Contents

CARDIAC vs RESPIRATORY – DIAGNOSTIC DILEMMAS

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Abstract

Small animal cases with cardiac or respiratory disease will often present with similar signs such as coughing or dyspnoea and rapid differentiation of these two categories of disease helps to guide further investigation and successful therapy.

During this lecture a number of case examples will be used to illustrate practical ways of differentiating these cases, emphasising key elements of the history and clinical examination before using further tests to attain a diagnosis and commence appropriate therapy.

Rapid Differentiation of Cardiac and Respiratory Disease

These cases will often present in a similar way but treatment of these fragile patients is very different and therefore rapid differentiation of cardiac and respiratory disease is required to ensure appropriate further investigation and correct treatment.

History

Features suggestive of chronic respiratory disease:

- Dogs will often be overweight
- Small breeds are over-represented
- Good exercise tolerance
- Harsh hacking cough with a terminal retch
- Coughing generally associated with exercise and excitement
- No weight loss and otherwise well
- Change in bark
- Contact with other coughing dogs
- Coughing in cats

Features suggestive of cardiac disease:

- In dogs - soft cough that tends to occur at rest however remember that cases with bronchial compression caused by left atrial enlargement can exhibit a harsh hacking cough with a terminal retch
- Reduced exercise tolerance
- Other signs of disease such as lethargy, loss of appetite and body condition
- Syncope
- Ascites

Clinical Examination

Features suggestive of respiratory disease:

- Normal or slow heart rate (can be marked sinus arrhythmia)
- Cough readily elicited by tracheal palpation
- Respiratory stridor or stertor
- Normal heart sounds
- Adventitious respiratory sounds may or may not be detectable

Features suggestive of cardiac disease:

- Tachycardia and loss of sinus arrhythmia
- Signs of reduced cardiac output such as weak femoral pulses, pale mucous membranes, pulse deficit
- Murmur or arrhythmia
- Ascites
- At risk breed such as CKCS, Boxer or Dobermann

Respiratory Emergencies

Recognition

- At risk breed e.g. brachycephalic
- History of a stressful event such as a hot day esp. after exertion,
- Open mouth breathing
- Noisy breathing (stridor / stertor)
- Dyspnoea / tachypnoea
- Orthopnoea – head and neck extended, elbows out
- Cyanosis of mucous membranes
- Anxious expression

- Standing or sternal recumbency rather than lateral

Common conditions

Common conditions include:

- Brachycephalic syndrome
- Laryngeal paralysis
- Tracheal collapse
- Feline asthma syndrome

Treatment

- Oxygen – flow by, mask, nasal prongs, oxygen tent
- Thoracocentesis if indicated (see later for more details)
- Sedation
- Albuterol ("Ventolin") inhaler for asthmatic cats
- Cooling – fan, wet towels, ice
- Tracheotomy (rarely required)
- Bronchodilators
- Palliative surgery after stabilisation for example arytenoid lateralisation for laryngeal paralysis; shorten soft palate and excise laryngeal saccules for brachycephalic syndrome

Monitoring

- Respiratory rate and effort
- Demeanour
- Body temperature
- Respiratory noise

Cardiac emergencies

Recognition of congestive heart failure

- At risk breed – Boxer, Dobermann, giant breeds
- History of coughing in dogs
- Dyspnoea
- Orthopnoea
- Froth from nostrils
- Anxious expression
- Ascites
- Cold extremities

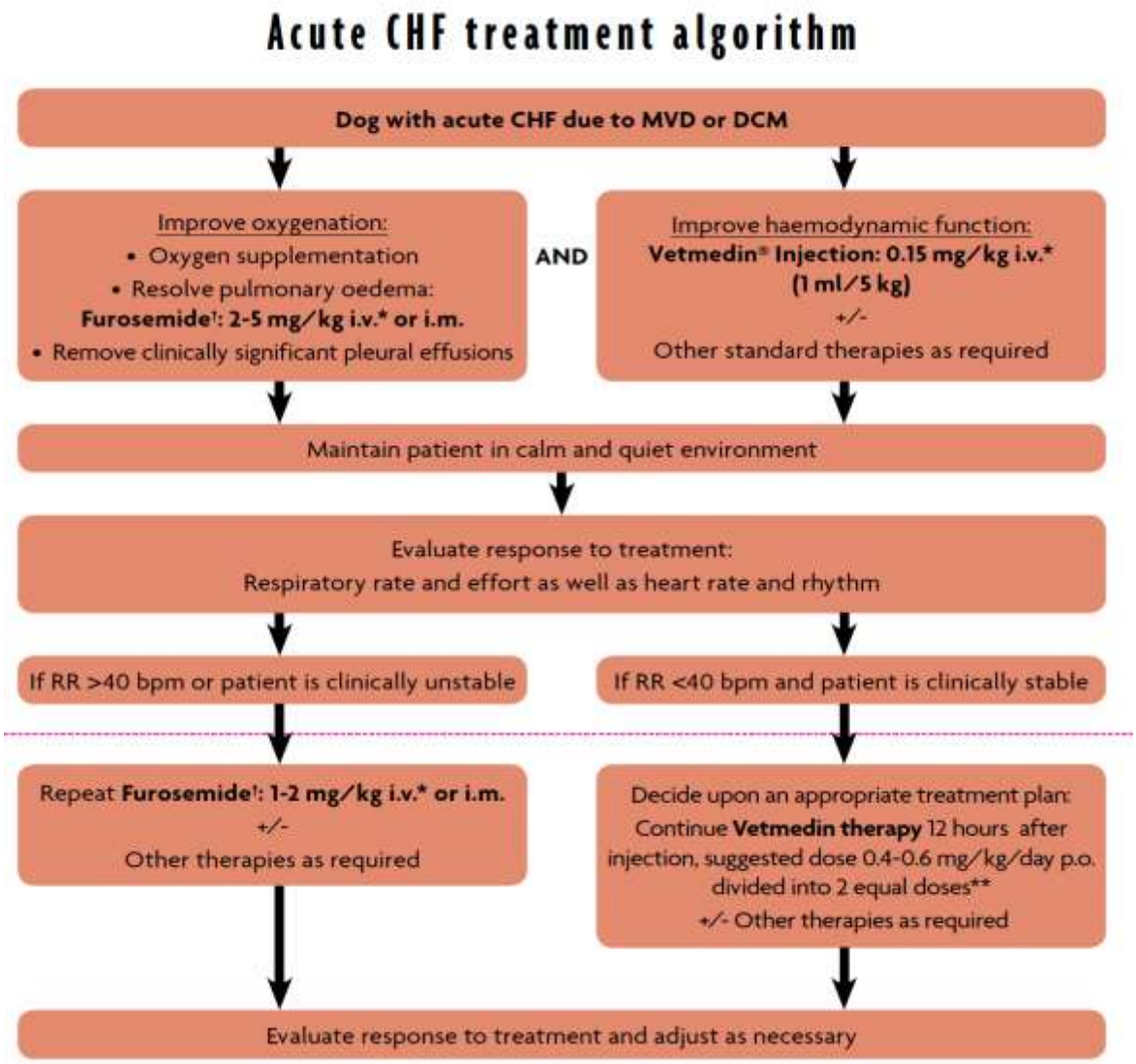
- Pale mucous membranes
- Weak pulses
- Rapid heart rate +/- arrhythmia
- Weight loss

Common conditions

- Dilated cardiomyopathy – generally large breed dogs
- Atrioventricular valve disease – generally small breed dogs
- Hypertrophic cardiomyopathy – cats
- Pericardial effusion – St Bernards, Golden Retrievers

Treatment

- Oxygen or cool air over face using a fan
- Intravenous cannula to allow administration of diuretics
- +/- Sedation
- Diuretics (unless pericardial effusion)
- Intravenous pimobendan
- Drainage of any clinically significant intrathoracic effusions
- Monitor and if necessary treat any haemodynamically significant dysrhythmias
- TLC – offer food / water
- Treatment of acute congestive heart failure in dogs may be summarised by the following algorithm:



Monitoring

- Heart rate (typically 100-150bpm in dogs with congestive heart failure, and 150-200bpm in cats with congestive heart failure)
- Respiratory rate and effort – typically decreases by 10bpm every hour from starting treatment
- Urination – should happen 1-2 hours after furosemide
- Body temperature – may be low at admission (especially in cats) and increases as circulation improves
- Demeanour - should become more alert
- Body position – able to lie down firstly in sternal and eventually in lateral
- Falling asleep is generally a good sign as cases have often been too breathless to sleep for several days
- ECG / Holter for monitoring arrhythmias
- Electrolytes and renal function should also be monitored.

Common acquired heart diseases in dogs

Dilated cardiomyopathy (DCM)

Aetiology:

- Genetic as tends to be seen in certain breeds and lines
- Taurine deficiency has been identified as a cause in American Cocker spaniels and may be involved in the disease process in Newfoundlands

Prevalence:

10% of cases of canine heart failure however if more sensitive screening tests such as echocardiography are employed then prevalence increases to 1:10 in Newfoundland dogs. The recently published PROECT study estimated a prevalence of 17% in Dobermans aged >6 years of age.

Clinical signs:

- Long pre-clinical phase thought to last from months to several years
- Arrhythmias may be present in both the pre-clinical and clinical stages especially in the large breeds
- Exercise intolerance, lethargy, coughing
- Weight loss
- Syncope or sudden death (Dobermans, Great Danes and Boxers)

Diagnosis:

- ECG – supraventricular dysrhythmias such as atrial fibrillation common in large breeds, ventricular dysrhythmias seen in Boxers and Dobermanns
- Radiography – progressive cardiomegaly usually with predominantly left sided enlargement, in the later stages signs of congestive heart failure will develop
- Echocardiography – indices of systolic function suboptimal; chamber enlargement, low velocity forward flow

Treatment of preclinical disease:

- Pimobendan – increasing use in pre-clinical disease
- Management of significant dysrhythmias such as complex ventricular arrhythmias in some breeds and/or atrial fibrillation. These cases are complex and treatment should be individualised – please contact a veterinary cardiologist for advice.

Treatment of clinical disease with documented heart failure:

- Diuretics
- Inodilator (pimobendan)
- ACE inhibitors
- Low salt diet in refractory cases
- Taurine supplementation in Newfoundlands and American Cocker spaniels (250mg PO q12h)
- Correct ratio of omega 3:omega 5 fatty acids ("Cardiguard")
- Antiarrhythmics depending on the results of ambulatory Holter monitoring

Myxomatous atrioventricular valve degeneration (MAVD)

Aetiology:

- Genetic, connective tissue disease?

Prevalence:

- Very common – almost all CKCS >8 years of age will have a systolic left apical murmur which is highly suggestive of this disease in this breed
- Also seen in many other small and medium sized breeds (e.g. Border collies), occasionally in the larger breeds
- Usually the mitral valve is affected but in approximately 30% of cases the tricuspid valve is also affected
- Disease tends to progress faster in males than in females

Clinical signs:

- Range from asymptomatic dog with a heart murmur to signs of congestive heart failure such as nocturnal coughing, weight loss, exercise intolerance, lethargy
- The signs are usually gradual in onset with the murmur being noted years before clinical signs develop but occasionally rapid deterioration will occur as a result of chordal rupture
- Syncope will occasionally occur
- Coughing may occur as a result of cardiac enlargement compressing the trachea, pulmonary oedema, pleural effusion or concurrent airway disease

Diagnosis:

- ECG – initially wide P waves suggestive of left atrial enlargement, the loss of sinus arrhythmia occurs with the rise in sympathetic tone in early congestive heart failure.

In the late stages of disease supraventricular tachydysrhythmias including atrial fibrillation may occur

- Radiographs – progressive enlargement of the left atrium, in the later stages also enlargement of the left ventricle. Signs of congestive heart failure starting with pulmonary venous congestion and leading to pulmonary oedema and/or pleural effusion in more advanced cases
- Echocardiography – colour and spectral Döppler will show the mitral and/or tricuspid regurgitation, enlargement of the associated atrium and ventricle, systolic function is preserved in the early stages but failure occurs in late disease. Systolic failure appears to occur more rapidly in the large breeds

Treatment:

- Diuretics are indicated if there are radiographic signs of congestive heart failure
- Pimobendan – improves systolic function, vasodilator, down regulates levels of some of the harmful cytokines
- ACE inhibitors – no proven advantage to use in asymptomatic stage (SVEP study) but many studies have demonstrated benefits in patients with congestive heart failure
- Methylxanthines (e.g. theophylline) may be beneficial in cases with coughing due to airway compression
- Anti-dysrhythmias are sometimes required (e.g. to control ventricular rate in atrial fibrillation but, especially in small patients, some of the drugs carry a significant risk of toxicity and therefore I would suggest contacting a veterinary cardiologist for advice.

Common acquired heart disease in cats

Hypertrophic cardiomyopathy

Aetiology

- Idiopathic, familial tendency demonstrated in Maine Coons and suspected in other breeds such as Ragdolls and British short hairs.

Prevalence

- Most common heart disease in cats

Clinical signs

- None!
- Murmur and/or dysrhythmia as incidental finding

- Weight loss, vomiting
- Signs of congestive heart failure
- Signs of systemic diseases that may result in heart failure in cats such as hyperthyroidism, hypertension, infiltrative disease

Diagnosis

- NT-pro-BNP assay
- All suspected cases should have T4 assay (if >6y.o.) and measurement of systolic blood pressure
- ECG – majority of cases in sinus rhythm
- Radiographs – may be normal or show cardiomegaly, signs of congestive failure. Cats with left sided failure more prone to pleural effusion than dogs
- Echocardiography – concentric hypertrophy of the left (+/- right) ventricle, left atrial enlargement suggestive of congestive heart failure, systolic anterior motion of the mitral valve, dynamic obstruction of the left ventricular outflow tract, evidence of diastolic dysfunction.

Treatment

- If no evidence of congestive failure then monitoring q6-12months using body weight, exercise ability, echocardiography and/or NTproBNP assays
- If congestive heart failure then furosemide and ACE inhibitor
- If at risk of thromboembolism then aspirin (75mg PO q3-7days) or dalteparin
- Supportive treatment – ensure adequate nutrition, potassium supplementation, TLC.

Screening scheme

- Initiated by Feline Advisory Bureau and Veterinary Cardiovascular Society
- Based on annual echocardiography examinations
- See websites for more information
- Genetic testing – for more information see:
    - http://www.langfordvets.co.uk/lab_pcr_felinehcm.htm

Overview of cardiac therapeutics

Diuretics

Furosemide

- Commonly used potent loop diuretic
- Rapid onset particularly if given intravenously

- Intravenous use may also result in venodilation
- Once the patient is stable then the dose should be reduced to the lowest effective dose
- Risk of electrolyte abnormalities including hypokalaemia especially if anorexic
- Risk of azotaemia
- Furosemide may inhibit the excretion of salicylates
- NSAIDs reduce the kidney's ability to respond to furosemide
- May exacerbate diabetic and hyperglycaemic states

Spironolactone

- Competitive aldosterone receptor antagonist in DCT
- Dose 1-2mg/kg PO q12-24h
- Weak diuretic but potassium sparing
- In CHF the concentration of aldosterone can be x20 due to increased production and reduced clearance
- ACEI do not fully suppress aldosterone secretion
- Aldosterone has growth promoting properties in non-epithelial cells → perivascular fibrosis
- Na overload → expression of isoforms of Na pumps which affects the contractility of myocytes, the reactivity of smooth muscle cells, the growth of fibroblasts and turnover of collagen

ACE inhibitors

- Dilate the efferent renal arteriole thereby reducing glomerular filtration pressure
- Peripheral vasodilation
- Increase sodium and water loss
    - Decreased aldosterone
    - Decreased vasopressin
- Decrease myocardial and vascular remodelling
- Improved baroreceptor function and enhanced sympathetic / parasympathetic tone
- Strong evidence for efficacy – numerous canine and human papers have shown that patients receiving ACEI improve clinically and also have prolonged lifespan.

There are few prospective, blinded, placebo controlled clinical trials in veterinary medicine but most of these have been related to ACEI. Two trials to note are:

- BENCH – benazepril administration resulted in a significant improvement in survival in dogs with MMVD and CHF
- LIVE – enalapril administration significantly increased the time to treatment failure in dogs with MMVD or DCM and CHF

Pimobendan

- Peripheral vasodilation via phosphodiesterase III and V inhibition, increased peripheral cAMP and cGMP
- Positive inotropic action via sensitising the contractile apparatus to calcium
- Evidence for efficacy:
    - Shown to prolong lifespan in Dobermans with DCM
- One study showed a reduced risk of an adverse outcome in dogs with early valvular disease
    - PITCH study – dogs treated with pimobendan, ACEI and furosemide had improved survival compared to dogs treated with placebo
    - QUEST study – survival times of two groups of dogs with CHF secondary to acquired atrioventricular valve disease showed that median survival for dogs receiving pimobendan was 267 days v benazepril 140days.
    - PROTECT – compared the time to sudden death or the onset of congestive heart failure in Dobermans with pre-clinical dilated cardiomyopathy treated either with pimobendan or a placebo and showed that pimobendan increased the median time to reaching endpoint by 9months.

Antidysrhythmics

- Digoxin, beta blockers and calcium channels are all used commonly and although there is general agreement as to efficacy, the evidence for this has been obtained from uncontrolled case series.

DISEASES OF THE RESPIRATORY SYSTEM

Laryngeal Paralysis (LP)

Condition characterised by a failure of the laryngeal cartilages to open during inspiration, creating a partial or complete upper airway obstruction.

Aetiology

- Idiopathic - primary dysfunction of the recurrent laryngeal nerve that controls the laryngeal musculature

- Congenital - reported for some breeds
- Secondary - localised polyneuropathy, myopathies and traumatic events that affects the laryngeal innervation or musculature. Systemic disease can also be associated with laryngeal paralysis (e.g. hypothyroidism).

Pathophysiology

Generally slowly progressive, until an acute exacerbation (i.e. hot weather) causes a life-threatening crisis. Aspiration pneumonia can also occur during violent gasping and gagging episodes

Signalment

- Breed
    - Idiopathic forms - Labradors and giant breeds.
    - Congenital: white GSDs
- Age for acquired form usually middle aged - older dogs

History

- High pitched bark
- Inspiratory dyspnoea and stridor
- Exercise intolerance and panting
- Cough and cyanosis are occasionally noted after exercise or excitement

Physical examination

- Often dog is normal at time of presentation; useful to examine the dog before & after exercise
- Cough
- Stridor
- Dyspnoea
- Skeletal muscle atrophy, particularly on the head, and / or other signs of poly-neuropathy or myopathy

Diagnosis

- History of gradually developing inspiratory stridor is strongly indicative of LP.

- Direct observation of arytenoid function during respiration under light general anaesthesia. When paralysed the laryngeal wall collapses during inspiration, closing the opening of the trachea and blocking the air flow.

Treatment

- Sedation may reduce the anxiety in most affected patients
- Treatment of the primary disorder if identified
- Corticosteroids (i.e. dexamethasone) iv reduce laryngeal oedema
- Oxygen supplementation
- Surgical arytenoid lateralisation is the palliative treatment of choice

Prognosis

Reasonable after surgical correction but risk of aspiration pneumonia

Tracheal Collapse

In some dogs the tracheal cartilage lacks structural rigidity and dorsal membrane is flaccid and wide. These anatomical deformities are more commonly seen in small toy breeds.

Aetiology

- ?Congenital predisposition
- Deficiency of glycoproteins or glycosaminoglycans
- Secondary traumatic collapse

Pathophysiology

- The tracheal rings are abnormal so that the individual rings become flattened. The extent of the affected trachea may include the cervical trachea all the way through the mainstem bronchi, although the problem is more often seen at the thoracic inlet.
- The cervical trachea collapses during inspiration, while the intrathoracic trachea collapses during expiration.
- The dynamic obstruction may cause mucosal inflammation → failure of the muco-ciliary escalator → lower airway diseases

Signalment

- Breed - Min. Poodles, Pomeranians, Yorkshire terriers
- Age - generally middle-aged adult dogs

History

- "Goose-honking", non-productive cough, associated with excitement or exercise
- Respiratory distress and cyanosis are occasionally seen

Physical examination

- Characteristic cough can be easily elicited by gentle palpation of the trachea

Diagnosis

Bronchoscopy

- Allows confirmation of diagnosis, grading of the severity of the disease (scale from 1 to 4) and determine the extent of the process.
- This procedure may also allow cytological examination and culture of BAL as many cases will have concurrent lower airway disease.

Radiography

- Ideally two radiographs (end-inspiration and end-expiration) should be taken to visualise different diameter of the trachea.

Treatment

Non-medical:

- Weight reduction
- Use of a harness rather than a collar
- Avoidance of excitement, overheating, strenuous exercise

Medical:

- Bronchodilators
- Corticosteroids (to control inflammation)
- Lomotil
- Cough suppressant - pholcodine

Surgical:

- May be considered for severely affected cases that do not respond to non surgical management.
- Historically high risk of complications.
- Tracheal stenting showing promise.

LOWER AIRWAY DISEASES

Chronic Tracheobronchial Syndrome (CTS)

It is a term that identifies a respiratory condition in dogs that have had a history of acute tracheobronchitis and have progressed to develop a mild chronic cough.

Clinical signs

- Mild chronic cough, usually stimulated by exercise, excitement or lead pulling
- Usually no other signs of respiratory or systemic illness

Diagnosis

- History of acute tracheobronchitis or contact with dogs affected by kennel cough
- Radiography: mild-moderate bronchial pattern
- Bronchoscopy: mild mucosal hyperaemia
- BAL: suggestive of low grade inflammation with mucus plugs, epithelial cells, neutrophils and macrophages may be observed on cytology. Usually no bacterial growth.

Therapy and management

- May resolve spontaneously
- Short term anti-inflammatory steroids for cases with severe inflammation
- Anti-tussives may help in controlling the nocturnal cough
- Use of a harness rather than a collar
- Avoidance of excessive exercise and excitement
- Avoidance of dusty / smoky environments

Feline asthma syndrome

Aetiology

Condition characterised by airway hypersensitivity resulting in intermittent bronchospasm. Mycoplasma infection increasingly noted and may be a part of the disease process.

Pathophysiology

- Intermittent airway bronchospasm result in severe obstructive dyspnoea
- Air trapping as air can enter the distal airways but then trapped
- Respiratory secretions get trapped in the narrowed airway thereby exacerbating the obstruction
- Chronic coughing between the acute flare ups of disease

Signalment

Seems to be more common in pedigree cats especially Siamese and Burmese but seen in all breeds.

Clinical Signs

- The disease is characterised by intermittent coughing with occasional episodes of acute dyspnoea.
- Normal cardiac output signs.
- Respiratory wheezes and crackles may be audible during flare ups of disease.
- Cyanosis and severe respiratory distress may occur.

Diagnosis

Clinical history may be sufficient in the acute setting and stabilisation is required prior to further testing to obtain a definitive diagnosis.

- Haematology / serum biochemistry – usually unremarkable
- Thoracic radiographs – normal cardiac silhouette, overinflation of the lungs, generalised increase in bronchial markings, +/- consolidation of the right middle lung lobe.
- Bronchoalveolar lavage – increased number of eosinophilis +/- background of inflammatory cells such as neutrophils and mucus. No evidence of parasitism. +/- PCR testing for Mycoplasma.

Treatment

Acute disease:

- Oxygen
- +/- sedation
- Albuterol
- Steroids

Chronic disease:

- Weight loss
- Minimise inhaled allergens/irritants
- Bronchodilators – oral or inhaled
- Steroids – oral or inhaled
- Doxycycline if Mycoplasma isolated

References and further reading

1. IMPROVE investigators. Acute and short term haemodynamic, echocardiographic and clinical effects of enalapril maleate in dogs with naturally acquired heart failure: results of the Invasive Multicentre, PROspective Veterinary Evaluation of Enalapril Study. J.Vet.Intern.Med. 9, 234-242. 1995.

2. BENCH study group. The effect of benazepril on survival times and clinical signs of dogs with congestive heart failure: results of a prospective, randomised, double-blinded placebo controlled long term clinical trial. J.Vet.Cardiol. 1, 7-18. 1999.

3. COVE study group. Controlled clinical evaluation of enalapril in dogs with heart failure:results of the Co-operative Veterinary Enalapril Study Group. J.Vet.Intern.Med. 9, 243-252. 1995.

4. Ettinger, SJ. Benitz A. M., Ericsson, G. F., Cifelli, S., Jernigan, A. D., Longhofer, S. L., Trimboli, W., and Hanson, P. D. Effects of enalapril maleate on survival of dogs with naturally acquired heart failure. J.Am.Vet.Med.Assoc. 213, 1573-1577. 1998.

5. Pitt, B, Zannad, F., et al, and for the RALES investigators. The effect of spironolactone on morbidity and mortality in patients with severe heart failure. N.Engl.J Med. 341, 709-717. 1999.

6. Luis-Fuentes, V., Kleeman, R., Justus, C. et al. The effect of a novel inodilator pimobendan on heart failure status in Cocker spaniels and Dobermanns with idiopathic dilated cardiomyopathy. Proceedings of British Small Animal Veterinary Association Congress 2000.

7. Smith, P.J., French, A.T., Van Israel, N., et al. Efficacy and safety of pimobendan in canine heart failure caused by myxomatous mitral valve disease. Journal of Small Animal Practice, 46, 121-130. 2005

8. Haggstrom, J., et al. QUEST Study. Journal of Veterinary Internal Medicine 22:5. 2008

9. Summerfield, N.J., Boswood, A. et al. Efficacy of pimobendan in the prevention of congestive heart failure or sudden death in Doberman Pinschers with pre-clinical dilated cardiomyopathy (The PROTECT Study). J. Vet. Int. Med 26: 1337-1349. 2012.

Notes page

APPROACH TO HEART MURMURS IN SMALL ANIMAL PATIENTS

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Abstract

Heart murmurs are a common finding in small animal patients and the key challenge is to differentiate cases which require further investigation and treatment from those where the heart murmur is likely to be an incidental finding. The aim of this lecture is to present a logical approach to heart murmurs in puppies, older dogs and also in cats. There will also be a review of some of the tests commonly used to screen for significant cardiac disease in general practice such as cardiac biomarkers and thoracic radiographs.

Does this murmur matter?

What is a murmur?

A heart murmur is an abnormal sound detected on auscultation generally associated with turbulent blood flow and/or vibration of cardiac structure(s).

Careful auscultation is ideally performed on a calm, relaxed patient in quiet surroundings with the patient breathing normally. A clinical examination is often performed prior to auscultation as cardiac output signs such as mucous membrane colour, capillary refill time and femoral pulse rate / quality give useful indications of global cardiac performance prior to auscultation.

Auscultation is performed starting at the left apex where the precordial impulse is palpable then moving craniodorsally before repeating on the right side. Auscultation of the sternal area using the bell of the stethoscope can be useful to detect gallop sounds. The lung fields are then auscultation to detect adventitious lung sounds such as crackles and wheezes. Abnormal cardiac sounds can be described according to their timing in the cardiac cycle, loudness and also point of maximal intensity.

Murmur classification - 1 - timing

Timing in the cardiac cycle is important:

- Systolic – "lub-sh-dub" or "swoosh"
- Diastolic – "lub dub whoooooo"
- Continuous – "swoosh whoooo"

Murmur classification - 2 – loudness

- Grading out of 6 or quiet, medium and loud
- Loudness is not always proportional to severity

Murmur classification - 3 – point of maximal intensity

- Murmurs of mitral regurgitation are often loudest over the left apex
- Murmurs associated with pulmonic and aortic valve stenosis are generally loudest over the left base
- Murmurs associated with tricuspid regurgitation are generally loudest over the right hemithorax

Sensitivity and specificity (acquired heart disease)

The sensitivity and specificity of auscultation for detecting significant heart disease depends on whether the patient is a cat or dog.

Cats:

- 21% of apparently healthy cats have murmurs.[1]
    - In 57 apparently healthy cats with murmurs, there was echocardiographic evidence of heart disease in 50%.[2]
- About 50% of cats with cardiomyopathy will have a murmur.[3]

Dogs:

- There is excellence co-relation between the presence of a murmur and the presence of the most common canine heart disease - chronic degenerative valvular heart disease. [6]

Repeatability of auscultation

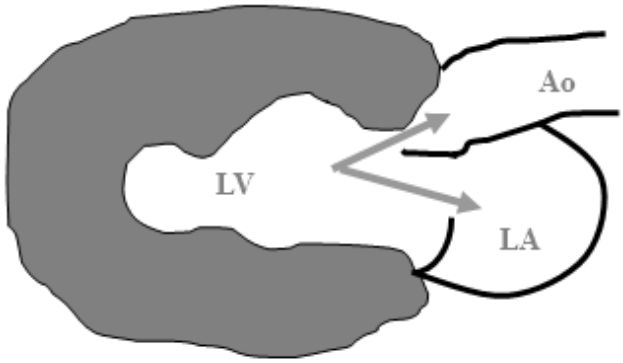
The repeatability of a technique refers to how consistently an abnormality is detected and is a comparison either between the same individual on multiple occasions or a comparison between different individuals.

- Cat murmurs may be variably present due to dynamic obstruction caused by intermittent systolic anterior motion of the mitral valve. This abnormal valve movement is present in some cats with hypertrophic cardiomyopathy and describes

the septal leaflet of the mitral valve being sucked into the left ventricular outflow tract during systole.

LVOT obstruction

Mitral regurgitation due to systolic anterior motion



- Using dogs with low intensity murmurs or dogs free of heart murmurs, inter-observer agreement was positively correlated to the level of experience at whilst the dogs were at rest but the agreement was poor after exercise.[9]

NTproBNP

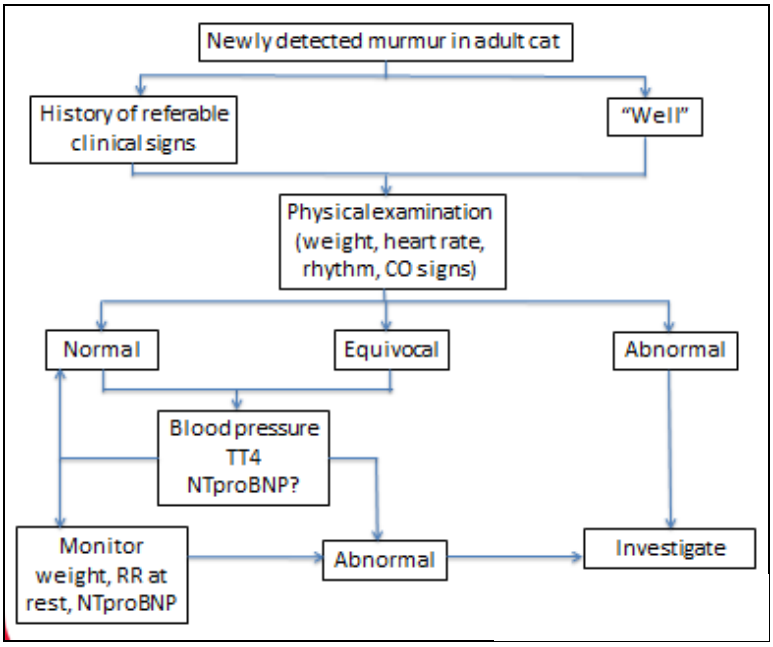
This is a commercially available assay ("Cardiopet"®) which has been marketed as a means of differentiating heart disease from heart failure and also cardiac versus respiratory disease.

- NT-proBNP is a peptide released from the ventricles in response to stretch
- Marker of volume load
- Increased in cases with CHF
- Sample handling is important – either collect into EDTA, separate serum within 30mins and then freeze OR use tubes containing a protease inhibitor. Sub-optimal sample handling tends to LOWER NTproBNP levels.
- Sensitivity / specificity is reasonably high but not 100%
- Concurrent disease (esp. azotaemic renal disease) will raise values

Cat murmurs

Heart murmurs are common in cats and due to their generally sedentary lifestyle it can be hard to differentiate cats with heart disease from cats with heart failure. However detection of cats that have reached, or are close to reaching, the "tipping point" separating heart disease from heart failure is pivotal to identifying which cases require further investigation and treatment.

This algorithm shown next shows a possible approach to heart murmurs in cats.



Primary v secondary disease

Whilst primary idiopathic hypertrophic cardiomyopathy is the most common heart disease affecting adult cats, heart disease may be secondary to systemic disease and conditions that can result in secondary cardiomyopathy include:

Primary cardiac disease	Systemic disease with secondary LV hypertrophy
Hypertrophic cardiomyopathy	Hypertension
Restrictive cardiomyopathy	Hyperthyroidism
Unclassified cardiomyopathy	Acromegaly
Dilated cardiomyopathy	Myocardial infiltration

Puppy Murmurs

Congenital heart disease- prevalence

Congenital heart disease is more common in dogs than in cats and is also strongly related to breed. To give an estimation of the prevalence of this problem:

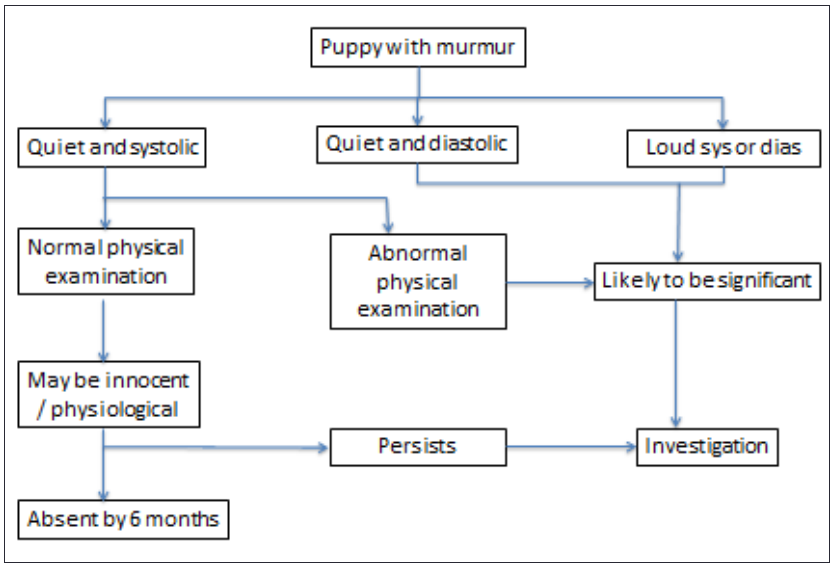
- In young boxers in Switzerland the overall prevalence of heart murmurs was 26.5 %.[7]
- Boxer puppies auscultation and echocardiography performed 7 times between the ages of 7 weeks and 36months. Presence and intensity of heart murmurs varied in the same dog and between dogs, but flow velocities did not change.[8]

Flow murmurs are common in puppies. Whilst precise aetiology is not known it has been postulated that lower PCV and serum protein level in puppies lower the velocity at which

turbulent flow will occur thereby creating audible murmurs. Flow murmurs are generally soft (grade 1-2/6), localised to the left heart base and are not audible by 4-6months of age.

It is important to note that even in puppies which severe congenital heart disease, clinical signs such and stunted growth and lethargy are rare in the first year of life and therefore the absence of signs referable to poor cardiac output and/or congestive heart failure should not preclude further investigation.

This algorithm below shows a possible approach to heart murmurs in puppies.



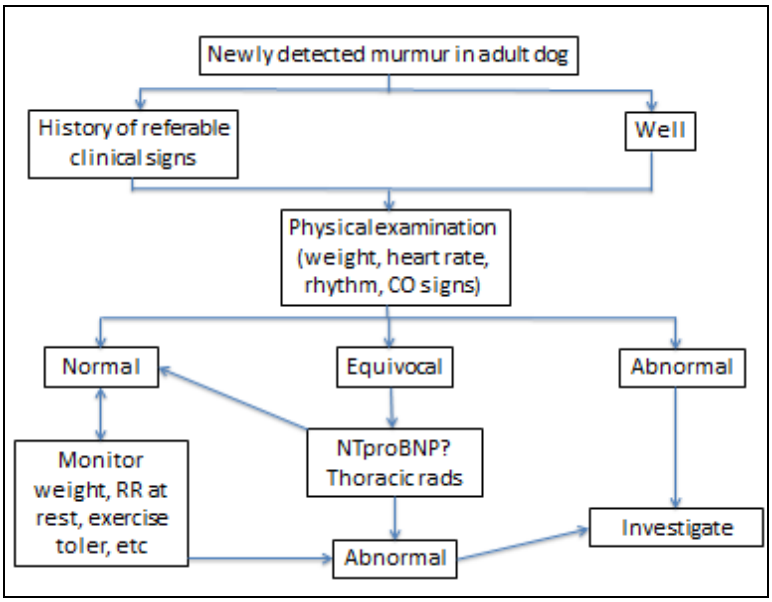
In summary refer early if:

- Loud systolic murmur
- Diastolic or continuous murmur
- Dog intended for breeding
- NB referable clinical signs are rare at <1y of age

Further information on congenital heart disease can be found in the BSAVA Manual of Cardiac and feline Cardiorespiratory medicine, 2nd edition. Eds Luis Fuentes V, Jonhson, L.R., Dennis, S. BSAVA Publishing 2010.

Acquired murmurs in adult dogs

Heart murmurs are commonly detected in adult dogs and this algorithm shows a possible approach to these cases.



At risk breeds

Some breeds are predisposed to developing heart murmurs for example:

- Mitral valve disease is more common in CKCS, small breed dogs and border collies
- Dilated cardiomyopathy is more common in large breeds, Boxers and Dobermans

Athletes

Human and canine athletes may develop heart murmurs secondary to cardiac adaptions to strenuous exercise. An athletic heart is more likely to be seen in young fit dogs and is characterised by enlargement of the ventricles (especially the left ventricle) and low contractility at rest. These individuals have excellent exercise tolerance; no abnormalities are detected on physical examination except a soft systolic murmur. Whilst human athletes are predisposed to developing atrial fibrillation, this does not seem to occur in dogs and cats.

Murmurs secondary to systemic disease

Dogs with severe systemic disease may develop heart murmurs. Conditions that tend to lower blood viscosity such as anaemia or hypoproteinaemia increase the likelihood of turbulent flow. High output states such as fever and sepsis can also potentially generate conditions that would result in turbulent flow.

Pre-breeding screening

Screening small animals for cardiac disease prior to breeding is increasingly common with the aim of reducing the frequency of heart disease in these breeds.

- Schemes exist for Boxers, Newfoundlands, CKCS and cats
- Based on auscultation +/- echocardiography
- Annual checks are required in some breeds

As the requirements vary for different breeds then please contact either myself or one of the other cardiologists on the Veterinary Cardiovascular Society breed screening panel for advice.

References

1. Assessment of the prevalence of heart murmurs in overtly healthy cats. Côté E, Manning AM, Emerson D, Laste NJ, Malakoff RL, Harpster NK. J Am Vet Med Assoc. 2004 Aug 1; 225(3):384-8.
2. Cause of heart murmurs in 57 apparently healthy cats. Dirven MJ, Cornelissen JM, Barendse MA, van Mook MC, Sterenborg. Tijdschr Diergeneeskd. 2010 Nov 15; 135(22):840-7.
3. Feline idiopathic cardiomyopathy: a retrospective study of 106 cats (1994-2001). Ferasin L, Sturgess CP, Cannon MJ, Caney SM, Gruffydd-Jones TJ, Wotton PR. J Feline Med Surg. 2003 Jun;5(3):151-9
4. Population characteristics and survival in 127 referred cats with hypertrophic cardiomyopathy (1997 to 2005). Payne J, Luis Fuentes V, Boswood A, Connolly D, Koffas H, Brodbelt D. J Small Anim Pract. 2010 Oct;51(10):540-7
5. Population and survival characteristics of cats with hypertrophic cardiomyopathy: 260 cases (1990-1999). Rush JE, Freeman LM, Fenollosa NK, Brown DJ. J Am Vet Med Assoc. 2002 Jan 15; 220(2):202-7.
6. Heart sounds and murmurs: changes related to severity of chronic valvular disease in the Cavalier King Charles spaniel. Häggström J, Kvart C, Hansson K. J Vet Intern Med. 1995 Mar-Apr;9(2):75-85
7. Prevalence of heart murmurs, aortic and pulmonic stenosis in boxers presented for pre-breeding exams in Switzerland. Höpfner R, Glaus T, Gardelle O, Amberger C, Glardon O, Doherr MG, Lombard CW. . Schweiz Arch Tierheilkd. 2010 Jul; 152(7):319-24.
8. A prospective study of systolic ejection murmurs and left ventricular outflow tract in boxers. Höglund K, Häggström J, Bussadori C, Kvart C. J Small Anim Pract. 2011 Jan;52(1):11-7
9. Low intensity heart murmurs in boxer dogs: inter-observer variation and effects of stress testing. Höglund K, French A, Dukes-McEwan J, Häggström J, Smith P, Corcoran B, Kvart C. J Small Anim Pract. 2004 Apr; 45(4):178-85.

Notes page

STIFLE SURGERY: CRANIAL CRUCIATE DISEASE AND MANAGEMENT OF PATELLAR LUXATIONS

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While some dogs rupture their CrCL as result of trauma, it is now recognised that most develop CrCL rupture as a result of progressive pathologic ligamentous failure, under conditions of normal loading. The majority of dogs present with unilateral rupture, although studies have reported 11-17% present with bilateral rupture. For dogs that present with unilateral rupture, 22 – 54% subsequently rupture the contra-lateral CrCL a median of 10 to 17 months after the initial diagnosis.

The number of recommended repair techniques for CrCL rupture runs well into three figures and shows no sign of abating. Intra-articular grafts, extra-capsular suture stabilisation and proximal tibial osteotomy techniques are currently recommended. Tibial osteotomy techniques do not attempt to provide stability of the stifle but alter joint geometry to eliminate cranial tibial thrust. Thus *functional* joint stability is achieved during weight bearing. It was universally accepted that whatever technique is employed, meniscal pathology must be addressed, but even that is now being questioned. It is also questionable whether some form of meniscal release technique is necessary, or advisable, when performing tibial osteotomy techniques.

An area of emerging interest, and perhaps one that will render the choice of surgical procedure irrelevant, is the post-operative management of these dogs. It should be an integral part of CrCL rupture treatment and the benefits of physiotherapy, including hydrotherapy in its various forms, needs to be scientifically evaluated.

Finally, the inevitable question has to be “which technique is best?” It has become fashionable to use evidence based medicine (EBM) to assist clinicians with the decision-making process for a variety of clinical problems. The definition of EBM was initially directed at the conscientious, explicit, and judicious use of current best evidence in making decisions about the care of individual patients. It has subsequently evolved to include the integration of individual clinical expertise, patient values, and the best available clinical evidence from systematic research. Level 1 evidence is likely to be optimally achieved through three

methods of evaluation: i) force plate analysis, ii) subjective and objective evaluation by the clinician and iii) subjective evaluation by the pet owner. However, while there is some agreement on what is an appropriate method for i), there is absolutely none for ii) and iii).

Aragon & Budsberg using EBM concluded in a somewhat damning fashion "there is not a single surgical procedure that has enough data to recommend it can consistently return dogs to normal function after CrCL injury"; this despite canine cruciate repair costing in excess of a billion dollars every year in the United States, and the incidence of cruciate surgery in dogs exceeding that in humans.

Extra-articular stabilisation techniques

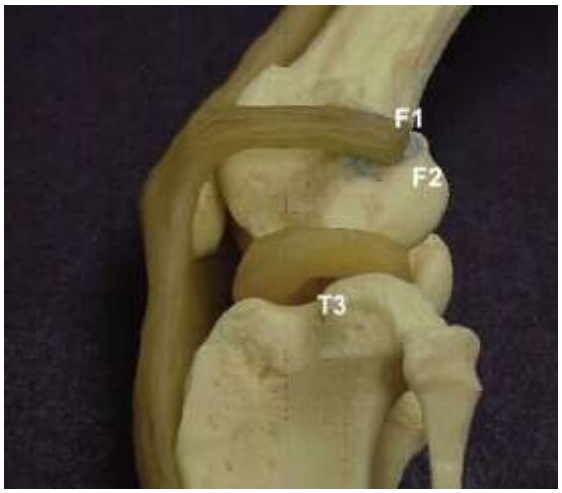
The placement of a lateral fabellar suture (LFS) was first described by DeAngelis and remains widely performed today, especially in smaller dogs.

The aim is to increase the tension in the peri-articular soft tissues and limit cranial instability. Tension is usually applied on the lateral aspect but there are techniques which involve both aspects of the joints. Tightening the lateral aspect will reduce cranial instability and limit internal rotation, although the changes in motion are not physiological and alter the stress patterns within the joint.

Though these techniques can be successful, both experimentally and clinically, there is likely to be joint laxity about 3 to 6 weeks after surgery. The clinical significance of this is unknown. All extra-articular techniques will limit joint motion to some degree. Suture materials should either be permanent or one of the longer lasting degradable materials. Orthopaedic wire has also been used for lateral sutures. Although it will break, failure occurs at a stage where it is probably no longer required.

The DeAngelis suture is tightened at the standing angle (140°) and lies between the joint capsule and the lateral fascia. Provision of early cranio-caudal stability has been suggested to encourage early return to limb function in the post-operative period. Over time, all materials utilized for this purpose appear to fail and ultimately stifle stability is maintained by peri-articular fibrosis. Failure of this procedure in the early p/o phase may lead to recurrence of joint instability, lameness, pain, and a more rapid progression of osteoarthritis. Causes of failure may include: premature failure of the suture, alterations in the attachment points, or entrapment of soft tissues at the initial time of surgery. Further, if the suture is placed in a position that is not isometric throughout the range of motion, it may overload the suture and its anchoring points.

Suture anchorage sites



There are two suture placement sites recommended for reconstruction of the CCL deficient stifle joint. The site adjacent to the femoro-fabellar ligament (F1) to the region of the caudal wall of the long digital extensor groove (T3) is one site. The second site is located at the caudoventral lateral femoral condyle at the level of the distal pole of the fabella (F2); the tibial site is the T3 site as described above.

Based on work by Roe et al Vet Comp Orthop Traumatol (2008) ***21,*** *215-220*

However, it has to be recognised that studies identifying the isometric points have been performed on a limited number of cadavers and variations in individual animals is likely. Further, none are truly isometric. Indeed, the term *quasi*-isometric is now being used!

Suture material

Efforts continue to identify the ideal material for use during extra-articular stabilization. The ideal properties for such a prosthetic should include: non-tiring strength, biologically inert, aseptic, inelastic, easily handled, inexpensive, excellent knot security, knot compactness and able to withstand cyclical and tensile loading.

Numerous studies have been published comparing and contrasting materials, fixation methods and sterilization methods of the suture. For many years monofilament nylon leader line had been a popular choice as it fulfils many of these requirements. However, nylon leader line is known to undergo significant elongation when knotted and when tied, forms bulky knots that may increase patient morbidity by causing increased tissue irritation. Knots may become more difficult to securely tie as the material increases in size. Knotting causes deformity and bending, which creates points of stress concentration and hence knotting may adversely affect the biomechanical properties of the suture loop. Crimping has the potential for allowing easier maintenance of initial tension and stiffness, it decreases loop elongation and eliminates the need for a bulky knot.

Several newer sutures have been developed that may closer fulfil the ideal requirements and offer significant advantages over nylon leader line. FiberWire, FiberTape and

OrthoFiber are non-absorbable, multifilament, polyethylene based orthopaedic sutures. These materials are thought to be stronger, stiffer and undergo less elongation than comparably sized monofilament sutures.

An in-vitro biomechanical test of suture loops, under monotonic tensile and cyclical loading until failure, compared FiberTape (FT), FiberWire (FW) and OrthoFiber (OF) with nylon leader line (NL).

Three main biomechanical properties were tested: elongation, stiffness and strength. Two knotting techniques, a square knot (SQ) and a slip knot (SL) and a crimp clamp (CR) system were also evaluated. Twenty loops of each of twelve combinations of fixation and suture underwent monotonic tensile and cyclical loading.

Knotted FT, FW and OF underwent less elongation than knotted NL under monotonic tensile and cyclical loading. Under monotonic tensile loading, knotted FT and OF were stiffer than knotted NL.

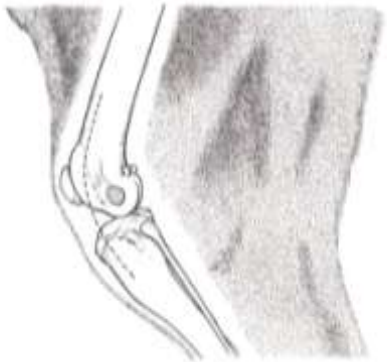
CR FT, CR FW and CR OF were stiffer than CR NL and CR FT, CR FW and CR OF were stiffer than knotted FT, FW and OF. FW and OF knotted loops were weaker than knotted NL. CR FT was stronger than CR NL. CR FT and CR OF were weaker than knotted FT and OF.

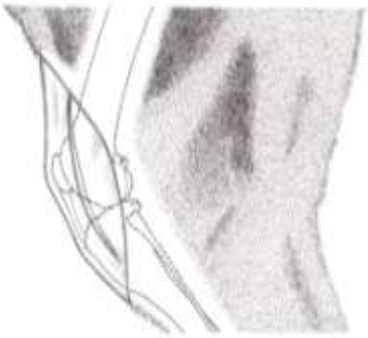
The conclusion was polyethylene sutures offer some advantages to nylon leader line including increased maximal load and stiffness and decreased elongation and that crimping the suture alters the biomechanical properties of the loop.

Multifilament materials have for many years been considered to induce a higher rate of “suture reactions”, largely due to the high surface area for irritation and bacterial adherence. A 10 to 25% incidence of draining tracts was reported in one study. Recently, Guénégo et al published a study following forty-two dogs with CrCL ruptures that were stabilized using a modified lateral extra-capsular technique using a braided polyester suture and bone anchor. With a mean follow-up of 18 months, there were no incidences of draining tract formation.

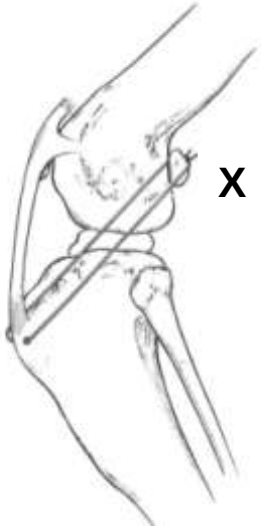
Surgical approach

Regardless of the type of prosthesis, the approach is similar using a lateral parapatellar incision.





The fabella is identified *before* the joint capsule is incised.



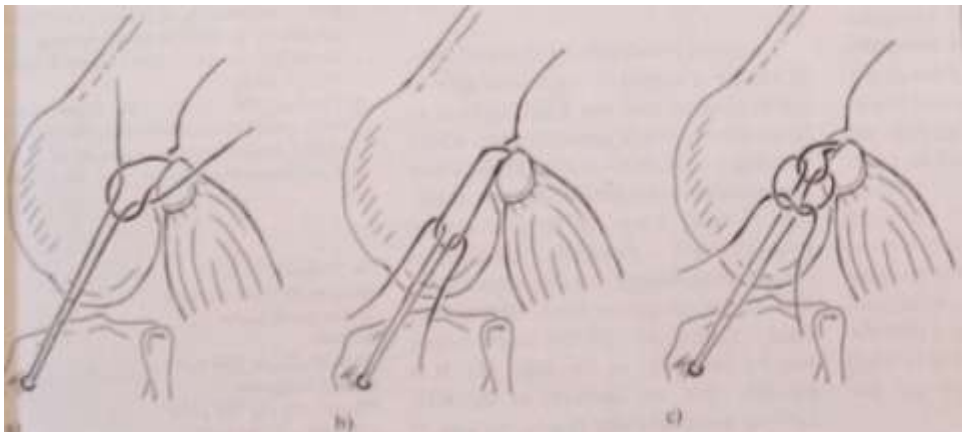
X It is important to anchor the suture in the dense femoro-fabellar ligament between the fabella and the femur rather than around the fabella as is depicted here

Methods of securing the suture

Crimp

The first central crimp can be made such that it grips the suture but there is still the ability to adjust the tension before applying the second and third crimps. The first crimp can then be completed. Compound action crimpers may make creation of the crimps easier.

If a crimping system is not employed, a self locking knot has been described to allow a surgeon to tie the suture single handed.



W. M. McKee, A. Miller Vet Comp Orthop Traumatol 1999; **12** 38-40

Outcome

Most studies report a success rate of 80 – 90 % if the outcome measures rely on owner satisfaction &/or clinical evaluation. Studies employing force plate gait analysis have shown less consistent results with one retrospective study showing good agreement between clinical and force plate analysis while a prospective study only showed a 40% improvement and a 15% return to normal function using force plate analysis.

The presence of OA in the stifle joint does not correlate with clinical function; radiographic outcome should be used cautiously as a predictor of clinical outcome.

