breeders with considerable knowledge and experience. Knowing what can be normal variation in the female reproductive cycle is a useful starting point (see table above right).

INTRODUCTION

3.8.1 Presentation

- Is the male a proven breeder?
  - Yes: Physical examination of male reproductive organs
  - No: Has there been a change in health status since the last successful mating?

- Has there been a change in health status since the last successful mating?
  - Yes: Is the female a proven breeder?
    - Yes: Has there been a normal oestrous cycle?
    - No: Failure to conceive
  - No: Is the female a proven breeder?
    - Yes: Has there been a normal oestrous cycle?
    - No: Failure to conceive
3.8 Reproduction

FEMALE REPRODUCTIVE PARAMETERS

<table>
<thead>
<tr>
<th></th>
<th>Dog</th>
<th>Cat</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age of reproductive</td>
<td>6–12 months</td>
<td>5–12 months</td>
</tr>
<tr>
<td>maturity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Oestrous cycle (freq.)</td>
<td>3.5–13 (6) months</td>
<td>Induced ovulation</td>
</tr>
<tr>
<td>Duration of oestrous cycle</td>
<td>7–35 (16) days</td>
<td></td>
</tr>
<tr>
<td>Gestation period</td>
<td>56–72 (63) days</td>
<td>56–71 (63) days</td>
</tr>
<tr>
<td>Litter size</td>
<td>2–15</td>
<td>1–9 (4)</td>
</tr>
<tr>
<td>Earliest age of weaning</td>
<td>4 weeks</td>
<td>4 weeks</td>
</tr>
</tbody>
</table>

Parameters vary with breed; figure in parentheses is the average

For an approach to infertility, see algorithm below. Inevitably, some mating will fail without evidence of clear cause, despite extensive investigation.
HISTORY AND PHYSICAL EXAMINATION

3.8.2 Vital history questions

- Neutering status should always be checked, as mistakes in records are relatively common.
- In intact patients, a full reproductive history is vital, as the approach to patients that have previously mated successfully is different to those that have not.
- It is assumed that patients that have previously mated successfully do not have hereditary or congenital anatomic or genetic abnormalities that would prevent conception. However, they may have acquired disease, e.g. endometritis; be incompatible with their chosen mate; or have developed issues with their reproductive cycle.
- In bitches, a description of their cycling frequency and length, and behaviour during oestrus should be obtained.

DIAGNOSIS

3.8.3 Routine tests

- Routine haematology and biochemistry in reproductive disease help to rule out intercurrent disease and pyometra.
- The state of the uterus and ovaries can be rapidly evaluated using ultrasound.

3.8.4 Monitoring the oestrous cycle in bitches

- The stage of the oestrous cycle can be monitored by plasma progesterone, or by vaginal cytology.
- Plasma progesterone (see table above right):
  - if rise from <1 ng/ml to 2–4 ng/ml, ovulation in about 2 days (increased frequency of samples increases accuracy).
  - 4–10 ng/ml indicates ovulation.
  - 10–25 ng/ml indicates fertilization.
- Vaginal cytology: see table below.
- See also individual conditions (3.8.5–3.8.7).

VAGINAL CYTOLOGY

<table>
<thead>
<tr>
<th>Cell type</th>
<th>Pro-oestrus EARLY</th>
<th>LATE</th>
<th>Oestrus EARLY</th>
<th>LATE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Parabasal</td>
<td>+++</td>
<td>+</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Intermediate</td>
<td>+++</td>
<td>++</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Superficial</td>
<td>+/-</td>
<td>++</td>
<td>+++</td>
<td>+++</td>
</tr>
<tr>
<td>RBCC</td>
<td>+</td>
<td>+++</td>
<td>++</td>
<td>–</td>
</tr>
<tr>
<td>Neutrophils</td>
<td>+</td>
<td>–</td>
<td>–</td>
<td>+</td>
</tr>
</tbody>
</table>
3.8 Reproduction

PROGESTERONE LEVELS

<table>
<thead>
<tr>
<th>Pro-oestrus</th>
<th>LH surge</th>
<th>Ovulation</th>
<th>Fertilization</th>
</tr>
</thead>
<tbody>
<tr>
<td>Progesterone (ng/ml)</td>
<td>&lt;1</td>
<td>2–4*</td>
<td>4–10</td>
</tr>
</tbody>
</table>

*ovulation around 2 days later

COMMON REPRODUCTIVE PROBLEMS

Pseudopregnancy (false pregnancy)

- Cats: can follow sterile mating, spontaneous ovulation or early embryonic death, leading to 40–50 days without oestrus.
- Dogs: common 6–14 weeks following oestrus.
- Common signs are mammary development, usually with milk, aggression, dullness, nesting/toy fixation, inappetence, vulval discharge (milky).
- Treatment unnecessary if signs are mild.
- Lactation can be suppressed by preventing the bitch from licking her mammary glands, exercising to reduce nesting behaviour, and conserving fluid (water deprivation overnight; furosemide).
- For more severely affected bitches, use prolactin inhibitors: cabergoline (5 μg/kg po q24hr for 5–10 days) suppresses lactation in 95% of cases.

Mismating or unwanted pregnancy

- Treatment: a variety of regimes is described; only oestradiol benzoate is licensed in the UK. Potential complications should be discussed with owners prior to treatment.

<table>
<thead>
<tr>
<th>Early metoestrus</th>
<th>Anoestrus</th>
<th>Vaginitis/pyometra</th>
</tr>
</thead>
<tbody>
<tr>
<td>–</td>
<td>++</td>
<td>–</td>
</tr>
<tr>
<td>+</td>
<td>+</td>
<td>–</td>
</tr>
<tr>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>+++</td>
<td>+</td>
<td>++++</td>
</tr>
</tbody>
</table>
3.8.7 Vaginal discharge and pyometra
- **Vaginitis**: patient is systemically well, haematology within normal limits. Juvenile vaginitis from 2–3 months old usually resolves with the first season. Adult vaginitis should prompt investigation of underlying cause.
- **Pyometra**: is a consequence of cystic endometrial hyperplasia allowing secondary infection during oestrus. Can occur in spayed bitches as a stump pyometra. Signs are usually apparent 6–8 weeks post-season. If pyometra is causing systemic toxicity, patients are typically dull, inappetent, with PU/PD. Vaginal discharge may be present.
- Confirm with haematology and imaging.
- Treatment: stabilize patient and then perform ovariohysterectomy. Medical management is possible but not recommended.

3.8.8 Phimosis and paraphimosis
- **Phimosis**: inability to extrude penis; can cause dribbling urination or infertility. Management is surgical.
- **Paraphimosis**: an engorged or oedematous penis that cannot be retracted. Obstruction of circulation to the penis will lead to desiccation, ischemia, urethral obstruction and gangrene.
- Paraphimosis is most commonly due to post-coital hair entanglement – a medical emergency.
- Treatment: Cleansing, sedation (ACP), lubricant and cold hypertonic dextrose applied to penis; surgical enlargement of preputial orifice and indwelling urinary catheter; ultimately amputation.

3.8.9 Caesarean section
- Indications:
  - Primary or secondary uterine inertia.
  - Abnormality of maternal pelvis or soft tissue of birth canal.
  - Relative/absolute foetal oversize or deficient/excess foetal fluid.
  - Foetal death with putrefaction, toxaemia or other maternal illness.
  - Dystocia due to malposition or neglect.
  - Previous history of prophylaxis, or breed-specific prophylaxis, e.g. bulldog.
- Anaesthesia has increased issues due to:
  - Reduced maternal respiratory capacity.
  - Increased sensitivity of respiratory centre to CO₂.
  - Acid/base disturbances (variable).
  - Blood and fluid loss at surgery.
  - Partial vena cava occlusion.
  - Foetal depression caused by anaesthetic agents.
Suitable anaesthetic regimes: a variety are appropriate; pre-oxygenation of the dam is important. In the majority of cases the practice’s standard anaesthetic protocols should be used.
- Premedication with low dose ACP, then diazemuls i/v immediately prior to induction, followed by the lowest dose of propofol or alfaxalone to allow intubation with inhalation maintenance.
- Ketamine/medetomidine as an i/m technique with atipamezole (one drop on the tongue) given to the neonate. Causes more respiratory depression and is less controlled than i/v techniques.
- Opioids can cause respiratory depression of neonates.
- CRI propofol is not recommended.

Post-parturient conditions 3.8.10
- Generally the bitch/queen recovers quickly from vaginal birth or caesarean. If this does not occur, possible causes are:
  - Uterine haemorrhage.
  - Retained placenta(s) or foetus(es).
  - Acute metritis.
  - Uterine rupture or prolapse.
  - Toxic milk syndrome.
  - Acute mastitis.
  - Galactostasis.
  - Eclampsia.

NUTRITION
Dietary management 3.8.11
- See lifestage nutrition (1.1.9)

SELECTED TOPIC
Neonatology 3.8.12
Fading puppy/kitten
- Reference ranges can be found elsewhere in this book for neonatal physiology (1.2.6) and for haematology and biochemistry (1.3.1, 1.3.2).
- A fading puppy or kitten is born apparently healthy, but gradually becomes inactive, loses its suckle reflex and may die in the first 2 weeks of life. Localizing signs are usually absent.
- This condition represents a clinical description rather than a diagnosis: it could be due to a multitude of infectious and non-infectious causes.
Evaluating the neonate

- History: include a breeding history of the household; kennel or cattery management (hygiene, worming, vaccination and so on); health of the dam during the pregnancy; health of the remainder of the litter; age of the puppy/kitten; and the pattern of the illness to date.
- Clinical examination is difficult. It becomes more rewarding as the patient gets older and begins to develop adult responses. Neonates show limited responses to disease, initially becoming agitated and crying, progressing to inactivity, hypothermia and loss of the suckling reflex. They deteriorate rapidly with severe infection. Weight gain can be a sensitive indicator of developing problems. Failure to gain weight over any 24 hour period should be investigated.
- Systematic approach to examination is essential.
  - External features: hair coat (amount, condition, parasites); hydration; signs of injury; umbilicus; dewclaws; (docking site); discharge from nose; urine staining (patent urachus); diarrhoea/rectal patency; congenital malformation.
  - Eyes: swelling under lids indicates pus (often *Staphylococcus* species); eyes open between 5 and 14 (cats) and 10 and 14 (dogs) days, pupillary light response present within 24 hours of the eyes opening, mild corneal cloudiness usually present as eyes open.
  - Ears: external auditory meatus opens between 6 and 14 days; check for mites; middle ear infection (bulging tympanum).
  - Mouth: mucous membrane colour; evidence of cleft palate.
  - Thorax: regular rhythm; heart murmurs may be functional (usually soft); lung sounds difficult to distinguish but should be present; check symmetry/malformation of the thoracic cavity.
  - Abdomen: should feel full, but not swollen or tight; liver and spleen not palpable; intestines soft, mobile and non-painful; urinary bladder freely movable.
  - Neurological assessment: alertness, response to stimulation, suckle reflex; other reflexes appropriate to age; gait (walking from 4 weeks old); posture. Flexor and extensor dominance appears more variable in kittens than puppies.
3.9 Skin

3.9.1 Skin anatomy and general function
A working knowledge of skin anatomy is important in interpreting the results of skin biopsies.

The skin is the largest organ in the body. It forms an anatomical and physiological barrier between the animal and the environment, along with other functions including:
- Thermoregulation.
- Indication of health and reproductive status.
- Camouflage and UV protection.
- Immunoregulation and immunosurveillance.
- Storage of water, fats, vitamins and other materials.
- Sensory perception.
- Vitamin D production.
- Secretion and excretion.

3.9.2 General approach to consultation
- Obtain a detailed history.
- Perform a thorough dermatological examination.
- On the basis of the foregoing, construct a differential list for discussion with the owner.
- Agree a course of action, which will lead on to options for further investigation and treatment (3.9.9).

It is essential to develop a long-term relationship with the owner that encourages open communication and a spirit of cooperation.
Taking a dermatological history is challenging as the condition may have been present for many years and been treated by multiple clinicians and practices.

Particularly important are the initial presenting signs reported by the owner and described by the vet, distribution, response to previous treatment, parasite control history and status of in-contact animals.

The following link may be useful: www.nuvacs.co.uk/pdf/AguidetotheveterinaryconsultationbasedontheCCmodel.pdf

3.9.3 Checklists

Dermatological history
- **Presentation:** identify presenting complaint and owner’s principal concerns.
  - Age of onset, initial clinical signs.
  - Duration of disease, progression of signs and seasonal variation.
- **General history:** full general history with special attention to changes in appetite/thirst, gastrointestinal disturbances, sexual status and history of previous illness or injury.
- **Related/in-contact animals and people:** parents, littermates, pets belonging to family/friends, casual contacts/dog walker.
  - Is there evidence of zoonotic spread to in-contact people?

Environment
- **Indoor:** sleeping area/bedding. Access to bedrooms.
- **Outdoor:** areas visited, swimming, other animals encountered. Check if outdoor access is regulated, on demand or free (cat or dog flap). Nature of surrounding environment.
- **Diet:** record all foods and liquids offered, occasional extras, leftovers, treats or bribes given with medication. Record time of meals and whether provided *ad lib* or in set meals.

Past management
- Have parasites been seen and how long ago?
- What parasite control measures have been administered, including the frequency and duration of treatment?
- What are the bathing and grooming practices?
- What therapy has been administered for other conditions?
- Prior investigation and treatment: record results and review response to any prior tests, trials or investigations performed.
  - Consider the drugs used and the dose and duration of treatment.
Dermatologic examination

- Record the general appearance, regional distribution, e.g. interdigital (see photo, below left), symmetry, nature, e.g. pustular (see photo, below right), number and distribution of clinical lesions. Highlight and explain lesions to owner.
- Check hairs for abnormalities in tip, shaft or root. Check follicles.
- Explain process of examination and solicit owner’s input.

**DIAGNOSIS**

Diagnostic tests

- Routine haematology, biochemistry, hormonal testing and imaging are appropriate to rule out systemic causes of skin disease.
- Local testing of the skin includes:
  - Wood’s lamp for fluorescent fungi (not all *M. canis*).
  - Coat brushing/wet paper test for flea dirt.
  - Hair plucking for damage, stage of the hair cycle, parasites and follicular plugs.
  - Tape impressions for surface bacteria and yeasts (see photo bottom left).
  - Scrapes for ectoparasites, bacterial/fungal culture.
  - Cytology aspirates of mass lesions.
  - Skin biopsy of lesions or masses.
  - Allergy testing: intradermal; serologic.

**Skin lesions.** Interdigital erythema in a dog with atopic dermatitis (above left); non-follicular pustule in a dog with bullous impetigo (above right).

**Yeast infection.** Budding *Malassezia* (oval shaped) in an impression smear from a dog with otitis externa (left).
3.9.5 Pruritis
- History of scratching, licking, rubbing or chewing.
- Clinical signs dominated by secondary pathology: hair shaft damage, lichenification, hyperpigmentation and excoriation.
- Establish age of onset, initial distribution, seasonality, response to previous treatment including ectoparasite control.
- For an approach to pruritis, see algorithm, below.

3.9.6 Pustules
- A small, well-defined, circumscribed elevation of the epidermis filled with pus. Can be sub- or intraepidermal, or follicular. The majority are bacterial in origin, but some are sterile and have an immune-mediated cause.
- For an approach to pustules, see algorithm, page 112.
Alopecia

- There are two basic mechanisms of hair loss: failure of the follicle to produce and damage to the hair shaft following emergence.
- Distribution may be diffuse, focal or multifocal; regional, symmetric or asymmetric.
- Accompanying clinical signs may include pruritus, self-trauma, erythema, papular or vesiculobullous eruptions and seborrhoea.
- For an approach to alopecia, see algorithm, page 114.
3.9.8 Diseases of the claw and claw fold

- Claw disease is usually immune mediated, commonly associated with lupoid onychodystrophy and pemphigus foliaceus. Claw disease has been reported with other causes, including Leishmania and distemper, as well as metastatic lung neoplasia in old cats. Idiopathic disease is described in some dog breeds, e.g. dachshund and Siberian husky.
- Diagnosis is by skin scrapes, hair plucks, radiography and biopsy.
- Asymmetric claw-fold disease is usually associated with bacterial infection secondary to bite wounds or trauma. Neoplasia (squamous cell carcinoma) and dermatophytosis are rare causes.
- Symmetrical claw-fold disorders result from bacterial infection of the claw fold secondary to:
  - Metabolic conditions (HAC, DM, hypothyroidism, necrolytic migratory erythema).
  - Immunosuppression, e.g. FeLV.
  - Immune-mediated disease.

* β-lactamase-producing bacteria: suitable antimicrobials include clindamycin 11mg/kg q24hr; cephalixin 25 mg/kg q12hr; enrofloxacin 5 mg/kg q24hr.
TREATMENT

Topical agents

- Topical agents are selected on the basis of detailed, accurate observation of presenting clinical characteristics, supported by cytology.
- Several companies produce similar topical agents and it is best to become familiar with a limited range.
- For ectoparasiticides, see 1.1.7.
- **General principles**: Topical agents must have effective skin contact. This may require long coats to be clipped.
  - Where there is marked scaling or oiliness, this should be removed with keratolytic and keratoplastic washes or degreasing shampoos. This enhances contact, removes surface irritants, antigens, organisms and the oily environment in which cutaneous pathogens and commensals flourish. Ensure adequate contact time and repeat frequently enough to prevent recurrence.
- **Dry scaling**: Common agents include sulphur, selenium sulphide, salicylic acid.
  - Usually a secondary phenomenon (rarely a primary keratinization defect).
- Use combination of keratoplastic/keratolytic washes followed by humectants or emollients.
- Essential fatty acid supplements or rinses may be useful.
- **Greasy seborrhoea**: Common agents include benzoyl peroxide, coal tar, selenium sulphide.
  - Prewash with degreasing shampoos followed by keratoplastic washes. May have to be repeated.
  - Once the severe problem is controlled, switch to milder agents.
  - Commensal overgrowths are common.
- **Pyoderma**: Common agents include chlorhexidine, benzoyl peroxide, mupirocin (local).
  - Use with systemic treatments or alone for long-term maintenance. In the latter role, benzoyl peroxide can be irritant and drying.
Commensal overgrowth: Common agents include chlorhexidine, benzoyl peroxide, miconazole, ketoconazole.
- Prewash with degreasing shampoo to remove the oily environment.
- Guided by cytology, wash with appropriate antimicrobial shampoo.