Complications

A 17.4% complication rate was reported in a retrospective study of 363 dogs. The only factors associated with a higher rate of complications were high body weight and young age. An infection/inflammation rate of 4.2% was reported in a retrospective study of 496 dogs. Factors associated with a lower rate were using suture material other than skin staples and p/o oral administration of antibiotics.

Is physiotherapy indicated?

A prospective study looking at the effects of p/o rehabilitation (massage, walking and swimming twice daily during weeks 3-7 for a total of 30 sessions) compared to restricted exercise (short leash walks up to 8 weeks, increasing to 16 weeks before unrestricted exercise) showed function of the operated limb in the rehab group was similar to the contra-lateral normal limb, in contrast to the limb in the exercise restricted group.

Is the tibial plateau angle predictive of clinical outcome?

Not according to one study where the TPA was 18.5 to 34.9 degrees.

Percutaneous placement of the LFS

The LRS was recently modified to include a percutaneous placement of the (p)LFS, following the trends for minimally invasive surgery and arthroscopic management of CrCL and meniscal disease. The (p)LFS technique involves two 2 cm incisions on the lateral aspect of the joint, one over the fabella and the other over the proximal tibia, and a third short incision on the medial aspect at the level of the proximal tibia.

Tightrope® (Arthrex Vet Systems, Naples, FL)

The Tightrope® technique was introduced to address a number of limitations of the LRS.

- the isometric points of insertion of the suture. (see previous comments!).
- the TR is also proposed as a stronger repair because of the biomechanical properties of its multifilament material (Fibertape)
- the repair relies on bone rather than soft tissue anchorage of the suture, further contributing to a decrease in implant elongation.
- the use of a toggle and a button allows minimally invasive placement of the filament.
- lastly, the TR repair does not require specialized equipment other than a cannulated drill bit provided in the kit

Is the Tightrope system (TR) easier than the (p)LRS?

A recent study identified the incidence and type of technical deviations during the training phase of TR and (p)LFS repairs, and evaluated the diagnostic value of post-operative radiographs.

Sixteen 3rd year veterinary students, 6 small animal surgical residents and a Diplomate of the American College of Veterinary Surgeons performed the TR and (p)LFS techniques on 10 paired limbs. Perceived level of difficulty, duration of surgery and technical deviations were assessed via questionnaire, radiographs and dissection. The TR procedure was perceived as more technically demanding than the (p)LFS by veterinary students and residents. Technical deviations were overall more common after TR than (p)LFS, and in limbs repaired by students, regardless of the procedure. The most difficult aspect of the TR consists of the bone tunnels. The most difficult step of the (p)LFS consists of passing the suture around the femoro-fabellar ligament.

Proximal tibial plateau osteotomies

Tibial osteotomies impart "dynamic stability" by altering the geometry of the stifle and thereby neutralizing the cranial tibiofemoral shear force. Studies have shown that dynamic stability can be achieved by decreasing the slope of the tibial plateau or by advancing the tibial tuberosity. The tibial plateau levelling osteotomy (TPLO) imparts cranio-caudal stability by reducing the tibial plateau angle (TPA) while the tibial tuberosity advancement (TTA) eliminates cranial tibial thrust by advancing the insertion of the patellar tendon, and modifying the angle between the medial tibial plateau and the patellar tendon which defines the patellar tendon angle (PTA). Despite the widespread use of these osteotomies, a global perspective of how these techniques affect both TPA and PTA has not been established.

Decreasing the tibial plateau angle counters the cranial tibial thrust force thereby reducing or eliminating the tibial thrust instability, but it has minimal effect on cranial drawer movement. It progressively loads the caudal cruciate ligament and alters the magnitude and direction of pull of the muscle groups around the stifle.

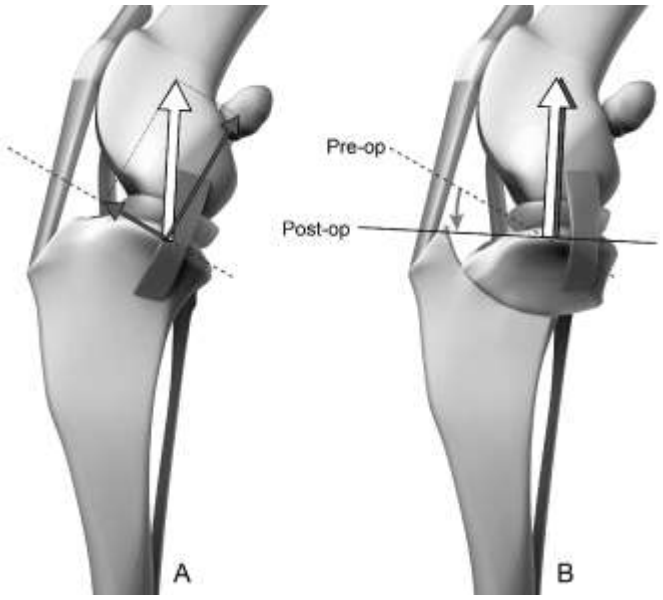
Tibial Plateau levelling Osteotomy

This is effective in neutralising cranial tibial thrust but not hyperextension or internal rotation of the tibia. Cadaveric studies have demonstrated that reduction of the TPA to 6.5 +/- 0.90 eliminates cranial tibial thrust, whilst any further reduction in TPA will increase the magnitude of caudal tibial thrust which is counteracted by the caudal cruciate ligament.

The alteration of the TPA by the TPLO procedure requires preoperative determination of the TPA, cylindrical osteotomy of the proximal tibia, rotation of the tibial plateau segment and stabilisation with a plate and screws. The position and radius of the cylindrical osteotomy is planned from the preoperative mediolateral radiographic projection. It is important to place the centroid of the osteotomy at the most proximal point of the tibial long axis, the intercondylar eminence. Geometric analysis and cadaveric studies have shown that failure to position the centroid of the cylindrical osteotomy over this point will result in translation of the proximal tibial fragment and imprecise alteration to TPA. Furthermore, inaccurate positioning and orientation of the osteotomy can induce angular limb deformity and increase the risk of tibial tuberosity fractures.

Surgical technique

106 dogs that underwent consecutive TPLOs using self tapping screws (STS), non self tapping screws (NSTS), STS-titanium or STS-stainless steel screws were compared by reviewing post-operative TPLO radiographs.



The resultant compressive force (large white arrow) across stifle joint is parallel to the tibial axis. Using the tibial plateau slope (TPS) as the baseline, whereby the femur can move along this surface if the CrCL is deficient, the resultant force can be broken down into its two orthogonal components (small shaded arrows), one perpendicular and one parallel to the tibial plateau. The latter represents the tibiofemoral shear force (resulting in cranial tibial thrust, CrTT). If the angle of the tibial plateau is reduced to zero, the tibiofemoral shear force vector becomes zero, and the joint compressive force and resultant force become one and the same.

Self tapping screws had a significantly higher incidence of trans-cortical fractures (18.0%) compared to non self tapping screws (0.8%). No difference was seen between stainless steel and titanium screws.

Locking screws in TPLO plates maintain the plateau position better than conventional screws.

The use of a jig is generally recommended although this is not a universally held view. One study showed none (0/8) of the jig-TPLO limbs and 75% (6/8) of jig-less-TPLO limbs showed fibular penetration, a difference that was statistically significant. Fibular penetration was most frequently associated with the most proximal screw.

The use of a saw guide has been recommended to allow more accurate placement of the osteotomy and more accurate levelling of the tibial plateau.

Outcome

Few publications record objective outcome measures. A retrospective longitudinal study of Labrador Retrievers found no association between p/o TPA and ground reaction forces where the TPA was 0-14 degrees.

Second-look arthroscopy has demonstrated some interesting results; stable joints with partial tears of the CrCL revealed the ligaments at a mean of 25 months were similar to

those seen at surgery, except that the torn fibres had resorbed. In contrast, joints with complete CrCL rupture or incompetent partial tears revealed modified Outerbridge grade 3 or 4 articular cartilage abrasion of the femoral condyles.

Is the LRS better than the TPLO?

In a study of Labrador Retrievers with unilateral CrCL injury, limb function was measured before surgery and 2 and 6 months after surgery using a force plate and compared to clinically normal Labrador Retrievers. Affected dogs had partial or complete medial meniscectomy and LRS or TPLO. Post/op rehabilitation was performed in nearly all the dogs.

No difference was found between the two groups; 14.9% of LRS and 10.9% of TPLO treated dogs had normal limb function. Improvement was seen in 34% treated via TPLO, and 40% treated via LRS. The conclusion was Labrador Retrievers treated with LRS or TPLO infrequently achieve normal function but the results of LRS and TPLO are similar.

Is the Tightrope system (TR) better than the TPLO?

The clinical evaluation of the TR is limited to one study of 47 dogs over 21 kg, where the TR provided similar clinical outcome with less complications than the TPLO.

Meniscal lesions

Dogs with chronic cruciate rupture often have associated damage to the medial meniscus. These animals will remain lame unless this is attended to.

Meniscal injuries are difficult to diagnose confidently without arthrotomy or arthroscopy. The “meniscal click” is described in the literature. This is a noise heard as the animal walks or the joint is manipulated. However, there can be both false positives and negatives. Meniscal click plus pain on stifle flexion has been reported as being more sensitive than a meniscal click alone.

A number of meniscal lesions are observed. Folding of the caudal horn is the commonest lesion observed. However, this may occur without detachment from the caudal attachments. Where feasible, management of meniscal lesions should involve partial rather than total meniscectomy. Sharp debridement of the damaged portion is required and it is essential to gain as much access to the medial compartment of the stifle as possible. The use of retractors is essential to perform this surgery adequately and the menisci should be palpated with a small hook as well as visualised.

Complications

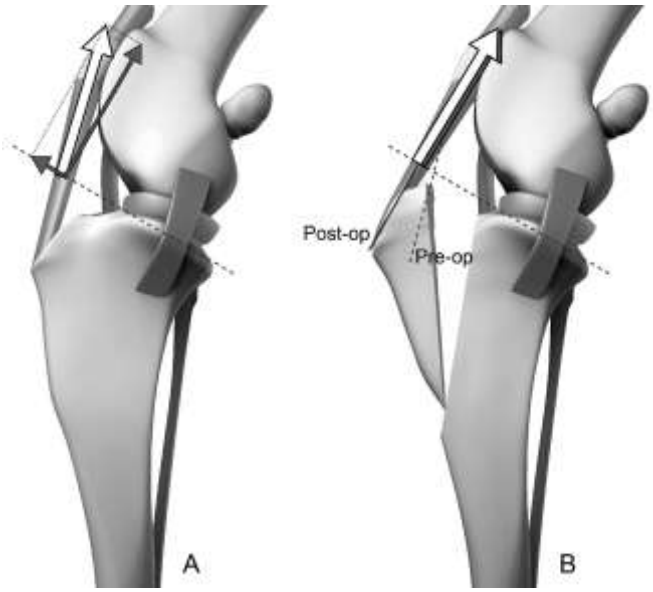
A retrospective case series of 305 dogs which underwent TPLO demonstrated 6 instances of patellar fractures (1.97%). 5/6 dogs were clinically lame when the fracture was diagnosed and all had radiographic evidence of patellar tendon thickening. All six dogs with TPLO related patellar fractures were treated non-surgically. In two dogs where follow up radiographs were available there was no evidence of fracture union. No dog had a persistent lameness suggesting conservative management can be successful.

Fifteen dogs weighing 20-45 kg with unilateral complete CrCL insufficiency had force-platform analysis, and lateral weight-bearing radiographs of the affected stifle taken preoperatively, 1, 3 and 6 months postoperatively. The distance between the origin and insertion of the CrCL (CrCLd) was measured on each radiograph and compared between each time point.

The authors concluded TPLO does not consistently resolve femoro-tibial subluxation during standing in dogs with CrCL insufficiency. Further, the medial meniscus appeared to be an important contributor to stifle stability in these TPLO joints.

Tibial Tuberosity Advancement

The TTA has been reported to functionally stabilize the stifle joint during weight bearing by neutralizing the cranial tibiofemoral shear force (cranial tibial thrust) by advancing the tibial tuberosity. This is accomplished by an osteotomy of the tuberosity in the frontal plane with advancement of this bone fragment. The mechanics of the TTA also have been validated in two experimental models.



Schematic representation in the stifle joint of the tibiofemoral forces, according to Tepic, before (A) and after (B) TTA. The resultant compressive force (large white arrow) across stifle joint is parallel to the patellar tendon. Using the tibial plateau slope (TPS) as the baseline, whereby the femur can move along this surface if the CrCL is deficient, the resultant force can be broken down into its two orthogonal components (small shaded arrows), one perpendicular and one parallel to the tibial plateau.

The latter represents the tibiofemoral shear force (resulting in CrTT). If the angle of the tibial tuberosity is advanced cranially until the patellar tendon angle (TPA: angle between the tibial plateau and the patellar tendon) is reduced to 90°, the tibiofemoral shear force vector becomes zero, and the joint compressive force and resultant force become one and the same.

A useful review reference is Tibial Plateau Levelling Osteotomy or Tibial Tuberosity Advancement? Boudrieau Vet Surg 2009 **38** 1-22

A comparison of TPLO and TTA:

INTRA-OP COMPLICATIONS	**TPLO**	**TTA**
Haemorrhage	possible	no
Fascial closure	easy	can be difficult
i/artic screw	yes	no
LDE damage	yes	yes
Tibial Fx	rare	yes
Torsion	yes	no

IMMEDIATE P/O	**TPLO**	**TTA**
Bruising	more	less
Swelling	more	less
Weight bearing	less	more
Pain	?	?

POST/OP	**TPLO**	**TTA**
Infection	5-7%	4%
Fibular Fx	yes	no
Tuberosity Fx	yes	yes
Patellar luxation	rare	rare
Tibial Fx	rare	yes
Overall rate	18-31%	31-56%
Meniscal injury rate	9%	7%

OUTCOME	**TPLO**	**TTA**
12-24 hours	less	more
10-14 days	less	more
6 weeks	similar	similar
10 weeks	similar	similar

Comparison of TPLO, TTA and TR

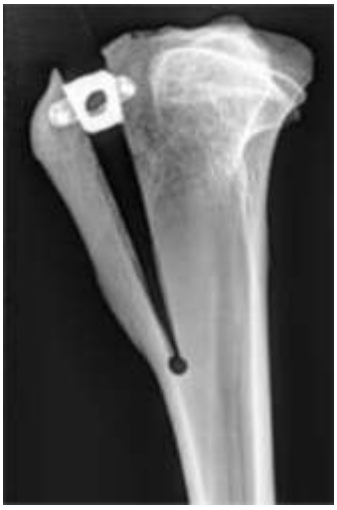
A retrospective clinical cohort study used medical records and completed owner questionnaires based on their assessment of their dog at least 1 year after surgery. Outcome questionnaires recorded return to function, presence and degree of pain, and complications.

TTA was associated with significantly ($p<0.03$) higher rates of major complications and subsequent meniscal tears than TPLO and TR, and TPLO had significantly higher rates of major complications and meniscal tears than TR. Percent of function >1 year after surgery was 93.1 + 10.0% for TPLO, 92.7 + 19.3% for TR, and 89.2 + 11.6% for TTA. Significantly ($p=0.016$) more TPLO and TR cases were classified as reaching full function than TTA. The highest levels, frequency, and severity of pain were noted in TTA cases; however, no significant differences were noted among groups.

The conclusion was that long term outcomes for TPLO and TR were superior to TTA based on subjective client and DVM assessments. Each technique was associated with a high long term success rate with TR showing the highest safety-to-efficacy ratio.

Modified Maquet Technique (MMT)

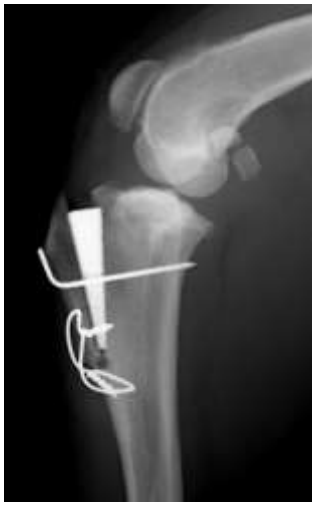
MMT is a variation on the TTA and TTO techniques.



If the hinge fails a tension band wire can be added.

A larger hole is preferable to a small hole since it reduces the likelihood of a hinge fracture.

Modified Maquet Procedure (MMP)



MMP utilises a titanium foam wedge called Orthofoam. This has been developed to support the osteotomy and allow osseous ingrowth.

Both are considered easier than the original TTA but long term outcomes are awaited.

Patellar instability

The patella can either luxate medially or laterally. Medial luxation is the more common clinical problem. Luxations are usually developmental but occasionally follow a traumatic

incident. Occasionally the patella is predisposed to luxation due to limb maldevelopment and luxation follows minimal trauma.

Medial patellar luxation

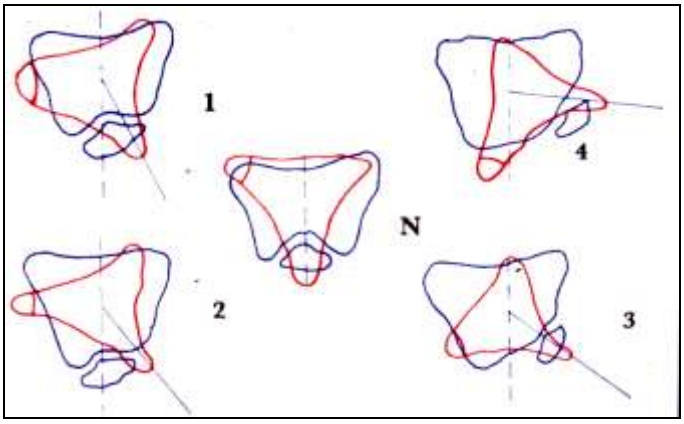
Medial patellar luxation is usually seen in small breed dogs with certain breeds being over represented, e.g. Pomeranian, Yorkshire Terrier, Chihuahua, Miniature and Toy Poodles. A significant hereditary component is suspected in these cases.

Developmental medial patellar luxation is associated with other anatomical abnormalities of the hindlimb.

These include:

- Lateral bowing of the distal femur
- Medial bowing of the proximal tibia
- Medial rotation of the tibial tuberosity
- Hypoplasia of the medial femoral condyle
- Increased femoral torsion.

Some authors have described changes in the hip. Whether it is displacement of the patella that results in the development of limb abnormalities or the converse is uncertain. Others have described a grading system from I-IV depending upon the degree of subluxation/luxation and the position of the tibial tubercle.



Singleton's classification based on the work originally performed by Putnam

Unilateral medial luxation often presents as an intermittent lameness. Traumatic luxations result in acute lameness. The lameness relates to the position of the patella. Sudden

displacement results in acute lameness, with the patient often carrying the limb for a number of strides. The patella will often spontaneously relocate when the problem resolves immediately. Some owners reduce the patella. This is either intentional or as a result of massaging the affected limb as a first aid measure.

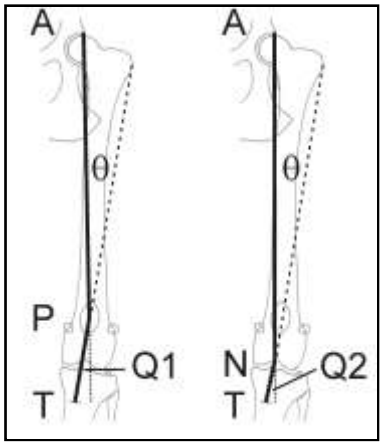
Bilateral developmental luxations present as a hind limb gait abnormality. Patients are young animals, usually under 6 months. The condition may be progressive and severely affected animals may have difficulty in standing. This can be misinterpreted as a neurological problem.

The condition is non-painful unless there has been significant damage to the patellar cartilage. This is uncommon in developmental luxation but more common in long standing instability following trauma.

Conservative management is indicated in asymptomatic cases in adult animals, some intermittent cases and those with mild non-progressive signs. This consists of exercise control, owner relocation of the patella if necessary and analgesics if required.

There are four major surgical procedures that can be used, either singly or in combination, to stabilise the patella:

- Imbrication
- Tibial tuberosity transposition
- Trochlear groove deepening
- Release of the medial fascia/joint capsule



The decision as to which of these to perform is generally made at the time of surgery rather than as a result of pre-operative planning. Attempts have been made to identify the Q angle using either the patella or the intercondylar eminence (Q1 & Q2 respectively) but they are not widely used.

Osteotomies of the femur and tibia are suggested in the literature for animals with associated major bony deformities but are rarely performed. Fibular head transposition has also been suggested as it will de-rotate the tibia to some extent.

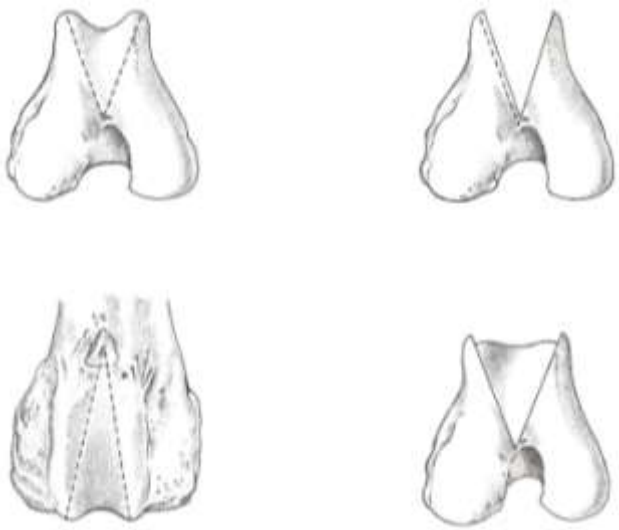
Although the four point clinical grading is useful in prognosis it is not an absolute indication for the selection of surgical techniques. It is likely, though, that animals with grades II-IV will require tibial tuberosity transposition. Imbrication alone is only indicated in traumatic luxations and the mildest of developmental abnormalities. The developmental problems usually require tibial tuberosity transposition and trochlear groove deepening. It is usual to provide imbrication following these techniques.

Imbrication is the most straight forward technique and if used in the correct situations is very successful. Essentially the tissues of the retinaculum are manipulated to increase the tension on the patella. Failure of imbrication techniques is usually due to wrong patient selection and/or use of inappropriate suture material.

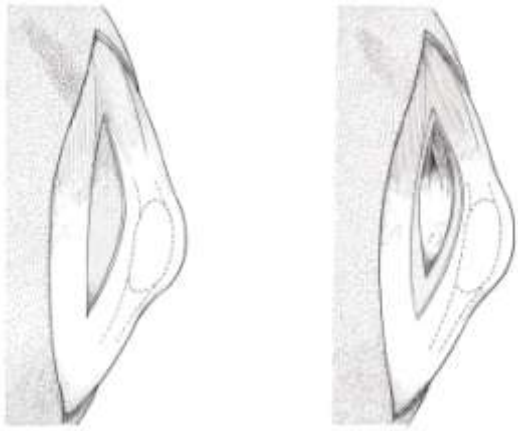
Trochlear groove deepening has been traditionally performed as a trochleoplasty, in which the required amount of cartilage and bone is removed, usually with a rasp. This leaves a raw cancellous surface as the articular surface for the patella. In time this becomes covered with a fibrocartilagenous layer but there is never a hyaline cartilage surface. In a few individuals this results in permanent discomfort in spite of the patella being stable. At surgery these individuals have erosions on the patellar articular surface presumably because of the abnormal articular surface.

Wedge sulcoplasty is a technique that deepens the trochlear groove and maintains the articular cartilage. An osteochondral wedge is removed, the defect deepened and the wedge replaced. This recesses the articular surface and though the walls have cancellous surfaces these are not important for articulation. The wedge is maintained in position by friction and fixation is not required or desirable. My impression is that postoperative progress is swifter, and morbidity lower, with this cartilage preserving technique.

Landmarks for a wedge sulcoplasty



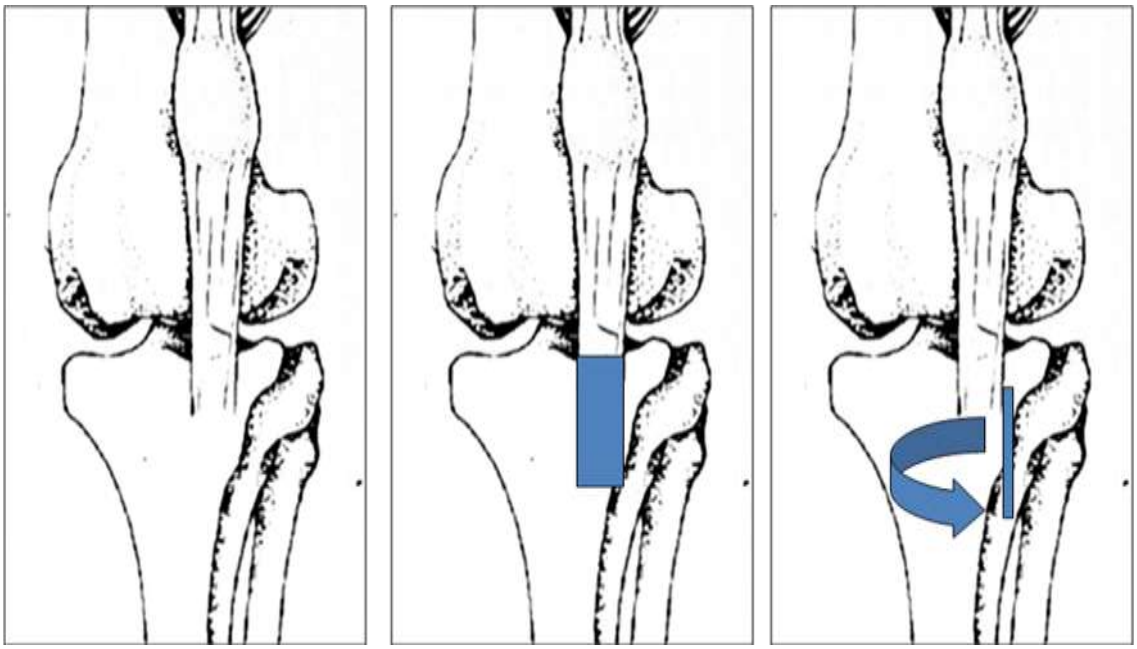
Release of the medial fascia/joint capsule



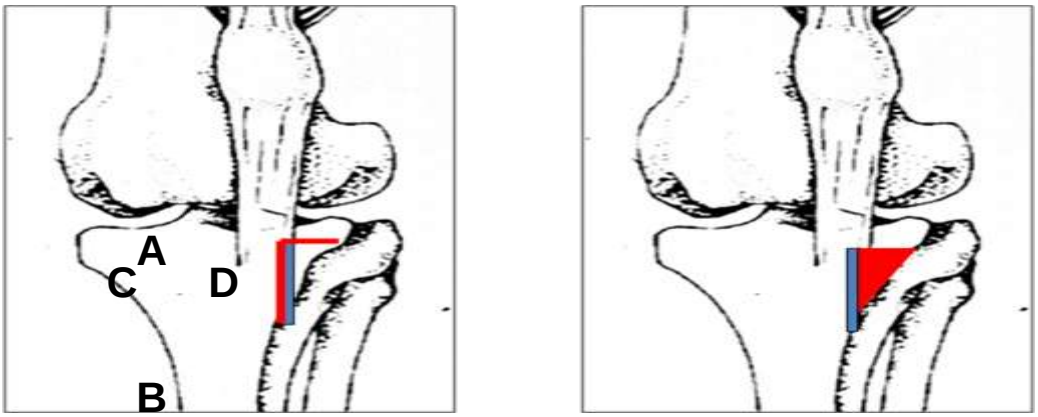
The medial fascia and joint capsule may be incised if necessary to release tension on the patella

Osteotomy of the tibial tuberosity is performed as described above. In skeletally mature dogs the tuberosity is freed completely but in younger patients, with a thicker periosteum, the distal soft tissue attachments are not incised so they may act as a tension band. The osteotomised tuberosity is moved laterally to its newly prepared bed and attached by K-wire(s). If the distal soft tissue attachment is not maintained a single K-wire should be re-inforced with a tension band. Alternatively, a horizontal mattress suture of stainless steel wire may be used to re-attach the tuberosity.

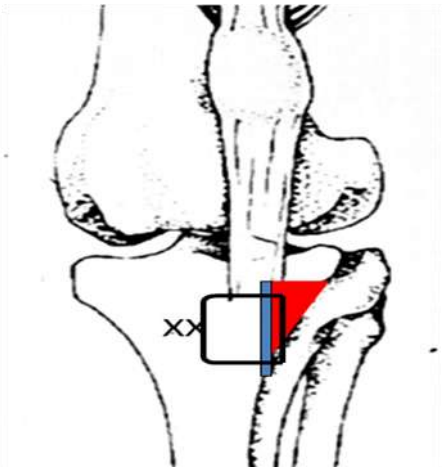
Tibial crest transposition in the adult dog



The crest is osteotomised with a saw blade or osteotome (larger dogs), rotated through 90 degrees and reattached laterally with a horizontal mattress suture of stainless steel wire.



To avoid the need for a tension band wire to counteract the distractive force of the quadriceps mechanism, the cranial tibialis muscle is retracted, and two osteotomies performed. The first removes the cortical bone from the lateral aspect of the crest (B-A) from distal to proximal. The second, from C-D, in a cranial to caudal direction, creates a “shelf” under which the transposed crest can sit. The shaded triangle indicates the removed bone.



The transposed crest is attached via a horizontal mattress suture of stainless steel wire. This is achieved by drilling two holes in the osteotomised crest and two in the residual crest.

Timing of intervention is important. Though it is uncertain whether it is the position of the patella that results in abnormal limb developmental or vice versa common sense suggests that there is no advantage in delaying intervention. Improvement in the transmission of forces within the limb can only encourage more normal development. Animals as young as 3 weeks of age have been operated on.

There are few reports of the management of patellar luxation in the veterinary literature and the common perception is that most cases do well. However, cases of medial patellar luxation are often discovered as incidental findings in animals with no related clinical problem and thus there should be some caution in interpreting the apparent success of techniques. In one retrospective study, reluxation of the patella was observed in approximately 50% of cases following surgery, but there was minor clinical manifestation. The incidence of degenerative joint disease in animals that underwent surgery at less than 6 months of age in this series was high; suggesting that restabilisation of the patella in deformed joints does not prevent the progression of joint disease.

Poor outcome in cases with marked anatomical deformities before surgery has been observed by other authors. Although limb function is usually improved by surgery the limb will remain an abnormal shape. If the tibial tuberosity is relocated there will be an exacerbation of the “toes in - hocks out” stance. Owners should be made aware of this before surgery.

The prognosis becomes more guarded with increasing deformity. It may not be possible to relocate the patella in some grade IV cases and alternative salvage procedures, such as arthrodesis, may be required. Chronic traumatic luxations will remain lame and will possibly

develop damage to the articular surface of the patella and the trochlear ridge. Thus surgical stabilisation should not be delayed unduly.

Notes page

ELBOW SURGERY: DYSPLASIA, CONDYLAR FRACTURES AND IOHC

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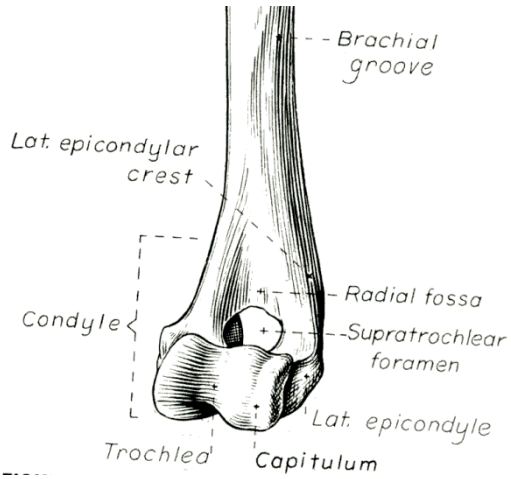
The elbow is a composite joint comprising the humero-radial, humero-ulnar and proximal radioulnar joints. The radius and ulna form a hinge with the humerus and are also capable of a degree of pronation and supination. Lateral movement is controlled by the strong collateral ligaments, and by the anconeal process which fits deeply within the olecranon fossa.



The condyle is the distal end of the humerus including the articular areas and adjacent fossa.

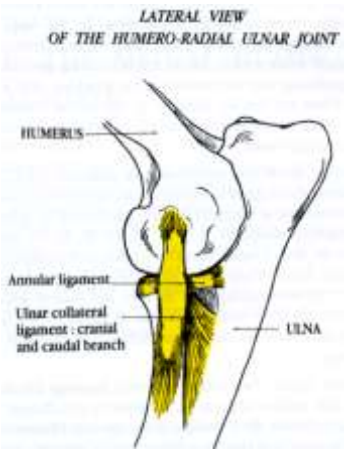
The capitulum is the small lateral articular area of the distal humerus which articulates with the radial head.

The trochlea is the much larger medially located pulley shaped part that extends proximally into the adjacent fossa. The trochlea articulates with the trochlear notch of the ulna.

The olecranon fossa is a deep excavation of the caudal part of the distal humerus. It receives the anconeal process when the elbow is extended. It is covered by the anconeus muscle.

Lateral approach

This approach provides access to the lateral aspect of the distal humerus and the lateral compartment of the elbow joint. It is the approach most frequently employed for the open reduction and fixation of lateral condyle fractures.



A curved skin incision is centred over the lateral epicondyle. After retracting the skin edges the lateral fascia over the underlying muscles is carefully divided. It is important at this stage to identify the radial nerve, which consists of the deep and superficial branches. It is most readily located by tracing the cranial border of the triceps muscle proximally. The nerve emerges from under the triceps muscle with the brachialis muscle, in the distal third of the humerus just proximal to the origin of the extensor muscles.

The next phase of the approach is to separate the tendon of origin of the extensor carpi radialis and the common digital extensor from the lateral epicondylar crest. The muscles are elevated by subperiosteal dissection to expose the joint capsule and the intercondylar fossa. The joint capsule is then incised in an inverted "L" shape to expose the articular surface. Further elevation and retraction of the extensor muscles can be performed to gain more exposure of the caudal aspect of the distal humerus. Closure is performed by apposing the soft tissues with interrupted sutures of an absorbable material.

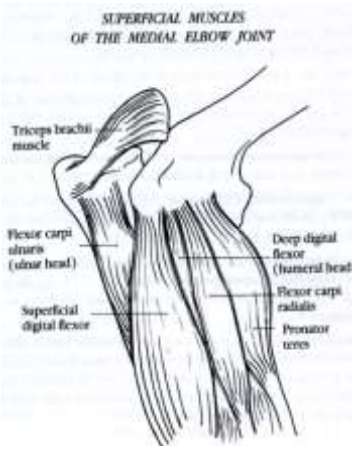
Caudo-lateral approach

This approach is used for the exposure and treatment of an ununited anconeal process. A curved skin incision is made behind the caudal edge of the lateral condyle of the humerus. Separation of the subcutaneous fascia will reveal the anconeus muscle, which is then divided across its fibres in its mid section. The caudal pouch of the lateral joint capsule is divided, usually in association with the muscle transection, to expose the caudal joint compartment, the lateral condyle of the humerus and the anconeal process. The joint capsule and the divided anconeus muscle are closed together with a series of interrupted absorbable sutures.

If internal fixation of the separated anconeal process is anticipated a modification of this approach is recommended to provide better access to the anconeal process in the flexed position. The area is exposed by separating between the lateral and long heads of the triceps muscle and the anconeus muscle is detached from its insertion on the ulna.

Medial approach

This approach is indicated for the treatment of developmental conditions of the medial aspect of the joint such as fragmentation of the medial coronoid process and osteochondritis dissecans. The patient is positioned in lateral recumbency with the affected leg down. A small sand-bag positioned under the elbow may help in later attempts to expose the inside of the joint



A curved 6-8 cm skin incision is centred over the medial epicondyle.

Numerous layers of subcutaneous fascia are divided to expose the medial epicondyle and the flexor muscles of the ante brachium.

The ulna nerve is located at the proximal end of the incision and care should be taken when the fat in this area is divided.

Exposure of the joint can be achieved in a number of ways. I prefer to separate between the pronator teres and the flexor carpi radialis with the option, if necessary, of transecting and reflecting the origin of the pronator teres downwards to provide better exposure. The proximity of the brachial artery and vein and the median nerve to the underside of the pronator muscle should be recognized. Other surgeons prefer to either split the flexor carpi radialis or to separate this muscle from the digital flexor, which lies caudal to it.

The joint capsule and if necessary, the medial collateral is divided parallel with the joint surface. Retraction of the joint capsule will reveal the medial humeral condyle and the outer aspect of the medial coronoid process. Better exposure of the deeper aspects of the joint can either be achieved by abduction and pronation of the radius and ulna or by flexing the carpus and using the sandbag under the joint as a fulcrum to lever the joint open. A small Hohmann retractor placed over the medial coronoid process can also be used to aid the exposure. The elbow joint is a close-fitting joint and this approach provides limited, but adequate exposure to identify and treat elbow osteochondrosis.

The joint capsule, collateral ligament and muscles are repaired with absorbable sutures. Slight elevation of the limb may facilitate tying the sutures. The fascial layers and skin are closed in separate layers with continuous absorbable sutures.

Caudal Approach

This approach is usually reserved for those cases where a wide exposure is required, for example for the open reduction and fixation of T and Y fractures of the distal humerus. Although in experienced hands it is possible to repair these fractures via a combined medial and lateral approach, it is often preferable to adopt the caudal approach by osteotomy of the olecranon, or tenotomy of the triceps, so that all aspects of the joint can be visualized, which aids reduction and fixation of these difficult fractures.

It may be preferable to position the patient in dorsal recumbency with the affected leg supported in extension. The position of the ulnar nerve should be determined before the triceps is reflected. The triceps insertion is very large and should be adequately defined before performing an ulnar osteotomy or a triceps tenotomy. If a lag screw is to be used to repair the osteotomy then the drill hole should be placed before the osteotomy is performed. The osteotomy is performed distal to the point of attachment of the triceps between the anconeal process and the olecranon tuberosities. The piece of bone should be large enough to facilitate later stabilization. The anconeus muscle and joint capsule are incised from the

medial epicondylar crest and reflected proximally with the triceps muscle. Exposure of the fracture is improved by flexion of the joint.

At closure no attempt is made to re-attach the anconeus muscle or the joint capsule. The olecranon is re-attached either with a lag screw, supported by a Figure of 8 wire, or with a tension band wire. The remaining tissues are closed in layers. The leg should be supported for about 7-10 days in a heavily padded bandage. Systemic antibiotics are recommended because of the length of the operation and the tissue damage.

ELBOW DYSPLASIA

Elbow dysplasia (ED) is a hereditary developmental problem commonly diagnosed in young large-breed dogs, such as the Bernese Mountain dog, Labrador and Golden Retriever, Rottweiler, and German Shepherd dog. Elbow dysplasia is a complex of joint disorders, and includes fragmented medial coronoid process (FCP), osteochondritis dissecans of the medial humeral condyle (OCD), ununited anconeal process, and elbow incongruity. While FCP is the most common cause of ED, a combination of different ED lesions within the same elbow is possible. Different degrees of FCP have been described, including fissures, displaced and non-displaced fragments, and chondromalacia- like lesions. Cartilage erosions in the region of the medial coronoid process and the medial aspect of the humeral condyle, in absence of coronoid fragmentation, have been reported. The term 'medial coronoid disease' has been introduced by some authors to cover this variety of lesions.

a) Ununited Anconeal Process

Normally the anconeal process develops as part of the ulnar diaphysis, but in certain breeds such as the German Shepherd Dog, it develops as a separate centre of ossification. This fourth ossification centre appears at about 10-13 weeks and is fused to the ulna by 18-20 weeks.

Ununited anconeal process (UAP) as a result of failure of fusion, is seen predominantly in the German Shepherd Dog, where it is probably an inherited defect. Traumatic separation can occur as a result of hyperextension of the elbow joint. In certain breeds, such as the Basset Hound, it occurs secondary to non-traumatic premature closure of the distal ulnar growth plate. The shortened ulna and the relative lengthening of the radius puts pressure on the trochlea of the humerus which in turn forces the humerus proximally, exerting sufficient pressure on the anconeal process at a critical stage of its development to result in its separation. The instability and irritation following separation of the anconeal process results in degenerative joint disease.

The condition can occur in one or both elbows. Affected animals are presented with a progressive forelimb lameness which usually starts between 4 and 5 months of age. Although the condition is seen most frequently in German Shepherd Dogs, it is also seen occasionally in Wolfhounds, Rottweilers, St. Bernards and Great Danes. Affected animals may have a strange gait with the elbow abducted.

This position may also be adopted when the animal is standing and the foot may be outwardly rotated. Palpation and manipulation of the joint will reveal a thickened joint with a varying amount of synovial fluid effusion. The range of joint movement will be reduced, with pain and sometimes crepitus on full flexion and extension.

The diagnosis is confirmed by taking a fully flexed lateral radiograph of the joint. The flexed view is important so that superimposition of the medial epicondyle on the olecranon can be avoided. A clear line of separation below the anconeal process is diagnostic, but in some cases there appears to be only partial separation. A varying amount of DJD will also be evident. In addition the congruency of the elbow joint should be assessed from radiographs taken in the extended lateral and cranio-caudal projections. Shortening of the olecranon has also been observed on the affected side.

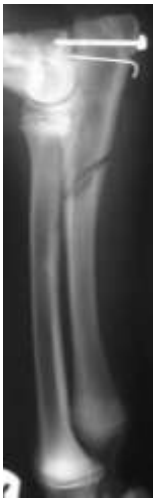
There are a number of choices of treatment. Removing the anconeal process is the most straight-forward and widely practised method and is the treatment of choice in chronic cases. However following surgery all the dogs develop degenerative changes with a varying amount of functional impairment.

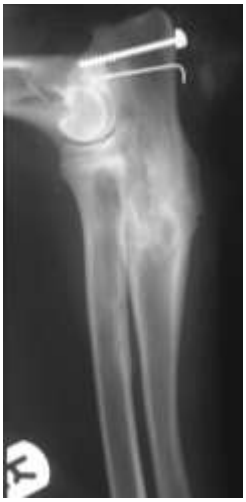
There is some disagreement about the timing of the operation. Olsson claims that early removal of the process causes more severe DJD and he advocates delaying surgery till the animal is almost mature. Irrespective of the timing of the operation the process is removed via a lateral approach through the anconeus muscle. The process is sometimes firmly attached to the ulna by fibrous tissue. A pair of sharp reduction forceps can be used to grasp the process, while its attachments are released with an osteotome. The joint should be immobilized in a padded bandage for about 7 days after surgery to reduce postoperative swelling.

Internal fixation with lag screws and/or K-wires has been described, performed via a caudo-lateral approach. This method offers the advantage of improved joint stability. The results appear to be superior to removing the process especially if the surgery is performed early,

but the long term results have not been well documented. Complications, such as implant failure, have been reported.

If subluxation of the joint is implicated in the pathogenesis, relief of the intra-articular pressure by an osteotomy of the diaphysis of the ulna may result in spontaneous union of the separated anconeal process. Olsson believes that the results of osteotomy of the proximal ulna appear to be superior to removal of the process. However Matis reports that healing may occur with the process in a displaced location which in turn results to a distorted trochlear notch. Possibly the best results will be achieved by combining internal fixation with a relieving osteotomy of the ulna especially if the osteotomy is performed early.





14 weeks p/o

b) Fragmentation of the medial coronoid process (or medial coronoid process disease [MCPD])

Currently fragmentation of the medial coronoid process of the ulna (FCP) is the commonest cause of elbow lameness in young, rapidly growing dogs of the large and giant breeds. Although this condition affects many breeds it is particularly prevalent in Rottweilers, Labradors and Bernese Mountain Dogs. Other breeds affected include German Shepherd Dogs, Golden Retrievers, St. Bernards, Chows, Rhodesian Ridgebacks and Newfoundlands. The condition has also been identified asymptomatically in small breeds of dogs.

There is no separate centre of ossification for the coronoid processes. Osteochondrosis seems to affect an area(s) of a joint which is subjected to shearing and traction forces. In the elbow these forces occur as a result of the combined action of pressure from the medial humeral condyle and traction from the annular ligament. In some cases there is evidence of

elbow incongruence secondary to asynchronous development of the radius and ulna. This results in a relative overgrowth of the ulna; causing retraction of the radial head from the elbow joint and exposure of the coronoid processes to abnormal shearing forces from the distal humerus. Wind studied FCP in Bernese Mountain Dogs and concluded that incongruence is the common denominator in the various manifestations of elbow osteochondrosis. Incongruence is due to abnormal development of the trochlear notch of the ulna resulting in a slightly elliptical articular surface with an arc of curvature with too small a radius to accommodate the humeral trochlea. This creates a joint with major contact points in the region of the anconeal process and the medial coronoid process, but not between the trochlear notch and the humeral trochlea.

One or more fragments of bone may fracture from either the inner aspect of the medial coronoid process immediately adjacent to the radial head, or from the apex of the process. The fragment(s) usually remain attached to the annular ligament. The fragments may project from the articular surface causing erosion (kissing lesion) of the adjacent medial humeral condyle. Rarely the coronoid remains attached to the surrounding cartilage, but the affected cartilage is thicker and therefore whiter than the normal cartilage and the underlying bone may be fractured. Other lesions that may be identified are chondromalacia and fissures of the coronoid process, erosion of articular cartilage in the trochlear notch and OCD of the distal humeral condyle. The end result is degenerative joint disease and the severity depends, in part, on the mobility of the fragments and the presence of other primary lesions such as OCD.

The first signs of lameness are usually noticed at about 4-5 months of age. The lameness is usually subtle at first, especially if both legs are affected. An early sign may be outward rotation of the feet, with the elbows held close into the body, giving the dog a "duck-footed" appearance. The lameness is usually worse following rest or heavy exercise. As the condition persists the secondary changes associated with DJD develop, resulting in a reduced range of flexion and extension. A painful response to manipulation, especially on external rotation and hyperextension is a consistent finding. In advanced cases there may be crepitus and thickening of the joint capsule particularly on the lateral side caudal to the humeral epicondyle. Joint fluid effusion is usually not pronounced unless there is a coexisting problem such as OCD or UAP.

The definitive diagnosis of FCP on conventional radiographs poses some problems because the location of the fragment(s) means that there is invariably superimposition of other structures. If the facilities are available the fragment can be visualized using computer axial

tomography. In advanced cases the fragment may be visible on the cranio-caudal or the cranio-caudal medial oblique projection. A tentative diagnosis is made on the basis of the presence of osteophytes, an increase in ulnar trochlear notch radiopacity and the elimination of all other known causes of degenerative joint disease. However it takes these osteophytes some weeks to develop and therefore they may not be evident on the initial radiographs, especially if the animal has been lame for less than 3-4 weeks. The first evidence of DJD will usually be found on the caudal, non-articular, surface of the anconeal process. Initially the osteophytes appear as a slight irregularity on the margin of the bone. It is therefore important to have good quality radiographs taken with the joint in a maximally flexed, but not rotated, lateral position. If the initial radiographs appear normal and the lameness persists, follow-up radiographs are recommended within 4-8 weeks. As the condition persists osteophytes will be found adjacent to the medial coronoid process and the radial head and there may be osteosclerosis in the proximal ulna. As well as the flexed lateral radiograph it is recommended that a cranio-caudal projection is taken to check for OCD lesions and an extended lateral projection to determine the congruency of the joint. Oblique craniocaudal projections are recommended in certain cases as they highlight the caudal and cranial aspects of the joint on the lateral and medial sides respectively. Arthroscopy is essential for the early diagnosis of MC D and is now widely used for treatment of elbow dysplasia.

Once positive radiographic evidence of joint irritation has been confirmed and all other causes have been eliminated, the medial aspect of the joint should be surgically explored and the loose piece(s) of bone and cartilage removed. The fragmented piece(s) of the coronoid are removed by elevation of the fragment(s) with a small chisel and transection of the attachments to the annular ligament. All visible articular surfaces are inspected for evidence of erosion and OCD lesions. If erosive and chondromalacia lesions are present then it may insufficient to just remove the fragmented coronoid. It is recommended to either drill holes in the diseased cartilage and exposed subchondral bone or to remove the surrounding cartilage and 0.5 to 1 mm of subchondral bone of the medial coronoid process in an attempt to shift the load away from this area onto the humero-radial side of the joint. The joint is flushed before closure and a pressure bandage applied for 5-7 days post-operatively. The owners are instructed to strictly restrict the dog's exercise for a further 4 weeks. Swelling of the soft tissues under the incision is an infrequent complication and may be associated with excessive postoperative exercise.

Early diagnosis and treatment gives the dog the best chance of returning to normal. However many cases remain lame and still continue to develop DJD after surgery and owners should be made aware of this before surgery. Bardet reported on the results of

treating these unsatisfactory cases by performing an oblique osteotomy of the proximal ulna. The osteotomy is performed below the joint at the junction of the proximal and middle third of the diaphysis, using a series of drill holes and a sharp osteotome to create the fracture. The oblique cut is positioned so that the proximal ulna is allowed to rotate slightly in a proximal and medial direction. No attempt is made to stabilize the osteotomy which heals by callus formation in 6-8 weeks. The results revealed a significant clinical improvement in 90% of the 40 joints operated on. DJD was progressive in 60% of the joints.

The results of a large retrospective and prospective study from Murdoch University of 130 cases of FCP, where 68 cases were treated surgically and 62 conservatively e.g. rest and anti-inflammatory drugs, revealed that there was no correlation between the severity of radiographic changes in the elbow and the type of lesion found at surgery. Surgical treatment did not significantly decrease the post treatment incidence of lameness, but surgically treated dogs were more active and less lame than those not receiving surgery. It was concluded that young dogs with mild lameness do not benefit from surgery, but dogs with chronic, moderate to severe lameness have a better prognosis with surgery. Overall about 75% of the owners with dogs that had surgery were pleased with the result whereas only 60% of the conservatively treated dogs were acceptable to the owners.

More recently the results of a prospective clinical trial using 20 dogs with unilaterally confirmed MCPD, have cast doubt on the value of arthroscopic treatment. One group of dogs (9) was treated conservatively (CT) while the other dogs (11) were treated arthroscopically with removal of coronoid fragments and burring of any associated chondromalacic cartridge (AT). All received a six week course of oral Tepoxalin on enrolment and outcome was evaluated using inverse dynamics gait analysis at the time of initial presentation and at four, eight, 26 and 52 weeks. The gait variables analysed were elbow moment, elbow power, total support moment (TSM) and total support moment ratio (TSMR) as a measure of thoracic limb asymmetry.

The results indicated affected peak elbow moment increased from 0.58 to 0.76 Nm/kg in the AT dogs, and from 0.66 to 0.81 Nm/kg in the CM dogs and there was no significant difference between the two groups. Affected peak elbow power increased marginally in the AT dogs, but was unchanged in the CM dogs and there was no significant difference between the two groups. TSM increased from 1.49 to 1.92 Nm/kg in the AT dogs and from 1.52 to 2.06 Nm/kg in the CM dogs and again there was no significant difference between the groups. TSMR was statistically different between treatment groups at one ($P = 0.003$) and two months ($P = 0.048$) with the AT group more asymmetric and hence more lame.

TSMR at 12 months was 0.83 (AT) and 0.86 (CM) implying a failure of return to soundness by either group.

The authors concluded that AT dogs had increased mechanical asymmetry at four, 29 and eight weeks compared to the CM group revealing surgery worsened limb function. There was no significant difference in mechanical symmetry between groups at 26 and 52 weeks, suggesting arthroscopic treatment of MCPD worsens limb function in the 26 weeks following surgery when compared to CM and is of no additional long term therapeutic benefit.

In conclusion, the prognosis for this condition is hard to predict. The pre- and post-operative use of polysulphated glycosaminoglycans, such as pentosan polysulphate sodium, has not in my experience made a significant change in either the short or long term results.

c) Osteochondritis dissecans (OCD) of the medial condyle of the humerus

OCD occurs with low frequency in Rottweilers and has its highest incidence in Labradors and Golden Retrievers. In common with OCD in other joints it has a high incidence of bilateral involvement. The lesion is usually found near the outer edge of the central weight bearing region of the articular surface of the medial humeral condyle.

The clinical signs are very similar to FCP. A flexed lateral radiograph will show the presence of DJD on the anconeal process and a cranio-caudal or cranio-caudal medial oblique projection will usually reveal a defect in the subchondral bone of the medial humeral condyle. Occasionally ossification of the cartilage flap will make it visible. In long-standing cases the flap may break off and become lodged in the caudal and medial aspect of the joint capsule. It may grow in this location forming a linear osteochondral ossicle, which may only be visible on a cranio-caudal lateral oblique projection.