Pruritus: Common agents include colloidal oatmeal, sodium lactate, coconut oil, essential fatty acids.
- Relief can be obtained simply by removing organic and inorganic debris, surface antigens, irritants and organisms, and the lipids that bacteria degrade to free fatty acids.
- Rehydrating the stratum corneum is helpful, as are topicals that replace missing waterproofing components.
3.9.10 Dietary approach to skin disease

- Skin and hair reflect the general metabolic health of a patient. In crisis, maintenance (especially of hair) is sacrificed for the essential body systems. Good hair and coat quality therefore usually reflects good nutritional status (see table above).
- Food-allergic skin disease: cases can be intensely pruritic with secondary damage to the hair/skin. They are managed with exclusion diets as for GIT, but response tends to be slower – at least 6–8 weeks.
- Dietary supplementation with essential fatty acid (EFA): diets should contain at least 3% (dog), 1.5% (cat) EFA on a DM basis. More than one-third of this should be linoleic acid. Supplementation is 40 mg omega-3/kg/day, aiming for an omega-6:omega-3 ratio in the diet of 5:1 – 10:1.
- Copper coat: many old dogs will develop copper coats over time. This pigment loss responds to tyrosine supplementation.
INTRODUCTION

Approach to nervous-system disease

■ Signs of nervous-system disease are common, but can be subtle (early cognitive dysfunction) or difficult to differentiate from behavioural disorders.
■ As with all body systems, disease can be associated with primary cause within that system, or secondary to another disease elsewhere, e.g. hepatic encephalopathy. See diagnostic algorithms for collapse (5.6) and seizures (5.8).

Decision making:
■ Is it disease of the nervous system or disease of another body system causing signs consistent with neurologic signs? (3.10.2)
■ If it is neurologic disease, what is the localization? (3.10.3 and 3.10.4)
■ What disease is causing these neurologic signs? (3.10.4)
■ How can I best approach management and treatment? (3.10.5, 3.10.6)
■ Finally, 3.10.7–3.10.10 deal with selected specific forms of nervous-system disease.

HISTORY AND PHYSICAL EXAMINATION

Performing a screening examination

■ A checklist for neurologic screening should include:

■ Mental state: behaviour, loss of learnt behaviour, alertness and responsiveness.

■ Cranial nerve (CN) function: symmetry of function, reflex testing, eyes. See table, next page.

■ Gait (walk and trot): lameness, tripping, ataxia.

■ Postural reactions: hopping, placing.

■ Reflexes: spinal, withdrawal, patellar. Note that biceps and triceps reflexes can be difficult to elicit in normal animals; only patellar reflex can be reliably obtained in all cases.

■ Muscle tone, size and symmetry.

*Cats tolerate neurologic examination poorly.*
Deficits identified within 24 hours of seizuring may be transient. Any such cases should be re-evaluated later. 

*Deficits need to be repeatable to be of significance.*

### 3.10.3 Localization of lesions within the CNS

- **Forebrain lesions** (cortex, thalamus and hypothalamus): seizures, behavioural changes, depression/ altered mentation, pacing, head pressing. Circling (same side as lesion), postural defects, hemiparesis, visual and menace defects indicate focal disease.

- **Brainstem lesions** (pons, midbrain, medulla) can lead to upper motor neuron (UMN) deficits CN III, V–XII, altered mentation, mild to severe tetraparesis; gait changes are usually more obvious/severe than with forebrain lesions. Lesion is ipsilateral to defect.

<table>
<thead>
<tr>
<th>Cranial nerves</th>
<th>Testing</th>
</tr>
</thead>
<tbody>
<tr>
<td>I Olfactory (S)</td>
<td>Difficult: smelling a non-irritant substance</td>
</tr>
<tr>
<td>II Optic (S)</td>
<td>Following dropped cotton wool; menace; PLR; negotiating obstacles</td>
</tr>
<tr>
<td>III Oculomotor (MP)</td>
<td>PLR; eye movement and position; induce horizontal nystagmus in both eyes when head moved sideways</td>
</tr>
<tr>
<td>IV Trochlear (M)</td>
<td>Eye movement and position</td>
</tr>
<tr>
<td>V Trigeminal (SM)</td>
<td>Facial and nasal sensation; corneal and palpebral reflex; jaw tone/movement</td>
</tr>
<tr>
<td>VI Abducens (M)</td>
<td>Retraction of globe on corneal reflex</td>
</tr>
<tr>
<td>VII Facial (SMP)</td>
<td>Facial expression; blink in response to corneal/palpebral reflex; skin pinch; tear production</td>
</tr>
<tr>
<td>VIII Vestibulocochlear (S)</td>
<td>Balance and head tilt; behavioural response to noise; nystagmus</td>
</tr>
<tr>
<td>IX Glossopharyngeal (SMP)</td>
<td>Gag or swallow reflex</td>
</tr>
<tr>
<td>X Vagus (SMP)</td>
<td>Gag or swallow reflex; laryngeal movement</td>
</tr>
<tr>
<td>XI Accessory (M)</td>
<td>Neck muscle mass – difficult to assess</td>
</tr>
<tr>
<td>XII Hypoglossal (M)</td>
<td>Tongue movement and atrophy; resistance to tongue extension</td>
</tr>
</tbody>
</table>

S = sensory; M = motor; P = parasympathetic
3.10 Nervous system

**CENTRAL VS. PERIPHERAL VESTIBULAR SIGNS**

<table>
<thead>
<tr>
<th>Head tilt/ataxia</th>
<th>Yes</th>
<th>Yes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Proprioceptive defects</td>
<td>No</td>
<td>Ipsilateral</td>
</tr>
<tr>
<td>Paresis</td>
<td>No</td>
<td>Ipsilateral</td>
</tr>
<tr>
<td>Nystagmus</td>
<td>Horizontal or rotary</td>
<td>Horizontal, rotary or vertical</td>
</tr>
</tbody>
</table>

**EFFECT OF SPINAL LESIONS**

<table>
<thead>
<tr>
<th>Site of lesion</th>
<th>C1–C5</th>
<th>C6–T2 = brachial plexus</th>
<th>T3–L3</th>
<th>L4–S3 = lumbosacral outflow</th>
</tr>
</thead>
<tbody>
<tr>
<td>Forelimb</td>
<td>UMN</td>
<td>LMN</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>Hind limb</td>
<td>UMN</td>
<td>UMN</td>
<td>UMN</td>
<td>LMN</td>
</tr>
</tbody>
</table>

**LMN damage**
- Loss of voluntary motor activity
- Loss of reflex motor activity
- Loss of resistance to passive movement (tone) resulting in flaccidity
- Rapid muscle atrophy
- Loss of sensation
- Loss of proprioception

**UMN damage**
- Limited voluntary movement
- Intact local reflexes which may be hyperactive (due to loss of higher control)
- Increased tone
- Occurrence of abnormal reflexes
- Loss of conscious sensation

- **Vestibular signs** (peripheral – semicircular canals; central – brain stem/cerebellum) are head tilt, ataxia, nystagmus and circling (see table, top of page). CN VII and VIII signs can be present with disease of the bullae.
- **Cerebellar lesions** lead to intention tremor, hypermetria, and ataxia.
- **Spinal lesions** can be localized by checking motor abnormalities in each limb separately (see table above). Differential signs between left and right limbs suggest lateralization.
- Upper (UMN) and lower (LMN) motor neuron damage result in different deficits (see table above).
  - Note that sensation, deep pain and proprioception are sensory and do not fit into the UMN/LMN scheme for localization.
- The presence of a flexor/withdrawal reflex is not the same as pain perception.
- **Bladder innervation:** see 3.7.1.
DIAGNOSIS

3.10.4 Diagnostic tests

- Good physical examination remains key to localizing the likely site of a lesion and directing further diagnostic tests.
- Routine haematology and biochemistry (including dynamic bile acids or ammonia) are important to exclude underlying systemic disease.
- Imaging: plain radiographs are generally of limited value.
  - Advanced imaging is generally necessary to further evaluate the spinal cord (myelography, CT +/- myelography, MRI) and brain (CT [gross lesions], MRI).
  - Scan design/acquisition is just as important as image analysis, because of the potential for creating artefacts, or for missing pathology.
- Other tests: cerebrospinal fluid evaluation, electromyelography, nerve and muscle biopsies may be required as appropriate.

TREATMENT

3.10.5 Approach to management of neurologic disease

- Management and therapy are heavily dependent on the location and underlying cause of the neurologic signs. Broadly, three options exist:
  - Surgery, to correct anatomic defects or remove structures (disc material, haemorrhage, tumours) that may be causing compression.
  - Medical therapy, most commonly used to manage inflammatory, infectious and neoplastic disease or seizures and to provide pain relief.
  - Physiotherapy, to maintain/improve function.

NUTRITION

3.10.6 Nutrition and neurologic disease

- For most neurologic disease, specific nutritional management is not required.
- Nutritional deficiencies causing neurologic signs are rare, e.g. thiamine deficiency, arginine in cats.
- Increasing antioxidant levels (vitamin E), mitochondrial cofactors (vitamin B6) and co-enzyme Q, as well as some nutraceuticals (Ginkgo biloba) may be helpful in managing cognitive dysfunction.
The most common presentation relates to difficulty in walking. This is usually due to depression/loss of voluntary movement, altered spinal reflexes, changes in muscle tone, muscle atrophy, or sensory dysfunction. Differential diagnoses for difficulty in walking include weakness/collapse associated with orthopaedic, cardio-respiratory, intoxicant, hypotensive, brain, muscular or neuromuscular disease or pain.

Major differential diagnoses for spinal cord disease are: intervertebral disc, atlantoaxial subluxation, neoplasia, infection (toxoplasma, neospora, distemper, FIP), inflammation (GME, steroid-responsive meningitis), fracture/luxation, fibro-cartilaginous embolism (FCE), ataxia due to proprioceptive deficits.

**Physical examination:** examine cranial nerves to rule out multifocal disease (see table, page 118); evaluate the spine (pain, muscle spasm, panniculus); evaluate limbs (muscle mass, tone, segmental reflexes, sensation and deep pain, voluntary movement, proprioception). See table, above.

**Diagnosis:** once the lesion has been localized, further diagnostics would include plain radiography and myelography, CSF evaluation, CT or MRI.

**Treatment** for chronic spinal disease depends on diagnosis, rate of progression of clinical signs and owner’s finances.

Options are surgical, conservative (strict cage rest, at home or in hospital depending on severity, continence, etc.) and medical (for spinal trauma) aimed at maintaining CNS perfusion.

Management of the paralysed patient:
- Bladder expression (manual or catheter).
- Keep clean and dry.
- Turn frequently.
- Ensure adequate fluid and calorie intake, as the patient may not be able to reach food/water.
- Enemas as required.
- Physiotherapy.
- Pain relief as required.
3.10.8 **Horner’s syndrome**
- Horner’s syndrome is classified as 1st, 2nd or 3rd order.
- **History and physical examination:** Miosis is always present. Other signs may include third-eyelid prolapse, enophthalmia and/or ptosis.
- **Diagnosis:** Most cases are 3rd order and idiopathic, but if necessary, pharmacologic testing with phenylephrine can be performed.
  - Localization of the lesion: varies from midbrain through spinal cord to T1–T3 up the vagosympathetic trunk and cranial cervical ganglion next to the tympanic bulla.

3.10.9 **Muscular disease**
- The majority of myopathies are acquired but some inherited conditions exist, e.g. centronuclear myopathy of Labrador retrievers, or muscular dystrophy in Siamese and Maine coon cats. See table above right for examples.
- **History and physical examination**
  - Myopathies are associated with muscle weakness or pain, reduced exercise tolerance and abnormal (stiff) gait.
  - Sensation is normal: reflexes may be slow due to decreased motor ability.
  - Due to the lack of a nuchal ligament, muscular weakness in cats commonly presents as neck ventroflexion.
- **Diagnosis** should be via history, including video of signs, and physical examination.
- Further tests include muscle enzymes (AST, CPK), electrolytes, triglyceride, cholesterol, retrovirus status, *Toxoplasma/Neospora* titres, 2M and acetyl choline receptor antibodies, muscle biopsy (check handling instructions), edrophonium tests and electromyelography.
- **Treatment** for neuromuscular disease is dependent on diagnosis.
- **Nutrition:** dietary support with antioxidants and nutraceuticals may be helpful in some diseases.

3.10.10 **Neuromuscular junction disease**
- Function of the neuromuscular junction can be affected by disease, by drugs such as aminoglycosides and by toxins, e.g. organophosphates, botulism or snake venom.
- In North America and Australia, tick paralysis is the result of neuromuscular junction dysfunction.
- Myasthenia gravis is the most common junctionopathy due to immune-mediated destruction of acetylcholine receptors (AChR).
Physical examination: findings may include stiff gait, pain, atrophy of muscles, weakness on exercise (myasthenia) or complete, flaccid paralysis (botulism) – may be generalized or focal.

Diagnosis can be difficult; consider serum cholinesterase activity, AChR antibody levels and electromyography.

Treatment is symptomatic and supportive: antitoxins and pyridostigmine, with immunosuppression for myasthenia.

### 3.11 Endocrine system

#### 3.11.1 Presentation

Endocrine disease is relatively common in cats (hyperthyroidism and diabetes mellitus) and dogs (diabetes mellitus, hypothyroidism, hyperadrenocorticism).

Each endocrine disease presents differently and requires different diagnostic testing and treatment.

The possibility of endocrine disease should be considered in patients with a wide range of clinical signs, such as PU/PD/PP (2.8), weight loss (2.7), inappetance, skin and coat changes (3.9); and especially if disease seems to be affecting a number of organ systems.
3.11.2 **DIABETES MELLITUS**

- Diabetes is the most common endocrinopathy in cats and dogs. It tends to affect middle-aged female dogs and male cats.
- Genetic predispositions exist in specific dog and cat breeds. Transient diabetes occurs in about 20% of cats and can be associated with oestrus in dogs.
- **Dogs**: nearly always type 1 diabetes due to a failure of pancreatic islet cell production of insulin.
- **Cats**: mix of type 1 and 2, i.e. reduced insulin production with increased end-organ resistance to insulin. Such cases still require insulin therapy.

**History and physical examination**

- PU/PD/PP is the most common presentation.
- Less commonly, cases present:
  - Collapsed with vomiting associated with diabetic ketoacidosis.
  - Having gone blind (dogs) due to cataracts.
  - In generally poor condition and with reduced ability to jump (cats).

**Diagnosis**

- Persistent hyperglycaemia and glycosuria need to be shown.
- Other causes of hyperglycaemia should be considered.
- Glycosuria in the absence of hyperglycaemia can occur in association with renal tubular damage.
- Hyperglycaemia needs to exceed the renal threshold (about 10 mmol/l) to cause typical diabetic clinical signs.

**Treatment**

- Virtually all patients require insulin therapy to manage their diabetes; lente insulin is an appropriate choice.
- The starting dose for a newly diagnosed diabetic: 0.25–0.5 iU/kg q12–24 hr.
- The dose should be revised no more frequently than every 5–7 days.
- Longer-acting insulins (protamine or glargine) are needed in some cases, particularly cats.
- Monitor the glucose curve, and fructosamine/glycosylated haemoglobin.
- Common causes of poor response to therapy include:
  - Administration problems – syringe filling, insulin quality, injection site.
  - Dose as yet inadequate.
  - Insulin resistance if dose >2 iU/kg: look for intercurrent disease, e.g. urinary infection, pancreatitis, inflammation, neoplasia.
  - Somogyi overswing.
There are several other adjunctive therapies, such as chromium, acarbose, metformin, sulphonylureas and herbs. They are rarely indicated and of limited efficacy.

**Nutritional management**

- **Dogs**: treat obesity, increase fibre and digestible carbohydrate. Fat and protein <30% of metabolizable energy; maintain consistent calorie intake; feed 30 minutes before insulin.
- **Cats**: treat obesity; maintain high-protein/high-fat diet.

**Selected topic**

- Basic management of diabetic ketoacidic crisis involves:
  - Baseline data from blood sample: urea/creatinine, glucose, electrolytes, phosphate, calcium, PCV, acid/base (if possible).
  - Fluid therapy: 0.9% NaCl with 40 mmol/l potassium.
  - Bicarbonate if $\text{HCO}_3^- < 12$ (dog), <10 (cat): 0.5 mmol/kg slow i/v.
  - Neutral insulin 0.2 iU/kg i/m repeat 0.1–0.2 iU/kg every 1–2 hours.
  - Parameters should be monitored and treatment revised depending on response.

**Hypothyroidism in Dogs**

**Introduction**

- Hypothyroidism can be congenital (rare) or acquired, secondary to immune-mediated thyroiditis.

**History and physical examination**

- The onset of clinical signs can be subtle and slow (see table below).