Surgical exploration/arthroscopy of the medial aspect of the joint is the treatment of choice. The flap of cartilage should be removed and the subchondral bone defect scraped to stimulate healing. The medial coronoid process should be checked at the same time and removed if found to be diseased.

With early diagnosis and treatment, uncomplicated cases of OCD have a better chance of making a full recovery than cases with medial coronoid process fragmentation.

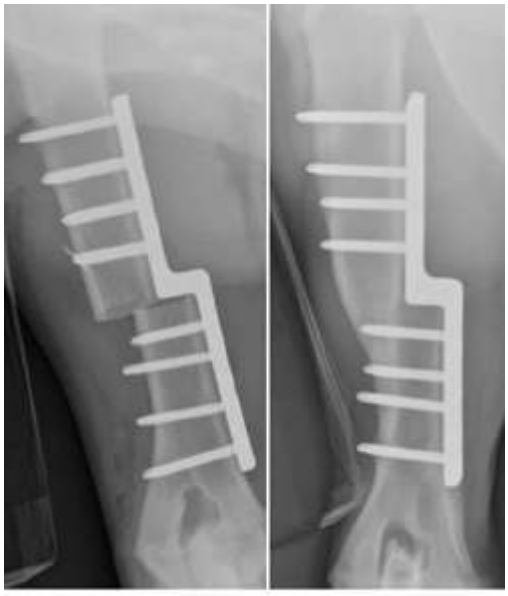
OCD of the shoulder joint will present with a similar history and clinical signs to osteochondrosis of the elbow, but careful examination of the animal should reveal that the pain is in the shoulder joint. Simultaneous OCD in the shoulder and elbow is unusual.

The pressures of the show-ring, the dictates of some breed societies and the current fashionable status of large breeds of dog have all contributed to an alarming increase in the incidence of orthopaedic problems in the elbow associated with abnormal development and rapid growth.

It is now clear that these conditions are inherited with an inheritance that is similar to hip dysplasia. It is strongly recommended that breeders of susceptible breeds not only subject their breeding stock to routine hip dysplasia radiographs prior to breeding, but also to radiographs of the elbow. Successful national schemes have been in operation for some years in Europe and an International Elbow Working Group was established in 1989 to help disseminate information and to establish a uniform classification system. Currently the BVA/KC Elbow Dysplasia Scheme requires two views of each elbow and each joint is graded from 0-3. The worst score of the two joints represents the dog's overall score.

Sliding humeral osteotomy

The sliding humeral osteotomy (SHO) procedure addresses medial compartment disease by shifting the load and weight bearing to the lateral (outer) portion of the joint. This unloads the medial compartment of the joint and reduces bone-on-bone contact. To accomplish this, an incision is made on the medial aspect of the humerus; the bone is cut and then shifted laterally.



Postoperative SHO Healed SHO

A specially designed plate with locking screws is used to stabilize the bone and shift the weight to the lateral part of the elbow. Healing usually takes about 8 to 10 weeks.

Data collected from 32 dogs included force plate data, radiographs and a 15 question owner survey regarding the soundness of their dog prior to SHO and at the final evaluation.

All dogs except for two exceeded their pre-operative ground reaction forces in the operated limb and there was a statistically significant upward trend in ground reaction forces as compared to the contralateral limb. Radiographic osteophytosis using the IEWG protocol had not progressed on the operated limb in any of the dogs except for one.

Ninety percent of owners felt that lameness had diminished at the final evaluation compared to preoperatively. Ten dogs had post-operative complications of which 6 were considered major requiring another surgery.

Proximal Abducting Ulnar Osteotomy (PAUL)

By performing an osteotomy of the ulna and applying a stepped plate designed by Kyon, the medial compartment of the elbow is unloaded.

The ulna is cut 3.5 to 4.0 cm distal to the elbow joint. A plate, which is a modified ALPS (Advanced Locking Plate-Kyon AG) plate with a 2 or 3mm step is applied to the lateral surface of the ulna.

Dr. Ingo Pfeil, Dresden, Germany, began clinical use of the PAUL technique in 2007.

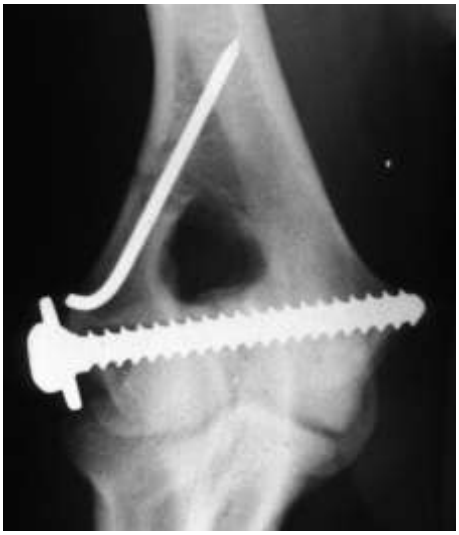
Controlled clinical release of the system was initiated in early 2010. Clinical experience in the United States, Europe and Japan has demonstrated reduced complexity and morbidity of the procedure in comparison to other corrective surgical techniques and is meeting expectations.

In most cases, complications have been related to technical errors and implant design.

Fractures of the distal humerus

Fractures of the distal humerus are common, particularly the Salter-Harris Type III and IV epiphyseal fractures of the lateral condyle in young dogs - whereas the bi-condylar T and Y fractures are more common in adult dogs, particularly Spaniels.

Lateral condylar fractures usually occur as a result of an indirect force following a jump or fall, which is transmitted, via the radius to the lateral condyle of the humerus. This condyle is not as well supported as the medial condyle and the force shears off the condyle through the articular surface, into the supratrochlear fossa and out through the lateral metaphysis. On palpation the most notable feature is the prominence of the medial epicondylar region of the humerus which tends to slip distally and medially once the support of the lateral condyle is removed as result of the fracture. The diagnosis is confirmed radiographically. Open reduction and internal fixation is indicated if malunion and DJD is to be avoided. Exposure of the intra-articular portion of the fracture via a lateral approach is important because precise reduction in this area is desirable. Reduction is achieved by traction and manipulation.



A pair of single or double sharp-pointed reduction forceps (Veterinary Instrument Co., UK or Synthes, Switzerland) are very useful in maintaining the reduction while the implants are inserted. Stabilization is normally achieved with a single transcondylar lag screw placed in the centre of the condyles. Accurate location of the screw is ensured by drilling the gliding hole first from the inside of the fracture surface to the outside.

Alternatively, following reduction of the fracture and temporary fixation with a K-wire, the epicondyles are used as landmarks, but the optimum position for the screw is just distal and cranial to these points. Rotation of the condyle is prevented by inserting a K-wire up the shaft of the epicondyle into metaphysis. The implants should not enter the supratrochlear fossa as they may impinge on the anconeal process.

Bicondylar distal humeral fractures (T or Y fractures) maybe caused by a fall or some other uncoordinated movement, or direct trauma from being hit by a car or from a gun-shot injury.

In one series of 133 condylar fractures over half the 45 bi-condylar fractures were caused by indirect trauma. Twenty-nine were classified as Y-fractures and 15 as T-fractures.

These are difficult fractures to treat successfully. This arises because the fracture is difficult to expose without undue additional soft tissue trauma, the intra-articular components of the fracture can be hard to reduce accurately and lastly, it is difficult to achieve rigid stabilization of the distal articular fragment to the diaphysis, because of the size shape and location of the fracture. Even in expert hands over 50% of the affected animals will end up with some degree of permanent lameness.

Postoperative physiotherapy should be started as soon as the bandage is removed but exercise should be strictly limited for at least 4 weeks. Some degree of long-term joint stiffness is to be expected.

Incomplete Ossification of the Humeral Condyle

Lameness caused by sagittal fissures spanning partially or completely across the humeral condyle has been recognised in dogs since 1989. Uncertainty regarding the etiopathogenesis of this condition remains, and as a result some authors describe the lesion as incomplete ossification of the humeral condyle (IOHC), whilst others describe it as an incomplete or stress fracture of the humeral condyle.

Prior to the identification of humeral intracondylar fissures (HIF) as a cause of lameness, a high incidence of humeral condylar fractures in spaniels had led to a suspicion of an inherent weakness or conformational abnormality in these dogs. The primary supporting presumption for IOHC rather than stress fracture is the fact that humeral condylar fractures and radiolucent fissure lines have an identical location to the cartilaginous remnant present between the lateral and medial centres of ossification in immature dogs. In normal dogs, these centres are reported to unite by 70 ± 14 days of age with completion of ossification by 32 weeks of age.

In many cases, fissures visible in clinically affected dogs extend proximal to the physis. This component of the HIF cannot be accounted for by incomplete ossification of the epiphysis. Thus, it has been proposed that these complete fissures may be the extension of a weakness created by the presence of IOHC. Support for this mechanism of fissure propagation is provided by the absence of documentation of dogs with complete ossification of the humeral condyle subsequently developing HIF.

A retrospective case series using the clinical records from dogs (n=77) undergoing prophylactic screw placement for the treatment of IOHC (n=53) or presenting for treatment of humeral condyle fracture with IOHC confirmed in the contralateral elbow (n=24) at six UK referral centres were reviewed. Patient signalment, presentation, surgical management, post-operative care and post-operative complications were recorded. Post-operative complications were divided into seroma, surgical site infections (SSIs) and implant complications.

Spaniel breeds and entire males were over-represented. The overall complication rate was 59.7%. Seroma (29.9%) and SSIs (23.4%) were the most commonly encountered post-operative complications. Implant failure occurred in 9.1% of cases. Increasing bodyweight was a significant risk factor for the development of post-operative complications (p=0.001). Placement of the transcondylar screw in a lagged manner rather than as a positional screw was the only factor identified as significantly reducing the incidence of complications (p=0.018).

Surgical management of incomplete ossification of the humeral condyle is associated with a high rate of post-operative complications. The transcondylar screw should be placed in a lagged manner wherever possible.

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ECGS AND ARRHYTHMIAS – WHEN TO WORRY

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Abstract

Arrhythmias are commonly detected during auscultation of small animal patients and may be physiological (such as sinus arrhythmia) or potentially serious and pathological. The goal of this lecture is to review common small animal arrhythmias and highlight the settings where a dysrhythmia is more likely to be significant and urgent intervention likely to be required. The potential applications for ambulatory ECGs in small animal practice will also be discussed using case examples.

Terminology

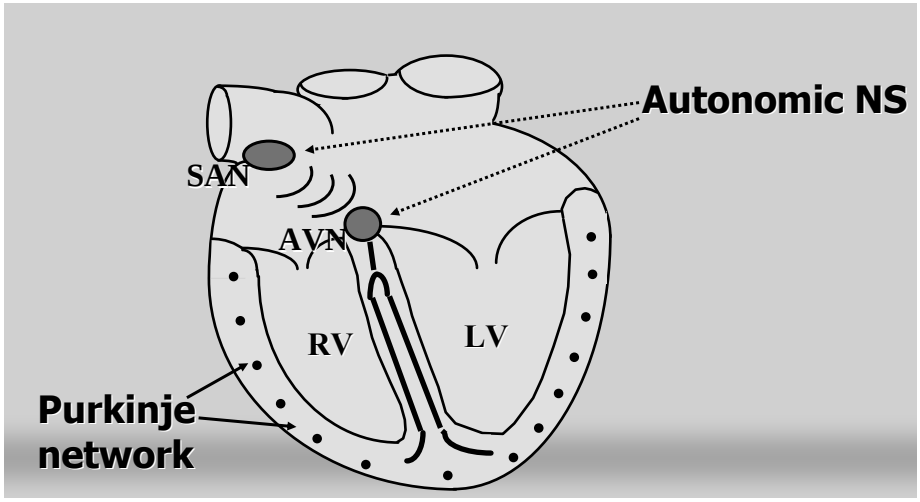
- Electrocardiography – process of recording electrical activity at the body surface. The electrical activity is generated by waves of depolarisation and then repolarisation of muscle creating small potential differences between one part of the heart and another.
- Electrocardiogram – device containing a galvanometer to record changes in potential difference across the heart muscle.
- Holter monitor – ambulatory ECG useful in the investigation of intermittent arrhythmias and drug monitoring
- Automaticity – the ability of cardiac myocytes to depolarise and thereby contract spontaneously
- Syncitium – cardiac myocytes have the ability to conduct an impulse from cell to cell resulting in sequential depolarisation and contraction

Uses of an ECG:

- Diagnosis of dysrhythmias
- Documenting heart rate and rhythm

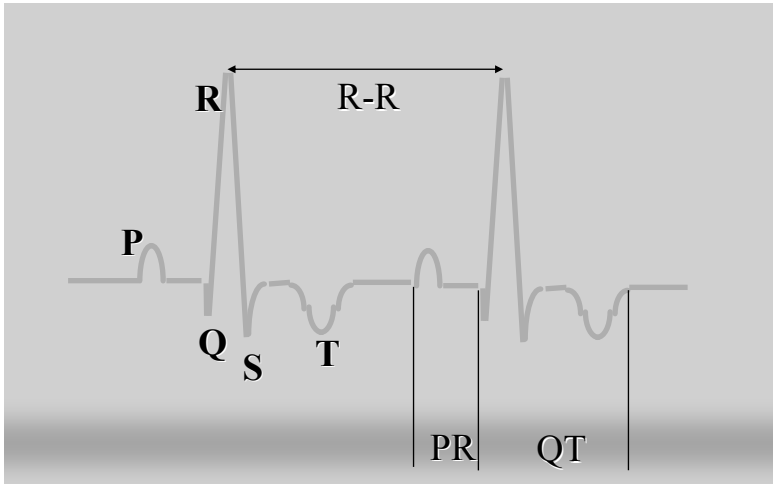
Formation of the QRS Complex

The conduction pathway



- Fibrous ring insulates ventricles from atria

ECG waveforms and intervals



- P wave starts in the sinoatrial node and spreads across the atria creating a small positive deflection on the surface ECG
- PR interval created by a short delay at the AVN. This allows time for the atria to contract thereby filling the ventricle before ventricular contraction.
- QRS complex created by depolarisation of the ventricles
- Q wave – 1st negative deflection created by depolarisation of the apical interventricular septum
- R wave – positive deflection created by depolarisation of the ventricles
- S wave – negative wave associated with depolarisation of the basilar part of ventricles and septum
- J point – isoelectric point at end of QRS complex

- ST segment – plateau of action potential prior to repolarisation
- T wave – ventricular repolarisation, can be +ve, -ve or biphasic. In some dogs polarity changes with activity and/or changes in body position.

Recording an ECG

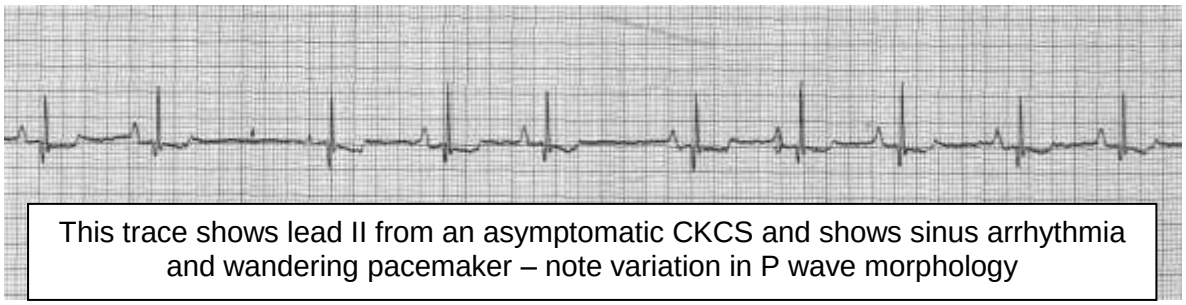
- Patient calm, still, unsedated and ideally in right lateral (cats may be in sternal)
- Good electrical contact – surgical spirit or ECG gel
- Clips on bony areas
- Electrically insulated surface
- Check sensitivity so that complexes are on trace
- Filter off
- 50mm/s leads I, II, III, aVL, aVR, aVF
- 25mm/s rhythm strip for 1-5minutes

Artefacts

- Movement – limbs or respiratory
- 50Hz AC – improve contact, check for sources of AC interference, blanket under patient

Interpretation

- Quality of recording (artefacts)
- Heart rate – interpret in light of temperament, breed and age. Count number of beats in 6 seconds and multiply by 10.
- Heart rhythm
    - Sinus
    - Sinus arrhythmia – caused by regular fluctuations in vagal tone often associated with respiration; can be very marked in brachycephalics and cases with respiratory disease
    - Wandering pacemaker – occurs due to dominant pacemaker shifting within the sinoatrial node resulting in differences in P wave morphology, usually associated with high vagal tone, more common in dog, need to distinguish from supraventricular premature complexes



This trace shows lead II from an asymptomatic CKCS and shows sinus arrhythmia and wandering pacemaker – note variation in P wave morphology

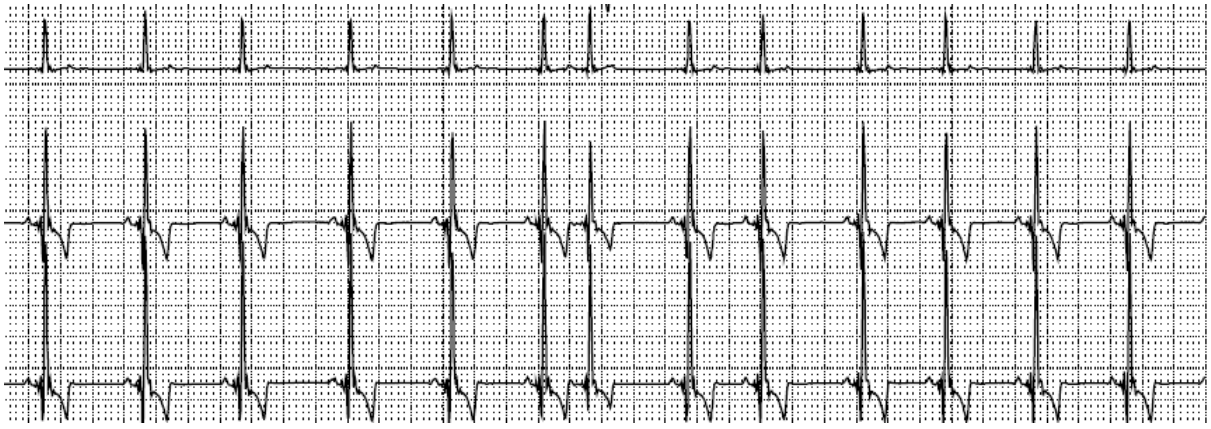
- P:QRS – should be 1:1
- Shape of QRS complexes – uniform or multiform
- Measurements of voltages & intervals
- Summarise and categorise your findings

ECG Abnormalities

Ectopic beats

Features of supraventricular premature beats (SPCs):

- Occur earlier than the normal R-R interval
- +/- P wave
- QRS complex appears similar to sinus beats

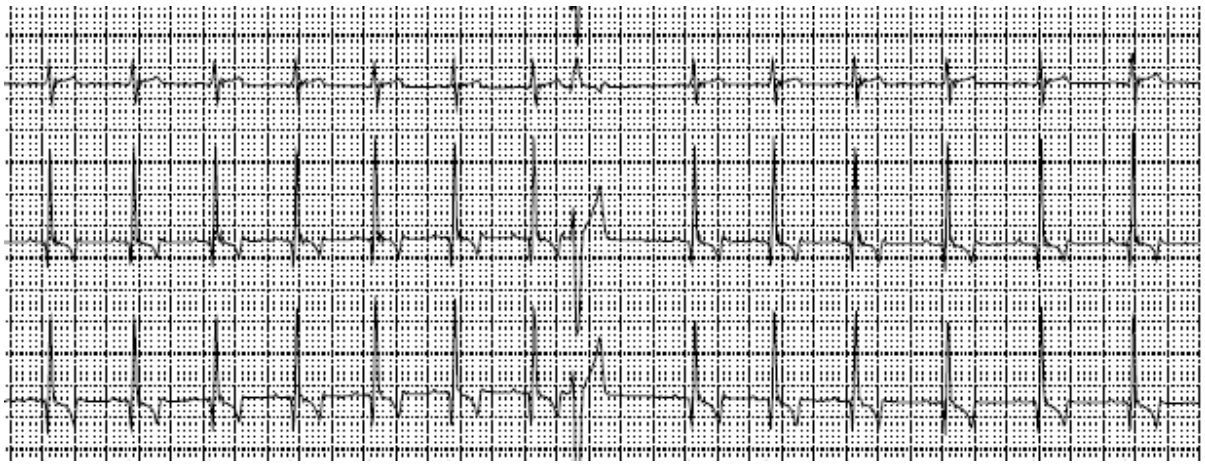


Significance of SPCs:

- More common in cases with atrial stretch and therefore presence suggests underlying heart disease
- More common in cases with supraventricular tachycardias and therefore ambulatory monitoring is indicated to assess heart rate and rhythm over a longer period
- Echocardiography is indicated to assess cardiac chamber size and function
- Single SPCs are unlikely to cause a significant drop in blood pressure and therefore specific anti-arrhythmic therapy may not be required but the underlying condition may require treatment and/or monitoring.

Features of ventricular premature beat (VPCs):

- Occur earlier than the normal R-R interval
- QRS complexes wide and bizarre
- Often have a compensatory pause after the ectopic beat
- T wave polarity usually opposite to sinus beats



Significance of VPCs

- Presence suggests underlying cardiac and/or systemic disease
- May be seen secondary to thoracic trauma
- More concerning in breeds predisposed to cardiac disease such as Boxers, Dobermans, Great Danes and, in these breeds, this finding would prompt further evaluation including echocardiography and Holter monitoring
- Whilst single VPCs are unlikely to require treatment, more sustained complex ventricular ectopy is more likely to require treatment (see section on ventricular tachycardia).

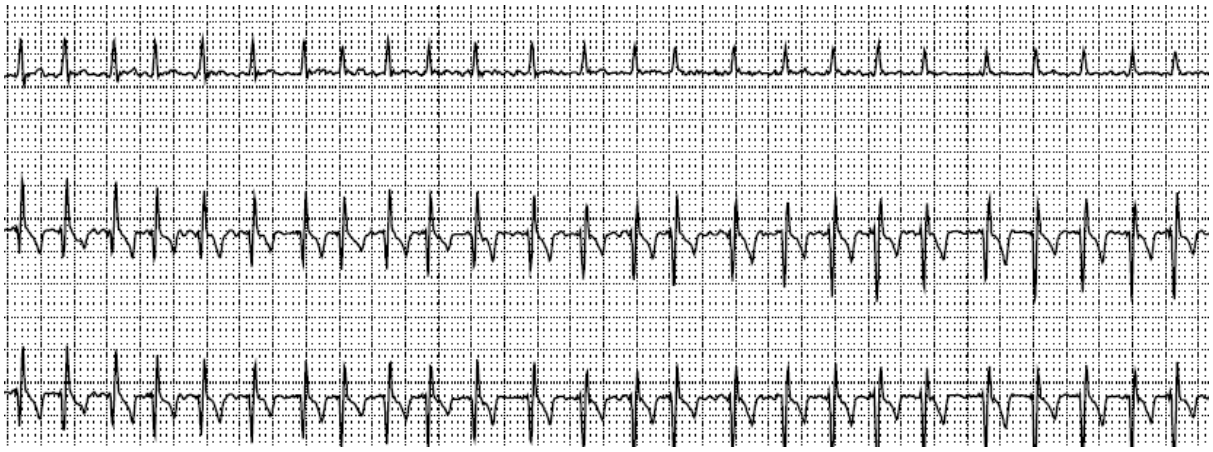
Common Tachyarrhythmias

Atrial fibrillation

Atrial fibrillation is most commonly seen in large breed dogs. It can be seen in smaller breed dogs and cats but in this setting generally occurs secondary to severe heart disease with marked atrial enlargement.

Atrial fibrillation is characterised by:

- Rapid irregularly irregular rhythm
- No obvious P waves preceding the QRS complexes
- Can be paroxysmal but generally sustained in dogs



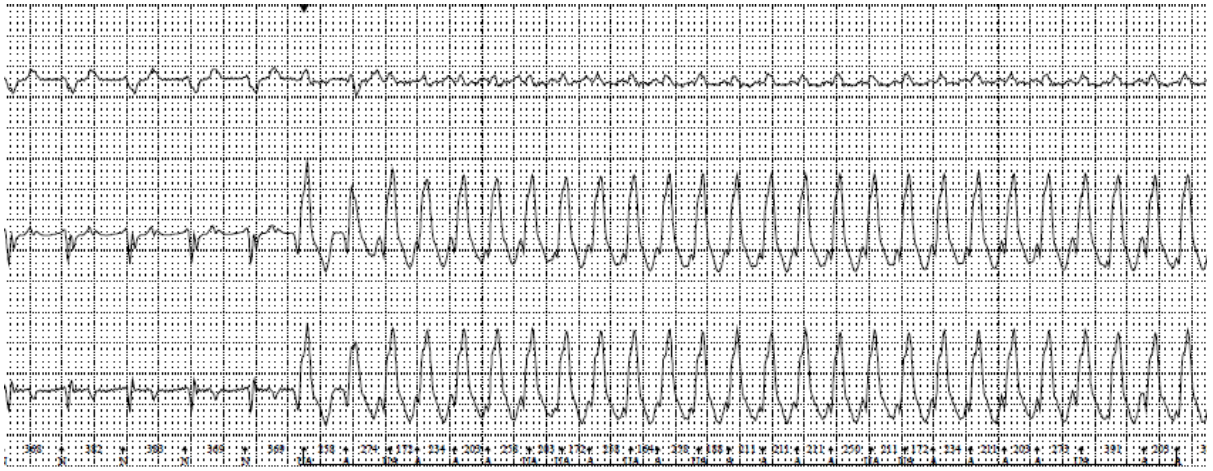
Significance:

- Implies that heart disease is likely to be present
- Further investigation required including echocardiography
- 24h Holter useful to determine mean heart rate before / after treatment
- Ventricular rate control is required in most cases but drug choice depends on underlying disease and whether congestive heart failure is present.

Rapid ventricular rhythms

Characteristics of ventricular tachycardia:

- Rapid (>180bpm)
- Generally regular
- More than 6 consecutive wide, bizarre QRS complexes



Significance:

- Seen in animals with severe cardiac and/or systemic disease (e.g. gastric dilation torsion, pancreatitis, splenic disease, IMIA, etc).

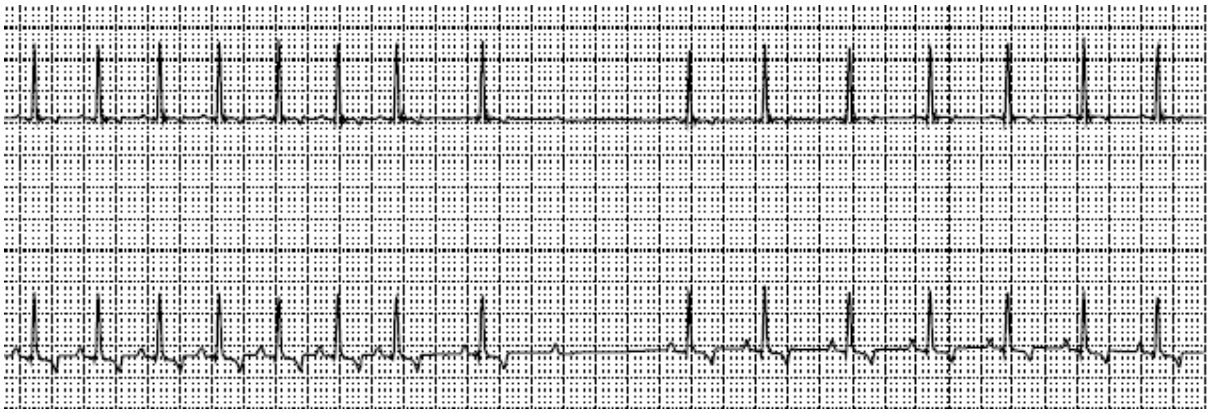
- Of particular concern in Boxers, Dobermanns and Great Danes as accompanies cardiomyopathy in these breeds
- Potentially a life threatening dysrhythmia as can degenerate into ventricular fibrillation
- Anti-arrhythmic treatment likely to be required urgently in addition the treating underlying disease if possible
- If dog is aerodynamically compromised and assuming electrolytes are within range then lignocaine 2mg/kg i/v may be an acceptable first line drug choice. See BSAVA formulary for more information.

Selected bradyarrhythmias

Second degree atrioventricular block

The features of this arrhythmia are:

- P wave without an ensuing QRS complex



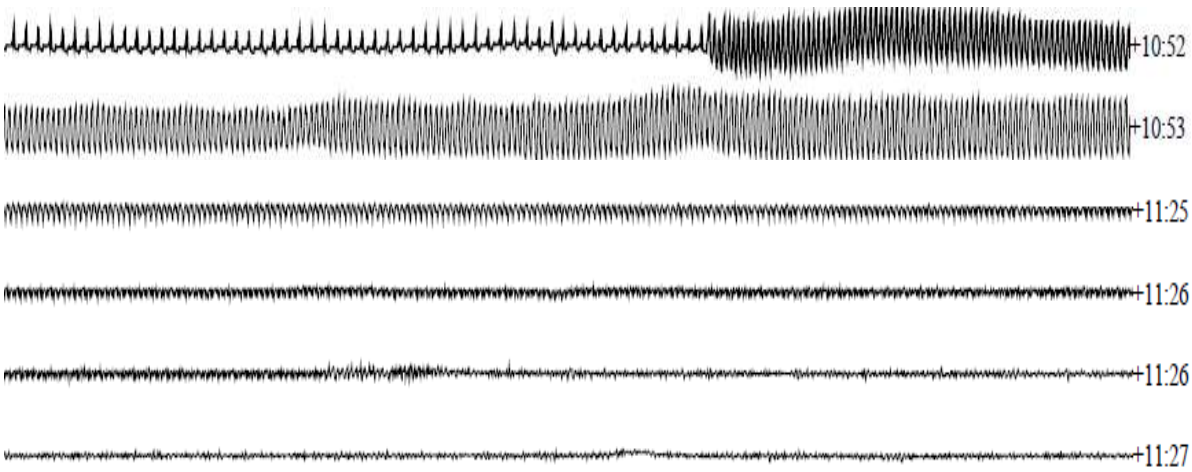
Significance of this dysrhythmia

- Commonly seen on ambulatory ECGs of normal dogs during periods of rest
- Abnormal if occurs during sinus rhythm
- Abnormal if there are multiple consecutive unconducted QRS complexes resulting in long periods of ventricular asystole
- More common in individuals with altered autonomic tone e.g. patients with respiratory, GIT and/or intracranial disease
- May also be seen in sick sinus syndrome
- Mild cases may respond to sympathomimetic therapy. Severe cases with high grade second degree atrioventricular block (as seen in the figure below) may require pacemaker implantation.

Terminal heart rhythms

- The number of ECG rhythms associated with cardiopulmonary arrest (CPA) in small animals is limited.
- In most situations the ECG will reveal one of the following arrest rhythms:
    - Sinus bradycardia – normal ECG but slow rate
    - Pulseless electrical activity (PEA) – normal cardiac electrical activity but no mechanical activity
    - Ventricular fibrillation (VF) – see figure below
    - Asystole – see figure below

The following figure was taken from an ambulatory ECG recording from a Boxer. The top line shows sinus rhythm which then converts to rapid ventricular tachycardia (VT) / ventricular fibrillation. Rapid coarse VF was sustained from 10:53 to 11:25 with the complexes becoming progressive lower in amplitude – likely fine VF. By 11:27 the trace shows asystole and, sadly, the dog was dead at this time.



Medical treatment

- Correct underling disease
- Consider electrical cardioversion if defibrillator available
- Lignocaine for ventricular fibrillation
- Adrenaline / atropine for bradyarrhythmias

Ambulatory ECGs

Holter monitors

Many of the ECGs shown in this presentation have been obtained from dogs wearing an ambulatory ECG or Holter monitor.

This device weighs about 150g and is placed inside a pocket inside a specially designed vest which the dog wears for 1-7days. The monitor is connected to the dog using 3 adhesive electrode pads. This photo shows my dog wearing a Holter monitor.

The device records continuously throughout the recording period and the data is then downloaded onto a computer and analysed using commercial software.

These monitors have been successfully used in many thousand cases including miniature and giant breed dogs and also in cats.

Indications for an ambulatory ECG include:

- Episodic collapse
- Unexplained exercise intolerance
- Arrhythmia detected on auscultation
- Pre-breeding screen in breeds at risk of dysrhythmias such as Boxers, Dobermans and Great Danes
- Investigation of whether ventricular rate control is adequate in atrial fibrillation
- Screening for dysrhythmias in dogs
- Assessing efficacy of anti-arrhythmic therapy.

Other forms of ambulatory ECG include an event recorder which continuously records a loop of ECG which is then over-written by the next loop. The device can be activated to store a loop by the owner pressing a button. The advantage of these devices is that they can potentially stay on the dog for up to a month. The disadvantage is that they require manual activation by the owner.

Reveal devices have also shown some promise as these loop recorders are small and implanted subcutaneously. However they also rely on owner activation and sedations / general anaesthesia is required to place and also remove the device.

Cases will be presented during the talk showing the clinical applications of ambulatory ECGs. Holter monitors are available for veterinary surgeons to hire – please contact www.holtermonitoring.co.uk for more information.

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A SURGICAL APPROACH TO HAEMOABDOMEN: THE SPLEEN AND BEYOND

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Haemoabdomen (haemoperitoneum) can be either traumatic or nontraumatic in origin. Although the spleen is always high on the list of potential causes of intra-abdominal bleeding, it is important to consider that the blood may arise from other parenchymal organs, a mass lesion, a vascular pedicle or be as a consequence of other conditions e.g. rodenticide toxicity. Although not every patient will require surgical intervention, those that do need a planned approach to limit further blood loss and treat the underlying problem effectively. This lecture will provide tips in how to proceed when the source of the bleeding is not the spleen.

Nontraumatic causes of haemoabdomen include rupture of intra-abdominal neoplasms, coagulation defects e.g. rodenticide poisoning and organ malposition/ischaemia e.g. GDV, liver lobe or splenic torsion. The source of bleeding is most commonly the spleen: in one study of 39 cases of non-traumatic haemoabdomen, most cases originated from the spleen with only one dog with a bleeding renal mass; two dogs with multiple hepatic masses and one that had multiple mesenteric bleeding masses leading to haemoabdomen.

Both blunt and penetrating trauma can cause haemoperitoneum, and road traffic collisions are recognised as the leading traumatic cause of bleeding. Rupture of an intra-abdominal neoplasm (splenic, hepatic, adrenal, renal, gastrointestinal and body wall) is probably the leading cause of nontraumatic haemoabdomen. Post-surgical haemorrhage is an important nontraumatic iatrogenic cause of bleeding and is of particular significance following ovariohysterectomy, splenectomy, hepatic lobectomy, nephrectomy or excision of mass lesions.

There are a number of situations where surgical intervention is not appropriate or may not be required e.g. rodenticide toxicity or self-limiting haemorrhage following trauma causing injury to the spleen or other organs etc.

Indications to pursue surgical management in dogs with haemoabdomen include:

- Penetrating abdominal trauma;

- Associated pneumoperitoneum (suggesting penetrating trauma or ruptured viscus);
- Associated septic or bile peritonitis;
- Organ ischaemia (GDV; splenic torsion; liver lobe torsion; mesenteric volvulus);
- Decreasing peripheral PCV in conjunction with increasing abdominal fluid PCV on serial samples;
- Inability to correct perfusion abnormalities with fluid resuscitation and transfusion therapy with evidence of continued bleeding;
- Persistent hypotension;
- Abdominal wall or diaphragmatic hernia.

It is beyond the scope of this lecture to deal with the medical stabilisation and anaesthesia of patients with haemoabdomen in detail. However, it is important to: have secured intravascular access; have in place suitable methods of fluid support including the possibility of blood transfusion or autotransfusion (see later); consider whether or not further investigation of the patient is warranted prior to surgery e.g. thoracic radiographs to rule out pulmonary metastatic disease; monitor the patient closely; provide adequate analgesia; minimise the impact of general anaesthesia on the patient and provide close continued support and monitoring postoperatively.

Prior to undertaking surgery on a patient with haemoabdomen it is essential that you have discussed the aims, complications, limitations and outcomes of surgery with the pet's owner. An estimate of the anticipated costs must also be provided. Depending on the expected aetiology or severity of the problem it may be appropriate to consider referral of the patient.

Surgical approach

A surgical assistant will be invaluable but it is recognised that one will not always be available.

Many patients can be partially pre-clipped before inducing anaesthesia. It can be useful both during preparation and subsequently during surgery to tilt the table (or use a board on the table top set on an incline). This will lower the abdomen slightly to help shift the blood within the abdomen away from the diaphragm to maintain or improve the functional residual capacity of the lungs. It will also help with surgical exposure if the source of the bleeding in the cranial abdomen.

When pre-clipping and prior to induction of anaesthesia oxygen supplementation can be provided, if it is not resented, using a face mask or a flow-by technique. The patient should be clipped and aseptically prepared for a complete abdominal exploration and any other procedures that may be required.

A ventral midline skin incision is appropriate. However, rather than simply extending this incision through the linea alba in the normal way, initially a more limited approach can be made if facilities for suction are available. Make a small incision, just large enough to allow insertion of a suction tip: ensure that you have entered through the peritoneum. A sump tip on the suction unit to reduce tissue grab and improve drainage is preferable as it facilitates more rapid evacuation of the blood from the abdomen e.g. Poole suction tip. Elevate the linea alba at the site of the incision to limit blood spillage, introduce the suction tip and suction blood from the abdominal cavity. Once most of the blood has been removed the incision may be extended in the normal way, using suction as required.

If suction is not available once inside the abdomen, all that you will see will be bowel loops swimming in a pool of blood. Empirical packing as described above can be performed but an alternative approach is to eviscerate the abdomen and start to rapidly evacuate the blood with sterile swabs, towels or catheter tip 60ml syringes. Evisceration converts the abdomen into a manageable workspace allowing improved exposure.

In the presence of ongoing bleeding that is not immediately apparent, and especially if you are operating on your own, the abdomen is packed with laparotomy swabs or sterile towels. I usually put one in each of the four quadrants, left and right cranial and caudal. The swabs are removed sequentially and all sources of haemorrhage are controlled at least temporarily. Temporary haemostasis can be achieved by placing haemostats on all vessels to be ligated (if atraumatic vascular occlusion is required e.g. Rumel tourniquet, atraumatic bulldog clamp or Satinsky vascular clamp. Once all sources of haemorrhage have been identified definitive control of the bleeding vessels and organs can be obtained. Another method of limiting ongoing haemorrhage is to compress the abdominal aorta at the level of the coeliac artery. This is achieved by sliding a hand dorsally along the left peritoneal wall, palpating the cranial pole of the left kidney and moving the hand cranial and medial to the left adrenal gland to compress the aorta digitally. This manoeuvre effectively controls arterial haemorrhage from the coeliac artery distally. Digital compression can be maintained until the haemorrhage is controlled.

Bipolar electrocautery is ideal for controlling haemorrhage from small arteries and veins < 1 and 2 mm in diameter respectively. Larger vessels must be ligated using suture or vascular clips of an appropriate size. If using vascular clips it is important to isolate the vessel from surrounding tissues and to choose a clip that is about 1/3-2/3 longer than the diameter of the vessel: once flattened by the clip, the circular vessel will become oval, and therefore longer! Feedback monitored bipolar or harmonic vessels sealing devices may also be used. Ligation of vascular pedicles or *en bloc* ligation of tissues needs to be performed carefully to prevent ligatures slipping. There a few recommendations to limit the possibility of ligatures slipping: always use a suture of suitable size for the task being undertaken; place the ligature perpendicular to the long axis of the vessel or tissue; use 'flashing' of the haemostat closest to the site of ligation or use a three clamp technique; tie secure knots and cut the suture ends to an appropriate length. Specifically 'flashing' of the haemostat refers to rapid loosening and tightening of the haemostatic forceps as the first throw of the ligature is placed. The manoeuvre is done such that one does not actually see the forceps loosening and the jaws of the haemostat do not move off the tissue. It is important to make sure the suture is not clamped. This technique removes tension from the vessel to be tied and the adjacent soft tissues making sure that the ligature can be tied securely. When vessels cannot be grasped easily with haemostats, 'stick ties' can be placed in a simple interrupted or cruciate pattern by placing a suture through the tissue surrounding the bleeding vessel: the soft tissue traps and helps occlude the vessel. Bleeding from most superficial lacerations in the liver, spleen, and kidney may be sufficiently controlled with direct pressure for 10–15 minutes. Apply only sufficient pressure to prevent bleeding as excessive pressure can delay or prevent coagulation at the site of injury. Superficial or minor lacerations that bleed despite application of direct pressure and deeper lacerations should be sutured with mattress sutures. If the mattress sutures are pulling through the capsule, a particular problem in the liver, consider using a product such a mesh or VetBioSIST to support the sutures. However, partial or complete liver lobectomy, partial or complete splenectomy, or partial or complete nephrectomy may be required to control ongoing haemorrhage. Another option for control of haemorrhage is the omentum, which has procoagulant properties and can be sutured into wounds in parenchymal organs. Topical haemostatic agents can also be placed into wounds that are oozing to help control capillary haemorrhage.

When all efforts at haemostasis fail to control haemorrhage from parenchymal organs and there remains a large amount of haemorrhage or ooze from multiple sites, the abdomen can be repacked with fresh sterile laparotomy pads or towels to provide direct pressure to oozing wounds and the abdomen is closed temporarily over the towels. The patient is recovered

from anaesthesia and hypothermia, acidosis and coagulation abnormalities are corrected. Reoperation is planned within 24–48 hours when the patient is more stable.

Splenic haemorrhage

Traditionally the spleen is removed by individually ligating the numerous branches of the splenic artery and vein at the splenic hilus. The procedure can be performed relatively quickly with a ligating and dividing stapling device or tissue sealing devices. Splenectomy by ligation of the splenic and short gastric arteries has been described as a faster and simpler method, particularly when adhesions and, or a mass are obscuring access to the hilus or edge of the spleen (modified or Hosgood technique).

With the latter technique, the spleen is lifted out of the abdomen and the omental bursa opened. The left limb of the pancreas is located and followed to identify the splenic artery and vein. The splenic artery is clamped and double ligated, taking care not to catch the pancreas in the ligature. It is important to check that the arterial supply to the left limb of the pancreas is not compromised. Next, the left gastroepiploic and the short gastric vessels are located near the fundus of the stomach. These vessels are also clamped and double ligated. The spleen is then removed. Remember to ligate any omental vessels of consequence. If omental adhesions to the mass make access to the splenic or short gastric vessels difficult, then clamp and ligate the omentum to improve access. While this technique is appealing, from personal experience it can be challenging to perform in cases where there are many omental adhesions to the mass. Furthermore, the site at which the arterial branch to the left limb of the pancreas arises is variable.

Liver haemorrhage

The liver is a highly vascular organ with a very thin collagenous capsule. There are a large number of significant vessels within the deep parenchyma that can be damaged and contribute to the bleeding: central branch of the portal vein; hepatic artery; hepatic veins, and the retrohepatic vena cava depending on the liver lobe(s) involved.

When you encounter significant ongoing haemorrhage from the liver try and institute the rule of the four p's:

- *Position*. Following blunt abdominal trauma the capsule of the liver may have fractured. Try and put the hepatic parenchyma back into a normal position. With normal coagulation, the formation of fibrin will help to hold the fractured parenchyma together.

- *Pringle manoeuvre*. A finger is placed in the epiploic foramen and of the hepaticoduodenal ligament compressed occluding the portal vein and the hepatic artery. A vascular clamp or modified Rumel tourniquet can be placed at the same location. This manoeuvre will control approximately 70% of the blood supply to the liver. Although these vessels can be occluded for 10-15 minutes, ideally a five minute on, two minute off technique with a total maximum occlusion time of 15 minutes is preferred. If possible simultaneous occlusion of the cranial mesenteric artery should be performed to prevent acute portal hypertension. If bleeding from the liver persists despite occlusion of the hepatic artery and portal vein, retrograde flow must be occurring from the hepatic veins or the vena cava as they pass through the liver. Their intrahepatic location makes isolation and visualisation of these vessels for ligation difficult and bleeding from this site carries a grave prognosis.
- *Pack*. Pack moistened laparotomy pads around the liver to stent any small capillary bleeding, and hold the liver lobes in place.
- *Peeking*. DON'T! If you get good haemostasis consider adopting a salvage surgical technique and close the abdomen in preparation for re-operation within 24-48 hours to inspect the damage and retrieve the pads.

Any subcapsular haematomata that are identified should not be explored. Debridement of parenchymal fissures is required with placement of sutures on each bleeding vessel. Deep, centrally placed sutures are not recommended because of the risk to the hepatic veins and vena cava. There is a risk of abscess formation and biloma following injury to the hepatic parenchyma. Liver lobectomy may be required if the wound involves the major part of a lobe.

Up to 70% of the liver may be resected although resection of the right and central divisional lobes is challenging. With an otherwise healthy liver hypertrophy of the remaining hepatic parenchyma can be expected within six months with an expected return of both hepatic volume and function.

There are a number of choices for partial liver lobectomy and lobectomy:

- Finger fracture (skeletalisation). This technique should be avoided near the hilus of the liver because it is not a precise technique and offers poor protection to the larger veins encountered in this area. Determine the line of separation between normal hepatic parenchyma and that to be removed, and sharply incise the liver capsule along the selected site. Bluntly fracture the liver with the fingers or the blunt end of a

scalpel handle, and expose the parenchymal vessels and biliary structures. Alternatively, a suction tip may be used to aspirate the hepatic parenchyma from around the vascular and biliary structures. In any event suction is useful to remove the parenchymal bleeding that will be encountered during this procedure. Electrocoagulate small bleeders encountered during the dissection and ligate or clip the ducts and vessels to prevent leakage and bleeding. Excise the hepatic parenchyma distal to the ligatures or clips.

- Mass (or bunch) ligation (including endoscopic loop snaring). The left lobes of the liver i.e., left lateral and left medial lobes maintain their separation near the hilus to a greater degree than the other lobes do. Therefore, the left lobes often can be removed in small dogs and cats by placing a single encircling ligature around the base of the lobe.
- Guillotine sutures. In small dogs and cats overlapping simple interrupted horizontal mattress sutures are placed through the liver parenchyma, along the margins of the lobe to be resected. The sutures crush through the parenchyma as they are tied and ligate the vascular and biliary elements. Be sure that the entire width of the hepatic parenchyma is included in the sutures. After tightening the sutures securely, use a sharp blade to cut the hepatic tissue distal to the ligature, allowing a stump of crushed tissue to remain with the ligature. The distal portion of the hepatic parenchyma is then resected.
- Surgical stapling. Thoraco-abdominal staplers are used in this technique. Ideally the 30V cartridge is used (three rows of overlapping staples) but in many cases this will not be large enough to span the pedicle. In that case a TA 55 or 90 can be used and a blue cartridge should be used in those circumstances.
- Feedback monitored bipolar or harmonic vessels sealing devices may also be used to achieve partial lobectomy.

A comparison of suction and vascular clip, suction and TA stapler, endoscopic loops, feedback monitored bipolar and harmonic scalpel for partial liver lobectomy showed that there were no significant differences between the techniques in terms of surgical time.

It is prudent, once control of bleeding has been achieved to carefully inspect the biliary tree for evidence for injury. Contrary to what you might expect it is usually the bile ducts that are injured rather than the gall bladder.

Renal haemorrhage

Bleeding from the kidney may occur following rupture of a mass lesion or following trauma. Trauma can result in a wide range of injuries to the kidney from simple contusion through laceration (parenchymal or pelvic); fracture / pulpifaction; rupture and injury to the vascular pedicle or ureter (avulsion or crush). The kidney is perhaps more likely to be crushed than ruptured because of its retroperitoneal location under the lumbar vertebrae and the firm capsule that surrounds its soft parenchyma: the exception of course being penetrating trauma. Rupture of the renal capsule can cause extensive retroperitoneal haemorrhage and may lead to ongoing urine extravasation and possibly the formation of urinomas. Minor capsular tears are associated with severe bruising of the renal parenchyma but do not usually require nephrectomy. Severe deep, multiple fractures and punctures of the kidney are best treated with nephrectomy. Kidneys are sometimes stripped out of their peritoneal beds and are found in the abdomen tethered only by their vascular and ureteral attachments. Such kidneys should be repositioned and secured in their normal lumbar beds. When positioning the kidney, care should be taken to ensure that the vascular and urethral conduits are not twisted. Kidneys that have been totally avulsed from their vascular attachments seldom survive. If an avulsed kidney is found during exploration, it should be removed and its vascular stumps located and ligated.

The kidneys lie in the retroperitoneal space lateral to the aorta and the caudal vena cava. They have a fibrous capsule and are held in position by subperitoneal connective tissue. The renal artery normally bifurcates into dorsal and ventral branches; however, variations in the renal arteries and veins are common. The ureter begins at the renal pelvis and enters the dorsal surface of the bladder obliquely by means of two slit like orifices.

Following a ventral midline abdominal approach the affected kidney is identified and isolated. The right kidney is more cranial than the left and relatively fixed in position. Using the mesoduodenum (on the right) or the mesocolon (on the left) as anatomic retractors will aid surgical exposure. If there is ongoing haemorrhage from the renal parenchyma or renal vessels, temporary occlusion of the renal artery will help to stem the flow. The easiest way to identify the renal artery is by retracting the affected kidney medially (see below).

Ureteronephrectomy

The kidney lies in the retroperitoneal space so firstly, the peritoneum over the kidney is incised. The kidney is freed from its sublumbar attachments, using a combination of careful blunt and sharp dissection. Retracting the kidney medially allows the renal artery and vein on the dorsal surface of the renal hilus to be identified. Double ligate the renal artery close to

the abdominal aorta to ensure that all branches have been ligated. I generally place a circumferential suture with a transfixing suture placed distally. Identify the renal vein and ligate it similarly. The left ovarian and testicular veins drain into the renal vein and should not be ligated in un-neutered dogs. Avoid ligating the renal artery and vein together to prevent an arteriovenous fistula forming. Double ligate and transect the ureter near the bladder. Remove the kidney and ureter as a single unit.

Partial nephrectomy

Partial nephrectomy is occasionally warranted although in most cases total nephrectomy is usually easier and carries less risk of postoperative haemorrhage. If partial nephrectomy is performed, electrocoagulation of bleeding vessels should be avoided because it causes excessive parenchymal damage. Avoid partial nephrectomy in animals with clinically significant coagulopathies because excessive blood loss may occur after this procedure. If possible, strip the renal capsule from the area of the kidney to be excised. Use absorbable suture with two long, straight needles attached. Thread the needles into the kidney at the proposed resection site. Tie the thread into three separate ligatures, but avoid damaging the renal vessels or ureter. Excise the renal tissue distal to these ligatures. Ligate any bleeders and suture the exposed diverticula with absorbable suture. Approximate the capsule over the end of the kidney, and anchor it to the sublumbar tissues to prevent rotation of the kidney. As an alternative, clamp the renal vessels with vascular forceps and excise the kidney parenchyma. Ligate the parenchymal vessels and close the renal pelvis and diverticula. Suture the capsule as described previously and remove the clamps from the renal vessels.

Other sources of abdominal haemorrhage

Slipped ligatures

Haemorrhage is perhaps the most common complication of ovariohysterectomy or spay-caesarean and can occur from many different sites. However, only bleeding from the ovarian pedicles, uterine vessels and broad ligament will be considered here. It is important when performing surgery to identify a slipped ligature or the source of on-going bleeding following ovariohysterectomy to extend the original incision to allow better surgical exposure and therefore more rapid isolation of the bleeding vessel. If the right ovarian pedicle is bleeding find the descending duodenum and reflect it to the left exposing the caudal pole of the right kidney and the right ovarian pedicle. If the left ovarian pedicle is bleeding find the descending colon, reflect it to the right exposing the caudal pole of the left kidney and the left ovarian pedicle. The safest way to exteriorise a bleeding ovarian pedicle initially is to reach in and with two fingers, grasp the pedicle and exteriorise it. Never apply haemostats blindly in the region of the source of the bleeding as you may inadvertently damage the ureter that

lies deep to the site of bleeding. Once the pedicle is exteriorised you can place two haemostats and then ligate in the crushed area of the most proximal haemostat. I would also place a transfixion ligature caudal to the circumferential ligature.

Signs associated with intra-abdominal haemorrhage

Cullen's sign: bruising around the umbilicus (periumbilical ecchymosis).
Turner's sign: bruising on the flank

Making a modified Rumel tourniquet

A modified Rumel tourniquet is made by sliding a ¼ or ½ inch Penrose drain around the vessel or pedicle and pulling the ends together. A haemostat is placed across the two ends of the tubing against the vessel to occlude it.