### Clinical Signs of Hypothyroidism in Dogs

<table>
<thead>
<tr>
<th>Metabolic</th>
<th>Lethargy; dullness; exercise intolerance; obesity; hypothermia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Skin</td>
<td>Seborrhoea; alopecia; dorsal scaling; coat is dull, bleached, brittle; refractory pyoderma; hyperpigmentation; comedones</td>
</tr>
<tr>
<td>Neuromuscular</td>
<td>Peripheral polyneuropathy; peripheral vestibular signs; ataxia; seizures; coma; myxoedema; myopathy</td>
</tr>
<tr>
<td>Rare</td>
<td>Cardiovascular; reproductive; ophthalmic; behavioural; skeletal (cretinism)</td>
</tr>
</tbody>
</table>
Diagnosis and testing

- Diagnosis is based on appropriate history, supporting clinical signs, biochemical changes (mild non-regenerative anaemia, raised cholesterol and triglycerides, elevated ALKP), consistent thyroid function tests and response to therapy.
- Total T\textsubscript{4} is lowered by non-thyroidal illness (NTI), drugs (corticosteroids, sulphonamides, barbiturates), breed (e.g. sight hounds), age and time of day.
- Free T\textsubscript{4} is less affected by non-thyroidal factors but needs to be measured by equilibrium dialysis.
- Interpretation of thyroid-stimulating hormone (cTSH) and TT\textsubscript{4} results (see also table above):
  - Low TT\textsubscript{4} and high cTSH is consistent with hypothyroidism.
  - 20–40% of dogs with low TT\textsubscript{4} and normal cTSH are hypothyroid.
  - High cTSH with normal TT\textsubscript{4} is associated with early hypothyroidism, recovery from NTI, or use of certain drugs.
- Dynamic testing is rarely necessary, but a TSH stimulation test is the most useful.
- Autoantibodies:
  - Thyroglobulin autoantibodies are a marker for subclinical lymphocytic thyroiditis and future hypothyroidism.
  - T\textsubscript{3} and T\textsubscript{4} autoantibodies give spuriously elevated radio-immunoassay results in dogs that may be hypothyroid.

Treatment

- Treatment is by levothyroxine hormone replacement, via liquid and tablet preparations.
  - The starting dose is 20 μg/kg q24hr or 10 μg/kg q12hr.
  - Monitor response (T\textsubscript{4} level 6 hours post medication) and alter dose as necessary. Full response can take 6–8 weeks. If there is no response at this stage, re-evaluate the diagnosis.
3.11.4 Hyperthyroidism in cats

**Introduction**
- Hyperthyroidism is a very common endocrinopathy in older cats, but there is marked regional variation.

**History and physical examination**
- Presenting clinical signs include: weight loss (90% of cases); polyphagia (60%); PU/PD (50%); hyperactivity (40%); GIT signs (40%); poor coat quality (35%); respiratory signs (25%); inappetence (15%); lethargy (10%); weakness (10%).
- Rare signs (<10% of cats) include tremors/seizures, heat intolerance, haematuria and neck ventroflexion.
- On physical examination, >90% have one or more of: a palpable goitre, tachycardia, gallop rhythm, skin changes, hyperthermia, dehydration/cachexia.

---

**Diagram:**
- **Hyperthyroidism possible**
  - T<sub>4</sub> >75 mmol/l → Diagnosis confirmed
  - T<sub>4</sub> 50–75 mmol/l → Repeat T<sub>4</sub> in more than one week’s time
  - T<sub>4</sub> 25–50 mmol/l → Clinical signs compatible but T<sub>4</sub> persistently low
  - T<sub>4</sub> <25 mmol/l → Seek an alternative diagnosis

  - T<sub>4</sub> still >25 and <75 mmol/l → No
    - Alternate diagnostics: Free T<sub>4</sub>, T<sub>3</sub> suppression, TSH stimulation, Scintigraphy
  - Yes → Suggests a second disease process is lowering T<sub>4</sub>. Further investigation required
Diagnosis and testing
- See algorithm, previous page.
- Common clinical pathology changes include elevated red cells with macrocytosis, leucocytosis–neutrophilia, eosinophilia; increased ALT, AST, ALKP, LDH, phosphate and glucose; and azotaemia.

Treatment
- Choice of treatment will depend not only on individual patient factors, but also on the owner’s finances and philosophy.
  - **Radioactive iodine**: generally curative with low side-effect rates, but extended hospitalization required; moderate risk of hypothyroidism.
  - **Surgery**: generally curative with moderate risks and recurrence rates.
  - **Anti-thyroid drugs**: moderate side-effects; needs to be continued indefinitely.
  - **Diet management**: may prove useful.

3.11.5 HYPERADRENOCORTISM (CUSHING’S DISEASE)

Introduction
- This is uncommon in cats, but occurs in pituitary and adrenal forms in dogs, with a female bias (poodles, dachshunds, terriers, beagles, GSD, boxers).

If clinical signs/pathology are poorly supportive, but adrenal function tests are abnormal, it is easy to make a false diagnosis of hyperadrenocorticism.

History and physical examination
- Clinical signs include PU/PD/PP, abdominal enlargement, exercise intolerance, panting, lethargy, obesity, alopecia, calcinosis cutis, anoestrus/testicular atrophy, comedones and thin skin.

Diagnosis and testing
- Clinical pathology includes stress leukogram, raised ALKP (can be marked), ALT, cholesterol, bile acids, glucose, low urine SG often with proteinuria and UTI.
- Adrenal function testing is unreliable: sensitivity and specificity are around 80–90%. There are no definitive tests and all can have false positive and negative results.
  - Consider the urine creatinine:cortisol ratio (for a negative rule-out), ACTH stimulation or low-dose dexamethasone suppression test (LDDST).
  - Distinguish between pituitary-dependent and adrenal-dependent forms of hyperadrenocortism (PDH and ADH) by endogenous ACTH level, or high-dose DST.
**Imaging:** radiographs show hepatomegaly, good abdominal contrast, pot-belly and adrenal calcification.
- Measurement of adrenal glands on ultrasound: if left >7.4 mm and/or right >8.1 mm), this is 70–80% specific and sensitive for hyperadrenocorticism.
- CT or MRI can be used to show adenoma of the pituitary.

**Treatment**
- Initial management: PDH therapy via trilostane (starting dose 2–4 mg/kg q24hr) or mitotane manages the clinical signs, but does not treat the disease.
- For ADH, perform surgery if possible, if not then treat as PDH.

**HYPOADRENOCORTISIM (ADDISON’S DISEASE)** 3.11.6

**Introduction**
- This is an immune-mediated disease, most common in middle-aged female dogs (poodles, West Highland white terrier, Great Dane, bearded collie, basset hound are predisposed). It is a rare disease in cats.

**History and physical examination**
- Clinical signs tend to be non-specific and to wax and wane. They include inappetence, lethargy/depression and weight loss.
- Other common signs include GIT signs, shaking, shivering, PU, and collapse.

**Diagnosis and testing**
- Clinical pathology is likely to show several of: lack of a stress leukogram, low sodium and elevated potassium, azotaemia, hypoglycaemia, non-ionized hypercalcaemia, low urine SG.
- The definitive test is ACTH stimulation of cortisol and aldosterone.

**Treatment**
- Initial management is by:
  - Fludrocortisone (0.01 mg/kg po q12hr).
  - Or, desoxycorticosterone pivalate (2 mg/kg i/m or s/c monthly) plus prednisolone (0.25–0.5 mg/kg po q24hr).
- Crisis management is by fluid therapy (0.9% NaCl).

**Nutritional management**
- Where sodium levels are hard to maintain, some patients benefit from salt supplementation to the diet – initially 0.5 mg/kg/day.

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Do not use ‘low salt’ type products, as these contain potassium chloride – fatalities have been reported.
3.11.7 Differential diagnoses of hyper- and hypocalcaemia

**Hypercalcaemia**
- Parathyroid disease: primary, secondary CRD.
- Toxic (hypervitaminosis D) – due to excess supplements, ingestion of vitamin D-based creams, calciferol rodenticide.
- Neoplastic: lymphoma, anal sac adenocarcinoma, myeloma, bone metastasis.
- Non-ionized hypercalcaemia seen with hyperalbuminaemia, haemoconcentration, hypoadrenocorticism.
- Granulomatous and fungal disease.
- Acidosis.

**Hypocalcaemia**
- Hypoalbuminaemia, hypomagnesaemia, spurious due EDTA/oxalate contamination.
- Renal secondary hyperparathyroidism.
- Hypoparathyroidism, parathyroidectomy.
- Diet – inadequate vitamin D, excess phosphorus (all lean meat).
- Excess phosphate – i/v administration, phosphate enema.
- Malabsorption, pancreatitis.
- Ethylene glycol poisoning.
- Eclampsia, alkalosis, C-cell thyroid tumours.

**Acute management** is required if the patient is agitated/hyperaesthetic by calcium gluconate (10%) 0.5–1.5 (dog) 1–1.5 (cat) ml/kg slow i/v over 20–30 minutes, then monitor.

**Chronic management** is by oral calcium 15–25 mg elemental calcium q6–8hr with vitamin D (calcitriol or alphacalcidol).

3.11.8 **INSULINOMA**
- Insulinoma is the most common pancreatic tumour in dogs, but is rare in cats.
- Cases present with weakness/exercise intolerance, perhaps with collapse or seizure.
- **Diagnosis** is based on a raised insulin/glucose ratio and ultrasound/CT of the pancreas.
- Other causes of hypoglycaemia should be considered, e.g. liver failure, septicaemia, delayed measurement.
- **Treatment:** initial stabilization is with glucocorticoids, and frequent small meals containing complex carbohydrate. After this, surgery carries the best prognosis, especially if there is no gross metastasis.
Obesity


INTRODUCTION

Definition and prevalence of obesity

- Pets are defined as overweight when they are 10–20% above their optimal weight.
- 15–20% of cats are estimated to be overweight, and 15–30% of dogs.
- Pets are defined as obese when they are more than 20% above their optimal weight.
- 10% of cats, and 10–15% of dogs, are estimated to be obese.
- Recommended weight ranges for various breeds exist, but the range can be wide, especially within breeds that have significant size variations, e.g. between working and show labradors.

HISTORY AND PHYSICAL EXAMINATION

Disease risks associated with obesity

- History and physical examination should focus on evidence of systemic disease. Disease risks associated with obesity include:
  - Degenerative joint disease.
  - Respiratory difficulties.
  - Decreased cardiac reserve.
  - Insulin resistance.
  - Poorer response to infectious disease.
  - Fatty infiltration of the liver.
  - Increased surgical/anaesthetic risk.
  - Feline lower urinary tract disease.

Body condition scoring

- It is relatively easy to teach owners to evaluate their pets; their score can be compared with yours at each consultation.
- The table on the next page shows a 5-point scoring system, but a 9-point system is also commonly used.

DIAGNOSIS

Testing for medical causes of obesity

- While physical examination is generally all that is required, if other intercurrent disease is suspected, routine haematology and biochemistry may be appropriate.
In a limited number of cases, specific endocrine testing may be appropriate for:

- Hypoadrenocorticism (3.11.6).
- Acromegaly.
- Hypothalamic disorders.
- Insulinoma (3.11.8).
- Hypothyroidism (3.11.3).
- Hypopituitarism.
Exercise helps to change the balance between calorie intake and expenditure. A patient’s ability to exercise may be affected by their obesity and concurrent disease, such as degenerative joint disease. Optimum regimes for weight loss have not been defined, but limited studies suggest that even short periods (5–10 minutes) of exercise twice daily can have a significant effect.

Drug management of obesity

Two medical treatments for obesity are licensed for use in dogs. Dirilotapide (Slentrol) and mitratapide (Yarvitan) reduce appetite and fat absorption through enterocytes. They are appropriate when initial attempts at dietary management have failed, or in some cases as an adjunct to diet.

Modifying pet and owner behaviour

Most owners know their pet is obese, but don’t know how to deal with it effectively. A combination of options should be considered, addressing causes of overfeeding in particular:

- Lack of adjustment to individual needs.
- Energy intake from snack/treats ignored.
- Eating encouraged as ‘a sign of good health’.
- Begging behaviour indulged.
- Food provided when the pet is left alone.
- Excessive use of food as a training aid.
- Association with owner’s attempts to diet.

Weight-loss diets are available that are nutritionally balanced, with low calorie density, to increase satiety when fed. These diets are easier for most owners than a reduction in the patient’s normal ration.

Owner support and regular follow-up increase success rate.

Weight-loss targets

Safe weight loss is 1–1.5% per week, to achieve a 15% loss in 15–18 weeks. An initial target loss of 15% should be set, with calorie intake at 60% of the maintenance energy requirement (MER) for the target weight (see 1.1.8 for calculation). If weight loss is not occurring, consider: lack of household compliance; alternative food sources; endocrinopathy; insufficient time allowed. If these can be ruled out, then re-calculate and decrease calorie intake by a further 15%.
3.13 **Infectious disease**

3.13.1 Scope of this section  
3.13.2 Feline infectious disease  
3.13.3 Canine infectious disease  
3.13.4 Overview of antimicrobial treatment  
3.13.5 Drug choice by body system  
3.13.6 Oral absorption of antimicrobials  
3.13.7 Patient considerations  
3.13.8 Side-effects and toxicity of antimicrobial groups  
3.13.9 Potential toxicity by body system

**INTRODUCTION**

3.13.1 Scope of this section

- This section focuses on common infectious diseases of dogs and cats. It is clearly not possible to cover all infectious diseases (see Further Reading, page 190). References to certain other infections appear under each body system as appropriate.

---

### COMMON FELINE INFECTIOUS DISEASES

<table>
<thead>
<tr>
<th>Disease</th>
<th>Leukaemia virus</th>
<th>Viral respiratory infection</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Organism</strong></td>
<td>Oncogenic retrovirus</td>
<td>Herpes or calcivirus</td>
</tr>
<tr>
<td><strong>Route of infection</strong></td>
<td>Close contact, saliva; in utero; nursing; contaminated blood</td>
<td>Oronasal, conjunctival</td>
</tr>
<tr>
<td><strong>Incubation</strong></td>
<td>2–4 wks: non-specific signs; 2 yrs: FeLV-related disease</td>
<td>2–5 days</td>
</tr>
<tr>
<td><strong>Infective period</strong></td>
<td>All the time the cat is viraemic: poor survival in the environment</td>
<td>During viraemic carrier state</td>
</tr>
<tr>
<td><strong>Clinical signs</strong></td>
<td>Lymphoma, leukaemia (rare); immunosuppression and PUO; auto-immune disease; bone-marrow suppression, especially anaemia; uveitis; gastroenteritis</td>
<td>Pyrexia, ptyalism, sneezing, ocular discharge, mouth ulceration, keratitis</td>
</tr>
<tr>
<td><strong>Diagnosis</strong></td>
<td>FeLV antigen ELISA; VI; PCR; immunofluorescence</td>
<td>Culture or PCR</td>
</tr>
<tr>
<td><strong>Treatment</strong></td>
<td>Supportive</td>
<td>Supportive</td>
</tr>
<tr>
<td><strong>Control</strong></td>
<td>Test and removal; vaccination; avoid exposure, even of vaccinated cats</td>
<td>Vaccination; environmental controls, e.g. sneeze barriers</td>
</tr>
<tr>
<td><strong>Prognosis</strong></td>
<td>Poor once clinical signs are apparent</td>
<td>Generally good</td>
</tr>
<tr>
<td><strong>Vaccine</strong></td>
<td>Moderately successful</td>
<td>Moderately successful</td>
</tr>
</tbody>
</table>
### COMMON DISEASES

#### Feline infectious disease
- Viral infectious disease is relatively common in cats (see table below), especially in kittens and cats held in large groups.
- Carrier states exist for a number of these infections with reactivation in periods of stress or reduced immune function.

#### Canine infectious disease
- Viral infections are comparatively rare in dogs in the UK, due to vaccination, but outbreaks do occur in unvaccinated groups. See table, next page.

<table>
<thead>
<tr>
<th>Immunodeficiency virus</th>
<th>Infectious peritonitis virus</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lentivirus</td>
<td>Coronavirus</td>
</tr>
<tr>
<td>Saliva (biting, fighting); perinatal</td>
<td>Oro-nasal; saliva; litter tray; in utero</td>
</tr>
<tr>
<td>3–6 wks: non-specific signs; 2–5 yrs: FIV-related disease</td>
<td>Experimentally: 5–10 days; Field: very variable; stress as a trigger</td>
</tr>
<tr>
<td>All the time cat is viraemic; poor survival in the environment</td>
<td>Sporadic occurrence in cat colonies, rarely epidemic</td>
</tr>
<tr>
<td>Chronic infection; ocular disease; stomatitis; wasting and diarrhoea; skin infection; renal failure</td>
<td>Can mimic almost any disease; cavity effusions (wet); granulomatous inflammatory disease (dry); uveitis; CNS signs; fever; renal; hepatic (jaundice); GIT</td>
</tr>
<tr>
<td>FIV antibody; ELISA; PCR</td>
<td>No definitive test available</td>
</tr>
<tr>
<td>Supportive; maintenance of general health</td>
<td>Supportive: antimicrobials, corticosteroids, interferon</td>
</tr>
<tr>
<td>Isolation of carriers; avoid saliva/blood transfer, e.g. via feeding bowls</td>
<td>Perinatal hygiene; avoid multiple stressors in kittens; review breeding lines with high incidence of FIP</td>
</tr>
<tr>
<td>Slow, chronic disease course</td>
<td>Very poor once clinical signs are apparent</td>
</tr>
<tr>
<td>USA: efficacy questionable</td>
<td>Europe: efficacy questionable</td>
</tr>
</tbody>
</table>
### COMMON CANINE INFECTIOUS DISEASES

<table>
<thead>
<tr>
<th>Disease</th>
<th>Parvovirus</th>
</tr>
</thead>
<tbody>
<tr>
<td>Organism</td>
<td>Canine parvovirus 2</td>
</tr>
<tr>
<td>Route of infection</td>
<td>Faecal–oral route; direct or indirect contact</td>
</tr>
<tr>
<td>Incubation</td>
<td>5–6 days</td>
</tr>
<tr>
<td>Infective period</td>
<td>3–6 days after recovery; viable virus 3–6 months</td>
</tr>
<tr>
<td>Clinical signs</td>
<td>Dull; anorexic; vomiting; haemorrhagic diarrhoea; puppies infected &lt;6 wks old – myocarditis and sudden death</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>Panleucopenia; serology; virus isolation; faeces; histopathology</td>
</tr>
<tr>
<td>Treatment</td>
<td>Symptomatic and supportive: fluid; nutrition; plasma; blood; antimicrobials; antiemetics; gastric protectants</td>
</tr>
<tr>
<td>Control</td>
<td>Highly infectious: cases need to be isolated</td>
</tr>
<tr>
<td>Prognosis</td>
<td>Good if aggressive early care</td>
</tr>
<tr>
<td>Vaccine</td>
<td>Highly effective</td>
</tr>
</tbody>
</table>

### LIPOPHILICITY OF ANTIMICROBIAL DRUGS

<table>
<thead>
<tr>
<th>Lipophilicity</th>
<th>Drug</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acidic, low lipophilicity drugs</td>
<td>Penicillins, cephalosporins</td>
</tr>
<tr>
<td>Basic, low lipophilicity</td>
<td>Aminoglycosides</td>
</tr>
<tr>
<td>Weak acid, moderate lipophilicity</td>
<td>Sulphonamides</td>
</tr>
<tr>
<td>Weak base, moderate lipophilicity</td>
<td>Lincosamides; erythromycin</td>
</tr>
<tr>
<td>Amphoteric, moderate lipophilicity</td>
<td>Tetracyclines</td>
</tr>
<tr>
<td>Highly lipophilic</td>
<td>Fluoroquinolones; doxycycline; chloramphenicol; metronidazole</td>
</tr>
</tbody>
</table>
3.13 Infectious disease

### Overview of antimicrobial treatment

- The type (especially off-licensed human antimicrobials) and frequency of use should be limited wherever practical.
- The main cause of apparent antimicrobial failure is that bacterial infection is not the primary pathology.
- Penetration to ‘protected’ sites requires movement across epithelial/mesothelial layers and is dependent on:
  - Lipid solubility: if highly lipid soluble, e.g. doxycycline, antimicrobial can easily cross membranes. See table, left.
  - Protein binding: affects drug availability to cross membranes, e.g. beta-lactams.
  - The ‘tightness’ of the barrier itself: the blood–CSF barrier is very tight, so only small molecules, lipid-soluble antimicrobials such as sulphonamides, or fluoroquinolones will penetrate into the CSF.
- Acid/base conditions of the site: basic antimicrobials, e.g. lincosamides, macrolides, are ion-trapped in the alkaline environment of the prostate.