Some notes about autotransfusion

Typically, the vast majority of blood transfusions in veterinary medicine are allogeneic (homologous), meaning an animal other than the intended recipient donates the blood. Autologous blood transfusion is the process by which blood is donated by the intended recipient of the blood. Autologous blood transfusion in veterinary medicine is rarely performed on an elective basis although it has been described.

When an animal's blood is collected from the abdomen, thorax or at the time of surgery for reinfusion into the same patient the term autotransfusion is used. Allogeneic blood transfusion in the blood typed (and if appropriate crossmatched) patient is preferred to autotransfusion of autologous blood; however when circumstances dictate autotransfusion of blood collected by abdominocentesis, thoracocentesis or during surgery may be lifesaving.

Contraindications to autotransfusion include spillage of enteric contents (urine/bile) (+++), bacterial contamination (+++), presence of sepsis (++) and the presence of a neoplastic process especially if neoplasm has ruptured (++). Autotransfusion of blood that has been present within the body cavity for some time (>24 hrs) should theoretically not be used due to the presence of microaggregates of leukocytes and red blood cell lysis that may precipitate a systemic inflammatory reaction. However, in life-threatening situations the benefit may outweigh the risk when no other blood product is immediately available.

Autologous transfusion does not decrease the risk of volume overload following transfusion and there is a risk of bacterial contamination during the collection procedure.

There are no studies documenting the metastatic risk with autotransfusion of blood from ruptured neoplasms in veterinary patients.

Blood may be collected from the peritoneal cavity in an aseptic manner by gravity flow using a peritoneal catheter or large gauge needle into a closed collection system or alternatively by slow aspiration into a 60 ml syringe. With the latter technique it is important to avoid high suction pressures and small bore needles to minimise the risk of damage to the red blood cells during collection. Anticoagulant is not generally required as in many cases defibrination will have occurred following contact of the blood with the peritoneal surface. In cases of acute haemorrhage 7ml CPDA-1 should be added to 60 ml of whole blood if blood is not collected directly into a blood collection bag.

Blood collected for autotransfusion should be administered through a blood administration set or in-line blood filter. The risk of transfusion reaction is much reduced in autotransfusion.

Suggested time limits for vascular occlusion in normothermic animals

Descending thoracic aorta	5-10 minutes
Portal triad (hepatic artery, portal vein, common bile duct)	10-15 minutes
Hepatic artery	30 minutes
Hepatic vein	Can be ligated*
Splenic artery and vein	15-20 minutes
Renal artery and vein	30 minutes
Abdominal aorta	30 minutes
Caudal vena cava (caudal to liver)	Can be ligated*
Iliac vessels	Can be ligated*
Femoral vessels	Can be ligated*

*with normal collateral circulation

Selected references

Lisciandro. Abdominal and thoracic focused assessment with sonography for trauma, triage, and monitoring in small animals *JVECC* 21: 104–122 (2011)

Pintar *et al.* Acute Nontraumatic Hemoabdomen in the Dog: A Retrospective Analysis of 39 Cases (1987-2001) *JAVMA 39*:518-522 (2003)

Whittemore *et al.* Nontraumatic rupture of an adrenal gland tumor causing intra-abdominal or retroperitoneal hemorrhage in four dogs *JAVMA* 219, 329-333 (2001)

Herold *et al*. Clinical evaluation and management of hemoperitoneum in dogs *JVECC* 18: 40–53 (2008)

Crowe. Simple autotransfusion in severe hemorrhage: success in 8 of 12 dogs

14: S1–S17 (2004)

Hirst and Adamantos. Autologous blood transfusion following red blood cell salvage for the management of blood loss in 3 dogs with hemoperitoneum *JVECC* 22: 355–360 (2012)

Kellett-Gregory et al. Autologous canine red blood cell transfusion using cell salvage devices *JVECC* accessed online Jan 2013 (2013)

MANAGING MASSES: PRINCIPLES OF SURGICAL ONCOLOGY

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Cancer treatment in cats and dogs is a rapidly changing and evolving discipline. The main treatment options at present are surgery, chemotherapy and radiation therapy.

Surgery is a pivotal tool in the management of many cancers in dogs and cats, and brings with it, more than any other treatment the potential for a cure. This is particularly the case for cancers where the risk of metastasis is low and there is a good chance of removing the entire tumour with histologically 'clean' margins. This lecture will discuss surgical planning for some of the common cancers encountered in general practice.

Introduction

The diagnostic evaluation of a patient with a suspected tumour requires a holistic approach. In simple terms, to treat a tumour in the best possible way we need to know firstly what it is we are dealing with i.e., a cytological or histological diagnosis is required; we need to know if it is 'simply' a local disease process or if it has spread; we need to know if there are an complications arising from the presence of the tumour and if there are any co-morbidities that may alter the patient's prognosis or ability to tolerate treatment.

Preoperative considerations

Signalment

The patient's age, gender, breed and weight are important considerations in the determination of recommendations with regard to surgery. Age is not necessarily a negative prognostic factor, but co-morbidities are more common in geriatric patients.

Staging

Staging is an important preoperative step for all patients presenting with tumours but unfortunately is often neglected – to the detriment of the patient. *Staging of the primary tumour* refers to the visible, palpable or microscopic extent of the tumour mass (see below under Surgical planning). *Staging for systemic metastasis* refers to surveillance for spread of the tumour to other regions. Unfortunately, microscopic metastasis may not be detectable

and are a common cause of treatment failures. Gross evidence of metastasis always significantly alters the prognosis.
Staging helps:

- To define the local, regional and distant extent of disease
- To help determine the optimum treatment for the disease
- To provide a baseline against which response to treatment can be assessed
- To provide prognostic information.

Staging diagnostics usually includes haematology, blood biochemistry, urinalysis, abdominal ultrasonography and thoracic radiography. Aspiration of sentinel lymph nodes may be performed during staging and this information can be invaluable when providing a prognosis. Advanced imaging may be required to assist not only in staging of the primary mass but also staging for systemic metastases (e.g. CT, MRI, nuclear scans etc.).

Pulmonary metastatic disease is common with many neoplastic diseases. In small animals, 3-view thoracic radiography, consisting of the right and left lateral and ventrodorsal or dorsoventral views, is considered the standard for detecting pulmonary metastases. In people, CT is considered the gold standard for detection of pulmonary nodules because CT is able to detect smaller nodules with greater frequency than survey radiography. The two main advantages of CT, compared with thoracic radiography, are elimination of superimposition by thoracic structures and superior contrast resolution. These allow for detection of small nodules that would otherwise go unnoticed. One of the main differences between thoracic CT performed in people and animals is the necessity of general anaesthesia for animals. The requirement of anaesthesia can cause poor aeration of the lung that can be problematic. In cats and dogs, three-view thoracic radiography will likely remain the standard for evaluation of pulmonary metastatic disease because of the lower cost and increased availability, compared with CT.

There are varying opinions with regard to the timing of staging investigations i.e., before or after the results of the histologic type (and grade) of the tumour is known but the default position should be preoperative assessment.

Neoadjuvant therapy

Neoadjuvant therapies (chemotherapy / radiation therapy) can be used prior to surgery in the case of large tumours or tumours located in anatomically challenging areas. An example would be chemotherapy administered prior to resection of a mast cell tumour (MCT). In some cases such therapy may reduce the final size of the surgical wound although there is

currently lively debate about whether the surgical margins should be taken from the size of the original mass or the size of the mass once the neoadjuvant therapy is complete.

Surgical planning

In surgical oncology the first surgical procedure that is performed has the best chance of achieving the best outcome for the patient. This is because of a number of factors including:

- Untreated tumours tend to have more normal local anatomy making intraoperative surgical decisions easier
- Tumours that have recurred may have spread to other previously unaffected tissue planes as result of the original surgery and consequently a larger field of excision may be required that necessitates more advanced reconstruction of the wound
- Failure to remove the tumour *en bloc* may leave behind that part of it that is most likely to be aggressive i.e., the leading edge.

Most mistakes in surgical oncology occur because of failure to identify the type and grade of tumour prior to surgery. This information allows appropriate staging of the disease prior to surgery (particularly when finances are limited). For example, for a subcutaneous mass on lateral thorax of a dog, the distinction between lipoma and other tumours such as haemangiopericytoma or mast cell tumour (MCT) is important because the extent of surgery required to achieve a cure is vastly different. Similarly for a dog presenting with a mass on the rostral mandible it is important to distinguish between a fibrosarcoma which requires a mandibulectomy and an epulid that may be cured using radiation therapy. With a cranial mediastinal mass the distinction between lymphoma and thymoma is important because lymphoma is not a surgical disease and is more appropriately treated with chemotherapy, while thymoma is a surgical disease.

Surgical biopsy prior to surgical excision should not be performed if the biopsy has greater risk than the definitive surgery, especially if the surgical excision is the same regardless of histologic diagnosis e.g. solitary lung, splenic and mammary masses.

Achieving a diagnosis

When diagnosing cancer, there are a number of techniques that may be of value in individual cases. *Impression smears* may be made of cutaneous masses that are exuding and the resulting slides submitted for cytology. Body fluids may be collected and similarly, submitted for cytology.

A *fine needle aspirate biopsy* (FNAB) is where a small needle is passed into the mass to obtain a sample of cells that are transferred to a slide for cytology. This technique is

preferred to attaching the needle to a syringe and aspirating material because the cells can be distorted and often more blood will be collected. When FNAB of masses within the abdominal or thoracic cavities is performed the needle may be attached to a syringe to prevent iatrogenic pneumothorax or pneumoperitoneum. Some tumours such as mammary tumours have a very complex architecture and FNABs are unlikely to yield a meaningful diagnosis. Overall, cytology is reported to have a sensitivity and specificity (89% and 100% respectively) with results of 89% and 98% for neoplasia, and there is good agreement (~90%) between cytology results generated following FNAB and histology in cutaneous and subcutaneous masses. FNAB may be guided by fluoroscopy, ultrasonography or CT and is particularly useful for masses deep within the body wall, neck abdominal or thoracic cavities. There is a small risk of tumour seeding along the needle tract.

A *needle core biopsy* will provide a small cylinder of tissue for histology and is suitable for soft tissue, visceral and thoracic masses. This technique will allow determination of histologic type and grade of tumour in many cases. Disadvantages are that a relatively small amount of tissue is submitted for histology, and may result in an incomplete or inaccurate diagnosis; it may be difficult to assess invasiveness into surrounding tissues. Biopsy tracts must be positioned so they can be excised during definitive diagnosis.

Punch biopsies are effective for cutaneous lesions as well as intraoperatively for biopsies of the liver, spleen, kidney and intestine. When using this technique for subcutaneous masses it is better to incise the skin overlying the mass.

Pinch biopsies (endoscopic biopsies) may be collected during endoscopic examination and may be submitted for histology. Laparoscopy and thoracoscopy will play an increasing role in staging veterinary patients as these techniques become more widely available.

Incisional biopsies provide a small block of tissue for histology. I prefer to sample more than one site if the mass is large, or position the biopsy to include a sample from both the centre of the mass and the periphery so that the degree of local invasion of blood vessels and lymphatics can be assessed. Incisional biopsy allows the determination of histologic type and grade of tumour in addition to the invasiveness. The disadvantages are that general anesthesia is usually required, poorly placed biopsy incisions can have a negative impact on definitive surgical excision and it is usually associated with higher cost to the client.

Excisional biopsies can be performed wherein the entire mass is excised before a diagnosis is made. In this case, the whole sample is the submitted for biopsy. The potential advantage

is that one procedure allows both diagnosis and treatment: the disadvantage is that it is very difficult to choose the appropriate surgical margin and in general excisional biopsies are overused. Ideally excisional biopsy should be used where wide surgical margins may be taken easily and without consequence to the animal, or where a second surgery can easily be performed if margins are found to be inadequate on histology. Excisional biopsies of masses on the extremities and head should be avoided. The use of surgical drains in biopsy sites should be avoided because the drain tract must be considered part of the contaminated field where incomplete margins are found on histology.

Lymph node biopsy is controversial. However, performing an FNAB or excising the sentinel lymph node can aid in staging the disease process.

Samples collected for cytology should be placed onto a labeled slide, smeared and air dried. Any fluid collected can also be submitted in EDTA for the clinical pathologist to prepare their own smears. Specimens for *histology* should be placed in 10% buffered formalin once they have been marked for purposes of alignment. Large samples should be sliced evenly to allow more rapid and complete fixation. It is important that an adequate volume of formalin is used relative to the size of the biopsy to ensure adequate fixing of the tissue (1:10).

When the results of cytology or histology are available the following should be considered:

- Does it provide adequate information with which to make an informed decision with regard to how to proceed with the case?
    - Is this mass neoplastic or is it some other process e.g. pyogranulomatous inflammation etc.
    - What is the histologic type and if relevant grade. Histologic grade has been shown to be prognostic in MCT, soft tissue sarcomas, chondrosarcomas, haemangiosarcomas, and melanomas. Immunohistochemistry may be required to enable the pathologist to confirm a histological diagnosis and / or to determine the cell of origin of poorly differentiated tumours.
    - What is the degree of malignancy i.e., cell morphology, mitotic index etc.
    - Are the surgical margins complete in cases of excisional biopsy?

Surgical excision of tumours

General surgical principles in surgical oncology

The following general principles should be adopted whenever possible:

- If more than one surgical procedure is planned under the same anaesthetic it is good practice to move from clean to more contaminated sites. A new set of drapes, instruments and gloves are nevertheless indicated between procedures.
- Avoid contact with ulcerated or open areas of the tumour with gloves or instruments. Stay sutures can be useful when handling tumours to limit the potential for shedding of cells from the mass during repeated handling.
- The proposed surgical excision including appropriate surgical margins is marked using a sterile skin marker for many cutaneous, subcutaneous and body wall masses. Alternatively, a standard approach is made to the relevant body cavity.
- A scalpel is generally used for skin incision and to cut the margins of the planned excision for cutaneous, subcutaneous and body wall masses.
- The application of equal and opposite tension along the margin of excision helps to identify tissue planes and make dissection easier.
- Meticulous haemostasis of the wound margin and wound bed is essential to limit the development of postoperative haematomas and seromas. Avoid using electrosurgery on the mass (particularly incisional and punch biopsy sites) to be submitted for histology as this may distort the gross and microscopic anatomy.
- The vascular supply to, and venous and lymphatic drainage from the tumour should be ligated as soon as possible during surgery, especially for tumours of ectodermal origin i.e., SCC, MCT where the probability of exfoliation is high.
- In body cavities the tumour should be shielded from surrounding organs using large moist laparotomy pads that may subsequently be discarded.
- If adhesions are encountered between the tumour and local tissues e.g. splenic masses as far as possible consider the adhesions as an extension of the tumour and resect them appropriately.
- Wound lavage may be performed to remove blood clots, foreign material and debris from the wound bed.
- If surgical drains are used, the drain should be located in an area that can be resected during any subsequent surgery, will not compromise subsequent radiation therapy and can be included in a radiation therapy field.
- Surgical gloves and instruments should be changed after mass resection and prior to wound closure that is performed using clean instruments that have not been used during resection of the tumour.
- Avoid tension on the wound edge during reconstruction.

- Removal of a second tumour at a distant site under the same procedure should be performed using a new set of drapes, instruments and gloves. In some cases a new surgical gown may be warranted.

Curative intent surgery (definitive excision)

This refers to the use of surgery as the sole therapy for the tumour. The surgical incision is made to include any previous surgical scars related to the tumour and any biopsy (or drain) tracts. With curative intent surgery, the tumour and a margin of grossly unaffected tissue will be excised. The margin required will depend on the histologic type and grade of tumour. Such surgical margins tend to be referred to in terms of distance i.e., 2 cm etc. but it is also important to consider the biological behaviour of the tumour.

During curative intent surgery the tumour should not be grossly apparent within the surgical field for cutaneous and subcutaneous masses. If there is gross evidence of the tumour in the surgical site, there are two main options. The first is to close the incision and approach the problem again from the beginning taking wider margins (this may include amputation): in this case it is important to repeat the aseptic preparation of the surgical site and the surgeon and use fresh drapes and clean instruments. The second is to complete the surgery as a marginal excision with a plan to perform adjunctive radiotherapy subsequently.

Local or marginal excision

With local or marginal excision (≤1 cm), the tumour with a minimal amount of surrounding tissue is removed. This technique is suitable for tumours such as lipomas, histocytomas, sebaceous adenomas and thyroid adenomas.

Wide local excision

With wide local resection the tumour with a significant (1-3 cm) predetermined amount of surrounding tissue is removed. In many cases, a deep margin is achieved by removal of one tissue plane deep to the last tissue plane the tumour touches e.g. a subcutaneous mass over the lateral aspect the thigh would be removed with the fasia lata (FL). However, if the tumour is attached to the FL, the muscle beneath the FL must be removed. The most effective natural tissue barrier to the spread of cancer are collagen rich and relatively avascular e.g. fascia, tendons, ligaments, cartilage.

Radical local excision

With radical local resection (>3 cm) the tumour with anatomically extensive margins of tissue are excised. Such resections are sometimes variably referred to as *radical resections* e.g. radical chest or abdominal wall resection for the removal of grade II-III soft tissue sarcomas, high grade MCTs or feline injection site sarcomas, or *compartmental or supraradical resections* e.g. pinnectomy for squamous cell carcinoma of the pinna; hemimandibulectomy for mandibular chondrosarcoma or osteosarcoma; limb amputation for appendicular osteosarcoma.

Second surgeries for incomplete margins

When treating patient with incomplete surgical margins, the entire incision must be considered contaminated. In addition, any drain tracts should be considered as contaminated. It may be appropriate to stage the patient again before surgical intervention depending on the length of time since the first surgery. The proposed surgical excision including appropriate surgical margins is marked using a sterile skin. As you can imagine this can result in a surgical wound of significant size and is why it is important to take appropriate surgical margins at the time of the first surgery.

Radiation therapy can be useful in treating recurrence of local disease in these situations as it is effective for treating microscopic disease of many tumour types e.g. soft tissue sarcoma, melanoma, MCT, ceruminous gland adenocarcinoma, many other sarcomas and carcinomas.

Cytoreductive surgery

Cytoreductive surgery may be required because the location of the tumour precludes definitive removal: this may be as a consequence of a previous attempt to remove the mass. Cytoreductive surgery is performed in the knowledge that other adjunctive therapies will be employed e.g. chemotherapy in the case of osteosarcomas and haemangiosarcomas or radiotherapy in the case of soft tissue sarcomas and MCTs, and it may can improve the efficacy of these therapies over that expected if they were used alone.

With cytoreductive surgery it is essential that the owners know that the intended surgery is unlikely to be curative.

Palliative surgery

It is reasonable to perform surgery to improve an animal's quality of life rather the longevity. Such procedures include limb amputation for appendicular osteosarcoma; splenectomy for a

bleeding haemangiosarcoma; tracheostomy for laryngeal tumours; placement of a cystostomy tube for tumours causing urethral obstruction etc. Other adjunctive therapies are often appropriate in this setting.

Adjuvant therapy

The best time to discuss adjuvant therapy is prior to surgery. This allows the owner to have a fuller understanding of the ongoing care of their pet.

Adjuvant chemotherapy is often used following surgery in tumours with a high metastatic risk e.g. appendicular osteosarcoma. Chemotherapy is usually administered after wound healing is complete and in any case within three weeks of surgery. Although it can be administered immediately following completion of surgery, there is the possibility that it may retard wound healing.

Radiation therapy may be administered prior to or after surgery. In general radiation therapy slows wound healing and if used prior to surgery it is imperative to ensure there is no tension on the wound.

Notes page

TRIAGE: PRIORITISATION IN THE FIRST HOUR

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Veterinary emergencies comprise a wide range of clinical problems ranging from those that are imminently life threatening to minor injuries and ailments. Triage is the process of rapidly classifying patients on the basis of their clinical priority allowing identification of those patients that need urgent life saving help and ensuring that this occurs immediately and before patients with less severe problems are dealt with.

All veterinary patients presenting as an emergency should be triaged within 5-10 minutes of arrival at the practice. Veterinary nurses as well as vets should be trained in triage and should work as a team such that they can focus their attention on the patients that need them the most. The process of triage involves synthesising information from the patient's history and initial clinical examination especially an assessment of their major body systems. Severe life threatening emergencies are those that involve significant disturbances in the major body systems where there is the potential for rapid deterioration and death.

The list of minor emergencies is long but includes problems such as minor wounds, mild vomiting or diarrhoea, polydipsia, "ain't doing right", skin lesions and weight bearing lameness. Although these animals are may be presented as emergencies by their owners, the triage process allows their stability to be identified; their full evaluation and treatment can then be delayed until after those patients with life threatening emergencies have been addressed.

Primary survey and major body systems assessment

The first part of the primary survey is aimed at recognition of actual or imminent respiratory or cardiorespiratory arrest so that appropriate therapy can be immediately provided. The familiar mnemonic ABC should be followed:

- Airway
- Breathing
- Circulation

If it appears the animal is undergoing or has recently undergone a cardiorespiratory arrest, CPR should be started and the reader is referred to standard texts for more information on how to perform this.

Level of consciousness should also be noted (conscious animals do not need CPCR, as least not yet!). Other imminently life threatening injuries that may need to be noted and addressed at this stage include:

- Severe arterial haemorrhage
    - Direct pressure should be applied (not tourniquets)
- High likelihood of spinal injury
    - Patient should be moved as little as possible whilst the major body systems examination is being performed and the severity of the injury confirmed.

This initial assessment should take 60 seconds or less to perform. Very few veterinary patients present in this severely compromised (i.e. for all intents and purposes dead!) state. In most patients it is appropriate to move directly on to the next stage of the assessment – major body systems evaluation.

Major body systems evaluation

The three major body systems are considered to be:

- Cardiovascular
- Respiratory
- Neurological

When triaging a patient, **these systems should always be examined first regardless of any other injuries**. Examination of these systems is a priority as dysfunction in any of these systems is potentially life threatening. If a patient dies, it is always the result of failure of one of these systems. Although other injuries may be more obvious, they are very unlikely to kill the patient unless they have a secondary effect on one of the major body systems. For example, consider the dog which has been hit by a car and has a fracture of the femur with a large open wound. Although this injury may appear very dramatic, it will not lead to the dog's death by itself. However the haemorrhage from the fracture site may lead to hypovolaemic shock, cardiovascular system compromise and death. The shock will be detected by examination of the cardiovascular system. Thus the major body systems assessment provides a means of assessing whether the patient's injuries are life threatening. All parameters should be recorded at the time they are measured.

Some authorities include the renal system as a major body system; however the renal system is difficult to assess fully by physical examination and ultimately leads to death of the patient via secondary metabolic disturbances (hyperkalaemia, acidosis) and the development of cardiovascular compromise; as such it should be identified as part of the CV system examination.

Identification of any major body systems abnormalities should lead the veterinary team to start stabilisation for these changes empirically whilst the rest of the physical examination (secondary survey) and other diagnostic tests proceed.

Cardiovascular system assessment

Evaluation of the cardiovascular system is the best way the veterinary team has of rapidly identifying whether a patient is suffering from shock and of giving information about the severity and nature of the shock. It also provides information on any concurrent primary cardiovascular disease. Shock is defined as poor tissue perfusion and if present should be addressed as a matter of urgency.

During the major body systems examination the following points should be noted:

- Heart rate
- Pulse quality (+ the presence of any pulse deficits)
- Mucous membrane colour
- Capillary refill time
- Cardiac auscultation

The first four of these parameters can be termed the “perfusion parameters” – it should be routine that when one of these is noted, all four are noted as the information provided by integrating these findings is much greater than that provided by any one parameter alone. Repeat assessment of the perfusion parameters is the most important (effective and cheap) way of monitoring response to therapy of shock.

Hypovolaemic shock is by far and away the commonest form of shock seen in veterinary patients with less common forms including distributive (septic) shock, cardiogenic shock and obstructive shock.

The degree of hypovolaemia is assessed by performing a careful examination of the cardiovascular system focusing on the perfusion parameters as described above. This has

the added advantage that it not only allows evaluation of hypovolaemia but also of other causes of shock that may co-exist in the same patient.

In uncomplicated hypovolaemic shock, these parameters follow a consistent course in dogs as outlined below.

	Mild	Moderate	Severe
Heart rate	130-150	150-170	170-220
Mucous membrane colour	Normal to pinker than normal	Pale pink	Very pale, grey or muddy
Capillary refill time	Rapid, < 1s	Almost normal 1-2s	Slow (>2s) or absent
Pulse amplitude	Increased	Moderate decrease	Severe decrease
Pulse duration	Mild decrease	Moderate decrease	Severe decrease
Metatarsal pulse	Easily palpable	Just palpable	Absent

Parameters should always be cross referenced, as findings which are inconsistent with the other cardiovascular parameters (according to the table above) strongly suggest that the cause of hypoperfusion is not simply uncomplicated hypovolaemia. This is especially important in the patient with abdominal disease as septic foci and other abnormalities such as hyperkalaemia are commonly on the diagnostic “rule-out” list. Suspected causes of hypoperfusion other than simple hypovolaemia should be aggressively investigated.

Examples of commonly encountered situations where perfusion parameters are inconsistent include:-

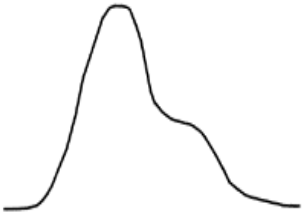
- Tachycardia greater than 220 (250 in cat) is strongly suggestive of a primary cardiac (arrhythmogenic) cause and warrants urgent ECG evaluation
- Tachycardia with poor pulses but bright red mucous membranes and a rapid CRT are strongly indicative of distributive shock (i.e. sepsis/SIRS) and an underlying cause for this should be sought
- Inappropriate bradycardia e.g. heart rate of 80 with poor pulses, slow CRT and pale mucous membranes. Alternative causes of the bradycardia such as hyperkalaemia should be evaluated.

Cats do not appear to follow the pattern of CV changes as outlined in the table above as reliably as dogs. Although in dogs close to death, the heart rate may slow just prior to cardiorespiratory arrest, in cats, bradycardia seems to occur at an earlier stage. A

hypothermic bradycardic cat should raise particular concern. Cats with sepsis commonly present with heart rates in the 120-140bpm range.

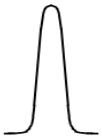
Cardiac auscultation should always be performed to identify any murmurs or other abnormalities. It should be remembered that in an animal with severe hypovolaemia, heart sounds are likely to be quieter than normal and it is easy to miss a murmur. Auscultation should always be repeated once the animal is euvolaemic.

The assessment of pulses is also worth further mention as it is an area muddled by terminology. Careful palpation of both the pulse height and width should allow the clinician to build up a mental picture of the pulse and its volume (see diagrams below). This does take practice (especially in cats) but is worth the effort as with experience repeated and frequent pulse palpation is a great (and cheap!) way of monitoring response to fluid therapy.



Normal pulse profile

Pulse in early hypovolaemia
"bounding", "hyperkinetic"



Pulse in late hypovolaemia
"weak", "thready"

Respiratory system assessment

Initial respiratory system assessment involves an evaluation of whether the patient has any increase in respiratory rate and effort and whether cyanosis (blue coloured mucous membranes) is present. If the patient is showing signs of respiratory distress, oxygen supplementation should be started (usually flow-by) and a more thorough examination of the respiratory system performed. The physical examination should include:

- Observation
    - Rate
    - Pattern – is the dyspnoea inspiratory, expiratory or mixed?
    - Is there any audible noise?
    - Stridor – whistling/squeaking inspiratory noise
    - Stertor – snoring noise

- Auscultation
    - Both sides of the chest should be listened to in multiple sites. Dependent on the size of the patient the chest wall should be split into a "noughts and crosses" board and the pattern of abnormal sounds noted. In a normal animal, lung sounds are bilaterally symmetrical and a little louder cranio-ventrally as there is greater lung mass there.
    - Sounds should be classified as
        - Decreased
        - Increased
        - Harsh – increased noise without specific crackles or wheezes
        - Wheezes – whistling noises typically heard on expiration and suggestive of lower airway disease
        - Crackles – "popping" noises typically heard on inspiration and typical of alveolar disease
    - When deciding whether sounds are increased or decreased, consideration must be given to the level of breathing effort the animal is making. For example a normal dog breathing hard (e.g. post exercise) will have louder lung sounds than it will at rest; a dog with a significant pneumothorax breathing hard may have a similar loudness of lung sounds as a normal dog breathing quietly.
    - Referred upper airway noise can be distinguished from lower airway noise by listening over the trachea.
    - You may need to find a quiet room to listen properly!
- Palpation (e.g. for cranial mediastinal masses) and percussion may be helpful in selected cases.

Further details on respiratory assessment and stabilisation can be found on the notes on Dyspnoea in these Proceedings.

Neurological system assessment

Initial neurological system assessment involves an evaluation of whether neurological disease forms a major part of the animal's presenting complaint. Specifically an evaluation of the patient's gait and mentation should be made – the terms below represent the best ways to describe initial neurological assessment.

Gait abnormalities should be described as:

- Paresis Weakness
- Plegia Paralysis (unable to move)
- Quadriplegia Paralysis of all four limbs
- Paraplegia Paralysis of any two limbs
- Hemiplegia Paralysis of one side of the body
- Hypermetria Exaggerated limb movements

In any paralysed animal it is also very important to note if the animal has deep pain (i.e. can feel its toes even if it cannot move them). This must not be confused with an intact withdrawal response that can be present even with a completely transected spinal cord.

The animal's mentation should be classified as:

- Alert
- Obtunded - mentally dull
- Stuporous - semi-conscious, rouseable by a painful stimulus only
- Coma - unconscious and unable to rouse.

If neurological disease is present, the following neurological features should also be noted at this time so as to provide a baseline for comparison should the patient deteriorate further:

- Pupil size and symmetry
- Presence or absence of pupillary light reflexes
- Presence or absence of palpebral reflex
- Facial asymmetry and any head tilt
- Nystagmus (abnormal flicking eye movements)
- Presence of gag reflex (in stuporous or comatose patients only)
- Anal tone (may be assessed when taking temperature)

Evaluation of these neurological parameters allows an initial assessment of severity of neurological injury to be made – a complete neurological examination should then be performed once the patient is stable to allow more accurate localisation of lesions.

Following triage - the complete physical examination

Once the major body system assessment is complete and empirical stabilisation for any major body system abnormalities has been started, a full physical examination including rectal temperature should be performed. During this part of the examination, a nose-to-tail

approach is recommended to ensure a logical approach and that no areas are missed. The importance of a full physical examination in emergency patients cannot be overemphasised – very often the clue we need to treat the patient adequately and effectively can be found during the physical exam. All physical examination findings should be recorded even if found to be normal.

INFECTIOUS GI DISEASE IN CATS

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INTRODUCTION

Causes of diarrhoea

- Diarrhoea is a common clinical sign in cats. It can be associated with a large number of different disorders, including primary gastrointestinal tract (GIT) diseases and extra-intestinal diseases (such as hyperthyroidism). There are many differentials for both acute and chronic diarrhoea **(Table 1 page 121).** However, cases are not always straightforward, and some differentials can cause both acute and chronic clinical signs.

Diagnosis

- A good **clinical history** is essential in gaining information to tailor the diagnostic and therapeutic approach. Careful questioning will help to indicate the likelihood of extra-intestinal disease, or highlight potential causes for the diarrhoea such as dietary change, environmental factors, infectious agents, drugs or toxins. In multi-cat households, the presence of clinical signs in multiple cats usually indicates an infectious or environmental aetiology. Unfortunately, the duration and frequency of clinical signs can be difficult to determine in cats that have access to the outdoors as can the differentiation between acute and chronic diarrhoea, and small and large intestinal diarrhoea. **Table 2 (p122)** details some of the clinical signs that can help to distinguish between diarrhoea of small intestinal and large intestinal origin. However, in cats, these differences are usually less marked than in dogs, and many of cats with diarrhoea have features of both. Interestingly, some cats with colonic inflammation present with constipation rather than diarrhoea.
- **Physical examination** should help to determine the presence of extra-intestinal disease (e.g. palpable thyroid nodule), or the need for more aggressive investigations or treatment (e.g. dehydration, tachycardia/bradycardia, weak peripheral pulses, abdominal discomfort, palpable abnormalities suggestive of a foreign body, mass or intussusception).

- **Where the investigation goes next is usually dependant on whether or not there is evidence of systemic disease or obvious findings on clinical examination** (e.g. a gut mass requiring surgery).
- **If there is no evidence of systemic disease or obvious findings on clinical examination:**
    - In cases of acute diarrhoea affecting only one cat, with no other associated clinical signs and no detectable abnormalities on physical examination, it is acceptable to instigate non-specific symptomatic therapy (i.e. 12 hour period of starvation followed small frequent meals of an easily digested diet e.g. cooked chicken, Hills i/d, Waltham/Royal Canin Sensitivity Control, or similar) without further investigations. It is also advisable to administer broad-spectrum worming treatments (e.g. Drontal Cat® {praziquantel and pyrantel embonate} Bayer; Milbemax® {milbemycin oxime and praziquantel} Novartis) particularly if recent worming history is poor.
    - If the diarrhoea has become chronic or if more than one animal in the household is affected, then full faecal analysis is the next diagnostic step.
- **Faecal parasitology** - It is important to examine fresh faeces (ideally <2 hours old) as with time any eggs, oocysts or larvae may not stay in a diagnostic state. In order to see the motile trophozoites of *T. foetus* faeces needs to be <1 hour old, ideally <10 minutes. It is also important to check with your laboratory to ascertain which infectious agents are being looked for and by which methodology. For example, sedimentation techniques are preferable for recovery of larvae that may be present in only small numbers, but this can result in more faecal debris, making it difficult to find very small organisms such as coccidia oocysts and Giardia cysts, which can be more readily detected using a floatation technique.
    - **Methods for detecting faecal parasites include:**
    - **Direct smears** - These are used to recover trophozoite stages of parasites (e.g. *T. foetus* and Giardia spp.). The smear should be made with saline as water can rupture the trophozoites. Negative smears are not uncommon if parasite levels are low. Smears can also be stained to look for bacteria.
    - **Faecal flotation** - This method is used to recover nematode ova, coccidia oocysts and Giardia cysts. A centrifugation flotation technique will markedly increase the sensitivity of detecting organisms. Fragile cysts can sometimes become too distorted to identify.
    - The ability to float depends on the organism having a lower specific gravity than the flotation medium. The use of zinc sulphate as the flotation medium

will improve detection of Giardia cysts. The method employed for flotation is very important and preparations need to be examined as soon as possible since any delay will distort delicate oocysts so that they are missed or incorrectly identified.

  - **Antigen detection tests** are available for a number of different organisms (see individual sections).

- **Faecal cultures** are often performed in cats presenting with diarrhoea, however they are usually of limited diagnostic value since potentially enteropathogenic bacteria are frequently isolated from healthy animals, making it difficult to interpret the clinical significance of any positive results. If an infectious aetiology is suspected, which is often based on the history or presence of acute onset haemorrhagic diarrhoea or concurrent evidence of sepsis, then faeces should be cultured for specific pathogens including *Salmonella* spp. *Campylobacter* spp. and *Clostridium perfringens* and *C. difficile.*
- **Where there is evidence of systemic disease or obvious findings on clinical examination** or there is persistence or recurrence of chronic diarrhoea despite dietary trial (see below) and exclusion of gastro-intestinal infectious agents:
  - Further investigations should include a **complete blood count and serum biochemistry** to investigate the possibility of underlying systemic diseases, and to look for potential consequences of intestinal disease such as anaemia from enteric blood loss, and hypoproteinaemia resulting from severe infiltrative diseases (e.g. intestinal lymphoma, severe inflammatory bowel disease (IBD)). In elderly cats serum thyroxine should also be measured to exclude hyperthyroidism. Serological screening for FeLV and FIV should also be performed. Serum should be sent for assessment of **serum folate and cobalamin** (B_{12}) levels and **feline trypsin-like immunoreactivity** (fTLI) and **feline pancreatic lipase immunoreactivity** (fPLI) activity.
  - **Survey abdominal radiographs** are of limited benefit in the majority of cats with diarrhoea but are important to perform if a foreign body, intussusception or mass is suspected.
  - **Abdominal ultrasonography** is useful for detecting masses, intestinal wall thickening and/or loss of layering, intussusceptions, mesenteric lymphadenopathy, liver disease and pancreatic involvement.
  - Occasionally, the use of **contrast radiography**, for example administration of barium impregnated polyspheres (BIPS) may be useful for demonstrating partial obstructions or motility disorders.
  - If no diagnosis has been made at this point (and the animal is not extremely

ill) an acceptable approach is be to **perform a dietary trial**. A large number (30-50%) of cats with chronic diarrhoea will respond well to an exclusion diet, e.g. boiled chicken or a hydrolyzed diet and water only for 2-3 weeks.

- If a diagnosis has not yet been reached following the above investigations, then gastrointestinal biopsies may be indicated. If the decision for **gastrointestinal biopsies** has been made, the next decision to make is whether endoscopic or full thickness biopsies are to be taken. Endoscopic biopsies are generally most appropriate initially, although full thickness biopsies may be needed at a later stage if a diagnosis is not reached and there is poor response to symptomatic therapy. If ultrasonographic abnormalities have been detected (e.g. mesenteric lymphadenopathy, focal intestinal thickenings, abnormalities of the submucosa/muscularis) then full thickness biopsies are more appropriate. When endoscopy is performed, both upper and lower endoscopy should ideally be performed, since small intestinal disease will often be present even if clinical signs are more suggestive of diarrhoea of large intestinal origin. Unfortunately, endoscopy misses disease located in the distal small intestine, which is a common site for IBD and intestinal lymphoma.

Treatment

- The first decision to make regarding treatment is whether or not the cat requires hospitalisation and more intensive treatment and monitoring. If diarrhoea is acute in onset and significant abnormalities are detected on physical examination, then hospitalisation may be required. In addition, very young animals, particularly those with profuse watery diarrhoea can very quickly become severely dehydrated, so it is preferable to hospitalize and administer intravenous fluid therapy early in the course of disease.
- A **12-24 hour period of starvation**, followed by the introduction of small frequent meals of highly digestible food (e.g. cooked chicken) is the commonest treatment option for simple acute diarrhoea where the cat is otherwise well. Oral water intake should be maintained and oral electrolyte/glutamine supplementation may also be beneficial.
- **Probiotics** are used to try to repopulate the intestine with beneficial bacteria e.g. *Bifidobacter, Lactobacilli* and *Enterococcus faecium.* **Prebiotics** (e.g. fructo-oligosaccarides [FOS] and inulin) are used to try to change the substrate of the intestinal flora and promote the growth of more beneficial populations. Several of

these types of products are now commercially available for use in dogs and cats. Whilst there is data to support their use, more studies are needed. They may be particularly useful when diarrhoea is caused by diet change (e.g. associated with rehoming, hunting, or introduction of a prescription diet), stress (rescue kennels, boarding cattery, or moving house), or when diarrhoea results from drug administration (e.g. antibiotics, chemotherapy).

- In critically ill or anorexic patients **microenteral nutrition** may be beneficial. This delivers small amounts of water, electrolytes and readily absorbable nutrients (glucose, amino acids and small peptides) to the intestinal tract to help preserve the integrity of the intestinal mucosal barrier and hence help to prevent bacterial translocation. Solutions containing glutamine may be particularly useful for this purpose (e.g. Glutalyte® Norbrook Companion Animal Range), since this is the preferred energy source for the intestinal epithelium.
- **Antibiotics** are not indicated unless a specific bacterial agent had been identified or if there is evidence of intestinal ulceration (i.e. haemorrhage), in which case antibiotics should be given to reduce the risk of septicaemia as a result of bacterial translocation through the damaged mucosal barrier.
- **Corticosteroids** are contraindicated, and in many cases (e.g. infectious diseases, pre-existing mucosal damage) will prove detrimental. They are generally only indicated where IBD is very strongly suspected and preferably when it has been confirmed on histopathology. Even then, many cats with mild to moderate IBD can be controlled with dietary therapy alone or, possibly, with the addition of metronidazole or tylosin.
- **Sulphasalazine** is a 5-aminosalicylic acid derivative that is frequently used as a treatment for acute colitis in dogs. Suphasalazine is a prodrug that in dogs is cleaved by colonic bacteria, releasing the active drug which acts locally in high concentrations in the colon as an anti inflammatory agent. However, in cats salicylates are readily absorbed (particularly if there is any small intestinal disease) and can induce toxicity so they should only be used with great caution.
- **Adsorbents** are frequently administered in acute diarrhoea to bind bacteria and toxins, to protect the intestinal mucosa, and potentially for an antisecretory effect. Commercially marketed adsorbents include kaolin, pectin and montmorillonite. In most products the adsorbent is combined with other agents e.g. Promax ® (montmorillonate, probiotics and glutamine), Diarsanyl® (montmorillonate, electrolytes), Kaobiotic® (kaolin and neomycin).
- **Motility modifiers** - anticholinergics (e.g. hysocine) are not recommended as they

can promote or exacerbate ileus. Opiates (e.g. diphenoxylate, loperamide) predominantly reduce intestinal secretions and promote absorption, in addition to stimulating segmental intestinal contractions, and so can on occasion be useful in the short-term management of acute diarrhoea. However, their use is not advisable in young kittens and they should not be used if an obstructive or infectious aetiology has not been excluded.

- **Treating and controlling infectious causes of diarrhoea** (general guidelines, but using *Giardia* as an example):
    - Treat all affected and in-contact animals
    - Use the least toxic drug options first; e.g. the most commonly used treatments for *Giardia* are fendendazole and/or metronidazole. Fenbendazole is recommended as a first line treatment, and if treatment fails with this alone, metronidazole can be added in.
    - Healthy uninfected cats should be separated from symptomatic cats and from infected but asymptomatic cats
    - Any new animals should also be kept separately
    - Faecal debris should be removed from any animals with diarrhoea
    - Litter trays should be cleaned and disinfected daily with quaternary ammonium compounds to kill cysts, or bleach (diluted 1:32).
    - Where possible, housing should also be treated with quaternary ammonium compounds
    - If there is a vaccine available, consider using it.

INTESTINAL INFECTIONS

Pathogenesis and clinical signs

- In most cases, for an infectious agent to cause diarrhoea the '**Epidemiological Triad'** involving **infectious agent** (number of organisms, presence of virulence factors, etc.), **host animal** (age, immune status, state of its GIT, stress, etc.) and **environmental pressure** (housing conditions, stocking density, diet changes, antibiotics, recent surgery, etc.) all come into play.
- **Asymptomatic carriage is common and disease is seen most frequently in young animals** (<6 months), especially if they are **group housed, stressed** (e.g. rescue or stray populations), and/or **fed raw food**. Infectious diarrhoea is therefore seen most commonly in young or immuno-compromised animals, or those housed in large numbers of unhygienic conditions. The prevalence of infections may also vary with geographic location, whether or not the cat is an indoor or outdoor animal, and

whether or not it hunts and eats its prey. Episodes of disease may be seen in kittens or cats of similar ages, and may be preceded by a stressful event such as an environment change, diet change, addition of new cats, or weaning, etc.

- Clinical signs vary but typically include **diarrhoea and vomiting**, and in some cases **anorexia** may also be present.
- **Asymptomatic carriage means potentially harmful organisms** can be found in many apparently healthy animals. For example, depending on the population investigated and the sensitivity of the test used ***Cryptosporidium*** **can be found in up to 12% of healthy cats,** ***Giardia*** **in up to 14%,** ***Salmonella*** **in up to 18% and** ***Campylobacter*** **in up to 45%.**
    - For infection to arise a number of steps have to occur:
    - Faecal contamination of environment, food, or water
    - Ingestion by host
    - Ability to overcome host defences, such as secretions (gastric acid, digestive enzymes), GI motility, mechanical barriers (mucus, enterocytes, tight junctions), immunoglobulins (IgA), competition with resident flora, and immune responses
    - Reach preferred site and attach to or invade cells
- **A number of different situations can lead to compromise of the host defences and therefore predispose to the development of infection, these include:**
    - Reduced digestive secretions, and/or reduced ability to digest and/or absorb nutrients e.g. with IBD or exocrine pancreatic insufficiency (EPI)
    - Motility disorders e.g. ileus
    - Presence of enterotoxins
    - Increased gut permeability or damage to enterocyte tight junctions
    - Reduced levels of immunoglobulins, especially IgA
    - Administration of antibiotics (which leads to alterations in resident microflora populations)
    - Immunosuppression can result from chemotherapy, corticosteroids, FeLV, FIV, uraemia, malnutrition, etc.
    - Presence of foreign body, obstruction, or abrasions

- Many infectious causes of diarrhoea are **potentially zoonotic** so when any animal presents with diarrhoea owners should be given appropriate advice regarding hygiene and told to prevent contact between the affected animal and any immuno-compromised individuals.

- Intestinal infections that can affect cats and may cause clinical signs such as weight loss and chronic diarrhoea and/or vomiting include **viruses** (parvovirus, enteric coronavirus [FCoV], intestinal FIP, FIV, FeLV, Toravirus, Astrovirus, Rotavirus), **bacteria** (*Campylobacter, Salmonella, Clostridium, E. coli, Yersinia, Mycobacteria*), **protozoans** (coccidians *Isospora felis*, *I. Rivolta*, *Cryptosporidium parvum* and *Cryptosporidium felis* and flagellates *Giardia lamblia* and *Tritrichomonas foetus*), **nematodes** (large ascarid roundworms *Toxocara cati* and *Toxascaris leonina*, and hookworms *Ancylostoma braziliense, A. tubaeforme* and *Uncinaria stenocephala*), and **cestodes** (tapeworms *Dipylidium caninum* and *Taenia taeninaeformis*).
- **Parvovirus, *T. cati* and *Isospora spp.* are the most common infections of kittens. *Campylobacter, Salmonella, G. lamblia, D. caninum* and *T. taeninaeformis* are the most common infections of adult cats.**
- **Feline panleukopenia** (FPV) is a highly contagious parvovirus infection of cats that can cause severe acute diarrhoea and death. It is very similar to canine parvovirus in terms of its pathogenicity and gastro-intestinal signs. Mortality in young kittens is very high, however it rarely causes chronic disease, and the wide spread use of vaccination has reduced the prevalence of FPV-associated diarrhoea. *(For more extensive notes on this infection please contact the author).*
- **Feline enteric coronavirus** (FCoV) is a ubiquitous virus which may cause self-limiting diarrhoea in infected kittens, and occasionally causes chronic disease in susceptible individuals. It is believed that mutation of enteric FCoV can result in the development of feline infectious peritonitis (FIP). Although diarrhoea is not usually a feature of FIP, isolated intestinal granulomas resulting in diarrhoea have been reported. *(For more extensive notes on this infection please contact the author).*
- **FIV** is often associated with chronic diarrhoea, but immunosuppression following infection also increases susceptibility to other infectious agents that may result in acute diarrhoea. **FeLV** has been associated with a fatal peracute enterocolitis and lymphocytic ileitis. *(For more extensive notes on these infections please contact the author).*
- **Toravirus** has been linked with 'third eye-lid prolapse and diarrhoea syndrome' where the diarrhoea is usually self limiting but can persist for weeks to months.
- Gastrointestinal infections with **potentially pathogenic bacteria** usually result in acute diarrhoea. However, they are also often isolated from animals with chronic diarrhoea, and in these situations the significance of infection is unclear, as they may also be isolated from clinically healthy animals (see above).
- Most ***Campylobacter*** *(C. jejuni* and *C. upsaliensis)* infections are asymptomatic, with

the organism being isolated in up to 45% of healthy cats and dogs. Clinical disease is usually restricted to young, parasitized, or immuno-compromised animals. More severe disease may be encountered if there is concurrent infection with other bacteria, viruses or parasites.

- Most ***Salmonella*** infections are asymptomatic, with up to 18% of healthy cats carrying the bacteria. Bacterial translocation from the intestinal tract may occur resulting in septicaemia and endotoxaemia. Infected cats may show vague mild signs without any evidence of gastrointestinal disease, but development of an acute febrile illness with or without diarrhoea has also been reported.
- Enterotoxin-producing ***Clostridium perfringens*** have been associated with both acute and chronic diarrhoea; however enterotoxin has also been detected in faeces of healthy animals so its role in causing diarrhoea is controversial. The clinical significance of ***C. difficile*** is also unknown but it has been incriminated as a cause of chronic diarrhoea in some cases.
- Many strains of ***E.coli*** are normal intestinal commensals, however some strains may result in acute diarrhoea, whilst other strains may caused chronic diarrhoea. Enterotoxigenic strains of *E.coli* have been associated with acute diarrhoea, but the identification of pathogenic strains requires specialised assays, so the significance of positive isolation alone is debatable.
- ***Yersinia pseudotuberculosis*** infection may occur in cats following ingestion of infected rodents or birds. Severe diarrhoea, weight loss, jaundice and mesenteric lymphadenopathy follows and the disease is often fatal. *Y. enterocolitica* can be a commensal of the gastrointestinal tract (GIT) but is rarely a cause of acute colitis.
- ***Mycobacterium*** spp. infection, especially *M. avium,* may occasionally involve the GIT resulting in diarrhoea, vomiting, mesenteric lymphadenopathy and weight loss. *(For more extensive notes on this infection please contact the author).*
- Adult ***T. cati*** and ***T. leonina*** live in the **small intestine** of cats. Eggs are passed into the environment with the faeces to be ingested by other cats. With *T. cati* these **migrate via the liver and lungs** to the small intestine. With *T. leonina* they **mature in the wall of the small intestine.**
    - While *T. cati* larvae can be **transmitted lactationally** to kittens, prenatal infection does not occur with either parasite.
    - Rodents can act as transport hosts.
    - *T. cati* (but not *T. leonine*a) can cause ***visceral larva migrans*** **in humans**.
    - Clinical signs are uncommon and in adult cats infections are usually subclinical. Young kittens with very heavy burdens can develop small bowel

diarrhoea, vomiting, abdominal discomfort, a pot bellied appearance, poor coat condition, a failure to thrive and, rarely, intestinal obstruction/intussusception.

- The prevalence of **hookworms** varies. ***Ancylostoma spp.*** **prefer warm, humid climates**, while ***U. stenocephala*** can live in **colder climates**. Severe hookworm infections are usually seen in warm, moist climates.
    - Adult hookworms live in the **small intestine** of cats. Eggs are passed into the environment with the faeces and then hatch. They can be ingested by other cats or infect them by **skin penetration**.
    - Lactational and prenatal infections do not occur.
    - Rodents can act as transport hosts.
    - Infection can cause ***cutaneous larva migrans*** **in humans.**
    - Infections cause less disease in cats than dogs, and most disease is seen in young adult cats that live in poorly-cleaned, crowded conditions. A heavy infection can cause weight loss, poor coat condition and melaena.
- Adult **tapeworms** live in the **small intestine** and both *D. caninum* and *T. taeninaeformis* require an **intermediate host** to complete their life cycle. Gravid proglottids containing many eggs are released from the adult worms. They may rupture within the intestines, or remain intact and pass out in the faeces and be seen around the cat's anus.
    - **Dog and cat fleas and lice** are the **intermediate hosts** for ***D. caninum***.
    - Rodents are the intermediate hosts for *T. taeninaeformis*.
    - Infections are usually subclinical, but heavy infections can occasionally cause anal pruritus, vomiting, diarrhoea, weight loss and, occasionally, intestinal obstruction.
- ***Isospora spp****.* are the commonest coccidial agents of cats *(I. neorivolta, I. felis, I. rivolta*). Up to 2% of feline faecal samples are infected. Infection occurs following ingestion of oocysts or paratenic hosts. They live in **intestine** of cats and shed oocysts into the environment via the faeces. Sporulated oocysts can be directly ingested by other cats.
    - Rodents harbouring cyst stages can act as transport hosts.
    - Infection and clinical disease (diarrhoea) is seen most commonly in kittens kept in large unhygienic groups.
    - Infection rarely causes clinical disease in adult cats unless they are stressed or immuno-compromised. In kittens, infections can range from subclinical through to severe haemorrhagic diarrhoea.

- ***Cryptosporidium parvum*** and ***C. felis*** live in the **small intestine** and sheds oocysts that can either break open to release sporozoites into the intestine resulting in chronic infection or be passed out into the environment where they can remain viable for many months.
    - In cats ***C. felis*** is the species seen most commonly.
    - Infection can cause severe disease in immuno-deficient animals and occasionally humans.
    - Infections are usually subclinical, but can also cause acute or chronic small bowel diarrhoea, or result in lymphocytic-plasmacytic duodenitis. This infection can be found in 4-8% of cat faecal samples, and 1.5-2% of canine samples.
- In cats, ***G. lamblia*** lives in the **jejunum and ileum**. Environmentally resistant cysts are shed in faeces, contaminate drinking water or food, and are then ingested by other cats.
    - Infection and clinical disease is seen most commonly in **cats kept in large unhygienic groups, and in cats that are <4 years old.**
    - The majority of infections are subclinical, but clinical signs can vary from acute to chronic small or large intestinal diarrhoea. The severity of disease is often determined by the presence of other intestinal pathogens or other concurrent intestinal disease. This infection can be found in up to 14% of feline faecal samples, and up to 39% of canine samples.
    - *Giardia spp.* show a degree of host-specificity. In cats the most commonly seen Assamblages are AI and F, with B, D and E being seen less commonly; AI is a common cause of disease in humans.
    - Infections can be subclinical, or cause acute, chronic or episodic small bowel diarrhoea, where intestinal malabsorption may result in mucoid, soft, foul-smelling faeces.
    - *Giardia* spp. cysts are most often found in the faeces of cats that a history of chronic diarrhoea (>2 weeks), and that are also found to have concurrent *Cryptosporidium spp.* oocysts and/or coccidial oocysts.
- In cats, ***Tritrichomonas foetus*** lives in the **colon** and sheds flagellated protozoa into the faeces.
    - Infection and clinical disease is seen most commonly in **cats kept in large unhygienic groups.** Although cats of all ages can be affected with diarrhoea, it is most commonly seen in young cats and kittens, the majority being under 12 months of age. Most of the affected cats have come from rescue shelters

and pedigree breeding colonies.

- Infection is presumably spread between cats by close and direct contact. There has been no evidence of spread from other species, or spread via food or water. Studies have shown that 31% of cats at a cat show in the USA were infected with this organism, compared to 14-31% in the UK; suggesting that this may be an important, common, and previously unrecognised cause of diarrhoea in cats.
- Infections can be subclinical, or result in chronic large bowel diarrhoea, with increased frequency of defecation, semi-formed to liquid faeces, and sometimes fresh blood or mucus in the faeces. With severe diarrhoea the anus may become inflamed and painful, and in some cases the cats may develop faecal incontinence. Although the diarrhoea may be persistent and severe, most affected cats are otherwise well, and do not show significant weight loss. *(For more extensive notes on this infection please contact the author).*

Diagnosis

- Intestinal infection should be **suspected** in any cat with diarrhoea, but especially those coming from a **poorly cleaned multi-animal environment in a geographic region with a high prevalence of infection.**
- Unfortunately, it can be very difficult to prove that diarrhoea in a particular cat is due to a specific organism. This is because **infectious agents can be present in normal animals, parasite eggs, protozoa, and bacteria are often excreted intermittently, some bacteria are difficult to culture, many tests are not validated in cats, and clinical signs of disease may spontaneously resolve before the test results come back.**
- Diagnosis is usually on the basis of faecal analysis and/or culture as these generally non-systemic infections rarely cause systemic changes. However, haematology may reveal an eosinophilia and, in severe cases, low serum proteins may be found on serum biochemistry.
- For more about faecal examination and culture see the sections in the Introduction. With the exception of tapeworm segments, most intestinal parasites are not noticed by owners in their cat's faeces.
- Faecal floatation techniques are used to diagnose most intestinal parasites (round worms, hook worms and *Isospora spp.).*

- **Special techniques may be necessary for some parasites:**
    - A few have been validated for cats, and they have variable sensitivity and specificity. While some PCR-based tests are now available, which are reliable, specific and sensitive, they are often expensive.
    - Faecal Parvovirus antigen ELISA is available for canine parvovirus and also detects feline parvovirus as they are antigenically very similar. Vaccination status should be considered when interpreting these tests as animals will shed virus particles following vaccination with live vaccine and so can lead to result in false positive results. A faecal Parvovirus PCR is available at a limited number of specialist centres.
    - Faecal Clostridium enterotoxin ELISA assays are available.
    - *C. parvum* and *C. felis* – special stains may be needed and faecal antigen tests are available e.g. Immunofluorescent antibody assay (IFA).
    - Giardia cysts are only excreted intermittently so it may be necessary to examine up to five fresh samples in order to detect them using direct saline faecal smears. Faecal antigen tests, including ELISA and IFA, are more sensitive and do not have to rely on the expertise of a technician in identifying the cysts.
    - *T. foetus.* – can be diagnosed by direct saline faecal smears, the 'In Pouch' culture system, or PCR.
- A number of laboratories now run PCR panels that look for a number of different pathogens; e.g. In the UK and US, Idexx now run a **Feline Diarrhoea PCR Panel** which looks for: *T. foetus,* Toxoplasmosis, FPV, FCoV, *Giardia, Cryptosporidium, Salmonella* and *Clostridium perfringes.*
- In some cases, rather than confirming the presence of an infection with faecal tests, a **therapeutic trial** with a suitable drug may be considered.
- Since some parasites can be transmitted lactationally *(T. cati),* and infections are frequently more severe in young cats, **all young cats should be evaluated for intestinal parasites or treated against the common parasites of the region**.

Differential diagnoses

- Differential diagnoses include most of the other causes of weight loss with a good appetite. **Inadequate nutrition** becomes the most likely differential when there are no signs other than weight loss. However, the variable presence of **gastrointestinal signs is more suggestive of some of the malassimilation syndromes such as IBD, EPI, or early alimentary lymphoma.**

Treatment

- The treatment of choice for **Campylobacter is erythromycin** (10-20 mg/kg q 8-12 hours PO), but vomiting is a common side effect which precludes its use in some cases. **Marbofloxacin** (2 mg/kg q 24 hours PO), or other fluoroquinolones, are appropriate alternatives in these cases.
- **Metronidazole** can be used in cases where **Clostridium** has been isolated (8-10 mg/kg q 12 hours PO).
- Antibiotics are contraindicated in cats where **Salmonella** has been isolated and the cat is asymptomatic or has diarrhoea without evidence of bacteremia. This is because treatment can promote bacterial resistance and a carrier state. If severe haemorrhagic diarrhoea, evidence of bacteraemia, or sepsis is present then parenteral antibiotics such as fluoroquinolones, should be initiated. The parenteral route should be used, rather than enteral route.
- **Roundworms and hookworms** may be treated with **pyrantel pamoate** (20 mg/kg/day - 2 doses need to be given 2-3 weeks apart) or **fenbendazole** (20-50 mg/kg/day usually given for 3-5 days, then repeated 2-3 weeks later) which are both safe and effective in cats.
- **Tapeworms** may be treated with **praziquantel** (3.5-7.5 mg/kg SC, PO) or **epsiprantel** (2.75 mg/kg PO). One dose is effective against *D. caninum* and *T. taeninaeformis*.
- ***Isospora spp.*** may be treated with **trimethoprim/sulphonamide** (15 mg/kg q 12 hours PO for 10-14 days), plus improved sanitation. A new treatment is toltrazuril (which is designed to treat coccidian in birds) but can also be used in cats and kittens (5-20mg/kg PO once or 7mg/kg PO for 2 days; vomiting is an occasional side effect). Ponazuril works in a very similar manner.
- ***C. parvum*** can be difficult to treat, and often relies on treating the underlying disease or associated infections; uncomplicated cases may resolve spontaneously. Consider using probiotics and adding fibre to the diet. Consider using **tylosin** (10-15 mg/kg q 12 hours PO for 28 days, but may need higher doses*), **azithromycin** (15 mg/kg q 12 hours PO for 10 days) may be effective, **paromycin** (125-165 mg/kg q 12 hours PO for 5 days) – but this should not be used in cats with bloody faeces as absorption can result in acute kidney failure and deafness, or nitazoxanide (25mg/kg q12h PO for 7-28d) is used in humans and is being investigated in cats; but it may cause vomiting.
- ***Giardia spp.*** may be treated with **fenbendazole** (20-50 mg/kg/day PO for 5 days) – safe except for rare cases of idiopathic hypersensitivity, but effective perhaps only

50% of the time, or **metronidazole** (10-25 mg/kg q12-24 hours PO for 5-7 days). Repeated treatment may be needed.

- ***T. foetus*** may be treated with **ronidazole** (10-30 mg/kg q 24 hours PO for 14 days). *(More information about T. foetus infection in cats is available on the FAB website www.fabcats.org.*

***Tylan**

Tylan soluble powder (for use in calves, chickens, etc… from Dunlops – it is very bitter)

Size 2 gelatine capsule (from Dunlops), large end level filled = 200mg

Size 4 gelatin capsule (from Dunlops), large end 3/4 filled = 100mg

Size 4 gelatin capsule (from Dunlops), large end 1/2 filled = 75mg

Usual dose 7-11mg/kg po q6-8h

Prevention

- Severe infections can usually be prevented by having a good preventative worming policy, giving prompt and effective treatment to any animals found to be infected or carrying these organisms, improving sanitation, and reducing stocking densities.

Prognosis

- If given the correct treatment, the prognosis for full recovery is usually good.
- Severe infections can occasionally result in permanent intestinal damage and chronic clinical signs.
- Resistant *C. parvum*, *Giardia spp.* and/or *T. foetus* infections can occasionally result in chronic disease.

Table 1: Causes of diarrhoea in cats

Intestinal Diseases:

- Dietary related (sudden change in diet, dietary indiscretion, intolerance, hypersensitivity)
- Infectious diseases
    - *Viruses* – parvovirus, enteric coronavirus, intestinal FIP, FIV, FeLV, Toravirus ('third eye-lid prolapse and diarrhoea syndrome'), Astrovirus, Rotavirus
    - *Bacteria* – Campylobacter, Salmonella, Clostridium, *E.coli*, Yersinia, Mycobacteria
    - *Protozoa* – coccidia (Isospora, Cryptosporidia), Giardia, *Tritrichomonas foetus*
    - *Helminths* – roundworm (Toxocara), tapeworm (Dipylidium), whipworm (Trichuris)

- Inflammatory bowel disease (IBD) e.g. plasmacytic-lymphocytic, eosinophilic, suppurative, etc.
- Neoplasia e.g. lymphoma, adenocarcinoma
- Intussusception
- Partial intestinal obstruction
- Short bowel syndrome (usually post surgical)
- Adynamic ileus and intestinal pseudo-obstruction

Extra-intestinal diseases:

- Polysystemic infection – bacteraemia, septicaemia, FeLV, FIP, FIV
- Liver disease
- Pancreatic disease
- Endocrine disease e.g. hyperthyroidism
- Renal disease
- Miscellaneous, such as toxaemias (e.g. peritonitis) or various toxins and drugs

Table 2: **Features that can help differentiate large and small intestinal diarrhoea**

	Small Intestinal disease	Large Intestinal disease
Faecal volume	Increases	Normal or decreased
Presence of mucus	Rarely present	Common
Melaena	Maybe present	Absent
Haematochezia	Absent except in acute haemorrhagic enteropathy	Can be present
Steatorrhoea	Present with malabsorption	Absent
Urgency in defaecating	Absent unless in acute	Usually but not invariably
Tenesmus	Absent	Frequent
Frequency of defaecation	2-3 times normal	>3 times normal
Dyschezia	Absent	Present in distal colon or rectal disease
Flatulence/ Borborygmi	May occur	Absent
Vomiting	Common in acute infectious disorders or as part of triaditis	May occur in 30-35% of acute colitis
Appetite	Usually normal but can be reduced	Usually remains normal
Weight loss	Usually only occurs as disease becomes chronic	Unusual
Faecal incontinence	Rare only associated with severe enteritis and large amounts of watery diarrhoea	May be present
Perianal Irritation	Absent	Occasionally present

Notes page

RENAL DISEASE IN CATS

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INTRODUCTION

Chronic kidney disease (CKD) is a common and important cause of morbidity and mortality in cats. It can be seen in cats of all ages, but occurs most commonly as an acquired disease of middle-aged to older cats. CKD is believed to be two to three times more common in cats than dogs. Increasingly, with the routine use of plasma biochemistry testing, more and more cats are being found to be azotaemic. Azotaemia refers to increased levels of plasma creatinine and urea. These begin to rise when the glomerular filtration rate (GFR) is no longer able to maintain normal excretory function. Unfortunately, the relationship between plasma creatinine and GFR is curvilinear. So the GFR may decrease rapidly in the early stages of CKD without incurring increases in creatinine, then, later on even small reductions in GFR may cause dramatic increases in creatinine. It is generally accepted that more than three-quarters of functioning renal tissue must be lost before azotaemia becomes apparent (Finco et al 1995; Squires 1996).

The classification of CKD into clinical stages can be very helpful. However, it is important to look at all of the abnormal clinical findings, and not concentrate on the urea and creatinine levels alone. That said, cats with CKD can usually be divided into:

- IRIS Stage I: Chronic kidney insufficiency (nonazotaemia renal failure) – ~67% of kidney function lost. Urine concentration ability is reduced, but urea and creatinine levels are still within normal limits. Serum creatinine concentration <140 umol/l (1.6 mg/dl). Indications of renal compromise may include abnormal renal palpation, imaging, and/or proteinuria of renal origin.
- IRIS Stage II: Azotaemia kidney failure – ~75% of kidney function lost. Urea and creatinine levels are raised. Serum creatinine concentration 140-250 umol/l (1.6-28 mg/dl). Phosphate levels are usually raised. However, initially the cat is not necessarily ill.
- IRIS Stage III: Uraemia kidney failure – >90% of kidney function lost. The urea, creatinine and other nitrogenous waste products are raised. Serum creatinine

concentration 250-440 umol/l (2.8-5.0 mg/dl). Synthesis of calcitriol and erythropoietin are usually impaired. The cat is systemically ill.

- IRIS Stage IV: End-stage kidney failure – >95% of kidney function lost. Serum creatinine concentration >440 umol/l (>5.0 mg/dl). Excessive ammonium generation with kidney. Life is not sustainable without dialysis or renal transplant.
- Further classification can then be made on the basis of presence of proteinuria and/or systemic hypertension (see later).

In 1998, with the support of Novartis Animal Health, the International Renal Interest Society (IRIS) was formed. It was established in recognition of the importance of renal disease in small animal practice, and aims to help veterinary practitioners to better understand, diagnose and treat renal disease in cats and dogs. (For more information on IRIS visit: www.iris-kidney.com).

This paper will very briefly discuss the aetiology, clinical signs and diagnosis of feline CKD, after which it will focus more deeply on possible long-term management options.

AETIOLOGY

While the underlying cause of most cases of feline CKD remains obscure, a number of different aetiologies have been documented (Table 1, page 142). The most common histopathological finding is of chronic interstitial nephritis (Lucke 1968), however, the cause of this is uncertain, but in some cases may involve chronic pyelonephritis or glomerulonephritis.

CLINICAL SIGNS

The clinical signs of cats with CKD are often non-specific, with dehydration, anorexia, lethargy and depression being seen most commonly (see Table 2, page 143) (Lulich et al 1992; Krawiec and Gelberg 1989; DiBartola et al 1987; Elliott and Barber 1998). Polyuria and polydipsia are seen less commonly than in the dog. This may result in part from poor recognition on the part of the owners, but also because cats with CKD often retain some degree of urine-concentrating ability. The presence of small kidneys cannot be relied on as an indicator of CKD as many cats have enlarged kidneys due renal lymphoma, polycystic kidney disease or peri-renal pseudocysts. Other manifestations of uraemia in cats include vomiting (due to uraemic gastritis, hypergastrinaemia, or the central effects of uraemia toxins), pale mucous membranes (due to anaemia – see below), and hypertensive retinopathy (see below).

DIAGNOSIS

Diagnosis of CKD is usually based on clinical signs plus the presence of azotaemia and inappropriately concentrated urine. However, because cats often retain some ability to concentrate their urine it is not necessary to document isosthenuria (SG ~ 1.010). In fact, while most cats with CKD fail to concentrate their urine above 1.035, isosthenuria is seen in only ~60% of cases (Barber and Elliott 1998; Lulich et al 1992; DiBartola et al 1987; Elliott and Barber 1998). It is important to know what the cat is eating as normal cats fed on a dry diet typically have a urine SG>1.050; therefore, a SG of 1.040 in a cat fed only on dry food would be abnormal.

Azotaemia is not always caused by CKD, and a single blood sample showing an increase in creatinine and/or urea should not be over-interpreted. In general, serum creatinine concentrations reflect renal function more accurately than urea concentrations. However, serum creatinine may also be increased because of dehydration (pre-renal azotaemia), intestinal absorption of exogenous creatinine (e.g. a cooked meat diet), catabolic conditions (e.g. starvation, fever, excessive exercise, infection, necrosis), or a marked increase in body muscle mass (IRIS 2000). Urea may be increased because of dehydration, intestinal absorption of exogenous protein (e.g. a high protein diet or gastrointestinal haemorrhage), certain catabolic states (starvation, hyperthyroidism, or the use of corticosteroids), or post-renal failure/obstruction. Once azotaemia has been detected its continued presence should be confirmed with further blood samples and a urine sample should be collected for assessment of its concentration.

Occasionally, the degree of azotaemia is not as severe as expected. This is seen most commonly in very thin cats which lack sufficient muscle mass to incur markedly raised creatinine levels, and occasionally in cats with severe liver dysfunction that are unable to produce urea.

In addition to azotaemia, a number of other clinicopathological changes are seen commonly in cats with CKD (Table 3, page 144) (Barber and Elliott 1998; Lulich et al 1992; DiBartola et al 1987; Elliott and Barber 1998). These include hyperphosphataemia (due to reduced GFR), acidosis (because the kidneys fail to excrete sufficient acid), hypokalaemia (due to inappropriate kaliuresis) and hypoproliferative anaemia (due to reduced erythropoietin production, reduced red blood cell survival times, uraemic suppression of erythropoiesis, and/or gastrointestinal bleeding). Other changes may relate to stress and/or dehydration (e.g. altered white blood cell numbers, hyperglycaemia, hyperproteinaemia).

In addition, proteinuria has recently been shown to be an independent risk factor for the progression of CKD in cats (Lees et al 2005; Syme et al 2006; Elliott and Syme 2006; Kuwahara et al 2006; Jepson et al 2007; King et al 2007). While only ~10% of cats with CKD have a urinary protein to creatinine ratio (UPC) of >1.0, recent studies have shown that in cats with CKD a UPC >0.4 (0.3-0.6) is in indicator of poor prognosis, and may warrant treatment (Lees et al 2005). One study found that mean survival times were 449 days in cats with a UPC <0.2, 224 days in cats with a UPC 0.2-0.8, and only 117 days in cats with a UPC >0.8 (King et al 2007). A separate study found that mean survival times were ~700 days with a UPC <0.43, but ~270 days with a UPC >0.43 (Syme et al 2006). While the presence of proteinuria typically indicates glomerular damage it is now suggested that proteins in the glomerular filtrate may be directly renotoxic contributing to progression of renal failure. IRIS now recommend that each cat is assessed for proteinuria by UPC and classified into the ranges <0.2 [non-proteinuria], 0.2-0.4 [borderline proteinuric] and >0.4 [proteinuria] (Elliott and Syme 2006; IRIS Guidelines).

While CKD is usually progressive, some cats may have long periods of relatively stable renal function in both experimental (Ross et al 1982; Adams et al 1994) and naturally occurring disease (Elliott and Barber 1998). Because of this it can be difficult to give an accurate prognosis for a particular cat with CKD. The plasma creatinine concentration is a weak prognostic indicator. In contrast, the presence of anaemia tends to indicate a poor prognosis. Also, cats in end-stage renal failure are more likely to be hyperkalaemic and/or acidotic, have lower urine specific gravity and more acidic urine (Elliott and Barber 1998). End-stage renal failure is also more likely to be associated with worsening renal secondary hyperparathyroidism (RHPTH), reduced levels of calcitriol and reduced levels of ionised calcium (Barber and Elliott 1998), and increased white blood cell counts (Kuwahara et al 2006; King et al 2007).

While it would be advantageous to detect CKD as soon as it develops, this can only be performed where GFR can be measured. Unfortunately, while a number of suitable techniques have been validated (e.g. measurement of GFR using the clearance of inulin, iohexol, Tc-DTPA, or exogenous creatinine) they are not currently routinely available.

It is essential when making a diagnosis of CKD that a full and thorough diagnostic investigation be made. The initial investigation and follow-up monitoring should include:

- Physical examination (including retinal examination)
- Bodyweight and body condition score (1 being very thin to 9 being obese), plus calculation of the percentage body weight change since the previous consultation

- Systolic and, where possible, diastolic blood pressure
- Haematology (looking for anaemia in particular)
- Serum biochemistry (urea, creatinine, potassium, sodium, calcium, phosphate, proteins, and serum thyroxin concentration)
- Urinalysis, UPC and urine bacterial culture
- Where possible, assessment of acid-base status
- Periodical assessment of iron status is also warranted, especially in anaemic CKD cats
- +/- parathyroid hormone status monitoring (especially in cats receiving calcitriol therapy)

It is important to remember that while young animals usually have only one disorder at a time, this is often not the case with the older patient. In older patients the diagnosis and treatment of CKD may be complicated by the concurrence of multiple interacting disease processes. It is only by detecting and treating the concurrent diseases at the same time as the CKD that the cat can best be managed.

The initial investigation should include a thorough physical examination (including ocular examination, measurement of body weight and systemic blood pressure), plus collection of a blood sample (for routine biochemical and haematological analysis, including serum thyroxin assessment), and a urine sample (for routine urinalysis, assessment of UPC ratio, and bacterial culture).

MEDICAL MANAGEMENT

Where an underlying cause for the CKD can be found this should be addressed. For example, bacterial nephritis or pyelonephritis should be treated with appropriate antibiotics, nephrotoxins should be removed (e.g. non-steroidal anti-inflammatory drugs, aminoglycoside antibiotics, exposure to ethylene glycol antifreeze, lilies or grapes), pre-renal complications should be corrected (e.g. dehydration or cardiac disease), and post-renal obstruction should be resolved. The rest of this section will consider the aetiology of some of the more important problems associated with CKD, and then discuss the pros, cons and practical application of possible treatment options. However, it is important to tailor the specific treatment plan to the individual cat, according to their specific needs and situation.

The importance of maintaining sufficient fluid intake

An inadequate fluid intake can result in dehydration, reduced renal perfusion, pre-renal azotaemia and exacerbation of CKD. Some cats may be presented with acute decompensation of their CKD, while others may experience chronic or recurrent dehydration. Maintaining an adequate fluid intake is therefore of prime importance. Owners should be made aware of the increase in obligate fluid loss that typically accompanies CKD and so ensure that their cat has constant access to fresh water. In addition, they should encourage further fluid consumption by feeding a moist diet, and offering tempting 'soups' (e.g. made from cat food diluted with warm water, not salty fish stock, human gravy that may contain sufficient onion powder to induce haemolytic anaemia in cats, or milk which contains high levels of phosphate).

Where this proves insufficient to meet the cat's needs many clinicians encourage the regular 'at home' administration of subcutaneous fluids by the owners. Lactated Ringer's solution (LRS) or normal saline are used most frequently, although their long-term use may lead to sodium accumulation which may exacerbate hypertension. This can be prevented by using fluid composed of two parts 5% dextrose to one part LRS, however, dextrose-containing fluids can cause pain and irritation on administration. The amount of fluid given can be adjusted according to need (~50-150 ml, given from daily to once a week with the aim of correcting dehydration *not* of inducing diuresis). In addition, as needed, the fluid can be supplemented with potassium chloride (10 - 20 mmol KCl per litre of fluids) or sodium bicarbonate (0.5 – 8 mmol per litre of fluids - see later for indications). While this can improve the cat's well being by reducing azotaemia, it should not be done to excess as over-diuresis may actually exacerbate CKD. In all cases, it is sensible to monitor serum electrolyte levels (especially sodium and potassium levels) and monitoring systemic blood pressure so that problems can be detected and corrected quickly. To ease the administration of the fluids an 'indwelling' subcutaneous catheter may be considered, as can a nasogastric tube or a percutaneously (PEG) or surgically placed gastrostomy tube.

The role of diet; including altering the levels of protein and phosphate

Dietary therapy represents the cornerstone of management for patients with CKD. This is because the list of factors within food that may exacerbate or protect against CKD is endless. Most work has concentrated on the roles of protein, phosphate, calcium, potassium, and acidification (see below). However, other studies have suggested that it may be beneficial to restrict sodium chloride (Dworkin et al 1996) although this has since been questioned (Buranakarl et al 2004); to change the lipid content of the diet and alter the balance of free fatty acids from omega-6 unsaturated fatty acids in favour of omega-3

unsaturated fatty acids (Brown et al 1996a and b; Finco et al 2000), where high eicopentaenoic acid content appears beneficial (Plantinga et al (2005); to adding extra water soluble vitamins (e.g. B-complex vitamins); or to adding antioxidants (e.g. Vitamins E and C and β-carotene) (Yu and Paetau-Robinson 2006).

The ideal 'renal diet' should therefore:

- Meet nutrient and energy requirements
- Reduce protein catabolism and alleviate clinical signs of uraemia
- Minimise electrolyte, vitamin and mineral disturbances
- Slow the progression of renal failure

Restriction of dietary protein

The clinical benefits of protein restriction in CKD have been demonstrated in a number of species (Harte et al 1994; Finco et al 1992; Levey et al 1999; Polzin et al 1991). The products of protein catabolism are believed to contribute significantly to the clinical signs associated with uraemia. Reducing the intake of non-essential protein may therefore help to reduce the production of nitrogenous waste and so reduce the severity of the anorexia, vomiting, weight loss, anaemia and lethargy.

Whether or not dietary protein restriction actually helps to reduce the progression of renal failure is more controversial. Experimental studies (mostly in rats and dogs) have shown that in the early stages of CKD a declining number of nephrons is compensated for by an increased GFR for each individual (single) nephron (SNGFR). This increase in SNGFR is achieved by glomerular hyperfiltration, glomerular hypertrophy and glomerular hypertension, and is associated with an increase in proteinuria. Together, these factors may lead to glomerular and tubulointerstitial sclerosis and progression of the CKD. In some experimental models protein restriction has minimised these changes and so retarded the progression of disease (Brown and Brown 1995; Polzin et al 1991). While these findings have been supported by a meta-analysis of several studies in humans (Pedrini et al 1996) there is still considerable debate as to whether or not protein restriction will truly limit the progression of CKD in naturally occurring CKD in most species.

A few studies have investigated the role of protein restriction in cats with CKD. Experimental studies appear to show that significant proteinuria and glomerular morphological injury may occur in cats fed a higher protein diet, however, the presence of increased protein and calorie intake made interpretation difficult (Adams et al 1994; Finco et al 1998). The difficulty

of separating out different dietary variables also proved a complicating factor in studies by Harte et al (1994), Elliott et al (2000), Plantinga et al (2005) and Ross et al (2006) where cats with naturally occurring CKD were fed diets restricted in protein and phosphorus (i.e. 'renal diets'). In these studies, the cats fed a 'renal diet' showed marked clinical improvement, less uraemic episodes, reduced levels of plasma urea and phosphate, and fewer renal-related deaths: on average the cats fed 'renal diets' lived about a year longer than those that were fed regular cat food. While the overall benefit of the restricted diets cannot be denied, the individual effects of the protein and phosphorus cannot be determined.

It is generally recommended that cats with CKD be fed a diet with moderate protein restriction; containing protein of ~20% of the caloric intake. Unfortunately, the exact requirements are unknown, and since cats have a naturally high protein requirement it is essential not too over restrict them (Polzin et al 1996), especially if they still have significant muscle mass. It is also important to ensure that the source of protein is of high biological value and contains all of the essential amino acids. A minimum protein content of 3.5 g/kg/day has been recommended.

Unfortunately, while feeding a moderately protein-restricted diet is recommended, the poor palatability of these diets may limit their acceptance. Because of this, it is often recommended that cats with CKD be gradually weaned onto these diets before they start becoming inappetent or anorexic.

Restriction of dietary phosphorus and use of phosphate binders

Hyperphosphataemia occurs in approximately two thirds of cats with CKD (Table 3, page 144) (Barber and Elliott 1998; Lulich et al 1992; DiBartola et al 1987; Elliott and Barber 1998), and is believed to contribute to the uraemic complication of CKD. The primary mechanism for hyperphosphataemia is phosphate retention due to reduced GFR. Because the regulation of phosphorus and calcium are intrinsically linked, the phosphorus retention leads to calcium-phosphate deposition in the tissues (metastatic mineralisation), and this, in turn, leads to a reduction in the concentration of plasma ionised calcium. The resultant hypocalcaemia, although subclinical, stimulates the release of parathyroid hormone (PTH). Phosphate retention, when combined with the loss of renal mass, leads to a decreased production and/or activity of renal 1-□-hydroxylase enzyme, and hence a reduction of 1,25 dihydroxyvitamin D (calcitriol). The hypocalcitriolaemia results in a further increase in PTH production and reduced intestinal absorption of calcium. Phosphorus retention is therefore

an important factor in the development of renal secondary hyperparathyroidism (RHPTH) (Chew et al 1992).

Secondary hyperparathyroidism occurs commonly in cats with CKD. In one study, 84% of cats with naturally occurring CKD were found to have RHPTH; with the severity and prevalence being highest in cats with end-stage renal failure (Barber and Elliott 1998).

Parathyroid hormone may be considered as a uraemic toxin. In excess, it has been associated with a variety of clinical abnormalities, including anaemia, neurotoxicity, osteodystrophy (resulting in low-grade bone pain), arthritis, glucose intolerance, hyperlipidaemia, pancreatitis, immunosuppression and soft tissue mineralisation. While it is clear that when soft tissue mineralisation involves the kidneys it can lead to progressive renal dysfunction, a more general role for PTH in the progression of CKD is still under debate (Chew and Nagode 1992).

Limiting phosphorus consumption appears to slow the progression of CKD. Experimentally, when cats with CKD were fed a diet restricted in phosphate, they developed less renal mineralisation, mononuclear cell infiltration and fibrosis than cats fed a normal diet (Ross et al 1982). Studies in dogs have also shown a beneficial effect to restricting dietary phosphorus once azotaemia develops (Finco et al 1992). As discussed above, (under 'Restriction of dietary protein'), feeding cats with naturally occurring CKD a diet restricted in both phosphate and protein resulted in a marked clinical improvement, plus reduction of plasma phosphorus and PTH (Barber et al 1999; Elliott et al 2000). Since PTH is believed to be a uraemic toxin reducing its concentration is likely to be beneficial (Barber et al 1999). Interestingly, RHPTH can occur prior to the development of overt hyperphosphataemia. However, the importance of starting phosphate restriction prior to the detection of increased circulating phosphate remains unclear (Barber et al 1999).

Restriction of dietary phosphate is an important part of CKD management. The aim is to normalise the serum phosphate concentration. This can initially be achieved by feeding a phosphate-restricted diet (most commercial 'renal diets' are low in protein and therefore also low in phosphorus). However, when that is no longer sufficient, intestinal phosphate binders will need to be added. Monitoring plasma phosphate is an efficient, if not very sensitive, method for the detection of RHPTH (Barber and Elliott 1998). That said, blood samples should be collected after a 12 hour fast and should be non-haemolysed. A more sensitive method is to directly assess PTH concentration (Barber and Elliott 1998), however, this

requires a fasted blood sample, 'frozen shipment' of serum and access to a species-validated test, which is usually expensive.

Intestinal phosphate binders are usually added once the fasting serum phosphorous is >2 mmol/l. Aluminium containing salts, such as aluminium hydroxide, aluminium carbonate or aluminium oxide have been commonly used (30-150 mg/kg/day, divided between meals, and adjusted according to response). Unfortunately, aluminium salt phosphate binders are often poorly palatable, messy to administer, and may lead to nausea, anorexia, or constipation. Of the many products available Alu-Caps™ (capsules containing 475mg of dried aluminium hydroxide; 3M Health care) are perhaps the most palatable, and can be given by mixing a proportion of the contents of a capsule into the cat's food. In humans, it has been shown that the aluminium may become deposited in bone, resulting in worsening renal osteopathy. While this has not been shown to occur in dogs (Finco et al 2000), the situation in cats is unknown. Because of this, some clinicians recommend the use of calcium salts e.g. calcium carbonate (20-100 mg/kg/day, divided between meals), or calcium acetate. However, they are less effective than aluminium salts, and they have the potential to induce hypercalcaemia. Because of this it is essential to normalise the calcium level before starting the medication, and to monitor it closely throughout therapy. A number of companies offer combined products e.g. Ipakitine™ from Vetoquinol; which combines calcium carbonate with chitosan (to reduce phosphate absorption from the intestines), and report significant plasma phosphate reduction in cats with CKD (Wagner et al 2004) (although the product also contains lactulose, which may cause diarrhoea in some cats). A number of newer products are now available; e.g. Renalzin™ from Bayer, which uses lanthanum carbonate, and so circumvents the potential risk of hypercalcaemia. Some clinicians have also used sevelamer hydrochloride, but anecdotally this appears less effective than lanthanum (Arnell and Ross 2009). Since hypophosphataemia can result in weakness and anaemia, it is important to monitor phosphate levels whichever type of phosphate binder is chosen.

Calcitriol therapy

Plasma calcitriol concentrations are reduced in cats with CKD (see above) (Barber and Elliott 1998). Since calcitriol therapy effectively reduces PTH levels it should, in theory, make a useful adjunct to the treatment of cats with CKD (Chew and Nagode 1992). Some clinicians, including the author, use calcitriol therapy extensively, and find that it improves their patients' appetite and general well being (1.5-3.5 ng/kg/day po, given separately from meals; remove the oil from a capsule, dilute in corn oil, then give the appropriate volume, and store for up to two weeks) (Nagode et al 1996). However, there are few controlled

studies showing beneficial long-term use and one study using this dosage showed no beneficial response (Hostutler et al 2006). Also, the difficulties associated with its administration and monitoring deter many clinicians from using it. Careful monitoring is essential because calcitriol administration can result in hypercalcaemia and resultant hypercalcaemic nephropathy. Calcium and phosphorous levels must be in the low-normal range before beginning treatment, and they should then be monitored every 2-4 weeks.

Control of hypokalaemia

Hypokalaemia, probably resulting from inappropriate kaliuresis, is a common finding in cats with CKD (Table 3) (Barber and Elliott 1998; Lulich et al 1992; DiBartola et al 1987; Elliott and Barber 1998). It is currently unclear whether hypokalaemia is usually a cause of CKD, a consequence of CKD, or both. However, there is good evidence to show that hypokalaemia can cause or exacerbate feline CKD (DiBartola et al 1993; Dow et al 1990), and potassium supplementation of hypokalaemic cats with CKD often results in improved renal function (Dow et al 1987).

While the most obvious sign of severe hypokalaemia is polymyopathy, with generalised muscle weakness and ventroflexion of the neck, this does not develop until there is severe potassium depletion. Other clinical signs of hypokalaemia can include anorexia, vomiting, weight loss, lethargy and cardiac arrhythmias (Arnell and Ross 2009). Routine assessment of serum potassium is therefore recommended, with supplementation where necessary. Since feeding acidifying, magnesium restricted, and/or high protein diets appears to increase the risk of hypokalaemia these should not be fed to cats with CKD. Instead, it is advisable to feed non-acidifying, protein-restricted diets, and supplementation is recommended if the serum potassium levels fall below 4 mmol/l. Potassium gluconate is used most frequently (initially at 1-4 mmol q12h po, reducing as required). However, potassium citrate may be preferable when the patient is also acidotic (75 mg/kg q12h po). Potassium chloride is used infrequently as it is unpalatable and may cause gastrointestinal irritation. Daily potassium supplementation of non-hypokalaemic cats with CKD does not appear to be beneficial (Theisen et al 1997). It is important to remember that all intravenous (and even subcutaneous) fluids need to be supplemented with potassium to prevent inducing hypokalaemia. Ideally, the amount of potassium added to the fluids is based on the serum potassium levels (Table 4).

Correction of acidosis

Reduced renal function leads to a decline in the renal capacity for acid excretion. Because of this, acidosis occurs fairly commonly in cats with CKD, particularly those with severe disease

(Table 3) (Barber and Elliott 1998; Lulich et al 1992; DiBartola et al 1987; Elliott and Barber 1998). While acidosis is believed to contribute to anorexia, nausea, vomiting, weight loss, lethargy, and hypokalaemia, its role in the progression of renal failure remains unclear (Polzin et al 2000). That said, in other species it is associated with increased protein catabolism, anorexia, and precipitation of uraemic crisis (Fettman et al 1992). In addition, enhanced renal ammoniagenesis can cause activation of the complement cascade and tubulointerstitial injury (Nath et al 1985).

It is advisable to monitor cats with CKD at regular intervals for their acid-base status (assess TCO_2 or plasma bicarbonate). Specific treatment should be considered when TCO_2 is < 15 mmol/l, and should aim to maintain the TCO_2 between 18-23 mmol/l (Finco et al 2000). Treatment most frequently consists of sodium bicarbonate (5-10 mg/kg q8-12h po) or potassium citrate (30 mg/kg q12h po). However, sodium bicarbonate should be used cautiously in hypertensive patients, and potassium citrate may be a better choice when hypokalaemia is also present.

Correction of hypoproliferative anaemia

Many cats with CKD develop progressive anaemia that results in a variety of clinical signs including lethargy, inappetence, weakness and weight loss (Barber and Elliott 1998; Lulich et al 1992; DiBartola et al 1987; Elliott and Barber 1998). The cause of the anaemia is multifactorial, and includes reduced erythropoietin production related to reduced renal mass, reduced red blood cell survival times, uraemic suppression of erythropoiesis, gastrointestinal bleeding and/or iron or folic acid deficiencies. The most commonly used treatment options include recombinant human erythropoietin (r-HuEPO) (or recombinant feline erythropoietin, if available), iron supplementation (if needed), and anabolic steroids.

A number of studies have shown that r-HuEPO can cause a dramatic reversal of anaemia in cats with CKD, along with a general improvement in well being (Cowgill 1994; Polzin et al 1992; Cowgill et al 1998). Treatment with r-HuEPO is usually started once the PCV has fallen below ~20%: 100 units/kg is given subcutaneously three times a week until the PCV reaches ~30%, after which the dosage interval can be extended. Longer-acting EPO is now available (darbepoietin 6.25ug/cat [0.45ug/kg] sq q7days) (Arnell and Ross 2009, Chalhoub et al 2011). Initially, the PCV and other red cell parameters should be monitored weekly, then once the cat is more stable this can be extended to perhaps once every three to four weeks. If further adjustments are needed the dose can be altered by 25-50 units/cat (Cowgill 1994). Complications to r-HuEPO therapy include poor response due to iron deficiency, hypertension, polycythaemia, induction of anti-r-HuEPO antibodies, and systemic or local

allergic reactions. To reduce the risk of iron deficiency it is sensible to assess serum iron levels and total iron binding capacity prior to starting treatment, and to continue to monitor these parameters while the cat is receiving r-HuEPO. (Serum iron concentration, % iron saturation and total iron binding capacity can be performed by Capital Diagnostics, Edinburgh, UK; while ferritin levels can be assessed by Kansas State University, USA). If iron supplementation is required ferrous sulphate (50-100 mg/cat q24h po) or ferrous fumarate (30-60 mg/cat q24h po) can be given. About 30% of cats treated with r-HuEPO eventually develop antibodies that prevent the r-HuEPO from inducing erythropoiesis and can, occasionally, result in transfusion dependent aplastic anaemia. Its relatively high cost, the risk of side effects, and the cost of the necessary monitoring often limit the use of r-HuEPO.

While some clinicians advocate the use of anabolic steroids (e.g. nandrolone decanoate 1-1.5 mg/kg weekly by intramuscular injection) experimental support for their use is generally poor (Polzin et al 1992), and some anabolic steroids have been shown to induce liver failure.

Support of adequate food intake: Control of nausea and vomiting, use of gut protectants, appetite stimulants, and intake supplementation.

Cats with CKD often have a reduced food intake. Their lack of appetite may be caused by:

i. uraemic gastritis (due to the effects of circulating uraemic toxins or hypergastrinaemia)
ii. gastrointestinal haemorrhage
iii. the central effects of uraemic toxins causing nausea and vomiting
iv. offering rather unpalatable ‘renal diets’,
v. the presence of constipation (resulting from chronic dehydration and exacerbated by some of the treatments e.g. sucralfate),
vi. anaemia
vii. metabolic acidosis and/or
viii. renal secondary hyperparathyroidism

Cats that do not maintain their food intake may incur protein malnutrition, endogenous protein catabolism, and metabolic acidosis.

Treatment options include the use of H_2-antagonists to reduce gastric acidity (e.g. famotidine 0.5-1.0 mg/kg q24-48h po [not iv], ranitidine 2-4 mg/kg q12h, iv or po [which also has a GI prokinetic effect], or cimetidine 2.5-5.0 mg/kg q8-12h, po, iv), sucralfate to help heal gastric

ulceration (250-500 mg/cat q8-12h po), centrally acting anti-emetics to help block the effects of uraemic toxins on the chemoreceptor trigger zone (e.g. metoclopramide 0.2-0.5 mg/kg q6-8h po, or 1-2 mg/kg q24h as a constant iv infusion) or maropitant (0.5-1.0 mg/kg q24h, po or sq), and lactulose (dosed to effect as a laxative, and it has the additional benefit of trapping urea in the bowel and reducing the azotaemia).

There are a number of different ways of encouraging cats to eat. These include the use of warmed or aromatic foods, and any intervention that improves the cat's sense of well being. Unfortunately, the use of chemical appetite stimulants is not without risk as diazepam can cause fatal hepatic necrosis (Center et al 1996), mirtazepine has been associated with excitation and even collapse so a lower dose is suggested (1/4-1/8 of a 15mg tablet every three days) (Feline Expert Panel observations), and cyproheptadine has very occasionally been associated with haemolytic anaemia (DGM personal observation). While anabolic steroids (e.g. nandrolone – see above) may appear to help in some case, few clinicians use them routinely. Where cats fail to maintain an adequate calorie (and/or fluid) intake, some clinicians will consider the long-term use of nasogastric or PEG tubes.

Systemic hypertension, antihypertensive drugs, and ACE inhibitors

While the relationship between hypertension as a cause versus an effect of CKD remains poorly defined, it is essential that all cats with CKD be assessed for its presence. This is because hypertension is found commonly in cats with CKD (from ~ 25% of cats with CKD seen in a first opinion practice up to 60-65% of similar cases in referral practice); the presence of untreated hypertension may lead to exacerbation of the CKD (Kobayaski et al 1990; Ross 1992; Littman 1994; Henik 1997; Mishina et al 1998; Brown et al 2000; Elliott et al 2001; Syme et al 2002; Jepson et al 2007). In a small survey of our own referral CKD cases 14/26 (56%) were found to be hypertensive (systolic blood pressure > 175 mmHg (Brown et al 2000; Elliott et al 2001; Henik 1997; Sparkes et al 1999). Six cases were diagnosed at the time of initial presentation, and a further eight developed hypertension within five years of being diagnosed with CKD. Our study, plus a number of others, noted that there is no correlation between the degree of azotaemia and the presence or severity of systemic hypertension (Elliott et al 2001; Kobayaski et al 1990).

While the exact aetiology of hypertension in CKD remains unclear a number of factors appear to be involved (Henik 1997; Kobayashi et al 1990; Ross 1992): Diseased kidneys may be unable to efficiently excrete sodium and water (resulting in extracellular expansion), while activation of the renin-angiotensin-aldosterone system (RAAS) leads to the production of angiotensin II (which produces vasoconstriction) and aldosterone (which promotes sodium

retention). Also, diseased kidneys may be unable to produce adequate amounts of vasodilator substances (e.g. prostaglandins and components of the kallilrein-kinin system), and autonomic dysfunction may result in increased circulating levels of catecholamines and an increased vascular responsiveness. While different types of renal disease may produce hypertension by different mechanisms, the presence of hypertension results in continually high glomerular filtration pressures that may worsen existing renal disease and contribute to further hypertensive injury and disease progression (Kobrin and Aradye 1997).

The most common changes consistent with persistent hypertension occur in the kidneys, eyes, heart and brain (Elliott et al 2001; Henik 1997). Unfortunately, hypertension is usually only suspected very late in the course of disease, once end-organ damage has already occurred. This is typically seen as exacerbation of renal failure, intraocular haemorrhage and/or blindness, left ventricular hypertrophy, and/or cerebral vascular accidents. In our own series ocular signs were present in 38%, and cardiac changes in 29% of the cases. Of the hypertensive cats, 71% had ocular evidence of hypertensive damage. Findings included anterior chamber, vitreal or retinal haemorrhage, retinal oedema or detachment, arterial tortuosity, alternating constriction and dilation of retinal primary venules, and/or glaucoma.

Blood pressure should therefore be evaluated as a routine part of all check-ups of CKD cats and anti-hypertensive therapy should be prescribed to those where the mean systolic blood pressure readings, taken with the cat in a calm state, are persistently above 170-180 mmHg or where there is evidence of hypertensive retinopathy (Stepien 2004). **IRIS Guidelines**: <150 mmHg [minimal risk], 150-160 mmHg [low risk], 160-180 mmHg [moderate risk], >180 mmHg [high risk].

Various indirect methods exist for the measurement of blood pressure. However, in cats, the Doppler method is believed to be most accurate, as oscillometric methods tend to underestimate blood pressure (Bartges et al 1996; Brown et al 2000). The only problem with this technique is that it is not always possible to measure the diastolic pressure. When it is possible, the diastolic blood pressure of normal cats should be less than 95 mmHg (Mishna et al 1998).

Although treatment of feline systemic hypertension has largely been extrapolated from human medicine, a number of studies have been performed on cats. A number of treatment regimes have been suggested, including the use of calcium channel blockers (CCBs) (e.g. amlodipine besylate; 0.625-1.25 mg/cat po q24h), and/or angiotensin converting enzyme (ACE) inhibitors (e.g. benazepril; 0.25-0.5 mg/kg po q24h). Also, where possible, any

underlying conditions should be treated. While other therapies have been suggested, including the use of beta adrenergic receptor antagonists (e.g. propranolol), alpha adrenergic receptor antagonists (e.g. prazosin), arteriolar vasodilators (e.g. hydralazine), diuretics (e.g. frusemide), or a low salt diet, they tend to be less reliable and/or effective (Bartges et al 1996).

Many people recommend **calcium channel blockers** (CCBs) (and amlodipine besylate in particular) as the single agent of choice for the treatment of systemic hypertension in cats (Bartges et al 1996; Henik 1997; Elliott et al 2001, Jepson et al 2007). CCBs may be of particular benefit in cats with CKD as they decrease systemic hypertension, dilate glomerular afferent arterioles, attenuate mitogenic effects of various growth factors, and attenuate mesangial entrapment of macromolecules (Epstein 1992). However, because of preferential afferent arteriolar dilation, elevated systemic blood pressure may be transmitted to the glomerulus, resulting in glomerular hypertension (Tolins and Raji 1991).

ACE inhibitors are now becoming a first line therapy for the treatment of systemic hypertension in humans with CKD (Jafar et al 2001; Maschio et al 1996). The positive effects of using ACE inhibitors to treat hypertension in CKD arise because, by inhibiting the conversion of angiotensin I to angiotensin II, ACE inhibitors decrease aldosterone secretion, decrease plasma and urine angiotensin II, increase urine concentration of prostaglandin E and bradykinin, reduce intra-glomerular capillary blood pressure (due to efferent arteriolar dilation), and reduce glomerular hyperfiltration and proteinuria (Allen et al 1987; Tolins and Raji 1991). However, ACE inhibitors may also lead to reduced renal perfusion and so cause tubular necrosis, resulting in progression of the renal failure (Amadio et al 1990). This may be more of a significant problem for those ACE inhibitors that are exclusively excreted though the kidney and their doses need to be adjusted in cases of CKD (Allen et al 1987). One advantage of benazepril is that most of its excretion is through the liver.

In cats, the beneficial effects of ACE inhibitors (and benazepril in particular), have been shown in a number of studies of experimental and naturally occurring CKD. Significant reductions in systemic blood pressure, glomerular capillary pressure, angiotensin II, aldosterone, and proteinuria have been documented (Brown et al 2001; Watanabe et al 1999 and 2007), along with delayed progression of disease and extended survival times (Mizutani et al 2006). Results from the BENRIC study support these findings, with the most significant effects being seen in proteinuric patients, and Persian cats (Gunn-Moore et al 2003; King et al 2007).

Is it better to treat cats with CKD with CCBs or ACE inhibitors?

i. Where systemic blood pressure is significantly raised (>200 mmHg) amlodipine is required because it is more effective in reducing very severe hypertension and it gives more predictable results (Brown and Henik 2000): the reduction in hypertension is associated with reduced proteinuria and increased survival (Jepson et al 2007). However, these cases may also benefit from the addition of an ACE inhibitor, particularly if their UPC ratio is at or above the high end of normal or if the hypertension is severe and/or refractory to CCB.

ii. There is now growing evidence to support the use of ACE inhibitors, not only in hypertensive cats, but more widely in normotensive individuals with CKD (Lefebvre and Toutain 2004, King et al 2006, Mizutani et al 2006). In addition, studies in human patients have suggested that ACE inhibitors are more effective at reducing the progression of renal failure, even in cases where no systemic hypertension is present (Jafar et al 2001). ACE inhibitors have been shown to be more renoprotective than CCBs in dogs with experimental CKD (due to induced diabetes mellitus) (Brown et al 1993). While ACE inhibitors are most effective in the treatment of CKD associated with mild to severe proteinuria or diabetes mellitus (Maschio et al 1996, King et al 2006), their beneficial effect in non-proteinuric cases has lead to the suggestion that they may have a positive effect beyond decreasing blood pressure and reducing urinary protein loss (Jafar et al 2001; King et al 2006).

iii. ACE inhibitors should not be given to cases of unstable or acute RF, and extreme care should be taken when considering starting them in cases of severe CKD (place the cat on IV fluids first and monitor the cat for deterioration or an increase in the plasma creatinine of >30% - if this occurs, stop the ACE inhibitor).

Urinary tract infections

Urinary tract infections (UTIs) occur commonly in cats with CKD, probably relating to the presence of dilute urine, but also because uraemia has inhibitory effects on neutrophil function. In a number of studies 25-35% of cases were found to have a UTI at some point during their illness (Demetriou et al, 1997; Barber person communication 2001; Mayer-Ronne et al, 2006). Interestingly, 75% of the UTIs occurred in female cats, and many of these cats had recurrent episodes of infection (Barber, personal communication, 2001). Unfortunately, while the presence of a UTI rarely results in specific clinical signs (e.g. typically of pollakiuria, renal pain and/or dysuria) it is highly likely to exacerbate the renal damage. It is therefore essential that cats with CKD be regularly assessed for the presence of a UTI. Unfortunately, pyuria and/or active urine sediment is not always present in cases of UTI associated with CKD in cats (or diabetes mellitus or hyperthyroidism) (Mayer-Ronne et

al, 2006). This may be because uraemic toxins (and hyperglycaemia) can reduce neutrophil function. Since diagnosis can only be confirmed by performing urinalysis *and* bacterial culture, urine samples need to be collected by cystocentesis.

Once a UTI has been confirmed, the chose of antibiotics is best made according to culture and sensitivity, and a prolonged course is required (4-8 weeks). Ideally, culture and sensitivity should be repeated after one week of treatment (to ensure the choice of antibiotic has been correct), and then again one week after completing the course (to confirm the treatment has been effective).

Long-term monitoring

Long-term monitoring is essential. Each examination should be as extensive as the initial examination (see above), and it should be repeated every one to six months, depending on the severity and extent of the clinical signs.

Further information for owners of cats with CKD can be found on the FAB website (www.fabcats.org). A very useful book designed to help owners of cats with CKD is available from www.catprofessional.com and the following site was designed by an owner of a cat with CKD: http://www.felineCKD.com/ which contains particularly useful information on administration of subcutaneous fluids and links to a variety of other useful web sites.

Table 1. Potential aetiologies of feline chronic renal failure

Chronic tubulointerstitial nephritis
Glomerulonephritis
Pyelonephritis
Polycystic renal disease – congenital or acquired
Amyloidosis – familial or acquired
Nephrotoxins – e.g. ethylene glycol, aminoglycoside antibiotics, lilies, grapes (raisons, currents), melamine/cyanuric acid
Hypercalcaemia
Hydronephrosis
Renal lymphoma
Eventual result of untreated pre-renal or post-renal failure

Table 2. Common clinical signs in 412 cases of CKD [a]

Clinical sign	**%**
Dehydration	62
Anorexia	62
Lethargy / depression	47
Weight loss	46
Polydipsia / polyuria	38
Vomiting	29
Large kidneys (1 or both)	25[b]
Small kidneys (1 or both)	19
Pale mucous membranes	10
Oral ulceration / discomfort	10
Also: Retinal detachment Poor coat Thin Halitosis Diarrhoea or constipation Haematuria / dysuria Bone pain osteodystrophy	

[a] Based on four studies (Lulich et al 1992; Krawiec and Gelberg 1989; DiBartola et al 1987; Elliott and Barber 1998). [b] Based on 337 cats from three studies (Lulich et al 1992; Krawiec and Gelberg 1989; DiBartola et al 1987).