### Distemper

<table>
<thead>
<tr>
<th>Morbillivirus</th>
<th>Leptospirosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aerosol or droplet exposure</td>
<td>Spirochete bacteria</td>
</tr>
<tr>
<td>3–10 days; 2nd fever spike 2 wks (max. 90 days) post-recovery</td>
<td>Ingestion; bite wounds; contaminated food or water</td>
</tr>
<tr>
<td>7 days post-infection until 1–2 wks (max. 90 days) post-recovery</td>
<td>Months–years following recovery. Killed by drying</td>
</tr>
<tr>
<td>Dull, anorexic; diarrhoea; cough and catarrhal oculonasal discharge; 50% develop neurologic signs</td>
<td>Peracute: death, shock, DIC Subacute: jaundice, DIC Chronic: progressive renal or hepatic disease</td>
</tr>
<tr>
<td>Leucopenia; inclusion bodies (conjunctiva); serology; virus isolation</td>
<td>Urine: dark-field microscopy or immunofluorescence Serology</td>
</tr>
<tr>
<td>Symptomatic and supportive: fluid; nutrition; antimicrobials; gastric protectants; antiemetics; antiseizure</td>
<td>Supportive: ensure adequate urine output; gastric protectants. Penicillin acutely; tetracyclines to prevent carrier</td>
</tr>
<tr>
<td>Highly infectious, but poor environmental survival of virus</td>
<td>Control of reservoir hosts</td>
</tr>
<tr>
<td>Guarded</td>
<td>Guarded</td>
</tr>
<tr>
<td>Highly effective</td>
<td>Moderate: only some serovars</td>
</tr>
</tbody>
</table>
3.13.5 Drug choice by body system
- A variety of alternatives is usually available for each body system: choice between them is based on the likely bacterium involved.
  - Bone: amoxyclav, cephalosporins, clindamycin, lincomycin.
  - Urinary tract: potentiated sulphonamides, penicillins, cephalosporins, tetracyclines.
  - Prostate: clindamycin, fluoroquinolone, doxycycline, trimethoprim.
  - Hepatobiliary: aminoglycosides, penicillins, cephalosporins, clindamycin, fluoroquinolone, metronidazole.
  - Respiratory: lipid-soluble drugs recommended.
  - CNS: chloramphenicol, potentiated sulphonamides, fluoroquinolone, metronidazole.
  - 'Four quadrant cover': for cases where there may be infection with a wide variety of bacteria that are both Gram-positive and negative, aerobes and anaerobes, e.g. septic peritonitis. This consists of a combination of:
    - Amoxyclav or cephalosporins (group I or II) or gentamicin.
    - Fluoroquinolone.
    - Metronidazole or group IV cephalosporins (cefoxitin).

3.13.6 Oral absorption of antimicrobials
- Poor or variable absorption may be noted for the following:
  - Ampicillin: decreased by feeding.
  - Erythromycin: may be a gastric irritant.
  - Tetracyclines: inhibited by milk.
  - Third-generation cephalosporins.
  - Aminoglycosides.

3.13.7 Patient considerations
- For neonates, increase initial dose and lengthen the interval.
- The presence of other disease may affect the appropriate antimicrobial, dose or frequency (see table, above right).

3.13.8 Side-effects and toxicity of antimicrobial groups
- Aminoglycosides: nephrotoxic, ototoxic, affect neuromuscular junction, e.g. GIT motility. Take care with streptomycin in cats.
- Tetracyclines: catabolic; may cause GIT upset, especially in cats; tooth discoloration; hepatotoxic – take care if patient is azotaemic; oesophageal stricture in cats.
- Quinolones: GIT upset; arthropathy; blindness; seizures; retinal degeneration in cats.
- Sulphonamides: keratoconjunctivitis sicca (KCS); polyarthropathies; skin eruptions; thrombocytopenia; Dobermans especially at risk.
3.13 Infectious disease

### Potential toxicity by body system

- **Nephrotoxicity:**
  - Proximal tubule – aminoglycosides, polymyxins, tetracyclines.
  - Crystalluria – sulphonamides.
- **Hepatotoxicity:**
  - Parenchymal damage – tetracyclines; erythromycin.
- **Neurotoxicity:**
  - Ototoxic – aminoglycosides; sulphonamides; lincosamides.
  - Neuromuscular blockade: aminoglycosides.
  - Blindness in cats: fluoroquinolones.
  - Seizures (in susceptible patients): fluoroquinolones, metronidazole.
- **Cardiovascular:** aminoglycosides; chloramphenicol; lincosamides; sulphonamides.
  - Tetracyclines cause transient depression of output and vasodilation, so avoid rapid i/v injection.
- **Gastrointestinal tract:**
  - Mucosal damage – ampicillin; lincomycin; clindamycin.
  - Vomiting – erythromycin.
  - Oesophageal stricture – doxycycline; clindamycin.
- **Bone marrow toxicity:** chloramphenicol.
- **Arthropathy:** fluoroquinolones.

### ANTIMICROBIALS AND OTHER DISEASE

<table>
<thead>
<tr>
<th>Renal disease</th>
<th>Hepatic disease</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Not affected</strong></td>
<td>Doxycycline; clindamycin; erythromycin</td>
</tr>
<tr>
<td><strong>Decrease dose</strong></td>
<td>Aminoglycosides; sulphonamides; quinolones; cephalosporins</td>
</tr>
<tr>
<td><strong>Double interval</strong></td>
<td>Penicillins; lincomycin</td>
</tr>
<tr>
<td><strong>Contraindicated</strong></td>
<td>Neomycin; tetracyclines; polymyxin; nitrofurantoin</td>
</tr>
</tbody>
</table>

- Erythromycin: may cause vomiting, so give with food, but note that this decreases absorption.
- Furantoin: GIT upset; hepatotoxic.
- Chloramphenicol: anaemia; dose-dependent; non-regenerative anaemia, especially in cats.
3.14 Oncology

3.14.1 Prevalence of neoplasia

Approximately 30% of sick medical cases have neoplasia as a cause of their presenting signs.

Neoplasia is the cause of death in 45% of dogs over 10 years of age.

3.14.2 Lumps

Owners should be asked about lumps/bumps they have noticed as they groom or stroke their pet, as these can be significant and are easily missed during physical examination. The history should be augmented by careful physical examination.

3.14.3 Required tests

Changes in routine haematology and biochemistry are not uncommon.

Imaging is crucial for evidence of non-cutaneous mass lesions.

Cytology/histology is required for definitive diagnosis.

The presence of a mass on palpation/imaging does not make that mass neoplastic, nor is it possible to palpate a skin lump to determine whether it is a lipoma.

3.14.4 Therapeutic modalities

For many tumours the most effective therapy requires a multimodality approach.

Surgery (see 4.3):

- For diagnosis and treatment of localized neoplasia.
- Can be curative, with appropriate margins.
- Maximum information (tumour type, extent, spread) prior to a first surgery gives the best outcome.
- Marking the edges of a removed tumour with tissue paints or variable suture numbers helps in assessing tumour-free margins.
- Note that post-operative analgesic requirements can be high.
- See also 4.3.4 – approach to a lump.
Radiation:
- Primarily for treatment of localized tumours.
- Side-effects are common – those seen depend on the area irradiated.
- Limited availability:
  - Dose needs to be fractionated, so repeat treatments required.
  - Multiple small fractions are more effective.
- Delivery of radiation is by orthovoltage units, radioactive sources, or linear accelerator (most common in UK). The optimal source will depend on type and position of the tumour being treated.
- Potential cases should be discussed with a radiation oncologist.

Chemotherapy has been tried in virtually every tumour described. Many tumours have limited sensitivity. The published data are often limited as to the true increase in survival that chemotherapy can deliver.
- Multi-agent treatment tends to be more effective than single-agent.
- Mild side-effects are common, severe side-effects are relatively rare.
- Metronomic therapy is repetitive, low-dose chemotherapy that aims to minimize toxicity.

Other therapies:
- COX-2 inhibitors, e.g. piroxicam, meloxicam, have been used in the management of transitional cell carcinomas. Their efficacy in other carcinomas is unknown.
- Tyrosine kinase receptor inhibitors are an alternative to chemotherapy for non-resectable mast cell tumours. Side-effects can be significant.
- Vaccines: xenogeneic DNA vaccine is available for certain melanoma types.
- Other therapies include immunotherapy, photodynamic, antiangiogenic, and gene therapy.

Patients with lymphoma that have been treated with prednisolone prior to diagnosis have shorter survival times.

Common chemotherapy drugs
- Suitably trained personnel and appropriate health and safety protocols should always be followed.
- Contaminated waste should be disposed of in specifically designated containers.
- Injectable drugs should be drawn up in a fume cupboard or using other safety devices, such as PhaSeal™. Otherwise, swabs should be wrapped around the neck of the bottle to contain aerosolized particles of drug.
Tablets should never be split.

Good record keeping is essential in case of adverse events.

**Prednisolone** is lymphocytolytic to neoplastic lymphocytes: initial dose rate 2 mg/kg or 40 mg/m². It may also reduce tumour-associated inflammation and, therefore, tumour size.

**Vincristine** interferes with microtubule assembly; used primarily to treat lymphoproliferative disorders. Its value in thrombocytopenia is questionable. Always give via i/v catheter.

- **Dose for lymphoma**: 0.5–0.75 mg/m² maximum once weekly.
- **Side-effects**: bone-marrow suppression usually mild; peripheral neuropathy; constipation; severe perivascular irritant.

**Cyclophosphamide** crosslinks DNA preventing synthesis and function; used to treat lympho- and myelo-proliferative and immune-mediated disease, and some sarcomas and carcinomas.

- **Dose**: dependent on protocol.
- **Side-effects**: myelosuppression (nadir 7–14 days); sterile haemorrhagic cystitis; GIT upset; hepato- and nephro-toxicity; reduced hair growth.

**Chlorambucil** is similar to, and a substitute for, cyclophosphamide but does not cause sterile cystitis; used as an immunosuppressive agent, for management of small cell lymphoma in cats, and for chronic lymphocytic leukaemia.

- **Dose**: dependent on disease.
- **Side-effects**: anorexia; nausea; vomiting; rarely, bone marrow suppression.

**Azathioprine** acts to inhibit purine synthesis necessary for cell proliferation in leucocytes; used as an immunosuppressive agent; potentially highly toxic in cats.

- **Dose**: in dogs, 2 mg/kg q24hr until remission, then 0.5–2 mg/kg q48hr.
- **Side-effects**: bone marrow suppression can be severe in some dogs, due to deficiency of an enzyme required for metabolism.

3.14.6 **Calculating the dose of chemotherapy agents**

Most chemotherapeutic agents have a relatively narrow therapeutic window.

Drug dosing by body weight tends to underestimate the dose in small patients and overestimate in large ones. Body surface area (BSA) is closer to the metabolic ability; it is based on body weight. See table, above right.

Adjustment should be made for obese or ascitic patients and a ‘guesstimate’ made of their ideal weight, or weight without fluid.
Dietary management of neoplasia

- Maintaining body weight improves survival times in patients with neoplasia.
- Omega-3 polyunsaturated fatty acids reduce tumour necrosis factor-α, which can be associated with weight loss.
- Inappetence can be a major factor, so highly-palatable, energy-dense diets are recommended.
- Energy is best provided as fats. Energy requirement can be twice that of a similarly-sized animal without neoplasia.
- Protein levels should be increased at 30–45% (dog), 35–45% (cat) of metabolizable energy. High-dose arginine supplementation (>2% on dry matter basis) may be helpful.
- A wide variety of other food ingredients and nutraceuticals have been recommended for cancer patients, but evidence of efficacy is lacking.
3.14.8 Paraneoplastic syndromes

- These are disease states that occur secondary to the metabolic, endocrine or haematologic effects of a tumour.
- These effects can be distant from the tumour itself and be responsible for the clinical presentation, e.g. hypercalcaemia associated with an anal sac carcinoma, causing a presentation for PU/PD.

**Haematologic**
- Anaemia due to chronic disease, bone marrow invasion, immune-mediated or microangiopathic haemolysis, GI and external loss, hypersplenism.
- Thrombocytopenia and altered coagulation (DIC).
- Hyperviscosity due to monoclonal gammopathy, polycythaemia.
- Leucocytosis and eosinophilia – rare.

**Metabolic and endocrine**
- Cancer cachexia involves anorexia, weight loss, fatigue and immune dysfunction; maintenance energy requirement can double in cancer cases. Cancer cachexia is caused by alterations in carbohydrate, protein and lipid metabolism, and it decreases survival times.
- Fever induced by cytokines accounts for about 10% of cases presenting with PUO.
- Hypercalcaemia due to release of parathyroid-related peptide (PTH-rP).
- Hypoglycaemia is observed with insulinomas, but also liver, pulmonary and muscle tumours. It can be secondary to sepsis, e.g. GI tumour rupture.
- Hyperhistaminaemia: release from mast cells causing local oedema, erythema and pruritus and distant GI ulceration.

**Neuromusculoskeletal**
- Demyelination and axonal degeneration; there is probably an underlying autoimmune mechanism.
- Myasthenia gravis; usually secondary to thymoma.
- Hypertrophic pulmonary osteopathy (Marie's disease).

**Dermatologic**
- Hepatocutaneous syndrome.
- Alopecia (cats).
- Exfoliative dermatitis (in cats with thymoma).
- Small, hyperplastic, dermal collagenous nodules.
ANAESTHESIA, ANALGESIA AND SURGERY

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4.2 Analgesia 150
4.3 Soft-tissue surgery 156
4.4 Orthopaedics 165
4.1 Anaesthesia

4.1.1 INTRODUCTION
- For any procedure, the choices of sedation or anaesthesia, and of which agent(s) to use are very situation-dependent. For a particular case consider:
  - The patient’s age, breed and temperament.
  - How still the patient is required to be.
  - The pain and duration of the procedure.
  - Whether a secondary procedure may become necessary.
  - Primary and intercurrent disease (if any).
- Heavy sedation with α2-agonists can cause profound cardiorespiratory depression. Light anaesthesia is often safer than heavy sedation.
- In some circumstances, local or regional anaesthesia should be considered, usually in combination with sedation or GA.

4.1.2 SEDATION AND PREMEDICATION
- Sedative choice will depend on the individual patient’s temperament, procedure being undertaken and underlying disease.
- Examples of protocols in routine use, with suggested dose rates, are shown in the table, right. The drug’s effect is generally more reliable if given by i/m rather than s/c injection. Use lower doses for light sedation, as a premedicant, or in sick patients.

4.1.3 USE OF INDUCTION AGENTS
- Induction (see table, page 148) should induce rapid, safe and stress-free unconsciousness.
- Induction should be sufficient to allow intubation, if used; and to allow the maintenance protocol to become effective.

Practice tips
- Don’t forget to pre-oxygenate where appropriate.
- To smooth the induction and reduce the dose of induction agent required, give diazemuls (0.5 mg/kg) i/v 20–30 minutes after premedication and before induction.

4.1.4 MAINTENANCE OF ANAESTHESIA
- Anaesthesia can be maintained by CRI or intermittent bolus injection of propofol or alfaxalone. Ideally a tube should be placed and oxygen given.
- The majority of anaesthetics are maintained by volatile gaseous agents.
### COMMON SEDATION AGENTS

<table>
<thead>
<tr>
<th>Agent and dose rate</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>ACP</strong> (0.01–0.02 mg/kg) <strong>+</strong> buprenorphine (0.01–0.02 mg/kg)</td>
<td>Light sedation for minor, non-painful procedures, e.g. ultrasound or premedicant for mild–moderately painful surgery ± NSAID</td>
</tr>
<tr>
<td><strong>ACP</strong> (0.01–0.02 mg/kg) <strong>+</strong> butorphanol (0.1–0.3 mg/kg)</td>
<td>More profound sedation than buprenorphine, premedicant for mildly painful procedures</td>
</tr>
<tr>
<td><strong>ACP</strong> (0.01–0.02 mg/kg) <strong>+</strong> methadone* (0.1–0.3 mg/kg)</td>
<td>Sedation similar to butorphanol but significantly better pain relief for moderate- to markedly-painful procedures</td>
</tr>
<tr>
<td>Medetomidine** (10–30 μg/kg) with opioid + ACP</td>
<td>10 μg/kg dose is useful for sedating cats. Mild to profound dose-dependent sedation. Level of pain relief associated with opioid use</td>
</tr>
<tr>
<td>Medetomidine (25–80 μg/kg) single agent</td>
<td>Can achieve profound sedation but also has marked cardiovascular effects. Sudden arousal from deep sedation can occur</td>
</tr>
<tr>
<td>Midazolam (0.25–0.5 mg/kg) <strong>+</strong> ketamine (2.5–5 mg/kg)</td>
<td>Primarily in cats. Can be given i/m or use 50:50 dose combination to effect i/v. Level of sedation unpredictable</td>
</tr>
<tr>
<td>Benzodiazepine (0.25–0.5 mg/kg) <strong>+</strong> opioid</td>
<td>Not for general use but relatively safe in critically ill animals. Give opioid first then benzodiazepine i/v 20–30 minutes later</td>
</tr>
</tbody>
</table>

* can use pethidine 2–10 mg/kg instead  
** dexmedetomidine given at half dose rate (same volume)

#### Reversal of sedation

Atipamezole reversal agent for α2-agonists is given at equal volume (dog); half volume (cat) of medetomidine or dexmedetomidine used in the original sedation.

- Isoflurane and sevoflurane (which gives quicker induction and recovery) are used most widely.
- Isoflurane maintenance for around 50% of individuals can be achieved at 1.5%; sevoflurane at 2.5%.
- Both can be used as induction agents (3–5% and 6–8%, respectively) with suitable scavenging. Induction is unpredictable and can be stressful if the gas is delivered by mask.
4.1.5 ANAESTHETIC CIRCUIT CHOICE

- Rebreathing circuits with carbon dioxide absorption (by soda lime) have recently become popular. They use much lower flow rates of oxygen and smaller amounts of volatile agent. The Humphrey ADE circuit, for example, is a multipurpose system:
  - Use without soda lime for dogs and cats weighing 7–10 kg, with free gas flow (FGF) rate of 70–100 ml/kg/min.
  - Use with soda lime for heavier dogs, with FGF rate of 30 ml/kg/min at induction and 10 ml/kg/min for maintenance.

- More conventional, commonly-used, semi-closed circuits are T-piece, Bain, Lack and MacGill (see illustration, right). They are chosen on the basis of patient size. FGF rates are much higher:
  - T-piece and Bain: 250–300 ml/kg/min.
  - Lack and MacGill: 150 ml/kg/min.
4.1 Anaesthesia

- 0.5, 1, 2, 3 and 5 litre bags are commonly used in veterinary practice: choose a bag that is approximately 0.1 l/kg body weight.

Using a thermovent can reduce the cooling effect of inhaling gaseous anaesthetics.
4.1.6 **MONITORING AN ANAESTHETIC**

- Good observational monitoring of anaesthesia is vital, especially in sick patients (see table, above).
- Manual counting of heart, pulse and respiratory rates serves to check machine values.
- Pulse quality and mucosal colour additionally evaluate the circulation.
- Single measurements are of relatively limited value; trend monitoring is much more useful.

4.2 **Analgesia**

4.2.1 **INTRODUCTION**

- Non-steroidal anti-inflammatory drugs (NSAIDs) are the mainstay for chronic pain relief in cats and dogs.
- Acute pain relief, especially in haemodynamically compromised patients, is often better delivered with opioids which can be used in combination with NSAIDs.

4.2.2 **OPIOID ANALGESICS**

- Opiates deliver relatively safe, low toxicity, effective pain relief. Many are scheduled drugs.
- See table on page 152. This is not an exhaustive list of opioid products; the majority are not licensed for use in cats and dogs.
- All opioids will have sedative effects and will cause some respiratory depression.
The duration of action and dose required can be quite variable between individuals. The dose required is usually lower in patients with hepatic disease.

A number of opioids can be given as epidural pain relief, or as continuous rate infusion (CRI) solutions. The latter are best in combination (4.2.3).

**CRI PAIN RELIEF**

- CRI cocktails are intended to follow on from loading-dose pain relief. Two useful formulations are:
  - Morphine, lidocaine and ketamine (MLK): 60 mg M + 1000 mg L + 60 mg K/l (0.9% NaCl) @ 1–3 ml/kg/hr; protect bag from light.
  - Fentanyl, lidocaine and ketamine (FLK): Fentanyl instead of morphine in the above MLK: 1.2 mg F/l (0.9% NaCl) @ 1–3 ml/kg/hr; protect bag from light.

**NON-Steroidal ANti-INFLAMMATory Drugs**

- NSAIDs moderate pain by blocking a variety of pathways (COX, LOX and thromboxane).
- See table, page 154, for a list of common NSAIDs. A variety of other NSAIDs have been used in dogs and cats, but their use is difficult to justify, given the wide range of licensed products available.
### OPIOID ANALGESICS

<table>
<thead>
<tr>
<th>Generic name</th>
<th>Site of action</th>
<th>Route of administration*</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Buprenorphine</strong></td>
<td>Partial agonist OP&lt;sub&gt;3&lt;/sub&gt; receptor</td>
<td>i/v, i/m, s/c</td>
</tr>
<tr>
<td><strong>Butorphanol</strong></td>
<td>Primarily OP&lt;sub&gt;2&lt;/sub&gt; agonist, OP&lt;sub&gt;3&lt;/sub&gt; antagonist</td>
<td>i/m, s/c (10/ml)</td>
</tr>
<tr>
<td><strong>Codeine</strong></td>
<td>Mechanism unclear, mu receptor?</td>
<td>po (15, 30, 60; 5/ml)</td>
</tr>
<tr>
<td><strong>Fentanyl</strong></td>
<td>Pure OP&lt;sub&gt;3&lt;/sub&gt; agonist</td>
<td>i/v (0.05/ml); patch (12, 25, 50, 75, 100 μg/hr)</td>
</tr>
<tr>
<td><strong>Methadone</strong></td>
<td>Pure OP&lt;sub&gt;3&lt;/sub&gt; agonist</td>
<td>i/v, i/m (10/ml)</td>
</tr>
<tr>
<td><strong>Morphine</strong></td>
<td>Pure OP&lt;sub&gt;3&lt;/sub&gt; agonist</td>
<td>i/v; i/m (usually 10/ml)</td>
</tr>
<tr>
<td><strong>Pentazocine</strong></td>
<td>Weak OP&lt;sub&gt;2&lt;/sub&gt;, antagonistic OP&lt;sub&gt;3&lt;/sub&gt;</td>
<td>po (25, 50)</td>
</tr>
<tr>
<td><strong>Pethidine</strong> (= meperidine)</td>
<td>Pure OP&lt;sub&gt;3&lt;/sub&gt; agonist</td>
<td>i/m, s/c (10–50)</td>
</tr>
<tr>
<td><strong>Tramadol</strong></td>
<td>All opioid receptors, esp. OP&lt;sub&gt;3&lt;/sub&gt;</td>
<td>po (50, 100, 200, 300, 5 /ml)</td>
</tr>
</tbody>
</table>

* Text in parentheses are injectable/oral solution strength (mg/ml) or tablet size (mg)

#### 4.2.5 REVERSAL AND MANAGEMENT OF OVERDOSE

- **Misoprostol** (Cytotec, 200 μg tablets): protects against NSAID-induced gastric ulceration. Can cause abdominal pain, diarrhoea, nausea, vomiting and abortion. Dosage is 2–7.5 μg/kg q8–12hr (dog), 5 μg/kg q8hr (cat).
- **Naloxone**: competitive antagonist for opioid receptor; use in accidental overdose or poisoning; 0.015–0.04 mg/kg i/v, i/m and s/c.
- **Atipamezole**: see table on page 148.

#### 4.2.6 OTHER APPROACHES TO PAIN RELIEF

- **Gabapentin**: used for neuropathic pain, mechanism of action is unknown. Dose up to 10 mg/kg po q8hr. Can cause mild sedation and ataxia.
- **Benzodiazepines** are used as muscle relaxants.
### 4.2 Analgesia

<table>
<thead>
<tr>
<th>Dose rate (mg/kg)</th>
<th>Legal category/comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Dog</strong></td>
<td><strong>Cat</strong></td>
</tr>
<tr>
<td>0.01–0.02</td>
<td>0.01–0.02</td>
</tr>
<tr>
<td>q6hr</td>
<td>q6hr</td>
</tr>
<tr>
<td>0.2–0.5</td>
<td>0.2–0.5</td>
</tr>
<tr>
<td>q6–12hr</td>
<td>q6–12hr</td>
</tr>
<tr>
<td>0.5–2</td>
<td>0.5–2</td>
</tr>
<tr>
<td>q12hr</td>
<td>q12hr</td>
</tr>
<tr>
<td>patch (4 μg/kg/hr)</td>
<td></td>
</tr>
<tr>
<td>or as FLK infusion</td>
<td>S2</td>
</tr>
<tr>
<td>0.1–0.5</td>
<td>0.1–0.3</td>
</tr>
<tr>
<td>i/m</td>
<td>q4–6hr</td>
</tr>
<tr>
<td>q4–6hr</td>
<td></td>
</tr>
<tr>
<td>0.5</td>
<td>0.1–0.4</td>
</tr>
<tr>
<td>i/m</td>
<td>q3–6hr</td>
</tr>
<tr>
<td>q3–4hr</td>
<td></td>
</tr>
<tr>
<td>2–6</td>
<td>–</td>
</tr>
<tr>
<td>q3–4hr</td>
<td></td>
</tr>
<tr>
<td>2–10</td>
<td>5–10</td>
</tr>
<tr>
<td>q1–2hr</td>
<td>q1–2hr</td>
</tr>
<tr>
<td>2–5</td>
<td>2–4</td>
</tr>
<tr>
<td>q12hr</td>
<td>q12hr</td>
</tr>
</tbody>
</table>

Legal categories: POM-V = prescription-only medicine (veterinary); S2 = schedule 2; S3 = schedule 3

- **Methocarbamol** (Robaxin): skeletal muscle relaxant. Dose 20–45 mg/kg po q8hr. Side-effects: salivation, vomiting, lethargy, weakness, ataxia and CNS depression.
- **Amantadine**: N-methyl-D-aspartate- (NMDA-) antagonist analgesic.
- **Amitriptyline**: NMDA-antagonist analgesic (also used for behavioural therapy, FLUTD and management of ureteroliths).
- **Non-drug modalities**: the value of physiotherapy, acupuncture and other therapies should not be underestimated.

⚠️ Early aggressive pain management reduces the ‘wind-up’ phenomenon and therefore the overall amount of pain relief required. In chronic cases, pain relief may actually reduce the rate of disease progression.
### NON-STERoidal ANTI-INFLAMMATORY DRUGS (NSAIDS)

<table>
<thead>
<tr>
<th>Generic name [Trade name]</th>
<th>Available preparations</th>
<th>COX selectivity</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Aspirin</strong> [various]</td>
<td>75, 300 mg (T)</td>
<td>Non-selective COX inhibitor</td>
<td>H, 1, 2</td>
</tr>
<tr>
<td><strong>Carprofen</strong> [Rimadyl*]</td>
<td>20, 50, 100 mg (T)</td>
<td>Preferentially inhibits COX-2</td>
<td>H, 1, 3</td>
</tr>
<tr>
<td><strong>Deracoxib</strong> [Deramaxx]</td>
<td>25, 100 mg (T)</td>
<td>Preferentially inhibits COX-2</td>
<td>H, 1, 3</td>
</tr>
<tr>
<td><strong>Etodolac</strong> [EtoGesic*]</td>
<td>150, 300 mg (T)</td>
<td>Preferentially inhibits COX-2</td>
<td>H, 1, 3, 4</td>
</tr>
<tr>
<td><strong>Firocoxib</strong> [Previcox]</td>
<td>57, 227 mg (T)</td>
<td>Preferentially inhibits COX-2</td>
<td>H, 1, 3</td>
</tr>
<tr>
<td><strong>Ketoprofen</strong> [Ketofen]</td>
<td>5, 20 mg (T) 1 mg/ml (IS)</td>
<td>Non-selective COX inhibitor</td>
<td>HR, 1, 2, 3, 8</td>
</tr>
<tr>
<td><strong>Mavacoxib</strong> [Trocoxil]</td>
<td>6, 20, 30, 75, 95 mg (T)</td>
<td>Preferentially inhibits COX-2</td>
<td>1, 3</td>
</tr>
<tr>
<td><strong>Meloxicam</strong> [Metacam*]</td>
<td>1, 2, 5 mg (T) 0.5, 1.5 mg/ml (OL) 2, 5 mg/ml (IS)</td>
<td>Preferentially inhibits COX-2</td>
<td>H, 1, 3</td>
</tr>
<tr>
<td><strong>Paracetamol</strong> [various]</td>
<td>120, 400, 500 mg (T) 25, 50 mg/ml (OL) 10 mg/ml (IS)</td>
<td>Antipyretic and analgesic via COX</td>
<td>H 1, 3, 7, 9</td>
</tr>
<tr>
<td><strong>Piroxicam</strong> [various]</td>
<td>10, 20 mg (T) + 20 mg dissolvable</td>
<td>Therapy for adenocarcinoma</td>
<td>HR, 1, 3, 5</td>
</tr>
<tr>
<td><strong>Robenacoxib</strong> [Onsior]</td>
<td>5, 6, 10, 20, 40 mg (T) 20 mg/ml (IS)</td>
<td>Preferentially inhibits COX-2</td>
<td>HR 1, 3</td>
</tr>
<tr>
<td><strong>Tepoxalin</strong> [Zubrin]</td>
<td>50, 100, 200 mg (T)</td>
<td>Anti-prostaglandin and leukotriene</td>
<td>H, 1, 3, 6</td>
</tr>
<tr>
<td><strong>Tolfenamic acid</strong> [Tolfedine]</td>
<td>6, 20, 60 mg (T) 40 mg/ml (IS)</td>
<td>COX selectivity uncertain</td>
<td>HR, 1, 2, 3</td>
</tr>
</tbody>
</table>

* Also produced by other manufacturers under different trade names

T = tablets; IS = Injectable solution; OL = oral liquid

COX = cyclooxygenase

H = hepatic metabolism with biliary excretion
HR = hepatic metabolism with renal excretion
## 4.2 Analgesia

<table>
<thead>
<tr>
<th>Administration route</th>
<th>Dose rate</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Dog</strong></td>
<td><strong>Cat</strong></td>
</tr>
<tr>
<td>po</td>
<td>10–20 mg/kg q12hr</td>
</tr>
<tr>
<td>i/v, s/c, po</td>
<td>Initially 4 mg/kg, then 2 mg/kg q24hr</td>
</tr>
<tr>
<td>s/c, po</td>
<td>5 mg/kg q24hr</td>
</tr>
<tr>
<td>s/c, po</td>
<td>Initially 2 mg/kg, then 0.25 mg/kg q24hr</td>
</tr>
<tr>
<td>s/c</td>
<td>0.3 mg/kg (single) or initially 0.1 mg/kg q24hr</td>
</tr>
<tr>
<td>po</td>
<td>2 mg/kg, 2nd dose @14d then monthly</td>
</tr>
<tr>
<td>po</td>
<td>10 mg/kg q12hr</td>
</tr>
<tr>
<td>po</td>
<td>10 mg/kg q24hr</td>
</tr>
<tr>
<td>s/c, i/m, s/c, po</td>
<td>Initially 2 mg/kg, then 0.1 mg/kg q24hr</td>
</tr>
<tr>
<td>s/c</td>
<td>4 mg/kg q24hr</td>
</tr>
<tr>
<td>po</td>
<td>0.3 mg/kg q24hr</td>
</tr>
<tr>
<td>po</td>
<td>10 mg/kg q24hr</td>
</tr>
<tr>
<td>s/c, i/m, s/c, po</td>
<td>Initially 2 mg/kg, then 1 mg/kg q24hr</td>
</tr>
<tr>
<td>po</td>
<td>0.3 mg/kg q24hr</td>
</tr>
<tr>
<td>po</td>
<td>4 mg/kg q24hr</td>
</tr>
</tbody>
</table>

**1.** Standard GI and hypotension, lowest effective dose
**2.** Lower dose as anticoagulant or has anti-platelet action
**3.** Small risk of precipitating CHF in patients with cardiovascular disease
**4.** Other sizes available for people
**5.** Value of Piroxicam over meloxicam unclear, efficacy so far only been documented for transitional cell carcinoma of bladder
**6.** 5-lipoxygenase inhibition may be associated with an improved GI safety profile
**7.** Injectable preparation for i/v use available for people, limited experience in dogs
**8.** Dogs’ dose schedule can be repeated weekly
**9.** Little anti-inflammatory activity
4.3 **Soft-tissue surgery**

4.3.1 *Introduction* 4.3.2 *Minimizing surgical infection* 4.3.3 *Haemostasis* 4.3.4 *Approach to a lump* 4.3.5 *Sutures* 4.3.6 *Bandages and dressings* 4.3.7 *Wound management* 4.3.8 *Post-operative care*

### 4.3.1 INTRODUCTION

- It is not possible within a text of this length to cover individual surgical procedures. The following is designed to address surgical issues that are common to most procedures.