Table 3. Common clinicopathological findings in 286 cases of CKD [a]

Finding	**%**
↑ plasma urea	98
↑ plasma creatinine	98
↑ plasma PTH	84[b]
Urine specific gravity < 1.030	75[c]
↑ plasma phosphate	63
↓ plasma TCO_2	55
Anaemia	37
↓ plasma calcitrol	36[b]
↓ plasma ionised calcium	25[b]
↓ plasma potassium	21
↑ plasma cholesterol	72
Urine protein:creatinine > 1.0	< 10[c]
Also ↑ plasma amylase ↓ lymphocytes ↑ plasma glucose ↑ white blood cells ↑ plasma ionised calcium	

[a] Based on four studies (Barber and Elliott 1998; Lulich et al 1992; DiBartola et al 1987; Elliott and Barber 1998). [b] Based on 80 cats (Elliott and Barber 1998). [c] Based on 52 cats (Elliott and Barber 1998).

Table 4. Amount of potassium that should be added to IV fluids

Serum potassium levels	**Amount of potassium to be added to 500ml fluids**
< 2 mmol/l	40 mmol
2.0 – 2.5 mmol/l	30 mmol
2.5 – 3.0 mmol/l	20 mmol
3.0 – 3.5 mmol/l	14 mmol
> 3.5 mmol/l	10 mmol ('maintenance' levels)

References available on request from the author.

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PRACTICAL USE OF FEEDING TUBES

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A lot of our hospitalised patients share common requirements; we provide them with intravenous fluids, analgesia and antibiotics while they are stabilised and their condition treated. Inappatence or anorexia commonly develop often due to pain or stress, but provision of nutrition is often given low priority against other needs. Nutrition is extremely important for the recovery of our patients, and if not addressed further complications are inevitable.

Patients that are not receiving their nutritional requirements have been shown to have increased mortality, longer hospital stays, prolonged recover, poor immune function, ineffective gut barrier and slower wound healing. By acting early and effectively, or preferably anticipating the need for intervention before a problem occurs, we can speed the recovery and improve the outcome for our patients.

Total parenteral nutrition (TPN), whereby an animal receives nutrition by a route other than via the alimentary tract, is rarely used in animals and may have associated risks. Broadly speaking, if the GIT is working it makes sense to use it, i.e. enteral nutrition should be used in preference. The intestinal mucosa needs glutamine and regular nutrients to maintain itself. Without this nutrition the gut barrier will become less effective, and potentially lead to bacterial translocation, and endotoxin absorption in critical patients.

In an anorexic patient we can try coax the animal to feed, or try chemical stimulation of appetite prior to bypassing the mouth with feeding tubes.

Coaxing the animal to eat, or hand feeding, may be a simple means of increasing an animal's food intake, but will only usually work in partially anorexic animals. Tempting with warmed, strong smelling food may encourage the animal to start feeding for itself. Force feeding is to be avoided as it causes stress to the animal, has the risk of aspiration, and can lead to "food aversion".

Chemical stimulation of appetite can be tried if food intake is still not adequate. Hopefully what we are achieving with these drugs is to remind the animal what they are missing, and 'kick-start' normal intakes. They should be regarded as a short term option just to get the animal fending for itself again.

When to Consider Tube Feeding

We can assess our patients to see if a feeding tube is currently required, or if a feeding tube is likely to be required in the near future:

Feeding tube required:

- Anorexia (total or partial) for over 5 days
- Significant weight loss (>10%)
- Increased nutritional losses
- Increase nutritional requirements
- Bypass specific parts of the alimentary tract.

Likely to be required:

- Animals undergoing surgery and anorexia likely
- Extra nutritional requirements but appetite likely to be poor

What type of tube to place then depends on a number of factors:

- Estimated length of time tube feeding required
- Ability to tolerate and anaesthetic
- Concurrent injuries
- Which area of GIT 'bypassing'
- Familiarity of team with technique

In general the more proximal the point of feeding, the more 'normal' it is physiologically so upsets are less likely, but if we are bypassing a specific problem (e.g. an oesophageal injury) then the point of feeding needs to be caudal to this (e.g. gastrostomy). In most circumstances, no tube should cross the lower oesophageal sphincter due to the risk of reflux and oesophagitis.

Naso-oesophageal tube. (3.5-5 Fr Cats. 3.5-8 Fr Dogs)

Advantages:

- Cheap
- Quick
- Non-invasive

- No need for anaesthesia

Disadvantages:

- Small tube size, so liquid diets
- Not all patients tolerate them
- Short term only (up to a week)

Soft flexible PVC or silicone tubes are easily placed into the nostril, sedation is rarely required, but using topical local anaesthesia makes things easier. The tube is directed into the ventral meatus, and a swallowing reflex checked for as the tube enters the pharynx. Pre-measure the tube to the 10th rib so that the end lies in the distal oesophagus and does not cross the oesophageal sphincter. Feed small frequent meals, or continuous infusion.

Do not use if: rhinitis or facial injuries involving the nares, persistent vomiting, unconscious, or abnormalities of the pharynx/larynx/oesophagus.

Pharyngostomy Tubes

High complication rate, NO LONGER USED

Popular after described in 1970, but frequently problems with airway obstruction, aspiration, infection and also haemorrhage during placement are encountered.

Oesphagostomy Tubes. (8-18 Fr)

Advantages:

- Inexpensive
- Larger bore tube than naso-oesophageal
- Quick, brief GA
- Tolerated well, can be used for months
- Minimal surgical skill involved

Disadvantages:

- Invasive
- Small risk of infection/fistula
- Needs to be dressed to prevent interference

Oesophagostomy tubes are placed into the mid-cervical oesophagus and tend to be tolerated well. Complications are minimal, the most common being infection at the entry site, but this should be avoided by correct care of the stoma. Several techniques are available for

placement, in all techniques the tube is anchored to the skin at the entry site, and terminates in the distal oesophagus.

Percutaneous placement - using an introducer needle with a peel away sheath (Van Noort system). The metal introducer is placed into the oesophagus and palpated through the skin; the needle is placed through the skin into the hollow tube at the end of the introducer, then withdrawn leaving the peel-away sheath in place. A pre-measured tube is inserted into the sheath, the sheath is then removed and the introducer withdrawn.

Surgical placement- long curved artery forceps (e.g. Carmalt forceps) are passed into the oesophagus and palpated through the skin, a skin incision is made over the tips of the forceps and the tips forced up through the incision- then either; a small stylet can be used to increase the rigidity of the tube, and it can be placed directly down the oesophagus, between the open jaws of the forceps, OR- the tip of the tube is grasped in the forceps and drawn back up into the mouth, where the tip is reversed and inserted back down the oesophagus.

Whichever technique is used: a) pre-measure the tube and mark it!
b) anchor the tube at the entry site.
c) confirm the position of the tube radiographically.

Do Not Use if: Vomiting, problems with oesophageal function/motility, laryngeal problems.

Gastrostomy Tubes. (14-20 Fr)

Advantages**:**

- Larger bore tube: less risk of blockage, can feed liquidised tinned food
- Easy to maintain
- Can be used for months

Disadvantages**:**

- General anaesthesia
- Some specialised equipment
- Must stay in place at least 14 days
- Risk of peritonitis

Gastrostomy tubes are useful in the longer term management of critical or recovering patients. Can be placed surgically (at laparotomy or flank incision), percutaneous endoscopic placement, or blind percutaneous placement. Silicone mushroom tipped catheters are marketed for this purpose, (ordinary Foley catheters are at risk of the balloon

perforating due to gastric acid erosion, followed by stomach contents leaking into the peritoneum) Other complications can include splenic laceration, infection and cellulitis.

Percutaneous Endoscopic Placement

the endoscope is passed into the stomach, with the dog lying in right lateral recumbency, the stomach is inflated, and an assistant passes a catheter through the body wall into the stomach, a thread of suture material is passed down the catheter and grasped with the mouth, a dilator is threaded onto the suture material, and then tied to the end of the feeding catheter. The suture material is then pulled by the assistant and the catheter is drawn down the oesophagus into the stomach, and out through the body wall until the mushroom tip is in contact with the stomach wall.

Surgical placement

Either during laparotomy or via a flank incision, a purse-string suture is placed in the stomach wall and the mushroom tip placed through a stab incision in the centre of the purse-string. The end of the catheter is then exited through the body wall. Omentum can be wrapped around the catheter between the stomach and the peritoneum to guard against leakage and form a pexy.

Blind percutaneous placement

Various commercial equipment is available, most involve a long hollow rigid stylet which is passed into the stomach via the oesophagus. There is a risk of damage to the oesophagus, stomach wall, and spleen.

Whichever technique is used for placement, in all cases;

- Confirm placement either endoscopically or radiographically.

Anchor the catheter at the body wall, but avoid excessive pressure under fixation discs etc this can lead to necrosis.

Enterostomy Tubes (or Jejunostomy tube) (5-8 Fr)

Advantages**:**

- Distal to the stomach/pancreas/biliary tract
- Well tolerated

Disadvantages**:**

- Cost
- Longer GA

- Peritonitis risk
- Narrow tube size
- Remain for at least 14 days

Used most frequently where bypass of the stomach, pancreas or biliary tract are required, e.g. in pancreatitis.

Most often placed during laparotomy, this feeding tube into the wall of the proximal jejunum through a purse string suture, and exited through a stab incision in the body wall. The main risk is leakage of bowel contents. Creating a serosal tunnel in the gut wall, and anchoring the gut to the body wall help prevent leakage. To avoid the risk of leakage, it is possible to place a gastrostomy tube and then have an enterostomy tube exiting from it and leaving the stomach via the pylorus. This can in cases be achieved endoscopically. This gives the advantage of being able to decompress the stomach by aspirating the gastrostomy tube, as well as being able to feed distal to the stomach.

Feeding concentrated liquid diets at this point of the alimentary tract can cause problems such as osmotic overload and diarrhoea.

Feeding requirements

The formulas for calculation of energy requirements of patients have varied over the years. Much of the earlier work was based on human requirements. For a healthy animal the energy requirements for basic body processes is referred to as the Resting Energy Requirement(RER), on top of this there will be energy expended for temperature regulation, and physical activity, this total gives the daily energy requirement. Critical patients are unlikely to be doing much physical activity; therefore the patient's daily energy expenditure is likely to be close to its RER. The complication is that it is thought certain disease processes greatly increase the RER, by multiplying RER by an 'illness factor' (ranging from 1.2-2.0) an Illness Energy Requirement is arrived. How large these illness factors should be remains controversial.

Though in some cases RER may not be greatly elevated, in most hospitalised patients the protein requirement is likely to be significantly increased. The dietary protein source needs to be highly digestible. In tube fed dogs protein should be 20-30% of calorie intake, and >30% of calories for cats. For example Hills a/d diet contains 33% of calories by protein.

Once a daily requirement has been calculated, on the first day of feeding a third of the requirement is given, on the second day two thirds, with the full ration being reached on the third day.

When using the feeding tube:

- Do not feed for first 24 hours to allow a seal to form (doesn't apply to naso-oesophageal tubes)
- Frequency of feeding is dictated by the type of tube used, and hence the volume that can be given at any one feed.
- Always flush the tube prior to feeding with water
- With gastrostomy tubes, prior to feeding each meal, aspirate the stomach, if more than half of the meal is still present, skip the next meal
- Warm food to body temperature.
- Watch for any signs of discomfort, retching etc- if noted slow down, or stop.
- Flush after feeding with water.

Avoiding Complications

The most common complications seen are tube dislodgement (patient interference, vomiting), infection at the entry site, or tube blockage.

In all tubes other than naso-oesophageal tubes, the tube entry site needs to be kept clean and dressed to prevent patient interference. Check and clean stoma, replace sterile dressing daily initially.

Blockage can be avoided by flushing before and after feeding, and keeping a column of water in the tube between feeds. In narrow bore tubes feed only commercial liquid diets, rather than liquidized foods. If the patient is being discharged into the owners care to continue tube feeding at home, demonstrate clearly to them what is expected. If a blocked tube cannot be flushed clear, cola can be left in the tube overnight, or sometimes pancreatic enzyme is used.

Vomiting, diarrhoea and nausea can occur if the daily requirement is fed straight away. Slow introduction of diet is required to avoid problems due to hyperosmolarity. On day one a third of the requirement diluted with water, split into 6 meals, day 2 two thirds and the full amount on the third day. The stomach capacity of a dog is 90ml/kg, whereas a cat is only 45ml/kg.

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CARE OF THE RECUMBENT PATIENT

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A recumbent patient is one that is lying down and unable to rise on its own. Obviously this may be a permanent condition, but hopefully in the majority of our cases it is temporary. Recumbency can be due to a wide variety of causes, hopefully while we are treating the cause, proper management will prevent further complications and life threatening problems. Commonly encountered conditions that lead to recumbency include:

- Trauma
- Iatrogenic
- Neurological
- Behavioural
- Weakness

Regardless of the initial cause of recumbency, these cases share common requirements for their day to day care.

Areas that need to be addressed and covered in care plans include:

Cardiovascular support	Blood sampling
Bedding	Respiratory
Urinary management	Faecal management
Eye Care	Oral Care
Nutrition	Analgesia
Hydration	Physiotherapy

Respiratory Care

Recumbent patients need regular monitoring and intervention to ensure they are adequately ventilating themselves. Most patients voluntarily lay in lateral recumbency, long periods of time spent in this position means the dependant lung has increased pressure contents of the chest, as well as pressure from abdominal contents. This pressure will decrease the amount that this lung is able to inflate and thus reduce availability functional lung fields and gas exchange abilities. Continued pressure leads to atelectasis (temporary collapse of the lung).

If the patient is in sternal recumbency, both lungs are able to expand fully, but leaving an immobile patient in one position for long periods of time may lead to pressure sores (decubital ulcers). A sensible compromise is to alternate the position of the patient every couple of hours, and record on the chart what position and when these changes are due. The patient is moved from left lateral, to sternal, to right lateral, to sternal, to left lateral recumbency. Turning a patient straight from left to right lateral recumbency runs the risk of both lungs being compromised at the same time.

Regular monitoring of the respiratory function will assess whether gas exchange is effective in the lungs of the patient. The gold standard is measurement of arterial blood gases, but useful information can be gathered from recording and spotting trends in respiratory rate and effort, heart rate, and oxygen saturation.

Concerns regarding a patient's ability to maintain blood oxygen levels will lead to the need for oxygen supplementation. The means of supplementation will be dictated by the patient's size, temperament, anticipated duration, and skills and equipment available.

Mucous secretions of the airways need consideration, recumbency may contribute to accumulation of mucous due to decreased clearance and small airway collapse. Nebulisation can be performed every 4 hours, either in an oxygen cage, or by trying to use a hand held mask. Coupage used regularly can help to loosen secretions, which are hopefully then coughed up by the patient.

Bedding

The surface the patient lies on is obviously going to have a large impact on comfort levels, but also on the likelihood of developing complications such as decubital ulcers. Ulcers commonly form over bony prominences such as the elbow, the hip and even the sternum.

They develop as a result of pressure compromising blood supply to the tissues, shearing forces acting on the tissues, and moisture. They occur most commonly in large breed dogs, but also chondrodystrophic breeds (e.g. Dachshunds) where they may be seen on the lateral aspect of the hock. Prevention of decubital ulcers is a lot easier than trying to treat them once they have occurred, they can often take 6 weeks or more to heal.

Treatment of decubital ulcers relies on reducing infection and contamination of the site, applying appropriate contact dressing layers, reducing the pressure of the area by using donut bandages and carefully positioned bedding.

Bedding we provide has to:

- Be comfortable!
- Provide support
- Wick moisture
- Aid in thermoregulation

Hydration Status and Fluid Balance

The fluid therapy plan needs to be considered as a flexible, ongoing situation, and will need adapting as the patients' status changes. The best way to monitor a patient's needs is by repeated physical examination- we should be able to assess the effect of the fluid therapy, and compare the actual effect to our desired effect. Remember that the animal's normal homeostasis may be impaired, so we need to monitor carefully to prevent overdosage or imbalances. The physical exam should check hydration and perfusion parameters, as well as checking for any complications of fluid therapy; such as oedema, phlebitis, extravasation of fluid.

- Hydration Parameters: Moisture of mucous membranes, skin turgor, retraction of the globe.
- Body Weight: sudden changes in body weight are usually due to changes in body water. When correcting dehydration, increasing bodyweight would be encouraging.
- Urine Output: Urine output can be measured by weighing wet bedding, catching urine in patients that can stand, or by indwelling urinary catheters. A urine output of 0.5ml-2.0ml/kg/hour is one of the goals of fluid resuscitation of hypoperfused animals.
- Central Venous Blood Pressure***:*** Can be measured if a central line is in place with the tip in the cranial vena cava, this gives us an idea of pre-load; the amount of blood returning to the heart to be pumped. CVP is a more useful assessment of overall vascular filling than arterial blood pressure.
- Blood Testing: When a patient is on IVF it is important to regularly check the PCV/TP and Electrolytes ideally every 12-24 hours depending on the critical nature of the patient. This will help to ensure that the balance of fluids and electrolytes is monitored closely. Hypokalaemia is a risk in patients on chronic fluid therapy, and measuring electrolytes can guide us regarding the requirement for supplementation.

Fluid Balance

When administering maintenance fluids to hospital inpatients, the best way to monitor volume status is by keeping track of the volumes of fluid going into a patient, compared with those coming out.

- Fluids in are easily measured; as well as volumes of intravenous fluids administered, measure any water drank, and record food eaten.
- Fluids out are less easy to measure. Urine output is easily measured if a urinary catheter is in place, otherwise weigh bedding, use non-absorbent cat litter, or catch urine with kidney dishes etc. When weighing cage liners, assume 1g is equal to 1ml of fluid. Any vomit or diarrhoea must be estimated.
- Calculating: measure every 6 hours, fluids in should be approximately 10% more than fluid out (some fluid is lost by sweating or evaporation from the respiratory tract). If the fluid out is larger than the fluid in, we need to increase the fluid rate. If the fluid in is much larger than the fluid out we need to think why: if the patient is still dehydrated, this would be normal, so does the patient still show signs of dehydration? Otherwise why is the patient absorbing extra fluid- exudates, oliguric renal failure, overhydration?

Signs of overhydration; if we administer too much fluid we can overload the body, and especially the interstitial space. Excessive fluid in the interstitial space may show itself as:

- Peripheral oedema- feet, legs, axilla, face etc.
- Chemosis
- Pulmonary oedema
- Cerebral oedema

Urinary Management

Prolonged distension of the bladder needs to be avoided, as well as causing discomfort and distress; it can lead increased risk of urinary tract infection, and detrusor muscle damage. Broadly speaking recumbent patients will fall into 3 categories when it comes to urination; those that can void the bladder if supported, those that are having the bladder manually expressed, and those that have an indwelling urinary catheter. Which method is suitable for the individual patient will be determined by the clinical team and noted on the treatment plan. Animals that are capable of voluntarily voiding the bladder will have to be observed closely for behavioural signs that they are ready to urinate, or the bladder should be palpated regularly.

Where an indwelling urinary catheter is placed, a closed collection system MUST be used. Leaving an open urinary catheter to drip urine runs the risk of urine scald to the skin and ascending infection.

An indwelling catheter connected to a closed collection system is also preferred to allow accurate measurement of urine output. Closed collection systems can either be commercially available, or an emptied intravenous fluid bag can be used (saline or Hartmanns, NOT glucose containing fluids), connected via a sterile giving set. The collection bag is placed below the patient to allow urine to drain by gravity, but avoid placing on the floor to reduce the risk of bacterial contamination. Closed systems should be 'broken' as infrequently as possible. If the bag needs to be emptied, or catheter disconnected, it should be done as aseptically as possible as this is the time of greatest risk for introduction of bacteria into the system. The external catheter should be wiped with dilute chlorhexidine every 4-6 hours to help prevent ascending infection.

Commercial collection bags have the advantage of having a built in measurement scale, and can usually be emptied via a tap at the bottom of the bag, this avoids disconnecting and connecting the system, as this is when there is greatest risk of contamination being introduced to the system. Some collecting bags also have an anti-reflux chamber to avoid backward flow from the bag to the bladder.

Hospital Acquired Infections (HAI)

Critical patients are at the highest risk of developing HAIs, due to the combination of indwelling devices (IV catheters, chest drains, tracheostomy tubes, feeding tubes, urinary catheters etc) and immune-compromise, so correct protocols are essential to minimise this risk.

Aseptic technique should be used, wearing gloves, when handling devices. Stomas should be cleaned and dressed daily, and the site checked for signs of inflammation and swelling. With giving sets and closed urinary collection systems the minimum of disconnections and connections should be made to minimise the risk of introducing contamination into the line.

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GETTING THAT RADIOGRAPHIC DIAGNOSIS: PITFALLS AND TIPS FOR EXCELLENCE

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The technical aspects of achieving a diagnostic radiographic study are easily overlooked, particularly when presented with an emergency situation or an uncooperative patient. However, if radiographs are of poor quality, through technical difficulties, poor positioning or patient factors, the end result can be a study that is of limited diagnostic use. At worse, critical lesions may be missed or a false diagnosis made. This can happen in general practice and in top end referral practice. This lecture provides information for veterinary surgeons and veterinary nurses on ensuring that diagnostic studies are obtained. It will be illustrated with many examples from clinical practice, as well as brief practical demonstrations aimed to avoid the pitfalls of radiology in general practice.

Get the patient ready

Even in an emergency the time taken to prepare the patient for the study will avoid the need to repeat the radiographs, or accept a non-diagnostic study. Collars or harnesses should be removed if covering the region of interest. Patient restraint is very important. If the patient is un-cooperative the chances are that the positioning will be poor and the study may be wasted. Sedation can greatly help in positioning, and in some cases (such as skull radiographs) general anesthesia may be necessary. However, sedation is not always an option for medical reasons, or due to client concerns. With many patients this would risk producing a non-diagnostic or misleading study. However, it is possible in some circumstances to position and restrain an awake patient and achieve good positioning, at least for studies of the thorax and abdomen, with a few positioning aids.

Positioning Aids

Accurate positioning for radiographs is arguably the most important part of achieving a diagnostic study, but may be the most challenging. Certain aids can massively reduce the difficulty in restraint and correct positioning. They can also make the patient more comfortable (reducing movement) and can help keep anesthetized patients warm! Radiolucent foam troughs and foam pads can be used in the region of interest, and

sandbags are extremely helpful in patient restraint. Ties can be used for limb positioning. This doesn't necessarily require much investment and homemade devices can work! With a co-operative assistant patient some methods of patient restraint will be demonstrated during the lecture.

Positioning

The exact position required depends on the study that you are taking. There are several excellent texts that relate to accurate positioning for each study, and I will not go in to detail here. The rules are that the patient should be as straight as possible, and the entire region of interest included, with the radiograph ideally collimated to that area. The region of interest, such as in musculoskeletal imaging, should be parallel to the x-ray beam. Clinical examples will be used to illustrate the effect that poor patient positioning can have on diagnosis. Many of these may result in a false diagnosis of pathology, such as fracture.

Technique

Everyone is familiar with underexposed or overexposed radiographs. It is easy to accept this as standard when it can result in poor quality radiographs. Although there is a wider range of techniques that result in acceptable digital radiographs, an overexposed digital radiograph is in some ways worse than an overexposed film screen radiograph, which can be reviewed using a bright light. Overexposure in digital systems results in complete loss of data which could be misinterpreted as free gas or lysis. Having a good technique chart for your system can avoid this fundamental error.

Kilovoltage peak (kVp) indicates the penetrating power of the x-ray. The milliamperage / time (mAs) determines the intensity of the primary beam. As a general rule the shortest time possible should be selected to avoid motion artifact. If it is not possible to select time and mA separately, an increased kV can be used to compensate for a lower mAs. A 15% increase in kVp is equivalent to approximately a 50% increase in mAs. Particularly for abdominal studies a lower kVp is ideal though, as this improves contrast resolution.

Grids should be used for any area thicker than 10cm. These reduce the amount of scatter radiation that reaches the film and improve the quality of the study. The use of a grid necessitates an increase in kVp.

Technique charts

Every practice should have a technique chart set up for their system. This should be available to anyone who is taking radiographs. If one is already in place, it is worth reviewing

it periodically to ensure that the best possible images are obtained. It is relatively easy to review your technique chart, and is strongly recommended if you are starting to see over-exposed (too dark) or under-exposed (too light) radiographs. As screens age it will be necessary to update your charts.

The following is an indication of how you can produce or update your technique chart:

1. Select your mAs. Approximate mAs may be:
    a. Extremity (table top, no grid) 2.5mAs
    b. Thorax 5mAs
    c. Abdomen 7.5mAs
    d. Spine 10mAs
2. This is for a medium screen/film combination. For a slow (detail) combination use double, and for a high speed combination approximately half.
3. Select your initial kVp. This is going to be approximately 2 x tissue thickness (cm) + FFD (film focus distance, usually 40) + grid factor. Most grids are 8:1 for which you add 8-10 kVp.
4. Expose a film. For an abdomen measuring 10cm thick you would use (2x10)+40+8 = 68kVp
5. If the radiograph is too dark increase the kVp by 15%
6. If the radiograph is too light decrease the kVp by 15%
7. Make a chart
    a. add 2kVp for each cm increase up to 80kVp
    b. subtract 2kVp for each cm decrease
    c. add 3kVp for each cm increase the places the kVp from 80-100
    d. add 4kVp for each kVp that places kVp over 100
8. Repeat for each type of study (Thorax, Abdomen, Extremity, Spine)

Digital systems

Setting up technique on the digital system is slightly more complex. As well as having appropriate exposure factors, the radiographs are also post processed according to the region imaged. The post-processing is typically setup by the manufacture at the time of installment but this can be reviewed, and it is important that the diagnostic result is not compromised, for example by a technique that results in very high contrast but inability to distinguish subtle lesions. If you are thinking of ‘going digital’ then your choice of system can have an enormous impact on the quality of your studies. Screen film does have a higher inherent spatial resolution than digital radiography, and a poor quality digital system could significantly reduce the diagnostic quality of your radiography.

Trouble shooting

If your radiographs are already too dark or too light, reviewing a technique chart could be very helpful. Other factors will play in to the apparently improperly exposed radiograph, and darkroom factors (for regular screen film radiography) could be a problem. The flow chart below may help you identify the source of a problem[1].

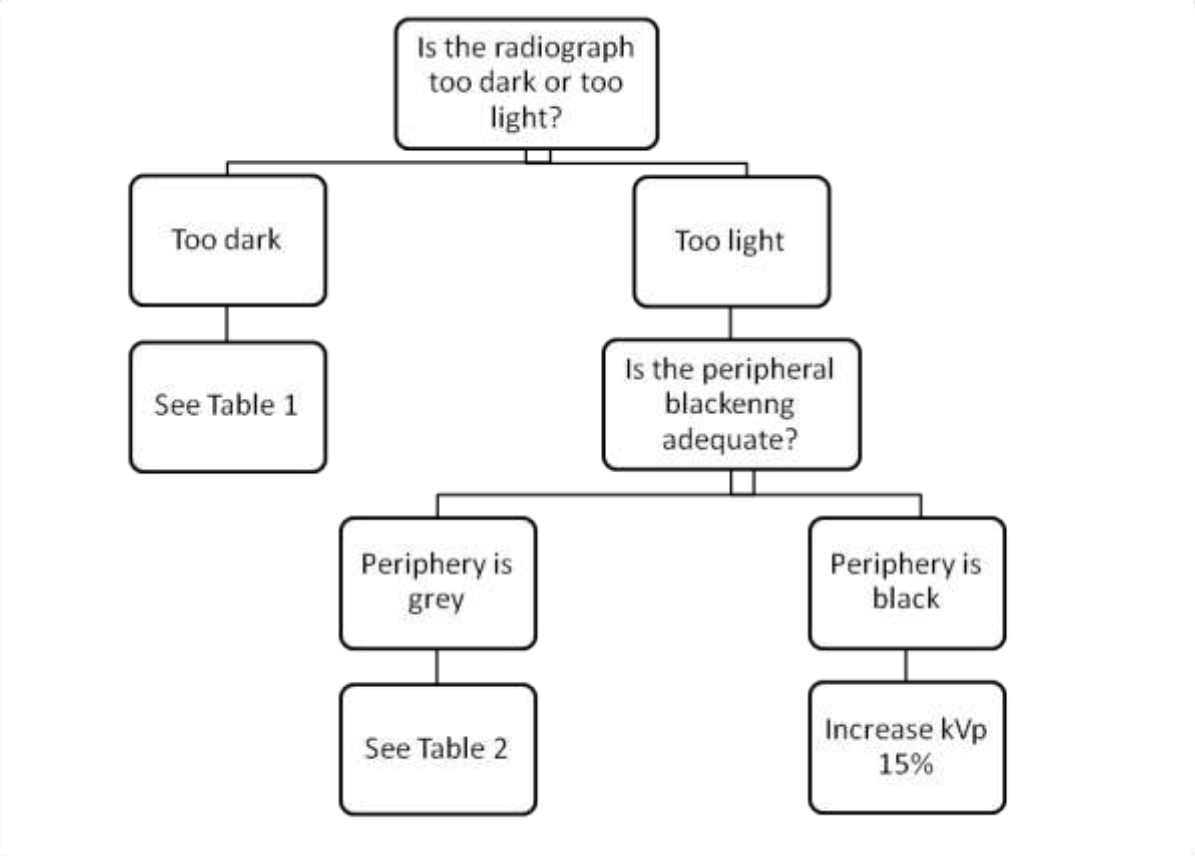

Table 1: Film is too dark

Common Causes	Processor and dark room problems (screen film)	Rare causes
Technique is too high Try decreasing mAs by 50% or kVp by 15%	Developer too strong	X-ray machine miscalibration
Double exposure	Developer Temperature too high	X-ray machine timer malfunction
Tube height too low	Processor timer malfunction	
	Light fog in the dark room	
	Safety light malfunction	

1 Adapted from a chart that is available on the Animal Insides website www.animalinsides.com by kind permission of Dr Matthew Wright

Table 2: Film is too light

Common causes	Processor problems (screen film)	Rare causes
Technique too low **Try 50% increase in mAs**	Developer exhausted	X-ray tube miscalibration
Wrong technique chart	Developer diluted	X-ray tube failure
Wrong patient measurement	Developer temperature too low	X-ray tube timer malfunction
X-ray tube too high	Processor timer malfunction	
X-ray tube not aligned to grid		

What constitutes a diagnostic study?

Once you have got the positioning and technique right, with each patient you need to determine which radiographs are necessary. There is a golden rule that applies to all but a select few cases – always, always take orthogonal views. The best radiologist in the world cannot isolate the location of a lesion from a single two dimensional radiograph, and a single lateral view of the thorax will only ever provide evaluation of the lung lobes on one side (potentially missing severe pneumonia or pulmonary metastases). A study of the thorax should always be two views (right or left lateral and VD or DV), and arguably three views, particularly if looking for metastases. An abdomen should also be two views (right or left lateral and a VD) so that any lesion can be localized and not missed. For musculoskeletal imaging, orthogonal views are essential and additional oblique or stressed views may be necessary in some circumstances. There are exceptions – a VD screening view of the hips, lateral view of the elbows, or lateral view of the abdomen to look for calculi within the urinary bladder – but these should be kept to a minimum. It can certainly be difficult to achieve this standard if you charge for each radiograph, and in many cases charging 'per study' can work out better without impacting the client. This is particularly true if you have a digital system, where the cost to the practice of taking an extra film is minimal.

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TOP TEN EASILY MISSED KEY RADIOGRAPHIC FINDINGS

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The importance of making a correct radiographic diagnosis, particularly in an emergency situation, cannot be under-estimated. Often radiographs are acquired to rule out or confirm suspected lesions, and it is essential that these are not missed or mis-interpreted. Unexpected diagnoses can completely change a treatment plan and may lead to emergency intervention that was not otherwise considered necessary. Unfortunately some of the most important radiographic diagnoses can be those that are most easily missed. This lecture will review a "Top ten" of these key radiographic findings with discussion of how best to improve diagnosis, illustrated by examples from clinical practice. The list is drawn from my experience of reading cases for general and emergency practice and through appreciating what is often missed by the attending clinician, even when experienced. It is of course not exhaustive, and could go on almost indefinitely, but within the limitations of this lecture I hope to provide some helpful advice to improve your ability to make the correct key radiographic diagnosis.

1. Pneumoperitoneum

Even very small amounts of free peritoneal gas can indicate a need for surgical intervention. The sooner a diagnosis of a ruptured viscus is made, the greater the chances that the patient will recover from surgical treatment of associated septic peritonitis. Free gas within the abdomen typically localizes to certain areas, and careful evaluation of these in a patient presenting with an acute abdomen can improve sensitivity to the diagnosis. Gas will almost always move to the non-dependent aspect of the abdomen. It is typically localized adjacent to the dorsal aspect of the diaphragm and along the dorsal margin of the liver lobes. This location is very typical for gastric perforation. With small intestinal perforation the gas may initially be localized to a focal area of the abdomen. Peritoneal gas pockets are initially often small and rounded and do not conform to the intestinal margins. Gas may also be seen along the body wall, and have a somewhat linear distribution. If in doubt, further imaging can help to confirm or dismiss a suspicion of free gas. In a patient that is clinically stable, follow up radiographs could be obtained in 2 hours. Increasing amounts of gas will almost always be present within that time frame. However, if possible, it would always be best to

use the position of the patient to help confirm the diagnosis. A horizontal beam radiograph obtained with the patient in left lateral recumbency is a much more sensitive way to look for free gas in the non-dependent aspect of the abdomen. If you cannot move the x-ray tube to obtain a horizontal beam, adding an additional view (left lateral for example) may help.

2. Unilateral pulmonary disease

The right middle lung lobe is a predilection site for bacterial bronchopneumonia, especially in cases of aspiration. Most standard protocols for thoracic imaging comprise a right lateral and ventrodorsal view. Even worse, a single view may be acquired. The dependent pulmonary parenchyma is not assessed on that lateral view. A right lateral view only provides evaluation of the left pulmonary parenchyma. Subtle changes within the right middle lung lobe can readily be missed on ventrodorsal or dorsoventral images, even by the most experienced radiologist. Adding a left lateral view to your protocol when pneumonia is suspected is an easy fix!

3. Peritoneal effusion

Effusion within the abdomen can have many origins. In the acute abdomen this is most often present as haemoabdomen associated with intra-abdominal neoplasia, peritonitis associated with pancreatitis or uroabdomen. Effusion can also be associated with right sided cardiac disease, portal hypertension, hypoalbuinaemia, carcinomatosis or other causes of peritonitis. Detecting effusion on radiographs can be key in making a correct diagnosis or adding an important differential. The diagnostic quality of your radiographs can be really important in the ability to detect subtle changes in serosal detail that indicate mild effusion. Evaluation for loss of delineation of the serosal margins of the intra-abdominal organs is an important part of abdominal radiograph evaluation and will be illustrated by case examples. Thin patients and young animals can have a normal decreased serosal delineation leading to mis-diagnosis. Abdominal ultrasound can be used to confirm suspected effusion, but is not always available in general practice.

4. Retroperitoneal effusion

The retroperitoneal space, housing the kidneys, adrenals, some lymph nodes and the great vessels, is separated from the peritoneal cavity by the peritoneal membrane. While retroperitoneal effusion is not very common, the ability to differentiate this from peritoneal disease can really help to narrow a list of differentials in a patient presenting with abdominal signs. Retroperitoneal effusion can be associated with haemorrhage, urine leakage, neoplasia and acute renal inflammation. Radiographically it is distributed dorsally on the lateral views, often with ventral displacement of the colon or small intestines. As it is

specifically retroperitoneal serosal detail that is limited, the margins of the kidneys will be indistinct on the affected side, but the serosal margins of the peritoneal organs should remain intact. If ultrasound is not available, further imaging such as an IVP study may be helpful in further evaluation.

5. Pneumothorax

Free pleural gas is both easily missed and easily over-diagnosed. It can readily be created artifactually by skin folds or over-exposure, but subtle pneumothorax is also easily under-interpreted. While a diagnosis of pneumothorax is often made on clinical examination in some circumstances this has not been possible or the diagnosis is unexpected. Pneumothorax associated with trauma is most common, and may require thoracocentesis. Spontaneous pneumothorax can occur secondary to rupture of pulmonary bullae, necrotic neoplastic lesions, abscesses or in association with severe parenchymal disease (including feline asthma). Evaluation for the underlying cause can be challenging on radiographs. Evaluation for pulmonary bullae or small volume pneumothorax is one circumstance when an expiratory rather than inspiratory radiograph can be helpful.

6. Linear foreign body

A linear foreign body is a surgical emergency. They may be more prone to perforation than simple mechanical obstruction. Carpet material, for example, is highly abrasive to the mucosal surface. They can also rapidly progress to involve the entire small intestine, as peristalsis draws the anchored foreign material distally. While some may be diagnosed by virtue of entrapment under or around the tongue, many linear foreign bodies can be anchored in the pylorus. Unlike simple mechanical obstruction, linear foreign material does not necessarily result in focal intestinal distension. The key is to appreciate the radiographic signs of plication. Elliptical gas lucencies, bunched intestine and the plicated serosal margin of intestine can be appreciated. Clinical examples will be used to illustrate this often challenging diagnosis. Beware the obese cat! Apparent bunching of intestine in the right abdomen can be a normal distribution. If in doubt a contrast study or ultrasound (in experienced hands) can confirm the diagnosis.

7. Rib fractures

We always look for rib fractures in trauma patients, but they are surprisingly easy to miss on radiographs, even when there is a strong clinical suspicion that they may be present. Fractured ribs can be extremely painful, so knowledge that they are there may affect pain management. The associated pain and damage to the intercostal muscles can also affect breathing patterns and may affect your interpretation of a dyspnoeic patient. Multiple

adjacent rib fractures can also result in a 'flail chest' where there is paradoxic movement of the thoracic wall. Fractured ribs are not confined to trauma cases either. They can occur secondary to dyspnea in asthmatic cats. Pathologic fractures associated with primary or metastatic neoplasia can also occur and may be unexpected. This important observation could lead to a change in diagnostics or patient management. A ventrodorsal or dorsoventral view is essential in evaluation for rib fractures. They are extremely difficult to appreciate on a lateral view. However, turning a lateral view 90º can be helpful. The abnormal orientation will lead you to focus on the ribs rather than the intra-thoracic structures!

8. Mediastinal lesion

The ability to differentiate a mediastinal from a pulmonary lesion or to appreciate a subtle mediastinal mass effect can significantly narrow your differentials. There are relatively few causes of a mediastinal mass, particularly cranioventrally. Lymphadenopathy, thymoma, ectopic thyroid carcinoma, abscess, granuloma or cyst are the typical differentials for a cranioventral mediastinal lesion. Other neoplasia can occur in the mediastinal space both dorsally and caudally. Occasionally haemorrhage may be confined to the mediastinum. Evaluation of both lateral and ventrodorsal / dorsoventral views is key in making the correct anatomic diagnosis. Mediastinal lesions are typically centrally located and will result in mediastinal widening. Larger lesions will cause displacement of other mediastinal structures such as the trachea or cardiac silhouette. Caudal displacement of the carina, for example, is characteristic of a large cranial mediastinal lesion. Subsequent evaluation could include ultrasound, biopsy and CT planning for surgery, but it is very helpful to make the initial diagnosis radiographically.

9. Cardiomegaly – over-interpretation

This isn't a missed finding, but a readily mis-interpreted one. Cardiomegaly and cardiac failure is frequently mis-diagnosed and can result in inappropriate patient management. This is often breed associated. Small breeds often have subjective cardiomegaly in the absence of cardiac disease. While increased sternal contact is associated with right sided cardiac disease this can be a normal finding in breeds such as Yorkshire Terriers. A radiograph that is not obtained on full inflation (not necessarily as a fault of technique – upper respiratory, pulmonary or pleural disease can result in under-inflation) will result in a cardiac silhouette that is relatively larger in comparison to the thoracic cavity. The vertebral heart scale can be useful as an objective measurement, but there are many canine breeds that have been found to have a normal cardiac size larger than the reported cut-off of 10.5. The Labrador Retriever is one of these breeds where subjective cardiac enlargement is a frequent finding,

and the vertebral heart score can be higher in normal animals. Conversely it is easy to under-estimate cardiac enlargement in feline patients and deep-chested canine breeds such as the Doberman. As both are pre-disposed to forms of cardiomyopathy this can be a real problem. Examples will be used to illustrate the variety of normal and abnormal cardiac size in certain breeds.

10. Pathologic fracture

Differentiating a primary traumatic from pathologic fracture can have enormous implication for patient management. While cortical destruction, 'sun-burst' periosteal proliferation and intramedullary lysis are typical, these changes can be subtle and variable and difficult to appreciate in the face of an already fractured bone. Evaluation for any evidence of periosteal new bone in an acute fracture, evidence of subtle cortical changes or medullary heterogeneity could lead to a need to biopsy rather than perform a simple fracture repair or stabilisation.

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ASSESSMENT OF THE DYSPNOEIC EMERGENCY PATIENT

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The patient presenting with dyspnoea represents both a diagnostic and therapeutic challenge. These patients are very fragile and the restraint necessary to perform diagnostic tests or administer therapy may precipitate sudden deterioration or even death. Both diagnostic and therapeutic interventions must be carefully considered in terms of their potential risk:benefit for any individual patient.

Bearing this in mind it is often necessary to institute empirical therapy with the aim of stabilising the patient prior to a complete diagnostic evaluation. Diagnostic tests such as radiography may be fatal and the clinician dealing with emergencies should aim to achieve as much information as possible from a careful physical examination. Empirical therapy should be chosen logically. A careful consideration of the patient's history coupled with a physical examination of the respiratory system however brief allows the clinician to choose therapy rationally and maximises the chance that the therapy will be effective. The primary aim when evaluating a dyspnoeic patient is to localise the anatomical level from which the dyspnoea originates as this will guide initial stabilisation.

The anatomical levels are:

- Upper airway (nose to carina)
- Lower airway
- Parenchymal
- Pleural space
- Ventilatory apparatus (chest wall, diaphragm)
- Miscellaneous
    - Neurological
    - Metabolic

Assessment of the dyspnoeic patient

As with all patients, assessment begins with a history and physical examination.

History

If the patient is severely dyspnoeic it may be advisable to remove the patient from the owner and place it in an oxygen enriched environment before the history is taken. However the animal's signalment and history may be helpful in making and ordering the differential diagnosis list so should be taken even if retrospectively.

Signalment rarely allows complete exclusion of differential diagnoses however may allow refinement of the differential list. For example pulmonary fibrosis would be a moderately common differential in an older Terrier (especially WHWT) whereas lungworm (*A. vasorum)* would be commoner in younger patients especially in the south (but spreading north!).

Important historical information includes any previous medical history especially episodes of breathlessness/exercise intolerance, recent unusual events especially any trauma or vomiting/regurgitation episodes, and the progression of the problem. The efficacy of any prior treatment is also worth noting.

Physical examination

The ability to interpret abnormalities of the respiratory system accurately relies on a good knowledge and experience database of the normal patient. Normal dogs and cats have a respiratory rate of 15-25 breaths per minutes with only a small amount of thoracic and even less abdominal movement. It is important to note however that the thorax and abdomen move together and in the same direction i.e. on inspiration the thoracic cavity expands with the ribcage moving outwards and the diaphragm flattens meaning the abdomen also moves outwards. Normal lung sounds are fairly quiet but can still be heard. They are slightly louder ventrally as there is more lung mass there although the sound of the heart must be "edited out" to appreciate this. Sounds should be bilaterally symmetrical.

Dyspnoeic patients have increased respiratory effort and probably rate. The physical examination can be used as subjective marker of the severity of respiratory distress. As respiratory effort increases, thoracic and abdominal wall movement become more obvious. Paradoxical abdominal movement may develop. This occurs with severe inspiratory effort where the diaphragm is effectively sucked forward by the strength of the intercostal muscle contraction - hence the abdomen moves in whilst the thoracic wall moves outwards. Paradoxical abdominal movement is most commonly seen with upper airway obstruction, severe parenchymal disease or severe chronic pleural effusions. Postural manifestations of dyspnoea such as open mouth breathing, extended neck, abducted elbows and an anxious facial expression may be seen. Some patients will appear distressed by the respiratory effort

whereas others may seem more behaviourally normal; the second group commonly have a more insidious onset to their respiratory signs.

Cyanosis may be detectable on examination of the mucous membranes. Detection of cyanosis requires a capillary deoxygenated haemoglobin level of approximately 5 g/dL. Considering dogs with normal haematocrits of 45% have a haemoglobin level of around 15g/dL, roughly one third of a dog's haemoglobin must be deoxygenated before cyanosis is detectable, thus it always represents severe hypoxia.

Before performing auscultation, the respiratory pattern should be observed and the dyspnoea categorised as inspiratory, expiratory or mixed. It should also be noted whether increased noise can be heard externally (i.e. without a stethoscope). Increased audible noise is invariably associated with upper respiratory tract problems such as laryngeal paralysis or brachycephalic obstructive airway syndrome. Auscultation of the chest should finally be performed, although in patients with severe dyspnoea, this may be delayed until the animal has spent some time receiving supplemental oxygen. Sounds should be categorised as to whether they are louder or quieter *than expected for the degree of respiratory effort.*

Increased lung sounds may be described as referred upper airway, harsh lung sounds, crackles or wheezes. The decision as to whether increased lung sounds simply reflect referred upper airway noise can be aided by listening over the cervical trachea. Decreased lung sounds are most commonly heard with pleural space disease although can occasionally be found with significant hypoventilation. It is really important that an assessment is made of whether the lung sounds are decreased relative to the effort the patient is making. For example a dog with pneumothorax and significantly increased respiratory effort may have lung sounds of similar audibility to a normal dog breathing normally. It is the relative quietness for the degree of effort that clues the clinician in to the presence of a pneumothorax rather than the absolute volume/loudness. The distribution of any abnormal noises should be noted.

Following a close observation of respiratory pattern and careful thoracic auscultation, it should be possible to define the most likely site of origin of the dyspnoea. This is vitally important for logical empirical treatment measures and is summarised in the table following.

Site	Respiratory pattern	Audible noise	Auscultation findings	Disease Example
Upper airway	Inspiratory effort	Yes	Referred upper airway noise	Laryngeal paralysis BOAS
Lower airways	Expiratory effort	No	Expiratory wheezes	Feline asthma
Pulmonary parenchyma	Mainly inspiratory but mixed patterns possible	No	Harsh sounds and/or crackles	Pulmonary oedema Pneumonia Pulmonary contusions
Pleural space	Short shallow inspiration	No	Dull lung sounds	Pleural effusion Pneumothorax

Assessing severity of dyspnoea

Further evaluation of the severity of the problem should include pulse oximetry and ideally arterial blood gas analysis. Imaging techniques are helpful in determining aetiology but are unfortunately of little use in assessing the functional status of the lung.

Pulse oximetry is widely available but has several limitations. Firstly, a good pulse wave is required for an accurate reading and many patients with severe dyspnoea have concurrent problems such as cardiac disease or hypovolaemic/distributive shock meaning a strong, regular pulse wave is not present in the peripheral circulation. Secondly, due to the sigmoid nature of the oxyhaemoglobin dissociation curve, a relatively comforting pulse oximetry reading of 90-93% corresponds to an arterial oxygen partial pressure (PaO_2) of around 60mmHg where a relatively small further drop in the PaO_2 will result in a rapid and precipitous drop in haemoglobin saturation and the pulse oximetry reading. Thirdly the presence of abnormal haemoglobin (e.g. methaemoglobin) may interfere with results. And finally, many of our patients either have pigmented skin or are unco-operative which makes it difficult to achieve an accurate value.

Arterial blood gas analysis is the most accurate way of assessing arterial oxygen levels. With the advent of relatively affordable in-house blood gas machines, more veterinary surgeons now have access to this and it is likely to continue to increase in the future. Arterial blood is usually obtained from the dorsal metatarsal artery in patients over 5kg and from the femoral artery in patients weighing less than this. The arterial partial pressure of oxygen (PaO_2) should be approximately five times the fractional inspired oxygen, thus on room air (21% oxygen), a normal animal should have a PaO_2 of 85-110 mmHg. Values less than 60mmHg

are cause for severe concern. Arterial blood gas also allows measurement of arterial CO_2 which allows assessment of ventilation as well as oxygenation status.

Further diagnostic tests

Further diagnostic testing is likely to be required at some point to pursue the specific diagnosis.

With pleural space disease, thoracocentesis is not only therapeutic but also often diagnostic. Full use should be made of any pleural fluid obtained. The fluid should be described grossly and a SG used to classify it as a transudate or modified transudate (SG < 1.018) or exudates. Cytology should be performed on both a direct smear and potentially a centrifuged sample. Although some diagnoses will require the input of a skilled clinical pathologist (e.g. neoplasia), emergency clinicians should be encouraged to evaluate their own cytology. Some diagnoses especially where the underlying process is septic can be easily made and prompt recognition allows early specific treatment. Fluid should also be submitted for culture if a septic process is suspected. If the clinician is unsure whether a pleural effusion is present or not, ultrasound provides a very useful and relatively safe way of confirming this. Ultrasound is recommended as opposed to radiography as it is simpler and quicker to perform and causes less stress to the patient.

Obtaining samples for airway cytology is often part of the diagnostic process in patients with suspected parenchymal or lower airway disease. Various techniques can be used including

- Transtracheal wash
- Endotracheal wash
- Bronchoscopy with bronchoalveolar lavage

Although endoscopy allows washes to be obtained from specific lung lobes it is not always practical or safe for this procedure to occur in emergency patients.

Transtracheal wash can be performed using equipment that is readily available in most practices. The patient is restrained in a sitting position and the neck clipped as if for a tracheostomy. The area is then prepped aseptically. The trachea is palpated and a local anaesthetic (commonly lignocaine) is infiltrated under the skin and down to the level of the tracheal rings. Various catheters can be used for the wash itself but we most commonly use a 14 or 16 Fr shore over the needle IV catheter through which we thread a 4 Fr long urinary catheter. The trachea is palpated and stabilised between the thumb and forefinger of one

hand. Using the other hand, the IV catheter is guided between two tracheal rings (the precise level is unimportant). The catheter can usually be felt “popping” into the tracheal lumen. The catheter is then angled distally, the catheter threaded off the stylet and the stylet withdrawn. A long urinary catheter is then threaded through the IV catheter. Once in the urinary catheter is in place, a small volume of sterile saline is injected through it and the chest is gently coupaged. The syringe used to inject the saline is left attached to the catheter and almost immediately negative pressure is applied to the plunger. The diagnostic sample will be sucked back into the syringe – it is very likely that only a small volume will be obtained. The process of injecting saline and then aspirating can be repeated 2-3 times. The volume of saline is variable dependent on patient size but as a rule I use 3-5ml saline in a 10ml syringe for small and medium sized patients and 5-8ml saline in a 20ml syringe for large patients. Following the procedure a light neck wrap is placed to reduce the risk of subcutaneous emphysema developing.

Transtracheal wash can be performed on most patients greater than 15kg in weight without sedation although may be technically more difficult in patients with fat necks. For patients less than 15kg a short anaesthetic with intubation and an endotracheal wash is recommended. Transtracheal wash is a useful technique and can be used to obtain diagnostic samples (e.g. for culture) before empirical treatment is started.

Imaging techniques are also commonly used in the diagnosis of dyspnoeic patients although it must be remembers their use should ideally be delayed until after initial stabilisation as they can be stressful to patients.

STABILISATION OF THE DYSPNOEIC EMERGENCY PATIENT

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Dyspnoeic patients are a particularly fragile patient population where empirical treatment may need to be started following a careful physical examination but before a definitive diagnosis is achieved. There are however several management strategies that should be adopted whatever the cause of the dyspnoea and other treatments that can be considered once the anatomical source of the dyspnoea is localised.

General treatment strategies

Minimise stress

Animals with severe hypoxaemia will limit their movement and therefore have sufficient oxygen delivery to support major organ systems. Any increase in non-essential tissue oxygen consumption may prove life threatening. Most especially, increased skeletal muscle activity, such as may occur with restraint or agitation, may precipitate cardiorespiratory arrest. All patients with severe lung disease should have their activity restricted and any stressful procedures such as catheter placement or radiography should be carried out cautiously and incrementally, allowing time for recovery between steps.