> Surgery should be avoided if you are unsure of the technique, if you have no ‘plan B’, or if there is inadequate post-operative/overnight care.

### 4.3.2 MINIMIZING SURGICAL INFECTION

**Environment**

- Use the operating theatre only for surgery.
- Clean every morning and evening (between every surgery if possible).
- Ideally, prepare patients (e.g. clipping, cleaning, anaesthetizing) in a separate room.
- Ensure adequate lighting and, ideally, filtered, positive pressure ventilation, especially for advanced orthopaedics.
- Plan the surgical list: cleanest surgery first, finishing the end of the list with the infected or ‘dirty’ surgeries.

**Preparation of the patient**

- Clip a wide area that allows for movement of skin into the surgical field during closure.
- Vacuum or brush the loose hairs off the coat and cover feet with a *waterproof* drape to prevent strikethrough.
- Use antiseptic scrub for skin preparation (chlorhexidine/povidone iodine), then alcohol-based antiseptic spray.

**Preparation of the surgeon**

- Use a surgical cap and also, ideally, a mask. Talking increases the risk of aerosol infection!
- Use a standard protocol to scrub hands, nail beds, arms; this takes 5 minutes.
- Use sterile gloves and gowns for every surgery.
- Change clothes used for consulting, house visits, or other procedures into clean scrubs and shoes for the operating area.
Surgical technique

- Drape the whole animal and cover the table and trolley; water-resistant drapes prevent strikethrough.
- If a surgery is difficult, make the incision longer and get an assistant to scrub in.
- **Swabs** dampened with sterile saline, for covering tissues and skin edges during long surgery, prevent desiccation.
  - Use swabs with x-ray-detectable threads for abdominal surgery.
  - Count your swabs onto the trolley and out of the animal.
- **Contamination:** if tissues become contaminated during surgery, lavage the surgical site with sterile saline prior to closure.
  - Warm sterile saline (large volumes) is needed for abdominal lavage; remove with suction prior to closure.
  - If the site is contaminated (e.g. strikethrough of drapes, GIT surgery), change gloves and drapes prior to closure.
  - Infected or contaminated surgery: change instruments for closure; don’t re-use suture materials used on GI/urinary tract.

**HAEMOSTASIS**

- Haemostasis is important, especially in invasive or complex surgery, in order to achieve:
  - Shorter surgery times.
  - A ‘dry’ surgical field, thereby improving visibility and reducing the risk of iatrogenic injury.
  - Reduced post-operative morbidity due to blood loss or hypotension.
  - Less risk of unidentified bleeding after abdominal or thoracic surgery.
  - Improved tissue healing, reduced post-operative oedema and less risk of infection.
  - Reduced post-operative mortality and re-operation rates.
- Whole blood and blood products are expensive and in relatively short supply and are not a substitute for haemostasis.
- **Ligatures:** Suture material should be small gauge with good knot security and handling characteristics – preferably a synthetic absorbable material.
- The surgeon should be familiar with the techniques used to place ligatures without the throws on the knot locking prior to tightening around the pedicle.
  - The sliding knot is used to place a secure ligature, deep within a body cavity, but the tissue incorporated in the knot should be carefully examined prior to tightening.
  - One-handed ties are also used to place secure ligatures on vessels in awkward positions.
**Haemostatic equipment** to seal small vessels during surgery includes:
- Electrosurgery (monopolar or bipolar).
- Laser.
- Ligasure™.

**Promotion of clotting:** in order to seal multiple small vessels or vessels that are difficult to access (e.g. friable surfaces [nasal turbinates, liver], bone surfaces, abraded skin/wound surfaces) techniques can be used to augment natural clotting. The method chosen must not delay healing or cause damage to adjacent structures. Two possibilities are:
- Reduce blood flow to the affected area allowing clot formation, using:
  - Tourniquet and pressure – temporarily, for first aid or intra-operatively. Minor haemorrhage should stop after 2–3 minutes.
  - Cold packs or cold sterile saline (reduce blood flow).
  - Topical vasoconstrictors (e.g. dilute chilled adrenaline).
- Topical haemostatic agents, though these are not licensed for use in animals, with little data on how they work. They are used to enhance clotting of friable surfaces.

4.3.4 **APPROACH TO A LUMP**

**Assessment and planning**
- All masses should be investigated and not ‘watched’.
- Not all lumps are neoplastic (see 3.14).
- For tumours before surgery, identify the tumour type, anatomy of primary tumour and metastasis (staging).
- Perform with minimal morbidity.
- Aim for cure in the first surgery; debulking is rarely indicated. Incompletely resected tumours are more likely to recur.
- Consider whether appropriate adjuvant therapy, e.g. radiation or chemotherapy, is indicated.
- Know when not to operate (when surgery is of no ultimate benefit to the patient), e.g. the presence of significant residual disease despite surgery.

**Tumour staging**
- Ascertain primary tumour type and grade. The most common superficial canine tumours are lipomas, mast cells or soft-tissue sarcomas.
- Fine-needle aspiration (FNA) is useful for investigating lipoma, lymphoma and mast-cell tumour; a non-diagnostic FNA suggests non-neoplastic disease or a sarcoma, so repeat FNA or obtain a biopsy.
- Tru-cut or incisional biopsy if FNA is non-diagnostic.
Lymph nodes: palpation alone is inaccurate. Consider FNA, biopsy or excision if there is lymphadenopathy.

Look for distant metastasis (thorax, abdomen) using radiography, ultrasound, or CT/MRI.

**Principles of oncologic surgery**

- Plan the extent of resection to remove the palpable tumour and a surrounding area of normal tissue, to ensure removal of all tumour cells (see diagram, above). Higher-grade tumours require a wider area of resection.
- A lateral skin margin of 1–5 cm (depending on tumour type and grade) and a deep fascial layer is mandatory. The deep margin is the most common margin for incomplete excision.
- Removing masses without FNA or biopsy may lead to choosing inappropriate margins of resection.
- ‘Shelling out’ will only remove the grossly visible tumour tissue and recurrence is likely.
- Wound reconstruction after tumour resection may require advanced surgical techniques. Early referral for definitive treatment is likely to have a better outcome than referral after resection with dirty margins.

**Sutures**

Suture materials

- Synthetic suture materials are preferred to organic suture (catgut) as they are removed by hydrolysis, leading to a gradual and predictable loss of tensile strength. The suture should be at least as strong as the target tissue (skin/fascia > intestines > bladder) and lose tensile strength at a similar rate to the tissue gaining it.
Monofilament sutures have minimal drag and form tight knots but are difficult to handle. Coated monofilament sutures are easier to handle, but may potentiate infection and will ‘drag’ through tissue.

**Absorbable** synthetic suture is generally used other than for skin sutures. A suture with prolonged tensile strength and slow absorption is appropriate for most situations, especially where early tissue separation could be problem, e.g. linea alba, blood vessel ligatures, intestines.

- Examples are polydioxanone (PDS, Ethicon) and polyglyconate (Maxon, Covidien).

**Subcuticular** sutures can have more rapid loss of tensile strength and absorption.
- Example: poliglecaprone (Monocryl, Ethicon).

**Permanent** synthetic suture is used for skin or when suture absorption is problematic, e.g. perineal rupture repair.
- Example: nylon (Monosof, Covidien).

**Suture size**

- Suture size selection depends on the size of animal and tissue to be sutured. Large-gauge suture is to be avoided, as knots are less secure. Appropriate size selection would be:
  - Skin/subcuticular tissue: 1.5–2 metric (4/0–3/0 USP).
  - Linea alba: 3–3.5 metric (2/0–0).
  - Vascular pedicles, e.g. ovary: 3–3.5 metric (2/0–0).
  - Intestines: 1.5 metric (4/0).

**Suture technique**

- Swaged-on needles are preferred. Reverse cutting needles used for skin, but tapercut or taperpoint needles for viscera and fascia.
- The knot is the weakest part of a suture. A minimum of 4 throws is required for polydioxanone and nylon, with 1 and 2–3 extra throws, respectively, at the beginning and end of a continuous suture. A square or surgeon’s knot is preferred and each throw must be securely tightened.
- Ligatures must be placed tightly, which may be aided with the use of a sliding/’slip’ knot using monofilament suture material.
- Appositional sutures in incisions should be placed so that the tissues are held in snug apposition without strangulation.
- An Aberdeen (self-locking) knot is a useful way to bury the end of a continuous subcuticular suture.
- Note that failure of a suture line is usually related to surgical technique, rather than the choice of suture material or size.
BANDAGES AND DRESSINGS

Wounds are dressed with a primary (contact) layer followed by a secondary layer (padding and absorption) and finally a tertiary (protective) layer.

- **Primary layers:** assess the wound and determine the effect you want for the contact layer – protective, debriding, or actively promoting healing.

- **Secondary layers:**
  - Use a soft woven bandage.
  - Wrap evenly, with about 50% overlap of each turn of the bandage. Depending on the level of support, this layer may be only 2–3 turns thick, or very heavy (e.g. a Robert Jones bandage).
  - Use an extendable, elastic, non-adherent bandage (e.g. TreatRap® or equivalent) to secure the padding layer.
  - The natural contours of the limb should be evened out, so that the bandage is roughly the same diameter all the way along; this reduces the risk of bandage injury.

- **Tertiary layers:**
  - Use a self-adherent, water-resistant material (e.g. Vetrap®).
  - Apply with even tension – not too tightly – up and down the limb.

- **Bandage injuries can cause massive tissue loss, amputation or death!**

- **Complications are caused by the bandage slipping or being applied too tightly or unevenly. The patient may chew at the dressing within 1–2 hours of application if it is causing restriction of circulation.**

- **All owners should be provided with written discharge instructions on bandage care, including instructions to seek veterinary attention immediately if the bandage becomes wet, slips or causes distress.**

Casts

- Splints may be incorporated into a bandage to provide more support, or a cast applied using a very thin layer of padding underneath.
- Choose the cast according to cost, strength and the size/age of the patient.
- The owner must have written instructions on cast care and the dangers of ignoring clinical signs: swelling of the toes, chewing at the cast, distress, pain or smell. They must check the cast twice daily.
The cast must be checked weekly by a veterinary surgeon. Casts used to support ligamentous injuries may be split after application and changed weekly. Casts used to support fracture repair are usually not changed for 6 weeks, but must still be checked. Casts placed on the limbs of growing animals may need to be completely reapplied at least weekly, to allow normal growth of the limb.

4.3.7 WOUND MANAGEMENT

- Wound healing is promoted by a moist environment and reduced inflammation.

Assess the injury:
- Take a history.
- Check for other organ injuries.
- Check for deep damage, e.g. crushing of muscle/bone in bite wounds.
- Probe penetrating injuries, especially over the chest and abdomen.
- Surgically explore bite wounds.

Control infection:
- Lavage with large volumes (1–5 l) of sterile fluid to remove debris and bacteria. Use an 18–21g needle and a 20–50 ml syringe attached to a 3-way tap-and-drip line and applied with firm pressure. Antiseptics are not needed.
- Surgically debride obviously necrotic tissue daily as required.
- Use wet-to-dry dressings for further debridement.

Control further wound contamination and necrosis:
- Cover with dressings at first presentation; keep dressed, creating a moist environment, until healthy granulation tissue covers the wound.
- Promote a viable vascular bed by continued debridement of wounds – surgical (as above), wet-dry dressings and active dressings.

Prevent further damage, particularly of healing (granulation) tissue.

Select an appropriate method of closure, e.g. primary closure, delayed primary closure (2–5 days) or secondary closure (>5 days) after open wound management, or second-intention healing.
4.3 Soft-tissue surgery

- General patient management:
  - Nutrition; consider oesophagostomy-tube feeding.
  - Analgesia; open wounds are very painful – use opioids and NSAIDs.
  - Antimicrobials are used initially, but are not a substitute for lavage and debridement. Stop antimicrobials once the wound is healthy and granulating.

- Wounds that are best closed:
  - Those that can be converted to a clean-contaminated wound by lavage and debridement.
  - Thoracic wounds.
  - Tissues that need normal function, e.g. near eyes, over joints.
  - Skin wounds over fractures are best closed within 3 days.

- Wounds that are best left open even if just for a few days:
  - Contaminated or dirty wounds where wound closure will probably result in abscess formation.
  - Suspected ongoing ischaemia/necrosis.
  - Distal limbs where there is not much available skin.

- Be aware of deep injury, e.g. necrosis in traumatic wounds such as bite wounds.

> If in doubt about the degree of contamination or ongoing necrosis, a wound should be left open. It can be closed in a few days if the wound is healthy, or can be left for longer if there are continuing doubts.

**POST-OPERATIVE CARE**

- **Nursing:** post-op patients require warmth, comfort, fluid therapy and analgesia. Delayed recovery may be due to hypothermia, hypotension, electrolyte imbalance, pain and fear, or dehydration.

- **Analgesia:** reduces stress and improves recovery. Opioids do not cause gastrointestinal ileus.

- **Nutrition:** early nutrition is essential; there is no indication to withhold food. Feeding should be encouraged as soon as possible after recovery, including after GIT surgery. Nausea post-anaesthetic/gastric surgery can be treated with prokinetics, antacid medication or central/peripheral antiemetics. At the end of major surgery, anorectic animals should have a feeding tube placed.
Postoperative complications

- Complications may be due to wound, patient or surgical factors.
- **Seroma**: a relatively common complication, especially when skin has been undermined, or in loose-skinned animals around the body folds or the neck. Most do not require treatment and will resolve over several weeks. Needle drainage is discouraged, as the seroma will re-form and there is a risk of iatrogenic infection. Large seromas may require surgical drain placement.
- **Surgical wound dehiscence**: usually due to excessive wound tension. Other causes include infection, tight sutures, inadequate analgesia, excessive movement or self-trauma. Most cases respond well to open wound management and healing by second intention. Resuturing is only performed if the underlying cause of dehiscence can be overcome and if tissue is healthy.
- **Infection**: multi-drug-resistant infections, including methicillin-resistant *Staphylococcus aureus* (MRSA), are occasionally encountered; their management is the same as for other infections. Use bacterial culture and sensitivity to choose antimicrobial therapy.
- **Haemorrhage** is usually due to poor surgical technique and inadequate haemostasis. Animals with haemorrhage in an abdominal cavity, e.g. after neutering, may present with weakness, collapse, pallor, slow recovery and tachycardia. Aspiration of blood via abdominocentesis is diagnostic. Following intravenous fluid therapy, emergency surgery will be required. Rarely, haemorrhage is due to clotting disorders; petechiae and bleeding from catheter/venipuncture sites may also be evident.
- **Peritonitis**: dehiscence of a gastrointestinal incision, if it occurs, is usually within 72 hours of surgery and will lead to septic peritonitis. Inappetance and vomiting are early presenting signs, and abdominocentesis yields fluid containing degenerate neutrophils and intracytoplasmic bacteria. Following intravenous fluid therapy, emergency surgery is required.
Orthopaedics

4.4.1 Scope of this section 4.4.2 Approach to the lame dog or cat 4.4.3 Diagnostic tests and imaging 4.4.4 Forelimb disease 4.4.5 Hind limb disease 4.4.6 Degenerative joint disease (DJD) 4.4.7 Managing the chronic orthopaedic patient 4.4.8 Emergency management of appendicular fractures

INTRODUCTION

Scope of this section

- The following is designed to address the approach to an orthopaedic case, with a brief discussion of common conditions.
- It is not possible within the scope of this text to cover individual orthopaedic procedures. For an approach to spinal disease see 3.10.

HISTORY AND EXAMINATION

Approach to the lame dog or cat

- The consultation should involve:
  - Gait analysis.
  - History taking.
  - Orthopaedic examination, including observation at rest.

  Gait analysis: it is easiest to evaluate dogs at walk and trot on a firm level surface. Cats can be given free range in a large secure room.

  Forelimb lameness (4.4.4) will result in head-nodding or head up on the painful limb.

  Hind-limb lameness (4.4.5) is suggested when the hock is higher, to reduce weight bearing; the leg may also be held away from the body. The pelvis may be rotated towards the sound side during the swing phase of movement, reducing joint excursion.

  Bilateral forelimb disease can be difficult to detect. Sufferers tend to shift their weight backwards and have a hunched back, crouched hind-limb stance and a very upright neck position, especially on rough surfaces.

  Bilateral hind-limb disease tends to cause a wobbling gait and a shift of weight forward.

- History: note duration, acuteness of onset, evidence of trauma, progression and persistence of the lameness.

  Inactivity stiffness suggests osteoarthritis, elbow dysplasia, or cruciate disease.

- Orthopaedic examination: it can be difficult to perform a full examination in some patients, especially cats and small dogs. Sedation may be necessary.

  Examine limb position, symmetry of weight bearing and musculature.
Examine ability to stand and stay standing.
Examine head, neck and tail carriage.
Firmly palpate the spinal column and manipulate the neck – but if cervical disease is suspected, do not perform this manipulation.
Forelimbs may be examined standing, sitting or in lateral recumbency; hind limbs, standing or in lateral recumbency.
All joints should be assessed for range of movement (ROM), discomfort, crepitus, thickening, instability or effusion. See table, above.
Examine soft tissues for change in size (swelling or atrophy), warmth and discomfort.
Examine paws, pads, claws and phalangeal joint for wear pattern, scuffing and pain.
Some dogs and cats will not be lame when presented for examination, as their lameness is intermittent and the excitement of the consultation may mask low-grade pain. If the presence or source of lameness cannot be identified then:
Hospitalize and repeat the examination later in the day.
Perform a full neurologic examination and rule out non-orthopaedic disease.
Ask the owner to obtain video evidence of lameness.
Ask the owner to keep a ‘lameness diary’, and revisit on a ‘bad’ day.

**DIAGNOSIS**

4.4.3 Diagnostic tests and imaging

- **Blood work** is generally unhelpful; it is most valuable in joint disease.
- Consider routine haematology (for evidence of inflammation or infection), muscle enzymes, serology/PCR for Lyme disease; serology for Neospora or Toxoplasma.
- Anti-nuclear antibody (ANA) and rheumatoid factor (RF) are of limited value and not primary tests.
Radiography is the most valuable initial investigation. Note that radiographic sensitivity is greatly improved if lameness has been localized.

- Patient should be sedated or anaesthetized.
- At least two views of each area are necessary, and preferably also the contralateral limb for comparison.
- Stressed, flexed and extended views may be helpful.
- Good positioning and radiographic technique are essential.
- Changes should be evaluated in conjunction with physical findings, age and breed.

Joint fluid analysis: see 4.4.6.

Ultrasound is increasingly used to examine soft-tissue structures supporting joints/muscles; interpretation requires experience.

Advanced imaging. CT and MRI offer advantages over radiography, but require specialist equipment and interpretation.

Arthroscopy/arthrotomy are necessary to fully evaluate some cases with joint disease; they also allow therapeutic intervention. They require specialist equipment and knowledge to be performed well.

Force plate with video analysis of gait measures the force as each limb is planted, to better evaluate lameness and response to treatment, while high-speed video cameras are used to analyse gait.

Scintigraphy: Technetium is used to look for focal ‘hot spots’ affecting the bone/support tissues, suggesting inflammation.

CAUSES OF LAMENESS

Forelimb disease

- Non-fracture, common causes of forelimb lameness include:
  - Carpal hyperflexion injuries – torn ligaments as a result of a fall or trauma.
  - For immature dogs:
    - Elbow dysplasia (coronoid, osteochondritis dissecans [OCD], ununited anconeal process, radioulnar incongruity).
    - Antebrachial growth deformity such as carpal valgus.
    - Shoulder OCD in large and giant breeds.
  - For adult dogs:
    - Elbow osteoarthritis (OA) – usually secondary to dysplasia.
    - Soft-tissue injury of the shoulder, such as ligament tear or tenosynovitis.
4.4.5 Hind-limb disease

- Non-fracture, common causes of hind-limb lameness include the following:

  **Immature dogs**
  - Hip dysplasia.
  - Medial patella luxation:
    - Grade I – manually displaces but spontaneously reduces.
    - Grade II – intermittently and spontaneously luxates, resulting in a skipping gait.
    - Grade III – permanently luxated but can be reduced.
    - Grade IV – permanently luxated and irreducible.
  - OCD of the stifle and hock.

  **Adult dogs**
  - Cruciate disease – usually cranial. Integrity of the cranial cruciate ligament (CCL) is evaluated using the tibial thrust test (above).
  - Hip OA.

- Common conditions affecting any limb include the following:

  **Immature dogs**
  - Metabolic disease – lameness and pain on palpation; may show systemic signs.
  - Metaphyseal osteopathy.

  **Adult dogs**
  - OA: signs will wax and wane; worse after rest.
  - Osteosarcoma, usually seen ‘away from the elbow and near the stifles’.

**Tibial thrust test.** The stifle is held in slight flexion. The index finger of one hand is placed over the tibial crest and the other hand flexes and extends the hock (1, 2). If the CCL is torn, the tibial tuberosity will move very slightly cranially (3), when the hock is in the flexed position.
JOINT DISEASE

Degenerative joint disease (DJD) 4.4.6

- Osteoarthritis associated with DJD is the most frequently occurring joint disease in dogs and cats. Diagnosis may require radiography or arthrocentesis (see below).

Differential diagnosis

- Traumatic OA usually affects a single joint with a history of trauma.
- Septic OA usually affects single joints.
- Polyarthritic (immune-mediated) OA:
  - Sufferers are usually systemically unwell, showing fever, leucocytosis, inappetence and/or reluctance to exercise/lethargy – the phenomenon of ‘walking on eggshells’.
  - Joint swelling and pain can be subtle.
  - Polyarthritic OA can be erosive or non-erosive:
    - Erosive polyarthritis is similar to rheumatoid arthritis in humans. In dogs, this is diagnosed from radiographs, as RF in dogs is insensitive and non-specific.
    - Non-erosive polyarthritis in its most common form is secondary to another underlying disease, and may be accompanied by pancreatitis, meningitis, etc. Infectious causes include tick-borne disease and chronic bacterial infection, e.g. prostatitis. Radiographs appear normal (with soft tissue swelling) and the condition is diagnosed via arthrocentesis.

Arthrocentesis procedure

- Arthrocentesis should be performed on 4–6 joints if polyarthritis is suspected (see illustrations, below).
- Use a 23g needle for cats, and for tight joints in dogs (15–25 mm); use 21g for larger joints in dogs (25–40 mm), except for the hip.

Anatomic approach to obtaining joint aspirates.
Releasing suction pressure prior to removing the needle reduces the likelihood of blood contamination.

- If only a few drops are obtained, smear onto a slide; place larger amounts in EDTA.
- For culture, place in a sterile glass container and add $9 \times$ volume of liquid culture medium (used for blood cultures).
- For interpretation of the joint fluid analysis, see values in the table above.

**TREATMENT**

*Managing the chronic orthopaedic patient*

- The majority of chronic orthopaedic cases involve degenerative joint disease (DJD) leading to OA.
- A wide variety of therapies exists, so a co-ordinated treatment plan must be developed. Such plans are complicated by the lack of clear evidence of medical efficacy. The options include:

  - **NSAIDs** (4.2.4). It is important to realize that the disease process continues regardless of its expression as clinical signs, therefore chronic lower-dose therapy may be more effective.
  - **Other pain relief** includes opioids, gabapentin, benzodiazepines, methocarbamol, amantadine and amitriptyline (4.2.6).
  - Glycosaminoglycans (pentosan polysulphate) have been shown to have benefit in reducing pain.
  - **Surgery**: the joint environment can be improved via:
    - Arthroscopy/arthrotomy procedures.
    - Replacement (hip or elbow).
    - Surgical arthrodesis.
  - **Nutraceuticals**: a variety of products is available, either as nutritional supplements or incorporated in special diets. They include antioxidants (PUFA), glucosamine, chondroitin, green-lipped mussel, lysine, carnitine, methionine, turmeric and *Boswellia* extract.
- **Weight loss** for patients with BCS of 7/9 or above.
- **Physiotherapy**: several modalities have been shown to reduce pain, control inflammation and improve balance and ROM. They include cryotherapy, passive ROM exercises, stretching exercises, balance and proprioceptive exercises, massage therapy, therapeutic ultrasound, laser, transcutaneous electrical nerve stimulation (TENS) and active exercise.
- Altering the home environment can be helpful, especially slippery flooring and stairs.
- A home-exercise programme for the owner to manage, with ‘dos and don’ts’ that can be simply followed to keep joints moving.
- Other modalities of therapy include acupuncture, homoeopathy, and magnets. The benefits of these are unproven.

### Emergency management of appendicular fractures

1. Fractures are rarely life threatening, but shock, pulmonary contusion, or rupture of the diaphragm/bladder can be. Ensure that these are covered by the initial patient assessment.

- Stabilize the patient before radiographing the fractures.
- Provide analgesia: methadone/morphine plus NSAID (if the patient is not hypotensive).
- If pain is non-responsive, consider neurologic injury, e.g. sciatic nerve entrapment.
- **Open fractures**
  - Open fractures can be classified as in the table overleaf; management will depend on the grade of the fracture.
  - Cover with sterile dressing and change daily until fixation.
  - Administer broad spectrum i/v antimicrobials – potentiated amoxicillin, cephalosporin – as the wound will be susceptible to hospital-acquired infection.

<table>
<thead>
<tr>
<th>NCC* ($\times 10^9/l$)</th>
<th>Mononuclear (%)</th>
<th>Neutrophils (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;3</td>
<td>≥90</td>
<td>≤10</td>
</tr>
<tr>
<td>&lt;5</td>
<td>≥90</td>
<td>≤10</td>
</tr>
<tr>
<td>&gt;3</td>
<td>≤75</td>
<td>≥25</td>
</tr>
<tr>
<td>&gt;3</td>
<td>≤85</td>
<td>≥20</td>
</tr>
</tbody>
</table>
Once the patient is stable, use the following procedure for wound cleaning and lavage.

- Give sedation or general anaesthetic.
- Preparation: wear sterile gloves, disinfect clipper blades, pack wound with sterile K-Y jelly, clean skin with chlorhexidine.
- Technique: use a 500 ml bag of warm sterile Hartmann’s solution; connect the bag via a giving set to a 3-way tap with a syringe and a 19g needle attached.
- Debride wound and take swabs after debridement.

**Temporary stabilization** is achieved differently for distal and proximal fractures:

- Distal limb fracture (below elbow/stifle) requires support dressing (a splint or Robert Jones bandages) that minimizes further tissue damage and provides pain relief. Extend the dressing above the elbow/stifle.
- Proximal limb, pelvic or spinal fracture requires strict cage rest. Dressing is usually counterproductive.

**Assessment UGA:**

- Radiograph the fracture site.
- All limbs should be assessed, including joints to evaluate instability, crepitus or luxation.
- Other areas may also need assessment, e.g. thorax, urinary tract.

Twelve percent of pelvic fractures compromise the integrity of the urinary system.

- Radiograph the whole bone with at least two orthogonal views, and assess for joint involvement, non-displaced fissure fractures and pre-existing pathology, e.g. lytic lesion.
- Radiography of contralateral bone helps planning for fixation.
5 CRITICAL CARE

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5.1 Fluid therapy

- Fluid therapy (see tables, below and right) replaces volume and can provide colloidal osmotic pressure (COP), as well as other benefits such as oxygen-carrying capacity.
- Correct choice maximizes these benefits; the tables attempt to summarize characteristics which affect choice.
- Ion concentrations: fluid choice should take account of the patient’s blood biochemistry, e.g. if potassium is high, then Hartmann’s or Haemocell is not first choice because they will increase potassium still further.
- Duration of action must be balanced against the maximum dose which can be given.

### FLUID THERAPY

<table>
<thead>
<tr>
<th>Crystalloids</th>
<th>Na⁺</th>
<th>K⁺</th>
<th>Cl⁻</th>
<th>Ca²⁺</th>
<th>HCO₃⁻</th>
</tr>
</thead>
<tbody>
<tr>
<td>7.2% NaCl</td>
<td>1232</td>
<td>0</td>
<td>1232</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td><strong>Rapid volume expansion lasts 30–60 min</strong> 4–7ml/kg (dog), 2–4 ml/kg (cat)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0.9% NaCl</td>
<td>150</td>
<td>0</td>
<td>150</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td><strong>General use, especially low Na⁺, high K⁺ or Ca²⁺ or alkalosis</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hartmann’s</td>
<td>131</td>
<td>5</td>
<td>111</td>
<td>2</td>
<td>29</td>
</tr>
<tr>
<td><strong>Standard maintenance, acidosis with functional liver</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0.18% NaCl</td>
<td>30</td>
<td>0</td>
<td>30</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td><strong>Hyperosmolar states, high Na⁺</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5% dextrose</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td><strong>Hyperosmolar states, drug diluents, never as a bolus</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Colloids</th>
<th>Na⁺</th>
<th>K⁺</th>
<th>Cl⁻</th>
<th>MW</th>
<th>COP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Haemacell⁹</td>
<td>145</td>
<td>5</td>
<td>145</td>
<td>30</td>
<td>25–28</td>
</tr>
<tr>
<td>Gelfusine⁹</td>
<td>77</td>
<td>0</td>
<td>62.5</td>
<td>35</td>
<td>33</td>
</tr>
<tr>
<td>Pentastarch 6%</td>
<td>150</td>
<td>0</td>
<td>150</td>
<td>200</td>
<td>32</td>
</tr>
</tbody>
</table>
| **Primary use is to expand blood volume with better duration than crystalloids and maintain i/v COP.**
* Gelatine-based; they last a few hours and have a poorer expansion than pentastarch (12–24hrs).
Up to 20 ml/kg (dogs) 10 ml/kg (cats) given as slow boluses; maintenance 1ml/kg/hr |

MW = average molecular weight (kD)
Ion concentrations are given in mmol/l
Whole blood or packed red blood cells

Management of anaemia, essential group matched in cats, subsequent transfusions cross-matched. Start at 1 ml/kg/hr for 30 minutes and monitor patient for adverse response, then give remainder of bag over 3–4 hrs. Fresh blood also contains plasma proteins, clotting factors (CF) and platelets.

Plasma

Fresh frozen (contains labile CF) or frozen, 10 ml/kg given as for blood; not volume replacing.

Cryoprecipitate

Thawed centrifuged plasma, precipitate – labile CF, supernatant – non-labile CF, albumin and immunoglobulin.

Albumin

Also provides COP. Human albumin can cause severe reactions (not seen in UK). 4 ml/kg of 20% solution raises albumin by 2.5 g/l.

Haemoglobin glutamer-200

(bovine) (oxyglobin)

Provides oxygen carrying capacity and COP. 30 ml/kg (dogs) lasts <72 hours in circulation; lower doses = shorter duration of action. 7 ml/kg maximum in cats, due to risk of pulmonary oedema.

Base crystalloid rate on body surface area and age, but:
- For immediate therapy 2 ml/kg/hr is a maintenance rate for adults and 4 ml/kg/hr for dogs/cats less than 4 months old.
- For shock, give 60–90 ml/kg/hr (dog), 40–70 ml/kg/hr (cat).

Haemorrhage: emergency management

5.2.1 Location and cause of bleeding 5.2.2 Controlling the bleeding 5.2.3 Emergency transfusion

Location and cause of bleeding
- Treatment is impossible unless the bleeding has been localized; this is aided by identifying the likely cause.
  - **Common causes** include trauma, neoplasia, thrombocytopenia, and inflammatory conditions (haemorrhagic gastroenteritis, cystitis, pericarditis and nasal disease).
  - **Less common causes** include *Angiostrongylus*, hypertension, and amyloidosis.
- **History** should focus on:
  - Likelihood of trauma, e.g. RTA, falls. Is the bleeding proportionate to any such trauma?
  - Previous responses following challenge to clotting, e.g. post-neutering.
  - Exposure to toxins (see 5.4), especially coumarins.
If the bleeding point is not obvious:
- Inspect carefully for ecchymoses or petechiation.
- Perform urinalysis (dipstick plus sediment exam if dipstick is positive).
- Image 3rd spaces (pleural, abdominal, pericardial).
- Image GIT: could there be GI bleeding?
- Check: is this DIC (disseminated intravascular coagulation – a complication of many severe disease processes and of trauma)?

5.2.2 Controlling the bleeding
- Once the bleeding point is obvious:
  - Do a PCV and platelet count – manual smear examination.
  - Image the area of bleeding.
  - Assess the clotting (see table above).
- If clotting is normal but there is vascular trauma:
  - Is the bleeding likely to stop without intervention?
  - Is local pressure going to be sufficient?
  - Is surgery appropriate? (see 4.3.3)
- If clotting is abnormal:
  - Is general supportive therapy going to be sufficient?
  - Will a transfusion be necessary? (5.2.3)

5.2.3 Emergency transfusion
- If clotting is defective, is it due to platelet or clotting factor deficiency (5.2.2)?
  - For platelet deficiency, give fresh, whole blood.
    - Platelets survive for <24hrs post transfusion.
  - For clotting factor deficiency, give plasma, vitamin K (5.4.3).
Trauma and RTA

- **History**: a detailed history of the incident should include:
  - How long ago?
  - Is the patient improving, stable or deteriorating?
  - Other medical problems, or current medication, e.g. NSAIDs.

- **Triage**: conduct a brief physical examination to assess:
  - Level of consciousness (LOC).
  - Respiratory rate and effort (5.5).
  - Heart rate and rhythm.
  - Perfusion: mucosal colour, pulse.
  - Temperature.

- **Personnel**: ideally three (leader, monitor, ‘gofer’).

- **Life-threatening conditions**: check ‘Airway, Breathing and Circulation’ (ABC).

- **Monitor**: begin a monitoring chart to evaluate trends:
  - Temperature, pulse, respiration.
  - Blood pressure and pulse oximetry.
  - Urine output.
  - PCV, TP, electrolytes, urea, blood gas.

- Establish i/v access as soon as possible.

- If LOC is declining: consider metabolic, hypoxia, hypotension, toxins, drugs, primary brain pathology.

- If patient also hyperexcitable, give sedative drugs, e.g. diazepam (5.8).

- Progression: alert → depressed → stupor → coma.

- The following actions should be considered:
  - Raise head and neck by up to 20°.
  - Secure airway against aspiration.
  - Try to maintain end-tidal CO₂ @ 30–35 mmHg.
  - O₂ therapy: maintain saturation >99%; pO₂>60%.
  - Avoid aggressive fluid therapy if possible; colloids are preferred to crystalloids.
  - If raised intracranial pressure is suspected, mannitol 0.25–1 g/kg can be given as a bolus over 20 minutes.
  - Glucocorticoids are of no proven benefit.

- **Heart rate and rhythm**: interpretation and response.

- Tachycardia: usually extracardiac, e.g. blood loss.
- Bradycardia: pressor, e.g. dobutamine 5–10 μg/kg/min CRI.
- Arrhythmia: only intervene if other parameters suggest it is having a significant effect on cardiac output – most arrhythmia are better untreated.

- Response to poor perfusion:
  - If pale, hypotensive, normal PCV – use fluid resuscitation:
    - Bolus colloid (5 ml/kg over 30min).
    - Crystalloid up to 90 ml/kg (dog), 70ml/kg (cat) in the first hour.
  - If PCV low – administer blood or blood substitute.
5.4 Poisoning

5.4.1 Poisons and common causes of poisoning
Poisonings are not common in dogs and cats. In general, dogs tend to ingest poisons while scavenging, whereas cats ingest them while grooming.
All pharmaceutical drugs, herbal products and nutraceuticals are potentially poisonous. See 3.13.8 for antimicrobial side effects, 4.2 for analgesics and individual body systems in PART 3.
Resources such as the Veterinary Poisons Information Service (VPIS [UK]) can offer online and telephone help.