Oxygen supplementation

All animals with severe lung disease will benefit from oxygen supplementation. Theoretically in patients with chronic hypercarbia and hypoxia, oxygen supplementation may lead to a lessening of their respiratory drive and precipitate worsening of their condition. This situation is very rare in clinical veterinary patients and should not be a reason to avoid oxygen supplementation. Oxygen can be supplemented in a variety of ways with the particular method used dependent on practice facilities and the patient. Care should be taken that the method employed does not cause undue stress to the patient.

Oxygen supplementation options include:

Flow-by – anaesthesia tubing with a relatively high oxygen flow rate is held close to the patient's nose or mouth. This technique is minimally invasive and is unlikely to cause significant stress to the patient; however the increases in fractional inspired oxygen are not great. This method is most commonly employed in the early stages after patient admission during initial examination and at later stages when procedures are being performed.

Mask – oxygen is delivered via a face mask designed for veterinary use. Similar to flow-by this method is most commonly used in the early stages of patient evaluation or in patients that are markedly mentally depressed/sedated. It is often not well tolerated especially by fully conscious patients – if the patient struggles its face should not be forcibly held in the mask. In this situation, flow-by is recommended.

Nasal cannulation – nasal prongs designed for human use can be utilised but often do not stay in the dogs' nares terribly well – I often find I am supplementing the dog's eyeballs with oxygen!!! An alternative is to place a nasal cannula. Any flexible tube such as a feeding tube is adequate. It is placed in the same way as a naso-oesophageal feeding tube but is measured such that the tube finishes in the nasopharynx. This can be approximated by premeasuring the tube to the level of the medial canthus. The end of the tube then needs to be connected to an oxygen source. Specific adapters are not generally available and a little ingenuity is required but a combination of male/female adapters and tape can usually be used. Relatively high fractional inspired oxygen levels of 40-60% can be achieved in this way. The factor limiting increased FiO_2 is the nasopharyngeal irritation caused by high flow rates – nasal catheters can be placed bilaterally such that flow rates can be maximised with minimal discomfort to the patient. The oxygen should be humidified if possible.

Oxygen cages - oxygen cages provide an oxygen enriched environment (up to 90%) with full temperature and humidity control and are the ideal way to supplement oxygen in a manner that is non stressful for the patient. They are however expensive and not widely available especially for larger patients. They also limit access to the patient which can make monitoring challenging. Improvised oxygen cages or hoods can be created using normal kennels or Elizabethan collars with cling film or similar over the front. The increase in fractional inspired oxygen (FiO_2) achieved with improvised cages is variable but it is possible that an FiO_2 of 60% may be reached. However "home made" oxygen cages are very prone to the build-up of temperature, humidity and CO_2 which makes them unsuitable for long term use.

Positive pressure ventilation

Positive pressure ventilation (PPV) may be necessary if arterial oxygenation cannot be maintained with less invasive methods of oxygen supplementation. Patients showing signs of severe hypoventilation or impending respiratory muscle fatigue would also be considered candidates for PPV. Generally speaking, PPV should be considered in animals with a PaO_2 less than 60mmHg (pulse oximetry reading of about 90%) on oxygen supplementation or a $PaCO_2$ greater than 60mmHg. The use of PPV allows the delivery of high fractional inspired

oxygen concentrations and the use of techniques such as positive end expiratory pressure (PEEP) to aid oxygenation. Whilst advanced intensive care facilities are required both in terms of personnel and equipment, in patients with severe yet reversible underlying disease, PPV can be very rewarding. A retrospective study of dogs with severe pulmonary contusions post trauma that required PPV showed a survival rate of 30%.

Positioning

Body position can significantly affect arterial oxygen concentration and there is a growing body of evidence from human clinical and animal experimental studies that prone positioning (sternal recumbency) can significantly improve PaO_2 in some patients. Placing or propping the animal in sternal recumbency may therefore be a simple yet effective way of increasing oxygenation.

Specific therapy

As discussed in the lecture on assessment of the dyspnoeic patient, the most important decision a clinician can make is the probable anatomical localisation of the dyspnoea. This allows rational empirical treatment with a good chance of aiding patient stabilisation to be employed.

Upper airway

Patients with dyspnoea secondary to upper airway obstruction frequently respond well to sedation (acepromazine 0.01-0.05 mg/kg iv) and oxygen supplementation. Patients develop a vicious circle of mild respiratory difficulty followed by increased effort that then causes worse airway collapse and worsening dyspnoea. Breaking this cycle with sedation can often result in successful (although temporary) resolution of the problem. It should also be remembered that the dog's main method of thermoregulation is via panting and thus these patients often develop a significant hyperthermia that then contributes to the problem – active cooling measures may need to be instituted. In rare cases, it is necessary to anaesthetise and intubate the dog temporarily. Many of these patients can then be recovered from anaesthesia slowly and, by breaking the cycle as described above, are much improved on recovery. Emergency tracheostomy is rarely necessary.

Lower airway

Feline asthma is the most important small airway disease that causes clinical signs of dyspnoea in the small animal patient. Although dogs may suffer from chronic bronchitis, this more commonly causes a cough as opposed to dyspnoea. If feline asthma is suspected, treatment with injectable (im or iv) corticosteroids and bronchodilators (e.g. terbutaline) is

recommended. Response may be rapid. Inhaled medications are also useful especially if the cat is familiar with this route – in patients presenting for the first time the stress of receiving inhaled medication makes it less useful.

Pleural space disease

Thoracocentesis is the recommended treatment for patients where pleural space disease is suspected and can provide a rapid improvement in the level of dyspnoea. It may also yield a sample that is helpful for diagnosis. Thoracocentesis is usually performed using a butterfly catheter in cats and small dogs and a needle or over-the-needle intravenous catheter in larger dogs. The precise site for needle introduction depends on the physical examination and where the clinician identifies areas of dullness, however the 7-10th intercostals rib spaces are commonly used. The needle should always be introduced off the cranial aspect of a rib to avoid traumatising the intercostal vessels and nerve. Most patients tolerate the procedure conscious although sedation or local anaesthesia may be necessary in more fractious animals.

Parenchymal disease

Parenchymal disease represents the most diffuse group of diseases and with mild parenchymal disease a specific diagnosis should be sought and specific treatment introduced. However with severe dyspnoea, some drugs may be given empirically, knowing that they are likely to be of benefit whatever the cause of the parenchymal disease. Frusemide is clearly the initial drug of choice for patients with cardiogenic oedema, however there is also increasing evidence that frusemide may be of benefit in patients with other forms of oedema including the permeability oedema seen with severe pneumonia, ARDS and pulmonary trauma. Although its principal action is diuresis, it also has beneficial vasoactive effects including venodilation, especially of the pulmonary veins, and it may increase perfusion to ventilated areas of the lung. An area of experimental investigation is the use of agents which speed alveolar fluid reabsorption. Drugs under investigation for this purpose include the β_2 agonists (e.g. dobutamine, salmeterol, terbutaline) and cAMP phosphodiesterase inhibitors. Finally, considering how detrimental stress can be, sedation may be indicated in these patients. Morphine represents a good choice of sedative agent as it also has venodilating properties and the respiratory depressant effects are minimal in veterinary small animal patients.

The rationale for intravenous fluid therapy should also be carefully considered in patients with severe parenchymal disease. Fluid therapy will tend to increase pulmonary capillary hydrostatic pressure. As this is the major determinant of pulmonary fluid extravasation, fluid

therapy has the potential to worsen dyspnoea. This effect is not restricted to animals suffering from cardiogenic oedema but will also occur in animals with other conditions such as pulmonary contusions or bacterial pneumonia. Some of these animals will have concurrent indications for intravenous fluid therapy such as hypovolaemia or dehydration. The rate and amount of fluids administered should be carefully tailored to both the cardiovascular and respiratory needs of the patient. This may mean giving fluids cautiously and at lower rates and volumes than might otherwise be chosen.

Other medical therapies such as antibiotics, anthelmintics or cardiac medications may well be necessary, however ideally should be used only once a diagnosis or at least diagnostic samples have been achieved.

The trauma patient with respiratory distress – some specific notes

Many patients that suffer significant trauma will have a degree of respiratory distress on presentation. The commonest causes of dyspnoea in trauma patients (in approximate order frequency) are:

- Pulmonary contusions
- Pneumothorax
- Chest wall injuries (rib fractures)
- Diaphragmatic rupture

And much less commonly:

- Major airway rupture
- Haemothorax

Rational stabilisation measures can be employed on the basis of the physical examination. Although contusions cannot be treated specifically recognition that they are present may inform the patient's fluid therapy plan – a les aggressive fluid therapy strategy will help protect from worsening of the contusions. The vast majority of traumatic pneumothoraces can be managed by thoracocentesis alone – very rarely it may be necessary to place a chest drain. As a general guide a chest drain should only be placed if multiple thoracocentesis within a short time frame (hours) are required to stabilise the patient. Chest wall injuries are generally managed by aggressive analgesia. Opioids or local anaesthetic blocks are recommended as they have minimal effect on the cardiovascular system. True flail chest requiring surgical management is very rare. Diaphragmatic rupture as the major cause of

dyspnoea is fairly uncommon and is rarely a surgical emergency the exception being if a gas filled abdominal viscera enter the thoracic cavity.

GETTING MORE OUT OF IMAGING: CONTRAST RADIOLOGY IN GENERAL PRACTICE

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The use of radiographic contrast can provide important diagnostic information above that of survey radiographs alone. Upper GI studies are the most frequently performed contrast study in general practice, often to rule out the presence of gastrointestinal obstruction. Contrast evaluation of the gastrointestinal tract, urinary tract and in some cases the musculoskeletal system are all possible in general practice without specialist equipment or expertise. The methods discussed in this lecture can be extremely useful if ultrasound is not available or if referral is not an option for the client. This lecture will discuss the indications for, methods and interpretation of contrast studies in general practice. The accompanying workshop will expand on this topic and provide further cases for interpretation and discussion. This is necessarily a limited overview, and further details on procedures and interpretation may be obtained from the references provided.

1. Contrast evaluation of the gastrointestinal tract

Upper GI study

The upper GI study is an excellent method for additional evaluation of the gastrointestinal tract when ultrasound expertise is not available. The interpretation of an upper GI study is arguably easier than ultrasound, and can be a more definitive means for excluding surgical obstruction in general practice. It is essential that the procedure is performed with an adequate volume of contrast and with orthogonal views obtained at regular intervals for this procedure to be diagnostic.

Indications

Suspected gastrointestinal obstruction is the foremost indication for an upper GI study in general practice. This can help determine if surgical intervention is necessary when survey radiographs are not definitive or difficult to interpret. If the patient is clinically stable and survey radiographs are inconclusive I would usually initially recommend follow up fasted survey radiographs in 4-12 hours after any necessary fluid therapy and medical management. Obstructive patterns are either static or progressive.

An upper GI study can also be used to investigate chronic vomiting or other upper gastrointestinal signs for which an underlying disease process has not been identified via bloodwork or dietary modification.

Method

The stomach should be empty of food prior to the study. If the procedure is elective then an initial 12 hour fast is recommended. An enema can also reduce the faecal material within the colon for elective studies. Sedation should be avoided due to the effects on gastrointestinal motility, but Acepromazine may be administered at 0.1-0.25mg/kg IM if necessary.

For canine patients 5-10ml of 30% weight per volume of micropulverised liquid barium should be administered by either orogastric tube (ideal) or oral syringe. If necessary, confirmation of the position of the tube can be obtained with a lateral radiograph. The larger dose would be recommended in smaller animals. For feline patients 12-20ml/kg of contrast is necessary for a diagnostic study. A lesser volume can markedly increase gastric emptying time and lead to a false diagnosis of delay and can limit gastric evaluation. Contrast should never be mixed with food for this study. This will also lead to delayed gastric emptying and there will be an irregular appearance to the contrast which can confound interpretation.

Immediately after contrast administration initial radiographs should be obtained. A lateral and ventrodorsal view is necessary as a minimum. If there is any concern for a gastric lesion dorsoventral and opposite lateral views will also be necessary as a filling defect associated with a lesion of the wall may only be visible on one view. Follow up lateral and ventrodorsal views are ideally recommended at 30 min, 1 hr, 2 hrs and until contrast has filled the colon and gastric emptying has occurred. Follow up radiographs obtained at 12 hours can help to confirm that all the contrast reaches the colon. If this sequence is not followed, a partially obstructive surgical lesion or focal intestinal abnormality could be missed. In cats gastrointestinal transit is more rapid and images should also be obtained at 15min.

Other contrast agents can be used. Iodinated contrast is typically recommended if gastrointestinal perforation is suspected. However this does not provide an ideal study. Ionic iodinated contrast is hyperosmolar and will draw water in to the intestines leading to gradual decreased opacification and intestinal dilation which can confuse interpretation. Non-ionic agents are better, but are very expensive for this purpose.

Interpretation

Gastrointestinal obstruction is characterized by focal distension of intestine that persists over multiple time points. The distension can be very focal, or more diffuse on the oral side of the obstruction. There will be a delay in passage of contrast beyond the point of obstruction. However, some fluid may pass, and the presence of contrast beyond this does not exclude an obstructive process. A persistent filling defect is frequently (but not always) seen in association with obstructive foreign material. With linear foreign material the focal distension is often not present. Instead there is plication of intestine rather than normal tubular contrast filled bowel. If obstruction is within the pyloric outflow tract there will be delayed gastric emptying, and if there is foreign material there is often a filling defect in the pyloric antrum.
Gastric outflow obstruction can also occur with neoplasia, pyloric hypertrophy or pylorospasm, in which case the outflow is persistently narrow on multiple views and can have a 'beak like' appearance.

Neoplastic or focal granulomatous inflammatory lesions of the stomach or intestines are characterised by static filling defects within the wall. In the stomach these may only be seen on the initial gastrogram phase of the study.

Normal canine gastric emptying occurs in 30-120 min. It can be delayed by systemic disease and certain medications in addition to gastrointestinal disease. Contrast typically reaches the colon by 30-120min and the small intestine empties in 180-300min. In cats normal gastric emptying occurs in 15-60min and normal small intestinal transit is 30-60min.

BIPS

Small spherical opaque structures called BIPS (barium impregnated polyethylene spheres) are available for use in evaluation of gastrointestinal disease. They come in a large and small size, and the pattern of passage of the BIPS can be associated with certain gastrointestinal disease. However, the use of these has generally fallen out of favour and I find them to be of limited use in evaluation of the gastrointestinal tract, as there are many factors that influence their passage. A barium upper GI study is considered more reliable in evaluating for obstructive disease.

Pneumocolonogram

Diagnosis of an obstructive pattern on survey radiographs relies on the differentiation of small intestine from colon, and this is not always easy. A pneumocolonogram involves the administration of gas (room air is fine) in to the colon. Mild distension of the colon with gas

will often confirm its location as compared to potentially distended bowel. Caution should be taken to avoid over-distension of the colon, which can result in damage.

Barium enema

Indications

A lesion of the colon may be suspected if there are clinical signs of colitis, tenesmus or haematechezia in the absence of infectious disease or evidence of prostatomegaly. Ultrasound is frequently used in evaluation of the colon wall, but when this is not available an enema can provide excellent evaluation for evidence of intussusception, strictures, mucosal abnormality or masses. This can also be used instead of a pneumocolonogram to differentiate small intestine from colon.

Method

It is essential that the colon is as empty as possible prior to a contrast study, as the presence of faecal material could interfere with interpretation. A 24 hour fast and warm water enema administration is typically required prior to an elective study.

The patient is placed in right lateral recumbency with the pelvis and rectum elevated using a foam wedge. Contrast can then be administered with a large Foley catheter and should be allowed to fill the colon in a gravity dependent manner without forced distension. This limits the chances of complication. A 20-25% weight/volume concentration of micropulverised Barium suspension is recommended. A starting volume of 11-15 ml/kg is used in the dog and 7-11 ml/kg in the cat. Lateral, oblique and ventrodorsal radiographs should be obtained immediately after filling of the colon with the catheter clamped to prevent leakage. An initial right lateral view will help to ensure that there is sufficient contrast filling.

Interpretation

An empty colon should fill evenly with contrast, though the contrast will not always reach the ascending colon and caecum in a normal animal. Focal narrowing of the lumen is an indication of intramural or extramural thickening and luminal constriction. This could occur secondary to neoplasia or less typically an inflammatory lesion. A neoplastic lesion will appear as a sessile filling defect, while intussusception has classic ‘coiled spring’ appearance. A more acutely life threatening abnormality is colon torsion. In this case the colon is typically distended with focal severe narrowing and displacement. These patients will present with severe and progressive clinical signs and the procedure may have been performed on an emergency basis.

Oesophagram

Indications

An oesophagram can be used in evaluation of suspected oesophageal dysmotility, obstruction or congenital anomaly. These patients often present with regurgitation as a primary clinical sign, but dependent on the suspected aetiology there may be additional gagging or associated neurologic deficits.

Survey thoracic radiographs should always be obtained in cases of suspected oesophageal disease. Bone foreign bodies within the oesophagus may be readily visible without further investigation. If diffuse megaoesophagus is present there is also no indication for an oesophagram, as this will only confirm the diffuse distension. However, a contrast study can be an excellent way to confirm oesophageal distension if you are not sure radiographically.

There is a risk of aspiration with oesophagography. If the patient has a history of aspiration the study is generally not recommended. However, barium is fairly inert in the lungs, and while aspiration of barium can look dramatic radiographically it is typically readily cleared to the regional lymph nodes. Aspiration of food is much more likely to result in pneumonia. Aspiration of ionic iodinated contrast can however be catastrophic. Hyperosmolar contrast draws fluid in to the interstitium and aspiration can rapidly result in severe morbidity and even death.

Method

The type of contrast recommended depends on the type of lesion suspected. Oesophageal motility disorders are best evaluated with barium fluid followed by barium soaked kibble (soaked for 10 minutes prior to the study). It is possible that either fluid or food will result in abnormal motility when the other type does not. This can also be true of diverticuli and oesophageal strictures (including those resulting from vascular ring anomalies).

Suspected oesophagitis is best evaluated with barium paste, which will adhere to inflamed mucosa. Barium paste can also be useful in delineating oesophageal foreign material and can be best for delineating a focal mass.

Non-ionic iodinated contrast media should be used if there is concern for oesophageal perforation. Ionic iodinated contrast media should *never* be used as the hyperosmolar contrast can lead to significant pulmonary oedema. If endoscopy is planned immediately after the contrast study this would also be an indication for non-ionic iodinated media.

Following survey orthogonal radiographs, contrast is administered orally (by feeding syringe for fluid/ paste or by hand for food) with the patient in right lateral recumbency. 60% weight per volume of barium sulphate suspension should be used for fluid studies. For small to medium dogs approximately 15 mL is recommended. 20-30 mL may be necessary for large dogs. Approximately 5 ml is recommended for cats. A similar volume of paste is recommended.

Immediately after contrast administration a lateral radiograph of the neck and thorax and ventrodorsal view of the thorax should be obtained. It may be necessary to repeat the study with an additional bolus. Given the midline location of the thoracic oesophagus, an oblique ventrodorsal view angled from left ventral to right dorsal can be helpful in evaluation of the intrathoracic oesophagus. If evaluating for motility disorders or oesophageal stricture, this process should be repeated with the barium soaked kibble.

All oesophageal studies must be performed with the patient awake, to limit aspiration and to ensure that motility is not affected pharmacologically. Some patients may not be suitable for oesophageal contrast studies.

Interpretation

An oesophageal foreign body, particularly if obstructive, will result in varying degrees of oesophageal distension oral to the lesion. The foreign body typically results in a focal filling defect. The foreign material can also become coated with contrast, and will remain static in appearance after the remainder of contrast has passed to the stomach.

Strictures result in focal narrowing which is persistently seen on multiple images, typically with distension oral to the lesion. It is possible that the stricture will limit passage of food but not fluid. Cranial to the carina and cardiac silhouette focal narrowing in immature patients may be associated with a vascular ring anomaly. Oesophageal stricture can occur secondary to scarring from a previous foreign body and association necrosis.

Oesophageal diverticuli are focal outpouchings of the oesophagus that can occur congenitally or secondary to prior foreign body trauma or inflammation. It is not uncommon to see a focal widening similar to a diverticulus within the cranial thoracic oesophagus of brachycephalic type breeds such as bulldogs, where this may be incidental.

Sliding hiatal hernias can appear transiently or persistently during an oesophagram study, and the displaced stomach is often delineated by contrast enhancement coating of the rugal

folds. Gastroesophageal intussusception is a more serious and acute condition, with a curved filling defect within the caudal distended oesophagus.

An oesophageal mass or abscess will result in a focal filling defect associated with the wall, static over several images.

It should be noted that the caudal feline oesophagus has a normal herringbone pattern that should not be confused with mucosal pathology.

2. Contrast imaging of the urinary system

Contrast imaging of the urinary bladder, urethra, kidneys and ureters is possible in general practice and can provide much additional information over survey radiographs, particularly when ultrasound is not available.

Cystography

Indications

Positive contrast and double contrast cystography can be used to evaluate a variety of suspected lesions of the urinary bladder. In trauma patients, positive contrast cystography can be very useful in evaluating for evidence of bladder rupture. This may also be used when rupture is suspected following outflow obstruction. Positive contrast cystography can also delineate a bladder mass or wall thickening. Double contrast cystography provides excellent evaluation of the luminal bladder contents and the bladder wall in patients presenting with haematuria or dysuria. This is more sensitive and specific than ultrasound or survey radiographs in determining the number and size of cystic calculi. It can identify calculi (urates for example) that are not evident on survey radiographs. It also provides excellent delineation of the bladder wall.

Methods

Only iodinated contrast media should be used for positive contrast cystography. If the colon is very full, fasting and a warm water enema may be necessary to avoid superimposition. Survey radiographs should initially be obtained. Deep sedation or anaesthesia is typically necessary.

The urinary catheter is placed aseptically into the bladder and as much urine as possible is withdrawn. A diluted solution of iodinated contrast (30mgI/ml) should be used. The volume of contrast that can be used depends on the urinary bladder. Most bladders will be filled with approximately 5mls/kg but if there is pathology of the bladder wall, as little as 1ml/kg can

result in bladder distension. The bladder should be palpated during filling to avoid over distension. Right lateral, oblique and ventrodorsal radiographs are obtained immediately after filling.

A double contrast cystogram can be performed immediately after a positive contrast cystogram with the withdrawal of the majority of the contrast and subsequent filling of the bladder with air. It is recommended that the patient is in left lateral recumbency for injection of air to limit the risk of air embolus. Double contrast cystography is more ideally obtained by initially administering a small volume of contrast, rolling the patient to coat the mucosa, followed by introduction of air to distend the urinary bladder. 1ml of contrast should be used for a cat and between 1 and 6ml depending on the size of the dog. Undiluted contrast can be used for double contrast cystography.

Interpretation

Initial leakage of contrast can appear as a small and indistinct plume adjacent to the urinary bladder. With time, there will be contrast filling of the abdomen. However, very small tears may not be appreciated without repositioning the patient, as compression of the lesion in lateral recumbency could limit leakage.

On double contrast cystography, calculi appear as filling defects within the centre of the contrast pool. They can be rounded or irregular. Gas bubbles move to the periphery of the pool. Blood clots may be central and peripheral, but are typically irregular shapes. Mucosal irregularity and bladder wall thickening can be appreciated in association with cystitis, typically toward the apex. A bladder wall mass would appear as a filling defect on positive contrast cystography and focal wall thickening on double contrast cystography.

Urethrogram

The urethra of the male canine or feline patient is relatively easily catheterized, and a urethrogram can be very useful method to evaluate for luminal calculi or strictures that would not otherwise be evident.

Indications

A history of urethral obstruction or stranguria can be evaluated with urethrography. In the acute stages of obstruction, passage of a urinary catheter is of paramount importance, but subsequent contrast evaluation can help determine the underlying cause or assist surgical planning. A urethrogram can more accurately determine the size, number and location of calculi within the urethra than would be evident radiographically. Radiolucent calculi, urethral

strictures, mass lesions or compression by an enlarged prostate can be evaluated. Evaluation of the female urethra is more challenging as it is much shorter, and localization is more limited. A positive contrast vaginocystourethrogram can be performed, but will not be discussed within this lecture.

Methods

Heavy sedation or light anaesthesia is necessary. Survey radiographs should be obtained, and in the male canine this should include a right lateral view with the legs pulled forward and centred on the region of the membranous urethra. A sterile Foley catheter is placed within the most distal aspect of the urethra. Iodinated contrast is injected into the urethra with a radiograph obtained immediately after finishing the injection. 5 ml is recommended the cat, and 10 ml, 20 ml or 30 ml for imaging of small, medium and large dogs respectively.

Approximately 50% of the contrast should be injected initially and a second radiograph obtained after injection of the remaining contrast. This helps to determine if filling defects represent static lesions or air bubbles.

Interpretation

Static luminal defects are most likely calculi. However, blood clots or mucosal plugs may also be present. Some contrast filling of the prostate can occur as a normal finding. However, narrowing of the urethra in the region of the prostate and irregular filling of the prostate can be an indication of prostatic neoplasia or prostatitis. Inflammatory or neoplastic lesions can result in focal or focally diffuse narrowing. Urethrospasm can occur as a clinical finding, but is also seen secondary to contrast administration in a less heavily sedated patient. This can be avoided with injection of 2 mL 2% lidocaine at the start of the study.

Intravenous pyelography (IVP)

This study is also known as intravenous urography (IVU). Intravenous contrast is filtered by the kidney and cleared by the ureters, and allows a degree of functional in addition to anatomic evaluation. The study requires radiographs be obtained in close succession.

Indications

If ultrasound is not available, an IVP study can provide useful information regarding the anatomic structure of the kidney. Differentials for renal enlargement can be better discriminated by this study. Suspected ectopic ureters can also be evaluated by this method, though interpretation can be challenging. The normal kidney will filter contrast within an expected time frame, and any change to this indicates abnormalities of renal function.

There is some risk with any intravenous contrast administration. Contrast associated acute renal failure has been described and the risk is limited by adequate hydration. This procedure is also contraindicated in patients with severe azotaemia. In patients with mild azotaemia an increase in the contrast dose may actually be necessary to perform a diagnostic study.

Methods

The patient must be adequately hydrated prior to the study, and dehydration should therefore be corrected. This study should not be performed if there is severe renal compromise.

Following survey radiographs an enema may be performed if there is sufficient faecal material within the colon to compromise evaluation. This is particularly true in evaluation of the distal ureters.

An intravenous catheter is necessary for contrast administration. A dose of 880 mgI/kg of iodinated contrast is recommended. This should be given as a rapid bolus. The dose can be increased by 10% in patients with mild azotaemia. Follow up right lateral and ventrodorsal radiographs should be obtained immediately and at 5 minutes, 15 minutes and 30 minutes. If the study is carried out in evaluation for ectopic ureters, initial air filling of the urinary bladder can help to visualize the ureterovesicular junction and additional oblique views are recommended.

Interpretation

The immediate vascular phase is often not appreciated and is rapidly followed by the nephrogram phase which results in enhancement of the renal parenchyma. This is followed (within minutes) by a pyelogram phase in which the renal pelvis and ureters are filled with contrast. There should be a gradual decrease in the opacity of the renal parenchyma.

The sequence of opacification can be abnormal with functional renal disease. Initial opacification followed by progressively increasing opacity can occur with systemic hypotension, renal obstruction or contrast induced renal failure for example.

Structural abnormalities can be differentiated. An irregular outline to an enlarged kidney on survey radiographs could be secondary to neoplasia or polycystic disease. A neoplastic lesion will typically results in an irregular area of abnormal contrast enhancement and

possible distortion or dilation of the renal pelvis and pelvic recesses. Polycystic disease results in multiple well-defined rounded filling defects that do not enhance.

A smoothly marginated enlarged kidney could be secondary to acute inflammation, FIP, a perirenal psuedocyst, hydronephrosis or neoplastic infiltration such as lymphoma. Acute pyelonephritis typically results in pelvic dilation and mild ureteral dilation, while chronic pyelonephritis causes blunting of the pelvic recesses in addition to mild ureteral dilation. Hydronephrosis secondary to ureteral obstruction typically results in severe pelvic dilation, dilation of the pelvic recesses and ureteral dilation to the level of obstruction. A perirenal pseudocyst appears as a large non-enhancing pocket around a typically small kidney. Other acute inflammation or FIP may not have specific changes.

Ectopic ureters are typically, but not always, dilated. They bypass the normal ureterovesicular entry to the urinary bladder and join the urethra or other more distal location. It can be difficult to differentiate intra-mural tunnelling ectopic ureters from the normal junction.

3. Musculoskeletal contrast studies

Fistulography

Most musculoskeletal structures are beyond the scope of this lecture and would be limited to referral practice. However, fistulography can be a very useful way of determining if there is a foreign body associated with a draining tract, or determining if a draining tract connects to a joint.

Fistulography indications

A draining tract can occur secondary to the presence of a foreign body that is not appreciated on survey radiographs. Use of contrast to evaluate the draining tract can help to determine if surgical intervention is indicated.

Methods

Ideally, a small Foley catheter placed in the periphery of the tract will prevent contrast leakage during the study. Injection of diluted iodinated contrast (50 mgI/ml) into the tract, immediately followed by orthogonal and oblique projections will allow evaluation, though repeat injection may be necessary if there is contrast leakage.

Interpretation

Foreign material is associated with a persistent and static filling defect. However, it should be remembered that contrast will typically take the gravity dependent path and it can be difficult to fill the entire extent of the tract.

References

1. The handbook of veterinary contrast radiology. Seth T. Wallack. 2003. Published by the author, San Diego Veterinary Imaging Inc., 361 North Sierra Avenue, Solana Beach, CA 92075
2. Textbook of veterinary diagnostic radiology, sixth edition. Donald E. Thrall. 2012. Elsevier.

RADIOLOGY OF THE MUSCULOSKELETAL SYSTEM

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Radiographic principles

A basic radiographic principle is to make a minimum of two orthogonal (at right angles to each other) projections of each area of interest is required. One should also include the joint above and below a fracture. The contralateral limb can be used for comparison, although it must be noted that many developmental diseases will show bilateral changes.

As a general rule, when radiographing joints, the radiograph should include about a third of the long bones proximal and distal to the joint, and when radiographing long bones, both proximal and distal joints should be included. Further radiographic views may be required to demonstrate different regions, depending on the area of interest. These include both standard (e.g. 45^{o} dorsomedio-plantarolateral obliques of the carpus and hock) and lesion-orientated oblique views, skyline views (e.g. cranioproximo-craniodistal views of the bicipital groove) and stressed views to demonstrate joint laxity in the dorsopalmar or craniocaudal and the mediolateral plane.

Exposures should be chosen to produce a range of grey tones to allow assessment of both the bones and the soft tissues, and both under and overexposure should be avoided. Good exposures will show trabecular detail within bones, with the cortical bone showing as dense white and the soft tissues as grey tones. Underexposure will present bones as homogeneous white structures with no internal detail visible, whereas overexposure will greatly reduce the visibility of the soft tissue structures.

Serial radiographic examinations are very useful in investigation of orthopaedic disease to assess dynamic change in the appearance of the bones and joints over time.

Arthrography

Arthrography allows visualisation of articular cartilage and identification of any non-mineralised bodies within the joint, such as cartilaginous joint mice. Positive, negative and double contrast techniques have been described, but positive contrast arthrography is the most commonly used technique. The shoulder is the commonest joint to be imaged with

arthrography, but the technique can be used in other joints. Survey orthogonal views of the joint should be taken first, and then repeated following arthrography. Dilute 300 mg iodine/ml (as in Omnipaque) with an equal volume of sterile water.

Radiological interpretation

All radiographs should be assessed according to Roentgen signs. These comprise changes in shape, position, size, contour, number, opacity and architecture. The radiograph should be assessed for both bone and soft tissue components of the musculoskeletal system. Presence of fat within joints or between fascial planes may allow interpretation of changes within joints or within surrounding musculature.

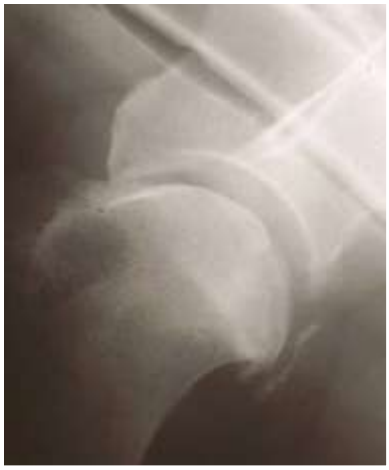
Radiology of the Joints of the Thoracic limb

Shoulder

Osteochondritis Dissecans (OCD)

This condition is part of the osteochondrosis complex. It is mainly a condition of giant breeds but may also be seen in border collies. The shoulder is the commonest site of OCD in the body. It affects the caudal third of the humeral head. The condition is often bilateral but clinical signs may be seen in only one limb.

In the initial stages there is thickening of the articular cartilage of the caudal third of the humeral head, so arthrography is required to demonstrate the increased width of the cartilage. In later stages the cartilage flap may become detached and result in a defect in the subchondral bone with accompanying sclerosis of the humeral head. The flap may become mineralised and thus visible on plain radiographs where it is known as a “joint mouse”.

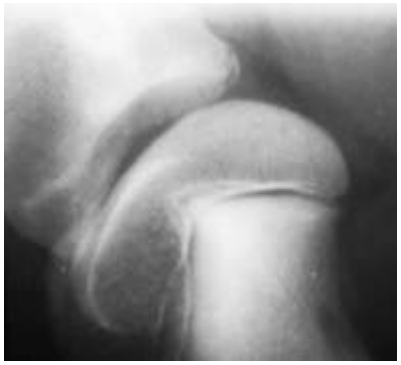


These mineralised bodies may continue to grow within the synovial fluid. Frequently they are within the caudal compartment of the joint capsule but occasionally they become trapped within the bicipital tendon sheath. Elliptical radiolucencies may be visible within the joint space. This is free gas within the joint caused by the vacuum phenomenon, when a reduction of volume within the joint caused by traction during position causes nitrogen to be pulled out of the blood stream and form a bubble within the joint.

Secondary degenerative joint disease may develop, with osteophytes on the caudal aspect of the humeral head and the caudal aspect of the glenoid.

Ossification of the glenoid rim

This is seen in juvenile dogs. A linear lucency is seen parallel and adjacent to the caudal rim of the glenoid. This fuses to the scapula as the animal matures.



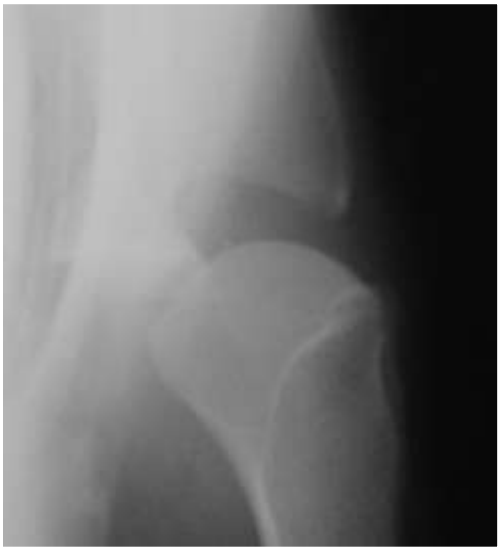
Rarely, is incomplete ossification of the caudal glenoid rim clinically significant.

Separation of the scapular tuberosity

This is an uncommon injury. It may be seen in association with a traumatic luxation of the shoulder or it may be part of the osteochodrosis complex. A lucent defect separates the scapular tuberosity from the cranial aspect of the scapula.

Luxation

This is a congenital condition in miniature or toy breeds. The luxation is usually medial and may be bilateral. The mediolateral projection reveals a flattened or convex glenoid surface and a relatively large and flattened humeral head. The caudocranial projection will reveal medial or lateral displacement of the humeral head relative to the glenoid.

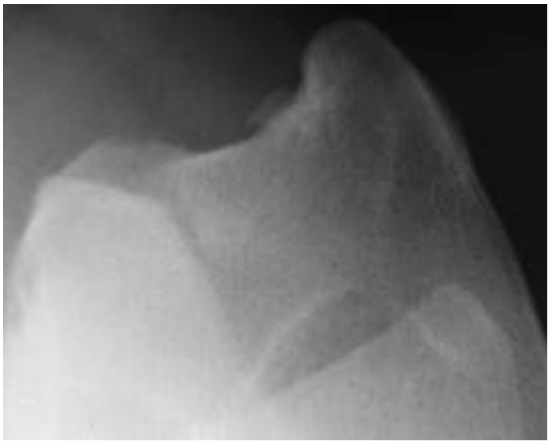


Traumatic luxations also occur and are also most frequently medial.

They can be differentiated from congenital shoulder luxations by the shape of the congruent articular surfaces.

Bicipital bursitis

Radiography is often of little use. Some increased radiopacity may be seen in the region of the bicipital groove.



A proximal-distal skyline view of the biciptal groove may increase the sensitivity of detection of small changes in this region.

A contrast study of the bicipital groove may also be performed using 3-4 ml of contrast agent. The contrast medium should outline a smooth tendon and the tendon sheath. Ultrasound is useful to see the echotexture of the tendons. Subtle irregularities of the bicipital groove may also be seen.

Shoulder instability

Rupture or tearing of the glenohumeral ligaments remains a contentious subject but some clinicians are convinced it is a significant cause of shoulder lameness in the mature sporting breed dogs. Radiography is often of little use but arthroscopy is excellent to visualise the pathology.

Fractures

Fractures of the scapula may be seen post trauma. The acromion and greater tubercles may fracture. The scapula may also fracture and this may easily be missed. It is difficult to radiograph the body of the scapula without superimposition of other structures.

Elbow

The elbow joint is a stable compound ginglymus or hinge joint, capable of flexion and extension and a limited amount of rotation. The articular surface of the distal humerus, called the humeral condyle, is positioned cranial to the long axis of the shaft and is shaped like an inclined cylinder. It is divided into a small lateral area called the capitulum humeri, which articulates with the radial head and the larger medially located trochlear humeri, which articulates with the trochlear notch of the ulna.

Congenital Luxations

This uncommon problem occurs in two distinct forms and accounts for about 15% of all non-traumatic elbow lameness in the dog.

Type 1

This type of congenital luxation results in a severe disability. It is associated with about 90 degree of outward rotation of the proximal ulna, removing the trochlear notch and the anconeal process from effective articulation with the humerus and displacement of the triceps tendon. This results in marked rotation and lateral deviation of the antebrachium, lateral rotation of the paw and marked reduction in elbow extension. The condition is usually recognised at birth or within the first 3 months of life. It can be uni- or bi-lateral.

Radiographs will demonstrate a varying degree of luxation. The cranio-caudal projection of the elbow may reveal a normal frontal view of the humerus, but a lateral view of the proximal ulna, whereas in the lateral projection, the situation will be reversed, reflecting the 90 degree rotation of the proximal ulna relative to the distal humerus. Other changes which may be present include hypoplasia or aplasia of the anconeal process, trochlear notch, humeral condyles and coronoid processes, plus distortion of the olecranon.

Type 2

In this type the relationship between the humerus and ulna is normal, but the proximal radius is displaced in a caudal and lateral direction. The proximal radial epiphysis may be deformed, but in general the degree of disability is not as severe as the Type 1. There is usually no limb deviation, but extension is reduced and the elbow thickened with a marked lateral swelling. There may be caudal bowing of the proximal ulna.

Osteochondrosis

Three forms of osteochondrosis occur in the elbow of young, rapidly growing and usually large breeds of dog. Each problem usually occurs in isolation, but any combination of the three is possible in one joint, although having all three problems in one joint is unlikely.

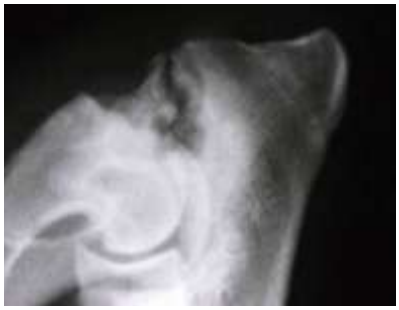
a) Ununited Anconeal Process

Normally the anconeal process develops as part of the ulnar diaphysis, but in certain breeds such as the German Shepherd Dog, it develops as a separate centre of ossification. This fourth ossification centre appears at about 10-13 weeks and is fused to the ulna by 18-20 weeks.

Ununited anconeal process (UAP) as a result of failure of fusion, is seen predominantly in the German Shepherd Dog, where it is probably an inherited defect. Traumatic separation can occur as a result of hyperextension of the elbow joint. In certain breeds, such as the Basset Hound, it occurs secondary to non-traumatic premature closure of the distal ulnar

growth plate. The shortened ulna and the relative lengthening of the radius puts pressure on the trochlea of the humerus which in turn forces the humerus proximally, exerting sufficient pressure on the anconeal process at a critical stage of its development to result in its separation. The instability and irritation following separation of the anconeal process results in degenerative joint disease.

The diagnosis is confirmed by taking a fully flexed lateral radiograph of the joint. The flexed view is important so that superimposition of the medial epicondyle on the olecranon can be avoided.



A clear line of separation below the anconeal process is diagnostic, but in some cases there appears to be only partial separation. A varying amount of DJD will also be evident.

In addition the congruency of the elbow joint should be assessed from radiographs taken in the extended lateral and cranio-caudal projections. Shortening of the olecranon has also been observed on the affected side.

b) Fragmentation of the medial coronoid process

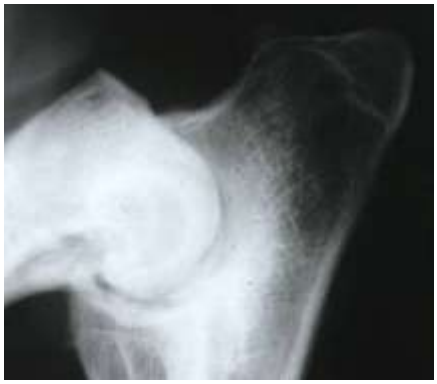
Currently fragmentation of the medial coronoid process of the ulna (FCP) is the commonest cause of elbow lameness in young, rapidly growing dogs of the large and giant breeds.

There is no separate centre of ossification for the coronoid processes. Osteochondrosis seems to affect an area(s) of a joint that is subjected to shearing and traction forces. In the elbow these forces occur as a result of the combined action of pressure from the medial humeral condyle and traction from the annular ligament. In some cases there is evidence of elbow incongruence secondary to asynchronous development of the radius and ulna. This results in a relative overgrowth of the ulna causing retraction of the radial head from the elbow joint and exposure of the coronoid processes to abnormal shearing forces from the distal humerus. Wind studied FCP in Bernese Mountain Dogs and concluded that incongruence is the common denominator in the various manifestations of elbow osteochondrosis. Incongruence is due to abnormal development of the trochlear notch of the ulna resulting in a slightly elliptical articular surface with an arc of curvature with too small a

radius to accommodate the humeral trochlea. This creates a joint with major contact points in the region of the anconeal process and the medial coronoid process, but not between the trochlear notch and the humeral trochlea.

The definitive diagnosis of FCP on conventional radiographs poses some problems because the location of the fragment(s) means that there is invariably superimposition of other structures. If the facilities are available the fragment can be visualized using computer axial tomography. In advanced cases the fragment may be visible on the cranio-caudal or the cranio-caudal medial oblique projection. A tentative diagnosis is made on the basis of the presence of osteophytes and the elimination of all other known causes of degenerative joint disease. However it takes these osteophytes some weeks to develop and therefore they may not be evident on the initial radiographs, especially if the animal has been lame for less than 3-4 weeks. The first evidence of DJD will usually be found on the caudal, non-articular, surface of the anconeal process.

Initially the osteophytes appear as a slight irregularity on the margin of the bone. It is therefore important to have good quality radiographs taken with the joint in a maximally flexed, but not rotated, lateral position.



If the initial radiographs appear normal and the lameness persists, follow-up radiographs are recommended within 4-8 weeks. As the condition persists osteophytes will be found adjacent to the medial coronoid process and the radial head and there may be osteosclerosis in the proximal ulna.

As well as the flexed lateral radiograph it is recommended that a cranio-caudal projection is taken to check for OCD lesions and an extended lateral projection to determine the congruency of the joint. Oblique craniocaudal projections are recommended in certain cases as they highlight the caudal and cranial aspects of the joint on the lateral and medial sides respectively.

c) Osteochondritis dissecans (OCD) of the medial condyle of the humerus

OCD occurs with low frequency in Rottweilers and has its highest incidence in Labradors and Golden Retrievers. In common with OCD in other joints it has a high incidence of

bilateral involvement. The lesion is usually found near the outer edge of the central weight bearing region of the articular surface of the medial humeral condyle.

The clinical signs are very similar to FCP. A flexed lateral radiograph will show the presence of DJD on the anconeal process and a cranio-caudal or cranio-caudal medial oblique projection will usually reveal a defect in the subchondral bone of the medial humeral condyle. Occasionally ossification of the cartilage flap will make it visible. In long-standing cases the flap may break off and become lodged in the caudal and medial aspect of the joint capsule. It may grow in this location forming a linear osteochondral ossicle, which may only be visible on a cranio-caudal lateral oblique projection.

Epiphyseal problems

Epiphyseal fractures and separation of the growth plates of the radius and ulna are common. The high incidence of growth impairment that occurs after these injuries may result in deviation of the limb and subluxation of the carpal and elbow joints.

Traumatic Luxation

Traumatic elbow luxation is an uncommon injury in the dog and cat and is usually seen in animals over one year of age. In dogs luxation is usually in a lateral direction because the large caudal projection of the medial epicondyle of the humerus prevents displacement of the radius and ulna in that direction. Luxation occurs when a lateral force is applied to the joint when the antebrachium is twisted and the elbow is flexed. Lateral displacement of the radius is only possible when the anconeal process is disengaged from the supracondylar fossa and this only occurs when the joint is flexed more than 90 degrees.

Two views should be taken as a lateral projection on its own can be misleading. The cranio-caudal projection will also reveal if there are any bone fragments associated with collateral ligament damage.

Medial epicondylar fractures

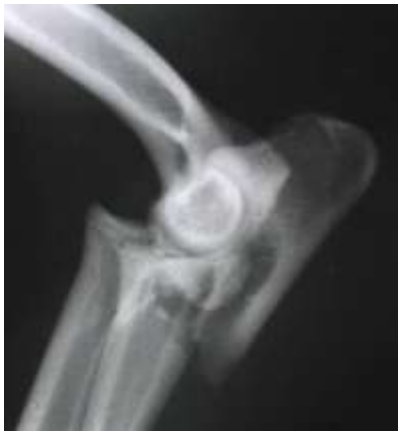
This uncommon injury is seen predominantly in young dogs. Affected animals may be presented with an acute onset of lameness after being hit by a car or a fall, or they may also be presented with a chronic lameness with an insidious onset. With an acute onset, the elbow will be painful and swollen; while in chronic cases a moveable bony mass may be palpable below the medial condyle. The diagnosis is confirmed radiographically.

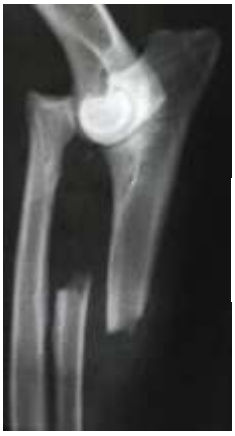
Olecranon fractures

Fractures of the proximal ulna are not uncommon. They can occur at any age and are usually associated with direct trauma. In the young animal the fracture may occur through the proximal ulnar growth plate, but in the mature individual the fracture is more likely to occur through the trochlear notch.

Ulnar fracture with radial head luxation

This uncommon injury is sometimes referred to as a Monteggia fracture. The ulnar fracture can occur at any level and the more distal the injury the more severe the damage is to the interosseous ligament. Luxation of the radial head usually results in rupture of the annular ligament.





Two examples of Monteggia fractures

Medial epicondylar lesions

Soft-tissue calcification adjacent to the medial epicondyle and bone spurs from the caudal edge of the epicondyle have been reported as a cause of lameness in the dog. It is likely that these two conditions are associated with either acute or chronic trauma to the tendon of origin of the various antebrachial flexor muscles. The condition is seen in both mature and immature dogs and responds well to surgical removal of either the bone fragments or to removal of bony spurs.

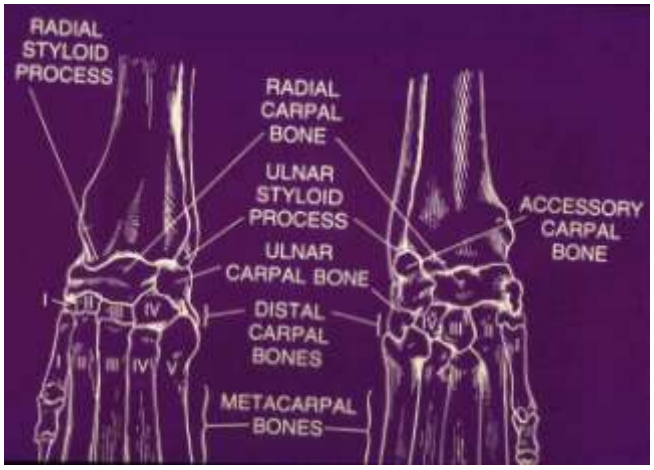
Carpus

The carpus comprises the antebrachiocarpal joint between the distal radius and ulna and the proximal row of carpal bones; the middle carpal joint between the proximal and distal rows of carpal bones and the carpometacarpal joint between the distal row of carpal bones and the heads of the metacarpals. Intercarpal joints also exist between individual carpal bones in each row.

The distal articular surface (trochlea) of the radius is concave and angled cranially by 10-15 degrees. A medial protruberance, the styloid process, is the origin of the radial collateral ligaments. The distal radius articulates with the styloid process of the ulna, which in turn articulates with the accessory and ulnar carpal bones.

There are seven named carpal bones. The radial, ulnar and accessory carpal bones form the proximal row. The distal row contains the first, second, third and fourth carpal bones. A small sesamoid is situated medial to the distal aspect of the radial carpal bone in the tendon of insertion of the abductor pollicis longus muscle.

The antebrachiocarpal joint normally allows 100 degrees of flexion, 10 degrees of hyperextension, five degrees of varus and 15 degrees of valgus angulation and accounts for 80-90% of carpal movement. The middle carpal joint is capable of 40 degrees of flexion only, representing 10% of carpal movement. Around 10 degrees of flexion is possible at the carpometacarpal articulation. Forty degrees of axial rotation is possible at the distal radioulnar joint.



No ligaments span the entire joint. The rows of bones are supported individually by short ligaments, like the links of a chain. Small intercarpal ligaments connect adjacent bones in each row.

Radial Carpal Bone Fracture

Saggital or dorsal slab fractures of the radial carpal bone are seen infrequently. Small medial fragments should be treated with caution as they may represent avulsions of the radial collateral ligament insertion. Palmar fractures may involve the insertion of the oblique portion of the short radial collateral ligament or the origin of the radiometacarpal ligament.

Spontaneous saggital fracture of the radial carpal bone occurs rarely, with the Boxer breed being over-represented. The radial carpal bone develops from three ossification centres and the fracture may represent a failure of these to fuse.

Accessory Carpal Bone Fracture

This injury is virtually exclusive to the racing Greyhound and to similar dogs such as Whippets and Lurchers. Fractures of the accessory carpal bone in the Greyhound usually affect the right carpus. The fractures are sprain-avulsion injuries, due to carpal hyperextension during racing. As such they represent the most obvious indicator of more extensive injury to the joint and, particularly, its soft tissue supporting elements.

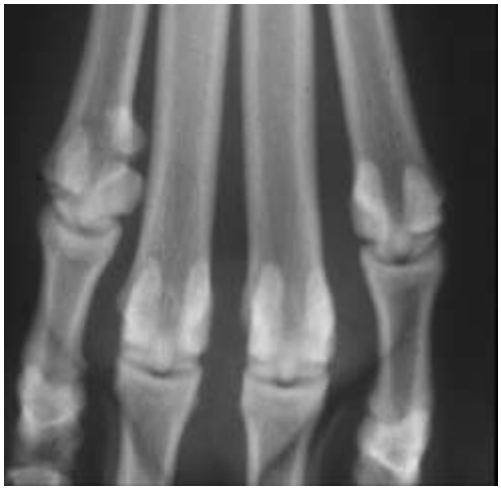
Five types of accessory carpal bone fracture have been described. Type I fracture is most common. Types I and II fractures are intra-articular. Type II and III fractures are usually accompanied by a Type I fracture. Type IV fractures represent avulsion of the flexor carpi ulnaris muscle.