5.4.2 History, physical examination and diagnosis
History is the key to appropriate therapy. Most poisonings have a clear history of known or likely exposure.
Poisonings in cats and dogs are generally acute. Cases can present with a variety of clinical signs and are therefore included in many differential lists.
Diagnosis: while specific diagnostic tests are available for some poisons, results are slow. Such tests are therefore confirmatory rather than therapeutically useful.
Routine haematology, coagulation profiles and biochemistry are helpful in establishing the specific poison, and how significantly affected the patient is.

5.4.3 Specific antidotes: vitamin K for coumarin poisoning
Few poisons have specific antidotes.
Coumarins in anticoagulant rodenticides (ACRs) work by blocking production of vitamin-K dependent clotting factors.
Initial vitamin K therapy: Use a small bore (23g) needle, with the vitamin K volume divided at several sites to which pressure can be applied if bleeding occurs.
For a known warfarin, first-generation coumarin, indandione, or an unknown coumarin: vitamin K 2.5 mg/kg s/c q8hr.
For a known second-generation coumarin, such as brodifacoum: vitamin K 5 mg/kg s/c q8hr.
General therapy for a known poison

- When the patient has been seen to contact/ingest toxin, or there is accidental overdose, the first action should be to prevent further absorption:
  - **Wash** skin with copious water. If using detergent, check that this will not increase the rate of absorption.
  - **Emetics**: administer within 2 hours of ingestion, provided that the poison is not a hydrocarbon or a caustic. Note that feeding the patient a small, moist meal improves chances of adequate emesis. Emetics include:
    - Washing soda crystal at back of tongue.
    - Salt (2 tsp) or mustard in cup of warm water.
    - Syrup of ipecacuanha 1:1 with water: 2–5 ml (cat); 10–20 ml (dog).
    - Apomorphine can cause intense vomiting: 0.1 mg/kg s/c (dog only).
    - Xylazine will cause sedation: 3 mg (cat); 1–3 mg/kg (dog) i/m.
    - 3% hydrogen peroxide: 2 ml/kg po.
- **Caustic/corrosive toxins**: use milk with demulcents via naso-oesophageal or orogastric tube.
- **Non-caustic/corrosive toxins**:
  - Gastric lavage with warm water/saline, under general anaesthesia.
  - Instil activated charcoal (1–3 g/kg), kaolin, chalk or barium.
  - Cathartics increase elimination of activated charcoal.
- Once absorption has been halted, the patient should be given symptomatic and supportive care appropriate to the toxic substance.

General therapy for suspected poisonings

- Ensure patient’s airway and adequate ventilation.
- Support cardiac output.
- Monitor heart rate and rhythm, and blood pressure.
- Check PCV, renal and hepatic parameters, electrolytes and acid/base status.
- Manage hydration using IVFT with crystalloids (Hartmann’s is usually appropriate) and colloids if BP is low.
- Treat CNS signs:
  - Manage seizures with benzodiazepines or barbiturates.
  - Manage excitation as for seizures, or with sedation (medetomidine, ACP/opioid).
- Maintain body temperature within normal range.
- Once the patient is stable, establish diagnosis and treat appropriately.
- See table, next page, for clinical signs and management of common poisonings.
### COMMON POISONINGS

<table>
<thead>
<tr>
<th>Poisoning</th>
<th>Abdominal pain</th>
<th>Anaemia/bruising</th>
<th>Blindness</th>
<th>Coma/collapse</th>
<th>Constipation</th>
<th>Convulsions</th>
<th>Depression/lethargy</th>
<th>Diarrhoea</th>
<th>Miosis/mydriasis</th>
<th>Dyspnoea</th>
<th>Excitement</th>
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<td>Xylitol (diet sugar)</td>
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* Signs depend on the type of battery, location, and whether leaking

† *Lilium* and *Hemerocallis* spp. (cats)
### 5.4 Poisoning

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Management</th>
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<tr>
<td>Fever</td>
<td>•</td>
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<tr>
<td>Haematuria</td>
<td>•</td>
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<tr>
<td>Inappetence</td>
<td>•</td>
</tr>
<tr>
<td>Incoordination/ataxia</td>
<td>•</td>
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<tr>
<td>Jaundice</td>
<td>•</td>
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<tr>
<td>Muscle tremor</td>
<td>•</td>
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<tr>
<td>Oliguria/anuria</td>
<td>•</td>
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<tr>
<td>Paralysis</td>
<td>•</td>
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<tr>
<td>Salivation</td>
<td>•</td>
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<tr>
<td>Thirst</td>
<td>•</td>
</tr>
<tr>
<td>Vomiting</td>
<td>•</td>
</tr>
</tbody>
</table>

1 = Emesis  
2 = Supportive  
3 = Activated charcoal and/or demulcent  
4 = Specific therapy  
5 = Removal surgically, endoscopically, or by lavage  
6 = Laxative

- **Fever**: 2, 3
- **Haematuria**: 1, 2, 3, 5
- **Inappetence**: 2, 4
- **Incoordination/ataxia**: 2
- **Jaundice**: 2
- **Muscle tremor**: 2, 3, 5
- **Oliguria/anuria**: 2, 4
- **Paralysis**: 2, 3
- **Salivation**: 2
- **Thirst**: 2, 3
- **Vomiting**: 1, 2, 5, 6

- **Fever**: 2, 3
- **Haematuria**: 1, 2, 3, 5
- **Inappetence**: 2, 4
- **Incoordination/ataxia**: 2, 3
- **Jaundice**: 1, 2, 3, 5
- **Muscle tremor**: 2
- **Oliguria/anuria**: 2
- **Paralysis**: 1, 2, 4, 5
- **Salivation**: wash, 2
- **Thirst**: 2
- **Vomiting**: flush mouth, 2
- **Thirst**: 2, 4

- **Fever**: 2, 3
- **Haematuria**: 1, 2, 3, 5
- **Inappetence**: 2, 4
- **Incoordination/ataxia**: 2, 3
- **Jaundice**: 1, 2, 3
- **Muscle tremor**: 2, 3
- **Oliguria/anuria**: 1, 2, 4, 5
- **Paralysis**: 2
- **Salivation**: wash, 2
- **Thirst**: 2
- **Vomiting**: flush mouth, 2
- **Thirst**: 2, 4
5.5 Dyspnoea: emergency management

**DYSPNOEA**

- **Exclude tachypnoea (see 2.3)**

- **Physical examination**
  - Mucosal colour;
  - Rectal temperature;
  - Pulse quality;
  - Nasal airflow;
  - Oral examination;
  - Cardiac rhythm;
  - Thoracic auscultation and percussion

- **History**
  - Trauma; previous cardiorespiratory disease; possibility of foreign body; intoxication; thromboembolic risk

- **Breathing character**
  - Marked inspiratory effort with increased chest excursion
    - **Obstructive disease**
      - Relieve/reduce obstruction; tracheostomy

  - Rapid shallow breathing
    - **Differentiate between pleural disease and pulmonary parenchymal disease**

  - Increased expiratory effort
    - **Small airway disease**
      - Inhaled bronchodilator: salbutamol 100 μg/actuation, repeat as needed; dexamethasone 0.1–0.5 mg/kg i/v; inhaled fluticasone 250 μg

- **Pulmonary parenchymal disease**
  - Oedema: furosemide 1–2 mg/kg q2–6 hrs or, better, 0.2–1 mg/kg/hr CRI
  - Pneumonia: aggressive i/v antimicrobials (3.13.4)

- **Pleural disease**
  - Drain carefully; sedate if necessary; stage if unstable – initially 10 ml/kg; chest drain if rapid air accumulation; repair diaphragmatic rupture
Emergency management of dyspnoea

- For a clinical approach to dyspnoea see 2.3.
- The key is to determine the level at which oxygenation is failing and then target treatment to that area:
  - Obstruction of airflow, e.g. tracheal collapse, feline asthma.
  - Failure of diffusion across the alveolar membrane, e.g. pneumonia, pulmonary oedema.
  - Failure of lung circulation, e.g. cardiac disease, pulmonary hypertension, pulmonary thromboembolism, anaemia.
  - Random therapy, e.g. giving furosemide to cases that do not have pulmonary oedema, can be counterproductive.
  - Dyspnoeic patients, especially cats, are very unstable and will die if stressed too far.
  - Patients with significant pulmonary parenchymal disease carry a poor prognosis.
Emergency management of collapse

- Collapse can be associated with the terminal stages of many diseases due to malnutrition, dehydration, cachexia and metabolic disturbances.
- Work from presenting signs to diagnosis, then treat the underlying disease.
In the majority of cases the following are rational initial steps while waiting for results of imaging and minimum database:

- Use of colloid.
- Conservative rates of crystalloids.
- Supplemental oxygen.
- Monitoring of temperature, pulse, respiratory and heart rate.
5.7 Urinary obstruction

**URINARY OBSTRUCTION**

- Male
- Risk of bladder rupture
  - No: Evaluate metabolic consequences
  - Yes: Emergency decompression: cystocentesis

- Female
- Causes of dysuria: UMN bladder, reflex dyssynergia, urethrospasm

- Emergency decompression: cystocentesis

* Potentially can cause fatal hypoglycaemia; monitor blood glucose every 30–60 minutes for 3 hours and use neutral insulin
† Use copious lubrication and infuse saline flushing while advancing the catheter to try and expand the urethra

5.8 Seizures

**PROLONGED OR MULTIPLE SEIZURES**

- History: previous episodes, duration, severity, progression, focal (asymmetric) or generalized, access to toxins
- Physical examination: TPR, mucosa, LOC
- Minimum database to exclude metabolic cause: PCV, electrolytes, glucose, calcium, BUN/creatinine, ALT, cholesterol and, ideally, bile acids or ammonia
- Multiple seizures
- Seizure >2 min
5.7 Urinary obstruction, 5.8 Seizures

**Management of hyperkalaemia**
Relieve obstruction; 10% calcium gluconate 0.5–1 ml/kg slow i/v; insulin and glucose*

**Post-renal azotaemia**
Yes

Minimize stress. Give small amounts of propofol, alfaxan or gaseous anaesthetic. Monitor and maintain BP. Dog: x-ray to assess level of obstruction

If catheterization fails, consider surgical options: urethrotomy (dog), perineal urethrostomy

**Hyperkalaemia (>7.5 mmol/l)**

Attempt to gently pass a catheter retrograde†; urethral massage; urohydropulsion

**Treat metabolic cause**
Glucose: 0.5 g/kg slow i/v bolus
Calcium gluconate (10%): 1 ml/kg over 20–30 min
Hyponatraemia: 0.9% saline
Hepatic encephalopathy: lactulose enema*, i/v ampicillin

Diazepam i/v or per rectum; start oral phenobarbitone

Diazepam (10 mg/2 ml): 0.5–1 ml/10 kg i/v or per rectum; repeat × 2–3 over 5–10 min
OR phenobarbitone: up to 30 min to effect, 16 mg/kg divided over 3–4 hrs

Poor response

Anaesthetize with thiopental or propofol; CRI propofol: 0.1–0.2 mg/kg/hr; diazepam† 0.1–0.5 mg/kg/hr

Poor response

* 3 parts lactulose to 7 of water; 1–2 ml/kg
† Diazepam is substantially absorbed on some plastics
5.9 **CPCR**

**CARDIO-PULMONARY CEREBRAL RESUSCITATION (CPCR)**

- **Pulse, no respiration**
  - Establish airway and ventilate with oxygen 6–12/min
  - Monitor for return of spontaneous breathing or for further deterioration into cardiac arrest

- **No heartbeat or respiration**
  - Ventilate at 6–12/min; cardiac compression 100/min
  - Fluid resuscitate if hypovolaemic: ≤50 ml/kg/hr (cat), <90 ml/kg/hr (dog); bolus of colloid 10 ml/kg
  - 1:1000 adrenaline 1 ml/10 kg; *intratracheal dose increase × 4

- **No respiration, slow or weak pulse**
  - Heartbeat returned
  - Consider direct cardiac compression

**Approach to CPCR**

- **Personnel** – ideally, four people are required to run resuscitation:
  - Person 1: ventilating the patient.
  - Person 2: performing cardiac compression.
  - Person 3: administering fluids and drugs.
  - Person 4: monitoring patient and keeping records of vital parameters, procedures and drugs given.
Procedure requires you to:

- Establish an airway using a cuffed endotracheal tube; if this is not possible, then perform tracheostomy to allow controlled ventilation.
- Establish i/v access.
- Maximize monitoring: pulse/heart rate and rhythm, respiration, temperature, mucosal colour, PLR, level of consciousness, ECG, pulse oximetry, end-tidal CO₂, electrolytes, blood gas and lactate.
Further reading

**Nutrition**

**General internal medicine**

**Ophthalmology**

**Cardiology**

**Respiratory medicine**

**Dermatology**

**Neurology**

**Endocrine and reproduction**

**Infectious disease**

**Oncology**

**Anaesthesia and analgesia**

**Soft-tissue surgery**
Units of measurement

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<tr>
<th>Measurement</th>
<th>SI units</th>
<th>Traditional units</th>
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* multiplication factors per unit
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<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
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<td>ACEi</td>
<td>angiotensin-converting enzyme inhibitors</td>
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<tr>
<td>ACTH</td>
<td>adrenocorticotropic hormone</td>
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<tr>
<td>ALKP</td>
<td>alkaline phosphatase</td>
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<tr>
<td>ALT</td>
<td>alanine aminotransferase</td>
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<tr>
<td>ARF</td>
<td>acute renal failure</td>
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<tr>
<td>BA</td>
<td>bile acids</td>
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<tr>
<td>BCS</td>
<td>body condition score</td>
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<tr>
<td>BSA</td>
<td>body surface area</td>
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<tr>
<td>CN</td>
<td>cranial nerve (CNI–CNXII)</td>
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<tr>
<td>COX</td>
<td>cyclooxygenase</td>
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<tr>
<td>CRD</td>
<td>chronic renal disease</td>
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<td>CRI</td>
<td>continuous rate infusion</td>
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<tr>
<td>CT</td>
<td>computed tomography</td>
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<tr>
<td>DIC</td>
<td>disseminated intravascular coagulopathy</td>
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<td>DM(B)</td>
<td>dry matter (basis)</td>
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<tr>
<td>ELISA</td>
<td>enzyme-linked immunosorbent assay</td>
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<tr>
<td>FCE</td>
<td>fibrocartilagenous embolus</td>
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<tr>
<td>FeLV</td>
<td>feline leukaemia virus</td>
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<td>FGF</td>
<td>free gas flow</td>
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<td>FIP</td>
<td>feline infectious peritonitis</td>
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<td>FIV</td>
<td>feline immuno-deficiency virus</td>
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<td>FLUTD</td>
<td>feline lower urinary tract disease</td>
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<td>FNA(B)</td>
<td>fine needle aspirate (biopsy)</td>
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<td>FORL</td>
<td>feline odontoclastic resorptive lesions</td>
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<tr>
<td>GFR</td>
<td>glomerular filtration rate</td>
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<tr>
<td>GI(T)</td>
<td>gastrointestinal (tract)</td>
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<tr>
<td>GME</td>
<td>granulomatous meningoencephalitis</td>
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<tr>
<td>HAC</td>
<td>hyperadrenocorticism</td>
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<td>HCM</td>
<td>hypertrophic cardiomyopathy</td>
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<td>H(L)</td>
<td>high (low)-dose dexamethasone suppression test</td>
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<td>IRIS</td>
<td>International Renal Interest Society</td>
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<tr>
<td>LMN</td>
<td>lower motor neurone</td>
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<tr>
<td>LOC</td>
<td>level of consciousness</td>
</tr>
<tr>
<td>MER</td>
<td>maintenance energy requirement</td>
</tr>
<tr>
<td>MRI</td>
<td>magnetic resonance imaging</td>
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<tr>
<td>NSAID</td>
<td>non-steroidal anti-inflammatory drug</td>
</tr>
<tr>
<td>NTI</td>
<td>non-thyroidal illness</td>
</tr>
<tr>
<td>NT-pro-BNP</td>
<td>N-terminal pro-brain natriuretic peptide</td>
</tr>
<tr>
<td>OA</td>
<td>osteoarthritis</td>
</tr>
<tr>
<td>OCD</td>
<td>osteochondritis dissecans</td>
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<tr>
<td>PCR</td>
<td>polymerase chain reaction</td>
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<tr>
<td>PDH</td>
<td>pituitary-dependent hyperadrenocorticism</td>
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<tr>
<td>PLi</td>
<td>pancreatic-specific lipase</td>
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<tr>
<td>PLR</td>
<td>pupillary light response</td>
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<tr>
<td>PP</td>
<td>polyphagia</td>
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<tr>
<td>PSS</td>
<td>portosystemic shunt</td>
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<tr>
<td>PTH(-RP)</td>
<td>parathyroid hormone (-related peptide)</td>
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<tr>
<td>PU/PD</td>
<td>polyuria/polydipsia</td>
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<tr>
<td>PUFA</td>
<td>polyunsaturated fatty acids</td>
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<tr>
<td>PUO</td>
<td>pyrexia of unknown origin</td>
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<tr>
<td>RER</td>
<td>resting energy requirement</td>
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<tr>
<td>ROM</td>
<td>range of movement</td>
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<tr>
<td>(T)T₄</td>
<td>(total) thyroxine</td>
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<tr>
<td>TLI</td>
<td>trypsin-like immunoreactivity</td>
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<tr>
<td>TSH</td>
<td>thyroid-stimulating hormone</td>
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<td>(U)GA</td>
<td>(under) general anaesthetic</td>
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<tr>
<td>UMN</td>
<td>upper motor neurone</td>
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<tr>
<td>UPC</td>
<td>urine protein: creatinine ratio</td>
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<td>UTI</td>
<td>urinary tract infection</td>
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<tr>
<td>VI</td>
<td>virus isolation</td>
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