The Pes

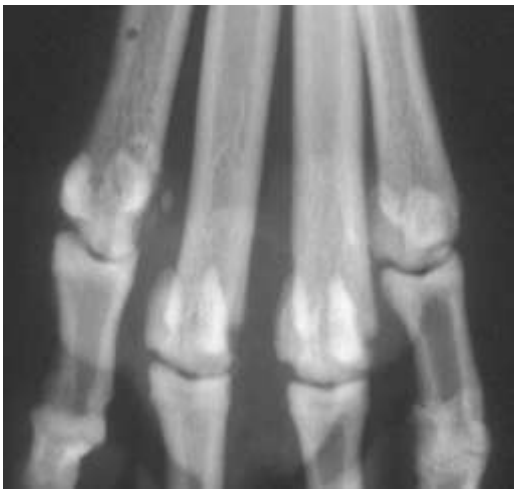
Fractures/luxations may follow crushing trauma and involve more than one digit. Subluxations and luxations are usually associated with fractures.

Multipartite sesamoids are incidental findings in Rottweilers and other large breeds, and usually affect the 2nd and 7th sesamoids. Sesamoid fractures have sharp, angular opposing surfaces and are associated with soft tissue swelling.

Neoplasia e.g. squamous cell carcinoma or melanoma commonly affects the paws of older dogs (they may metastasise to other paws). Neoplasia is characterised by soft tissue swelling with bone lysis (bony reaction is minimal). The major differential of neoplasia is infection which tends to result in marked periosteal reaction.



Multipartite sesamoid



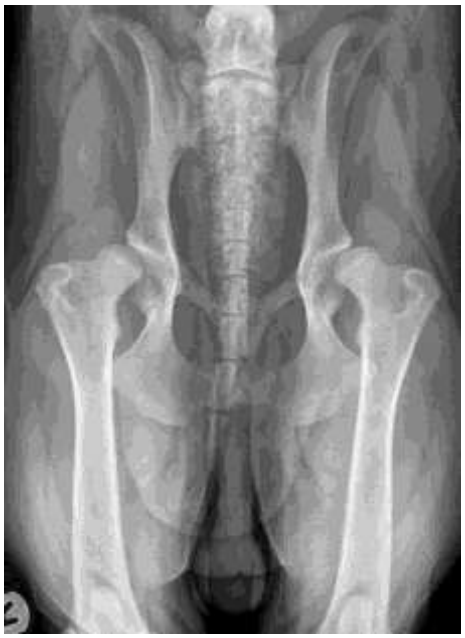
Fractured sesamoid

Radiology of the Joints of the Pelvic limb

Hip

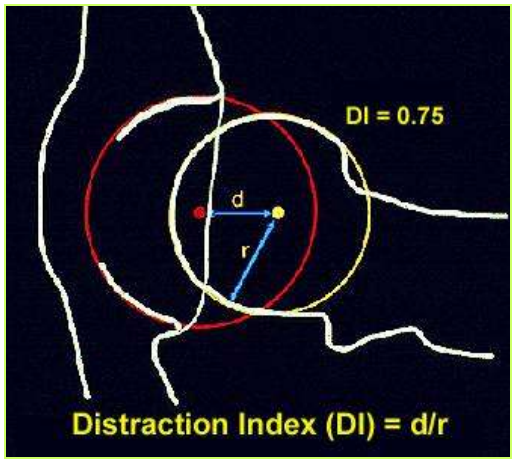
Hip dysplasia

Hip dysplasia is an abnormal development of the hip joint and is usually bilateral. It is manifested by varying degrees of instability of the joint, laxity of the surrounding soft tissues, malformation of the femoral head and acetabulum and secondary degenerative joint disease. It has a multifactorial aetiology, including polygenic predisposition and environmental factors.



The ventrodorsal projection of the pelvis is the most useful. There are degrees of severity of the disease, ranging from mild coxofemoral subluxation to severe subluxation with extensive remodelling of the acetabulum and femoral head. Varying degrees of periarticular osteophyte formation are seen as the secondary changes progress. New bone develops on all aspects of the acetabulum and on the femoral head and neck. Wastage of the hip and thigh muscles may also be appreciated.

Stress radiography as in the Penn hip scheme utilises the so called distraction index to determine the degree of joint laxity. Currently, there is a move to introduce this to the UK.



The distraction index (DI) used in the PennHip method serves as a measurement of passive hip laxity, the degree of looseness of the hip joint when the dog's hips are completely relaxed. Dogs with a DI of 0.3 have tighter hips and are less likely to develop DJD, while those with looser hips whose DI values approach 0.7 or more are at greater risk.

Avascular necrosis of the femoral head (Legg-Calve-Perthes disease)

This is an aseptic necrosis of the femoral head and neck in small breed dogs, thought to be secondary to ischaemia. The femoral head and neck undergo necrosis and deformation, with collapse of the subchondral bone, leading to hip joint incongruity and instability. Severe secondary degenerative joint disease will develop. Toy breeds and terriers are most susceptible and the condition is frequently bilateral.

Tthe femoral head may appear mottled and of heterogeneous radiopacity, with focal regions of radiolucency. There is flattening and distortion of the femoral head which results in secondary remodelling of the acetabulum, which becomes shallower. Secondary degenerative joint disease develops rapidly.

The Stifle

Four sesamoid bones are present in the stifle, the patella in the tendon of insertion of the quadriceps, the sesamoid bones of the gastrocnemius (fabellae, medial and lateral) and the popliteal sesamoid in the tendon of the popliteus muscle.

Avulsion of the tibial tuberosity

This tends to occur in the immature dog and cat (tibial tuberosity joins the tibia at 6-8mo and completely fuses by 8-12mo). Radiographically there may be widening and irregularity of the physis of the tuberosity. It is good practice to radiograph both stifles to compare. There may also be cranial or proximal displacement of the tibial tuberosity and soft tissue swelling cranial to the proximal tibia.

Patellar Fractures

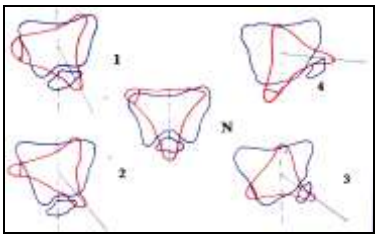
These may be seen in association with patellar ligament rupture. A sharply demarcated fracture line separates the patella in two of more fragments. The proximal fragment or fragments may be displaced proximal to the trochlear groove. A joint effusion and soft tissue swelling will be present.

Bipartite patellas have been seen but with these the fragments would be rounded and there will be no associated soft tissue swelling.

Patellar luxation

This may be traumatic and be associated with damage to the medial/lateral patellar ligaments or it may be a congenital or developmental condition. Congenital/developmental patella luxation tends to affect toy breeds of dog and is associated with genu varus (bow legged) and a shallow trochlear groove. It is predominantly medial and the patella can be

seen overlying the femoral condyles on the lateral view. A skyline view is required to demonstrate the depth of the trochlear groove but is rarely indicated.



Singleton classification of patellar luxation

Rupture of the patellar ligament

This is an acute injury. The rupture occurs either proximal or distal to the patella. A fracture of the patella or avulsion of the tibial tuberosity may also be seen. On a flexed mediolateral view there is proximal displacement of the patella (radiograph the other limb for comparison). There will be pronounced soft tissue swelling and a reduction in the size of the infrapatellar fat pad.

Cranial cruciate injury

This is an injury seen mainly in dogs. In the earliest stages there will be a joint effusion (reduction in size of the infrapatellar fat pad). The tibia may be displaced cranially with respect to the femur (normally the spine of the tibia should lie at the midpoint of the femoral condylar articular surface. Occasionally a small fragment of bone from the cranioproximal tibial plateau will be avulsed. This is the site of insertion of the cranial cruciate ligament. Chronically radiographic signs of osteoarthritis develop.

Collateral ligament injury

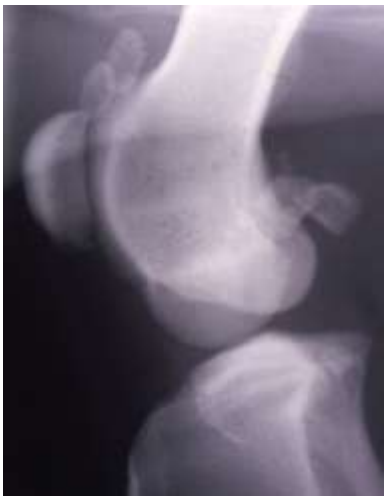
Soft tissue swelling may be apparent over the affected ligament on the craniocaudal view. Other traumatic injuries may also be apparent. If stressed views are performed then widening of the joint may be seen either medially or laterally.

Avulsion of the long digital extensor

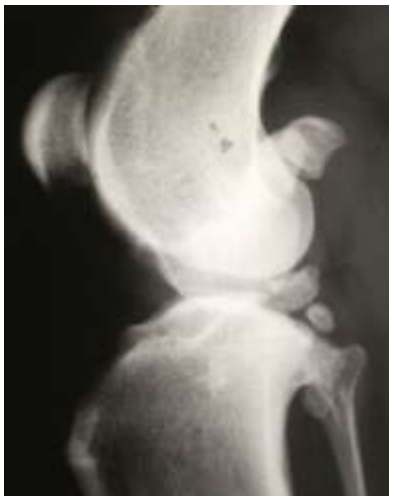
This is an uncommon condition. Radiographically an avulsed fragment of bone from the lateral femoral condyle may be seen lateral to the joint. A joint effusion may also be present. A radiolucent defect may be apparent at the site of the avulsion. Very early in the presentation the avulsed fragment may be relatively radiolucent.

Osteochondritis dissecans

This is a condition of giant and large breeds. The age of presentation is usually 6-10 months. Either the lateral or medial femoral condyle may be affected. Soft tissue swelling may be apparent. There may be flattening of the affected condyle. The mineralised flap may be evident adjacent to the condyle or a fragment may be evident within the joint (either the supra-patellar or caudal joint pouch).



supra-patellar joint mouse



joint mouse in joint compartment

Hock

The hock is a compound joint comprising seven tarsal bones, the distal tibia and fibula, and the proximal metatarsals. The talocrural joint is a trochlear joint between the tibia and fibula, and the talus (tibial tarsal bone). It permits flexion and extension from 35 to 180 degrees. The distal tibia contributes medial malleolus, intermediate ridge and part of the lateral cochlea. The distal fibula forms the lateral malleolus.

The proximal row of tarsal bones comprises the talus, and the calcaneus (fibular tarsal bone) which provides a lever arm for the insertion of the Achilles apparatus. On the medial aspect of the distal tarsus, there are two bone layers. The central tarsal bone (Tc) has an intermediate position between the talus and tarsal bones 1-3. On the lateral aspect the fourth tarsal bone alone lies between the calcaneus and the metatarsus.

The proximal intertarsal joint (PITJ) has two parts: medially, the talocalcaneocentral joint, and laterally the calcaneoquartal joint. The centrodistal joint is the joint between Tc and T 1-3. The tarsometatarsal joint lies between the numbered tarsal and metatarsal bones. The

limited flexion/extension and gliding movement of the intertarsal joints is restricted by the ligamentar scaffold of the tarsus. Parasagittal intertarsal joints have considerable rigidity.

tarsocrural joint (tibiotarsal)

calcaneoquartal } (proximal intertarsal joint)
talocalcaneocentral }

centrodistal (distal intertarsal joint)

tarso-metatarsal joint

Pes varus

Pes varus, resulting from distal tibial deformity, has been specifically described in the Dachshund. It typically manifests at 5-6 months and probably reflects tibial dysplasia rather than growth plate trauma. An underlying genetic (autosomal recessive) component has been suggested.

Lateral torsion and tarsal valgus deformity

A lateral torsion and tarsal valgus deformity of unknown aetiology is described in large and giant breed dogs (e.g. Rottweiler, St. Bernard, Bernese Mountain Dog). The deformity, which is untreatable, arises within the tarsus. Although rarely a significant clinical problem, the resultant poor conformation is a cosmetic fault.

Osteochondrosis

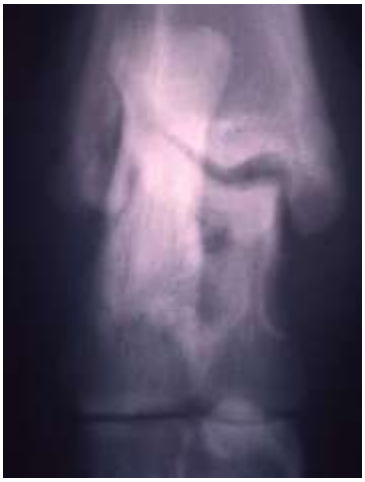
The manifestation of the disease in the hock is invariably osteochondritis dissecans (OCD) of the medial or lateral trochlear ridge of the talus, with lesions of the proximal medial trochlear ridge predominating.

Clinical signs are typically seen from 4-6 months onwards. Lameness is variable, perhaps becoming intermittently non-weight bearing. Pain and crepitus are evident on joint manipulation. Range of talocrural joint movement is reduced, and there is talocrural joint effusion. The disease may be bilateral and is often associated with an abnormally upright stifle and hock conformation.

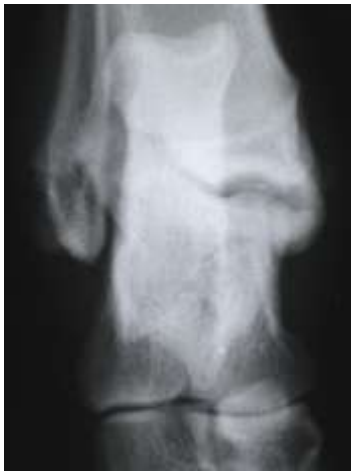
Standard lateral and dorsoplantar radiographic views are usually adequate to demonstrate lesions of the medial trochlear ridge. Superimposition of the calcaneus complicates

interpretation of the lateral trochlear ridge, and a flexed dorsoplantar skyline view, or dorsoplantar oblique views may be necessary.

A number of radiological abnormalities may be present. Widening of the talocrural joint space, with flattening of the affected trochlear ridge and subchondral sclerosis of the tibial cochlea and talar trochlea are usually evident. Osteochondral fragments are variable in size and may be displaced or non-displaced. Joint effusion may manifest as soft tissue swelling in early cases. With time, there is an inevitable progression of degenerative joint disease, and massive peri-articular new bone formation and remodelling may be seen.



Widening of the talocrural joint space, with flattening of the affected trochlear ridge



Degenerative joint disease secondary to ocd

Distal tibial fractures

Distal tibial growth plate fractures are an uncommon injury of the skeletally immature dog. The fracture is most frequently Salter-Harris Type I and is associated with fibular fracture and displacement. Intra-articular extension of tibial diaphyseal fracture is not common. Spontaneous fractures of the caudal distal articular margin of the tibia in racing Greyhounds have been described. Such fractures may be associated with malleolar fractures.

Talocrural luxation and subluxation

Talocrural luxation and subluxation are invariably associated with disruption of collateral support. This may occur as a result of closed malleolar fracture, open malleolar fracture/shearing injury, collateral ligament rupture or collateral ligament avulsion.

Malleolar fractures

Malleolar fractures involve the articular surface and disrupt the origin of the collateral ligaments. Medial and lateral collateral fractures permit valgus and varus deformity respectively. Careful manipulation of the talocrural joint in flexion and extension can disclose selective involvement of the long and short collateral ligament components. Radiographic studies should include appropriate medial and lateral stressed views as talocrural subluxation may not be apparent on neutral studies.

Malleolar shearing injury

Shearing injury is not an infrequent complication of road traffic accidents. The injury occurs as the limb is abraded along the road surface beneath a locked wheel, causing loss of skin, underlying soft tissue and bone. The subcutaneous prominences of the medial (most commonly) and lateral malleoli are vulnerable to such injury. Medial malleolar shear is the more severe injury due to the normal valgus configuration of the pes.

Collateral ligament rupture

Collateral ligament rupture which is not associated with malleolar avulsion or shearing injury is less common. Such injuries are likely to be an incomplete rupture of the collateral ligament. Despite the relatively mild talocrural laxity, severe lameness is evident. Radiography is indicated to rule out avulsion fracture, e.g. intra-articular fracture of the medial trochlear ridge at the insertion of the short medial collateral ligament.

Intertarsal luxations

Talocalcaneal luxation

This is an uncommon injury of the dog and cat. It is associated with blunt trauma and causes a non-weight bearing lameness and hock deformity. Rupture of the talocalcaneal ligaments can result in dorsal displacement of the talar head with talocentral luxation, plantar displacement of the distal calcaneus, or massive distal luxation of the talus. This may occur as an isolated injury, or in association with talar neck fracture.

Proximal intertarsal luxation

There are three basic forms of PITJ dislocation which should be distinguished by clinical and radiographic examination.

PITJ subluxation with plantar instability

Calcaneoquartal subluxation due to plantar tarsal ligament rupture is a common injury of the hock. Two types of dog are predisposed; racing/coursing dogs and middle aged collie type

dogs, particularly the Shetland Sheepdog. While breakdown in the athlete occurs during racing, the majority of pet dogs do not present with a history of trauma. Bilateral plantar ligament rupture is not uncommon.

Radiography, including stressed flexed views, confirms plantar subluxation. Deviation from the normally straight plantar axis of the tarsus, from calcaneus to metatarsus, should be evaluated. Enthesiophyte formation in the middle plantar ligament is a common finding in the companion animal, whereas avulsion fracture of the origin of the plantar ligament is more common in the athlete.

PITJ subluxation with dorsal instability

This is a less common injury than PITJ hyperextension, and is associated with rupture of the small ligaments of the dorsal aspect of the proximal intertarsal joint. Medial or lateral instability is often present. The tarsus is generally stable during the weight bearing phase of the stride, and functional impairment is therefore relatively mild.

PITJ luxation with dorsoplantar and collateral instability

This is the most serious, but the least common form of PITJ disruption, which may also be complicated by tarsal bone fracture.

Centrodistal luxation

This injury can occur in isolation, but is more frequently seen as a complication of calcaneoquartal subluxation with plantar instability. The isolated injury presents with valgus deformity due to dorsomedial ligament rupture.

Tarsometatarsal luxation

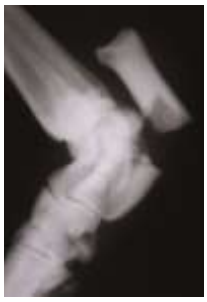
This is a less common injury than talocrural or proximal intertarsal luxation. Again, subluxation with plantar instability is a more disabling injury than that which involves dorsal or dorsomedial instability.

Calcaneal fractures

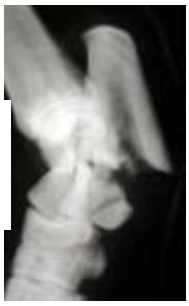
Fractures of the calcaneus are not uncommon in the racing Greyhound. The forces generated in cornering to the left predispose the right hock to injury. Loss of the lever arm for the common calcaneal tendon results in a plantigrade stance. The pull of the gastrocnemius tendon can cause considerable displacement of the fragments.

There are two distinct mechanisms of calcaneal fracture in the racing Greyhound. Most are associated with central tarsal bone fracture which permits talocalcaneal subluxation. As the

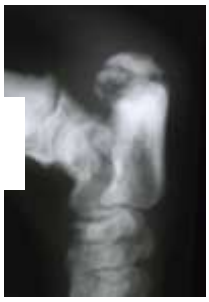
calcaneus tilts dorsally and medially, relief of the accumulating forces may involve either calcaneal fracture, or compression fracture of the fourth tarsal bone. Such calcaneal fractures are either mid-body shaft fractures or tarsal ligament strain avulsion fractures e.g. dorsomedial slab or lateral sagittal slab.



Mid-body fracture



Basilar fracture



Physeal fracture

Calcaneal fractures which are not associated with central tarsal bone fracture are caused by extreme tension along the plantar aspect of the bone. Fracture of the plantar distal process or base of the calcaneus occurs, and invariably causes proximal intertarsal subluxation.

Talar (tibial tarsal bone) fractures

Fractures of the tibial tarsal bone are not common and are usefully classified as articular fractures of the body, or non-articular fractures of the neck or head. Osteochondral fragments of the trochlear ridges may be associated with avulsion of the insertion of the short collateral ligaments.

Central tarsal bone fractures

Tc fracture in the non athletic dog is rare, and generally involves avulsion fracture of the plantar process. Tc fracture is very common in the racing Greyhound, occurring most frequently in the right (outer) hock due to the stresses imposed by cornering to the left. Clinical findings are of pain and crepitus on Tc palpation, and variable soft tissue swelling. The degree of lameness may be mild, and dogs can run on to finish races. With severe fracture, tarsal varus and plantar convexity may be apparent.

Tc fractures are classified according to fracture configuration (Boudrieau).

- Type I Dorsal slab with no displacement
- Type II Dorsal slab with displacement
- Type III Sagittal fracture with displacement of the medial fragment
- Type IV Both dorsal and medial slab fractures, with displacement
- Type V Severe comminution and displacement

Type IV fractures are by far the most common. While Tc fracture is the most common injury of the greyhound hock, isolated Tc fracture is rare. The incidence of secondary fractures increases with the severity of Tc fracture. The most common concomitant fractures are T4 compression fracture with calcaneal fracture, and T4 fracture with avulsion of the lateral base of metatarsal V. These other fractures are a result of dissipation of forces following collapse of the dorsomedial buttress of the tarsus, and distal migration of the talus.

Fractures of T2, T3, T4

Fractures of the numbered tarsal bones are almost invariably associated with Tc fracture in the racing greyhound. Compression fracture of T4 is most commonly observed. Dorsal slab fractures of T3 can occur in isolation.

Gastrocnemius tendon avulsion

Avulsion of the gastrocnemius tendon from the calcaneus with an intact SDFT results in variable hyperflexion of the hock and digits. This may not present as an acute injury and marked tendon thickening is common. Large breed dogs, particularly the Dobermann, appear most susceptible. Radiography reveals variable dystrophic calcification within the soft tissue, and proliferative new bone on the tuber calcanei.

Superficial digital flexor tendon luxation

Displacement of the SDFT from the groove of the calcaneus is an uncommon injury in the dog. Lateral luxation occurs most frequently, with a predisposition among Shetland Sheepdogs and racing Greyhounds. Lameness is acute in onset and moderate in degree, with intermittent non-weight bearing and hock hyperflexion during weight bearing. Calcaneal bursal effusion is invariably present. Radiography is rarely helpful.

Notes page

SALVAGE SURGERY: FEMORAL HEAD AND NECK EXCISION ARTHROPLASTY, CARPAL AND HOCK ARTHRODESIS TECHNIQUES

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Excision arthroplasty is a salvage procedure that can be used for a variety of hip joint diseases. By removing the femoral head and neck the hip joint is converted to a pseudoarthrosis. The length of the operated hind limb is effectively reduced by this procedure and the femoral shaft tends to lie dorsal to its normal position.

Function depends on the integrity of the surrounding soft tissues. If much scar tissue is present pre-operatively, range of movement of the pseudoarthrosis will be poor and the animal may remain lame. Pressure on the sciatic nerve may occur in some patients and result in postoperative lameness.

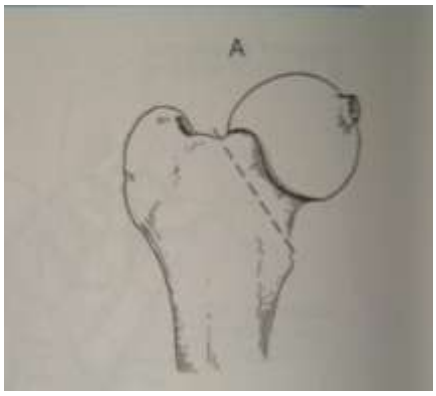
Immature animals respond to femoral head and neck resection better than adult animals. The procedure can be carried out bilaterally and simultaneously if required. Unilateral surgery will often result in residual dysfunction or lameness because of the post-operative asymmetry. Early use of the limb must be promoted postoperatively to avoid joint stiffness.

Minimal surgical dissection should be employed to reduce scar formation. A cranio-lateral approach will allow sufficient exposure of the femoral neck. This should be transected cleanly, level with the shaft of the femur. The angle of transection is vertical and parallel with the cranio-caudal plane of the femur. An osteotome, bone cutting forceps, rongeurs, or oscillating saw may be used. Excising the head at right angles to the cranial face of the femoral neck will leave a spike of bone caudally that may cause subsequent problems due to impingement against the acetabulum. If present, this spike of bone should be removed at the time of surgery. Accurate removal of the femoral head and neck must be checked before final wound closure and p/o radiographs should confirm this.

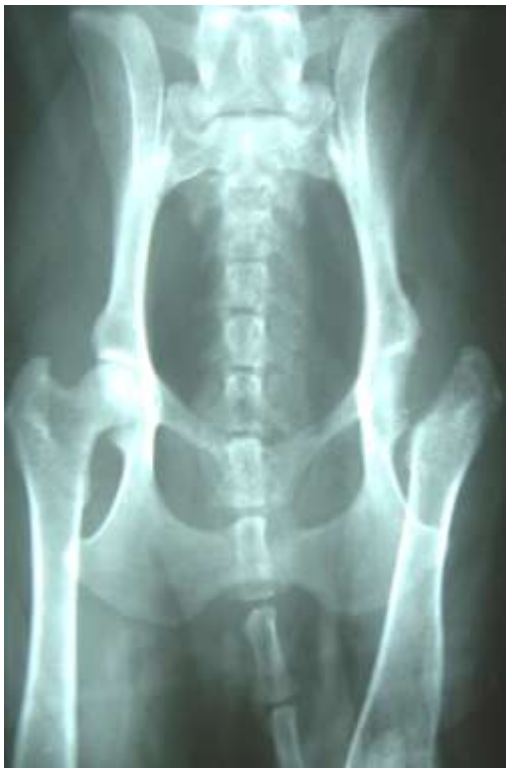
A cranio-lateral approach is used. Incise *both* leaves of the tensor fascia lata and retract this muscle cranially while retracting the biceps femoris caudally. Partially tenotomise the deep gluteal tendon, make a linear incision into the joint capsule along the femoral neck and into the origin of vastus lateralis.

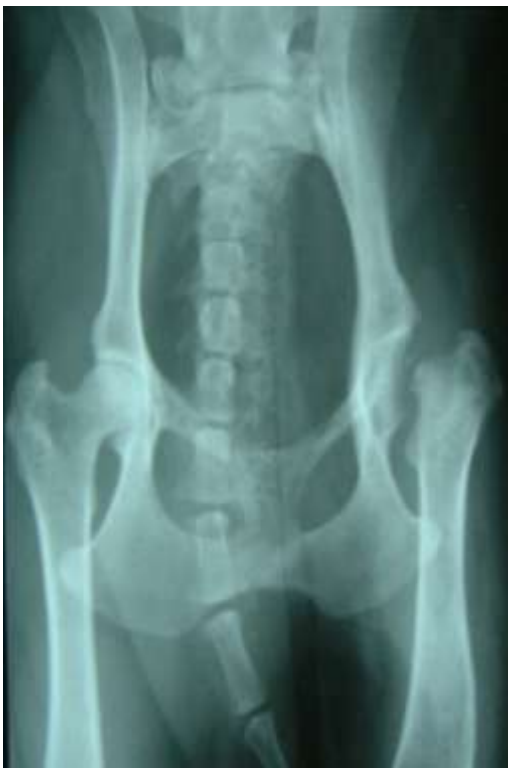
Ensure the teres ligament is completely cut and elevate the joint capsule from the femoral neck. **Rotate the stifle so that the patella is vertical.**

Remove the calcar region by osteotomising along the dashed line, using the lesser trochanter as a landmark.



Close the joint capsule and repair the tenotomised deep gluteal tendon. The remainder of the repair is routine.

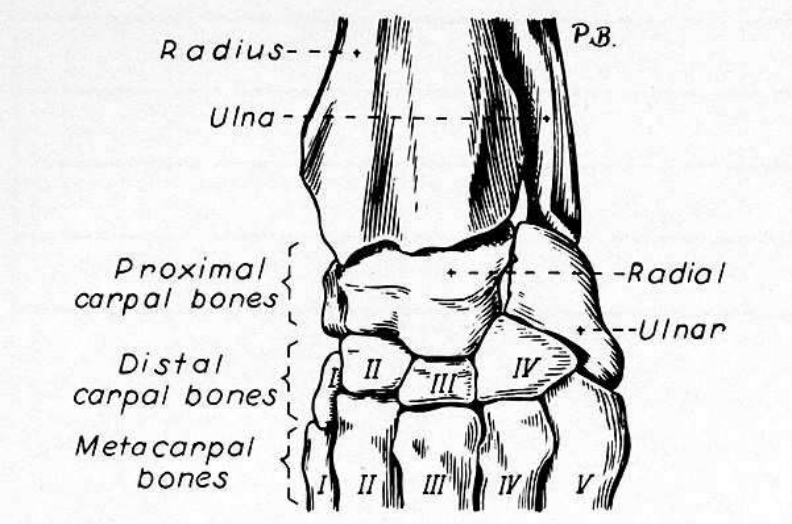




Post operative radiography is mandatory. A satisfactory osteotomy is seen in the left radiograph while the right radiograph demonstrates further surgery is required.

Carpal arthrodesis

The carpus is a composite joint with three rows of joints, the antebrachio-carpal, the middle carpal and the carpo-metacarpal joints. The intercarpal joints are in the sagittal plane.



Surgical approaches

Surgical approaches to the carpus are usually made over the area of interest. The collateral ligaments are approached directly via overlying skin incisions. The dorsal approach to the joint is the one most frequently used. In this approach the common and lateral digital extensor tendons are retracted laterally and the extensor carpi radialis medially. The approach is best made lateral to the cephalic vein, allowing it to be retracted medially. Transection of the extensor retinaculum and the tendon of insertion of the abductor pollicis longus muscle will be required if wide exposure of the carpus or distal radius is required. Transverse arthrotomies allow inspection of the carpal joints and bones.

The palmar surface of the joint is considerably more difficult to expose, although this approach has been used for palmar plate application in arthrodesis of the carpus.

Exsanguination of the distal limb using an Esmarch bandage and careful use of a tourniquet allow a bloodless surgical field and facilitate visualisation during carpal surgery. Postoperative swelling, which may be increased by use of a tourniquet, is reduced by application of a Robert Jones bandage for three to five days.

CARPAL SOFT TISSUE INJURIES

Injury to the soft tissue supporting elements of the carpus is common, particularly in Collie types and working dogs. Soft tissue injury is usually caused by a fall from a height, road

traffic accident or during working (e.g. racing Greyhound, working Sheepdog). Carpal soft tissue injuries can be divided into collateral ligament rupture, luxation of individual joints or bones, and hyperextension injury.

External support is usually an unsuccessful treatment for carpal ligamentous injury severe enough to cause joint instability. Temporary external support may be justified in cases that are obviously destined for arthrodesis, to allow acute swelling and inflammation to subside before surgery is attempted, but this must be weighed against the potential complications of external support, such as skin necrosis.

Collateral Ligament Rupture

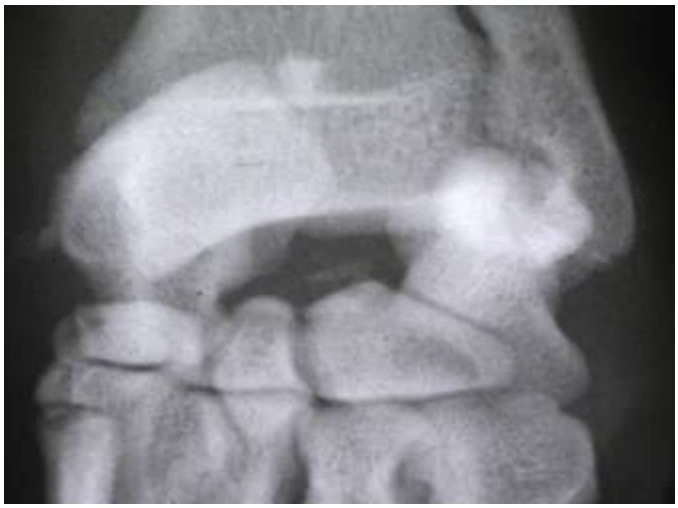
The carpus is hyperextended 10 degrees and has up to 15 degrees of valgus deviation during weight bearing. These factors, along with the cranial angulation of the distal radius, increase tension on the radial collateral ligament and predispose it to injury. Rupture of the ulnar collateral ligament is rare.

Radial collateral ligament rupture causes medial instability of the antebrachiocarpal joint. The radius may be rotated externally on palpation and the antebrachiocarpal joint space opened medially. Stressed radiography is helpful in confirming the abnormal instability.

Carpal Luxations

Luxation can affect any joint of the carpus. Luxations are usually associated with a fall or vehicular trauma, but can occur during work. Hyperextension injury is common in association with luxation. Luxation is usually associated with obvious joint instability and can be confirmed by plain and stressed radiographic examination. Depending upon the severity of soft tissue injury and the degree of joint instability, reduction and ligamentous reconstruction or arthrodesis may be the treatment of choice. Arthrodesis is indicated if hyperextension is present.

Luxation of the radial carpal bone is uncommon. The radial collateral ligament and other dorsal and intercarpal ligaments rupture and the radial carpal bone rotates caudal to the radius. The palmar ligaments are spared injury and hyperextension is usually not present.



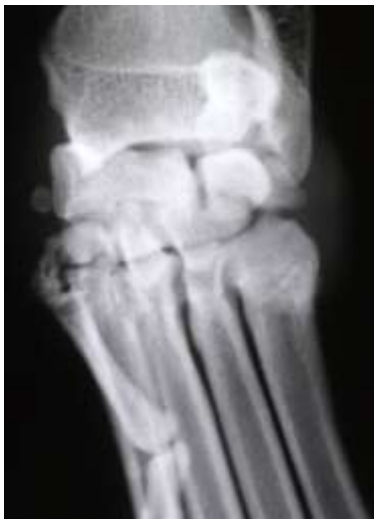
Treatment involves open reduction of the luxation and repair of the ruptured radial collateral ligament. The prognosis is fair and arthrodesis may be required if instability persists.

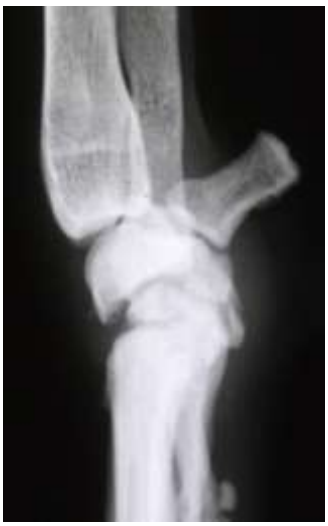
Carpal Hyperextension

Carpal hyperextension injury is common in dogs. Most cases involve either a fall from a height or occur at work. Traumatic hyperextension of the joint causes disruption of the palmar soft tissue supporting elements, causing subluxation or luxation and abnormal joint posture.

Some Rough Collies suffer bilateral spontaneous progressive carpal hyperextension, with degeneration of the palmar ligaments and the fibrocartilaginous pad. Occasionally, middle aged cats are seen with spontaneous bilateral hyperextension, presumably due to ligamentous degeneration or laxity.

Hyperextension is associated with pain, non-weight bearing lameness and soft tissue swelling if the injury is acute. Chronic or degenerative injuries often appear relatively painless and affected animals bear weight with an abnormal hyperextended joint posture.
Stressed radiographs must be made in order to determine the level of instability.





Surgical repair of the ligaments, replacement with autologous tissue and support with orthopaedic wire have also been described but are rarely successful. The most reliable form of treatment is pan- or partial arthrodesis. If the antebrachiocarpal joint is normal, fusion of the middle and carpometacarpal joints only may be attempted to retain some carpal mobility. However, partial fusion appears to have a higher failure rate. If the antebrachiocarpal joint is affected panarthrodesis should be performed. Fusion of the antebrachiocarpal joint alone leads to progressive degenerative changes in the other joints in the future.

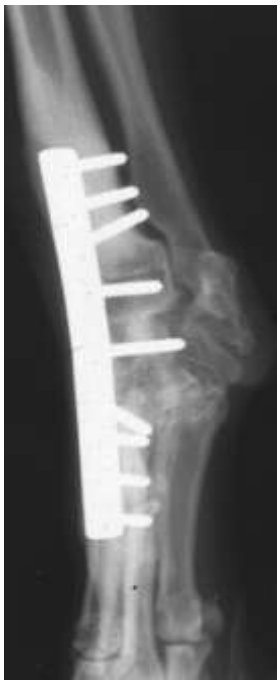
Indications for arthrodesis

The primary indication for carpal arthrodesis is a hyperextension injury with severe palmar ligament and fibrocartilage damage, and resultant instability and subluxation. These injuries are rarely managed successfully by external coaptation since the random collagen repair process is very slow and cannot withstand the stress of weight bearing. Additional conditions that may necessitate carpal fusion include shear injury, luxation (e.g. radial carpal bone), irreparable articular fracture, DJD and immune mediated arthritis.

Two basic types of arthrodesis may be performed. Pancarpal arthrodesis involves the fusion of all three joint levels and is indicated when the entire carpus or the antebrachiocarpal joint is involved. Partial carpal arthrodesis involves fusion of the middle carpal and carpometacarpal joints, with sparing of the antebrachiocarpal joint. Selective fusion of the antebrachiocarpal joint is not advocated because the middle and distal joints have minimal motion and with increased stress may subluxate and develop DJD. With hyperextension injuries, stressed lateral radiographs with the carpus in maximum extension, are necessary to detect the area of sprain and instability. Disruption of the supporting structures of the accessory carpal bone with resultant proximal displacement is not a contra-indication for partial carpal arthrodesis.

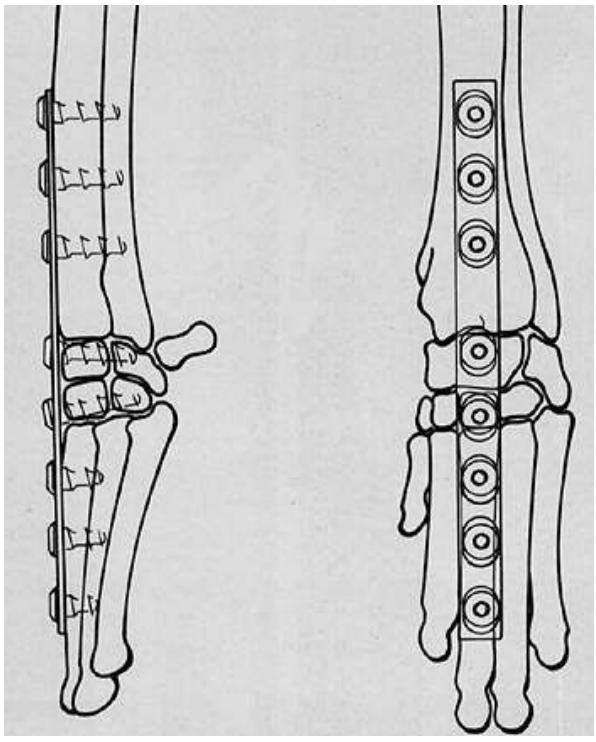
PANCARPAL ARTHRODESIS

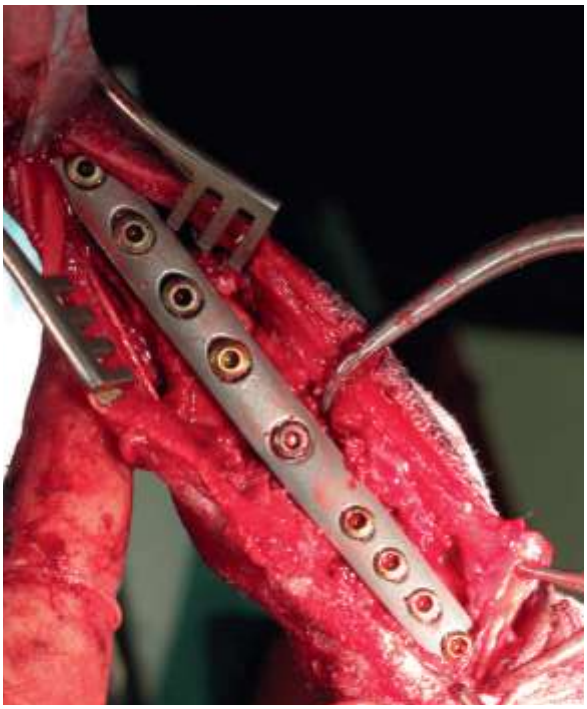
Bone plate and screw fixation is the technique of choice. Dorsal plating is generally preferred since it is technically easier to perform than palmar plating. The latter is, however, biomechanically more sound since the plate is positioned on the tension side of the carpus. The use of cross pins, two dorsal plates, medial plating and plate rod have also been described. External fixators are extremely useful when managing open injuries with extensive bone and soft tissue loss. The fixator allows wound management while providing rigid fixation with minimal implants at the site of arthrodesis which is frequently infected.



An Esmarch bandage is used to exanguinate the distal limb and a tourniquet is applied at the elbow. The carpus is exposed by a dorsal incision lateral to the cephalic vein and the tendons of the extensor carpi radialis are severed at metacarpals II and III. The remaining carpal and digital extensor tendons are reflected laterally. The dorsal joint capsule is excised, the joint flexed, and the articular cartilage removed from all exposed surfaces. A small Hohmann retractor may be used to aid exposure. A compression plate, usually 10-hole, is contoured to ensure approximately 10 degrees of extension. In some dogs with particularly straight limbs bending the plate is not necessary.

The first screw hole to be drilled and tapped is in the radial carpal bone, followed by the distal screw hole in metacarpal III. Cancellous bone is collected from the proximal humerus and packed into the joint spaces. The graft is secured in place by the plate using the two prepared holes and the remaining screws are placed to provide compression. Smaller diameter screws may be used in the metacarpal bone to reduce the chance of fracture by interposing a washer between the screw head and the plate. Wound closure is routine.
Alternatively, a hybrid plate may be used.





A Robert Jones dressing is applied for a few days, and after swelling has subsided, a coaptation cast is applied. Support is necessary for six to eight weeks until there is radiographic evidence of fusion.

A CastLess PCA plate is available which utilises 2 metacarpal bones. A 3.5/2.7 and 2.7/2.0 version is available.

A retrospective analysis of the clinical records from a heterogenous group of 15 dogs and three cats, (a total of 20 pancarpal arthrodesis) using this plate without external coaptation demonstrated two out of 17 arthrodesis in the 15 dogs to have serious postoperative complications (plate deformation, wound infection) which required revision surgery.

Fistula formation (8-16 weeks post surgery) was seen in six arthrodeses. These all resolved after medical therapy.

In cats, all three arthrodeses showed postoperative complications (bone resorption under the plate n=1, paw swelling n=2).*

Long-term evaluation by telephone interview with the owners (17 owners, 6-21 months after surgery, one case lost in follow-up) revealed good or very good results for 17 of 19 arthrodesis.

*Due to the size of the available implants complications have to be expected in cats.

A study to determine the mechanical differences between the Veterinary Instrumentation Hybrid Dynamic Compression Plate (HDCP), and the OrthoMed CastLess Arthrodesis Plate (CLP) used single-cycle load to failure using a materials-testing machine and cyclic loading between 38 and 380 N ± 5% to simulate estimated *in vivo* loads until failure or 10^6 cycles.

Single-cycle to failure: bending stiffness was significantly higher for the HDCP (2269 ± 175 N/mm) than CLP (1754 ± 88 N/mm; $P < .001$). Bending structural stiffness was higher for the HDCP (3.8 ± 0.3 Nm^2) versus CLP (2.9 ± 0.2 Nm^2; P= 0.0022). A difference between the 2 plates for bending strength was not demonstrated; HDCP = 13.9 ± 1.4 Nm, CLP13.2 ± 0.5 Nm (P= 0.24). No failures occurred with either plate type when plates were cycled to 10^6 cycles. The conclusion was there is no mechanical advantage in bending resistance

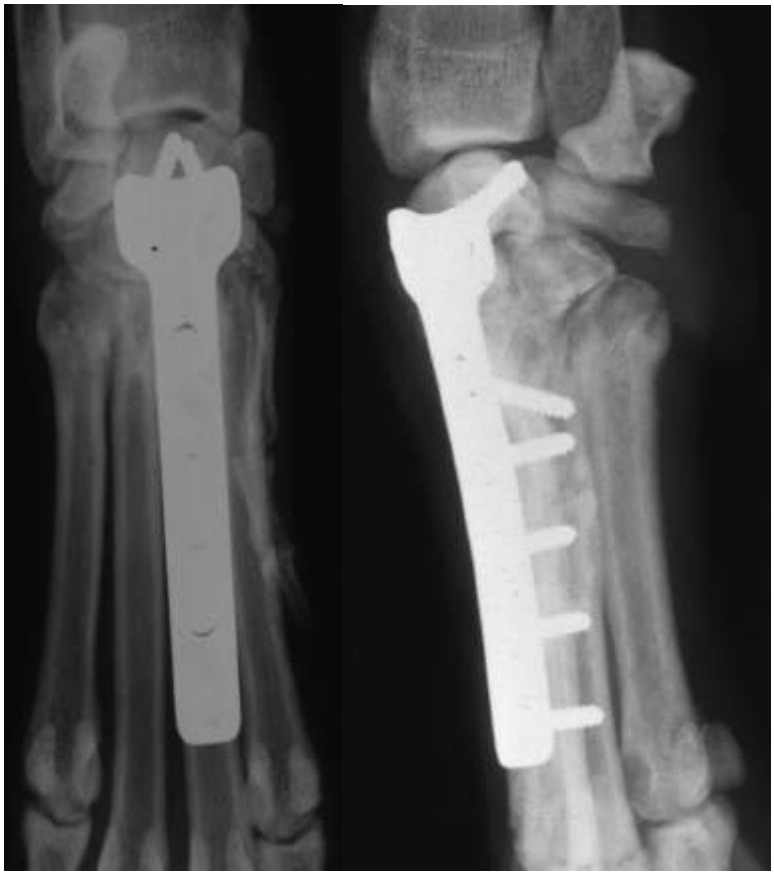
afforded by the CLP over the HDCP. Fatigue failure of either plate during the convalescent period of an estimated 150,000–250,000 cycles is unlikely.

Results and complications

The results of pancarpal arthrodesis are generally very good with the majority of dogs regaining full limb function within four months. Complications are minimal and are usually implant related. The most common problem is loosening of the distal metacarpal screws and occasional fracture of metacarpal III distal to the plate. Other complications include plate breakage and soft tissue reactions associated with low-grade infection or temperature changes. Removal of the implants in the latter cases will generally resolve the problem. Pancarpal arthrodesis seldom results in undue stress on the elbow and phalangeal joints.

PARTIAL CARPAL ARTHRODESIS

Intramedullary pin or bone plate fixation have been described.



T plate fixation

Patient positioning and limb preparation are the same as for pancarpal arthrodesis. A dorsal approach is made to the carpus with the incision extending distally to the level of the metacarpophalangeal joints. Care is taken to preserve the insertions of the extensor carpi radialis tendons. The dorsal joint capsule is incised and the articular cartilage removed from the middle carpal, intercarpal and carpometacarpal joints.

Care should be taken to avoid impingement of the proximal border of the plate on the distal radius. Veterinary Instrumentation market a plate with holes very close to the proximal border to assist in this respect.

Alternatively, fixation can be achieved using Kirschner wires driven up the metacarpal bones. Slots are made in the dorsal cortex of metacarpals III and IV in the distal third of the shaft. Kirschner wires are introduced into the medullary canal and driven proximally to the level of the carpometacarpal joint. Fracture of the metacarpal bones may result if the slots are of inadequate length or pins of too large a diameter are used. Cancellous bone is packed in the prepared joint spaces. The wires are driven through the distal carpal bones and into the radial carpal bone as far as possible without penetrating the proximal articular surface. Flexing the carpus to 90 degrees and applying palmar and proximal pressure on the metacarpal bones will aid pin positioning and prevent fixation in a hyperextended position. Nevertheless, this is much easier to describe than to perform. After both pins are seated the ends are bent and cut off. Wound closure is routine. Postoperative management is the same as described for pancarpal arthrodesis.

Results and complications

Dogs with a partial carpal arthrodesis have approximately half the normal range of motion in the carpus. The results of arthrodesis using pin fixation are comparable to those for pancarpal arthrodesis. In one report, 70% of cases had normal limb function at follow-up. This is in contrast to the results in a study of 10 dogs, where partial carpal arthrodesis was performed using straight bone plates where only 50 per cent of cases regained full limb function. Interference with movement of the antebrachiocarpal joint, causing significant DJD and lameness is a particular problem with straight plates which cannot be seated as far distal on the radial carpal bone as T plates. Other potential complications include pin migration and interference with the movement of the metacarpophalangeal joints. DJD of the antebrachiocarpal joint has been reported to develop in approximately 15 per cent of cases. Whether this is secondary to abnormal stresses transmitted to this joint following a partial fusion, or due to undiagnosed ligamentous injuries, is debatable.

Following arthrodesis, cast support is mandatory until radiographic evidence of joint fusion is seen. It is common for the carpus to lose some of its range of flexion following surgical intervention or prolonged external support. This does not appear to affect joint function significantly.

Proximal intertarsal luxation with plantar instability

Calcaneoquartal subluxation due to plantar tarsal ligament rupture is a common injury of the hock. Two types of dog are predisposed; racing/coursing dogs and middle aged collie type dogs, particularly the Shetland Sheepdog. While breakdown in the athlete occurs during racing, the majority of pet dogs do not present with a history of trauma. Bilateral plantar ligament rupture is not uncommon.

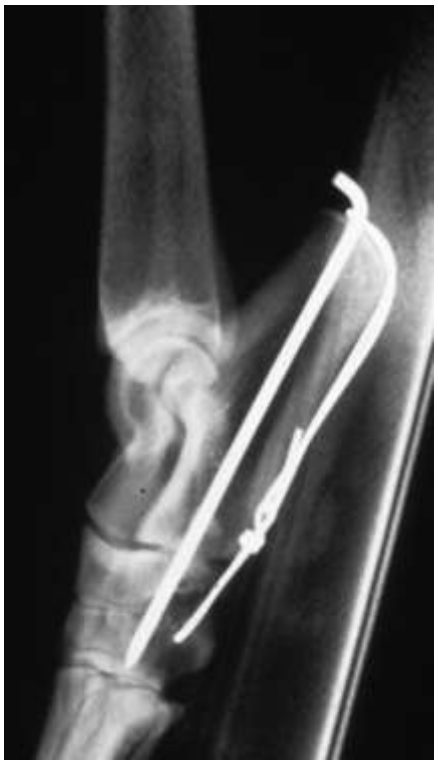
Plantar ligament breakdown is usually an isolated injury, but it may be associated with other tarsal fracture/luxation, particularly centrodistal luxation. A plantigrade stance with proximal intertarsal hyperextension characterises the condition. Plantar instability is easily demonstrated by manipulation of the calcaneus and metatarsus. Soft tissue swelling in the acute case, and pain, are not major clinical features.

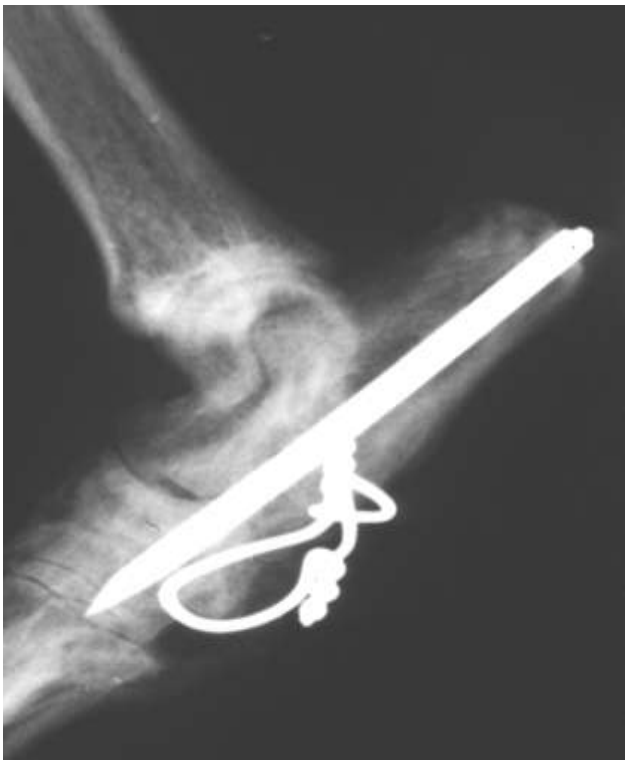
Radiography, including stressed flexed views, confirms plantar subluxation. Deviation from the normally straight plantar axis of the tarsus, from calcaneus to metatarsus, should be evaluated. Enthesophyte formation in the middle plantar ligament is a common finding in the companion animal, whereas avulsion fracture of the origin of the plantar ligament is more common in the athlete.

Conservative management will not restore plantar stability and coaptation should not be considered as effective treatment. The incidence of complications of repair is also greater with inappropriate cast/splint management. Primary suture reconstruction of the plantar ligament will not produce an adequate repair. Calcaneoquartal arthrodesis is therefore the treatment of choice.

Calcaneoquartal arthrodesis

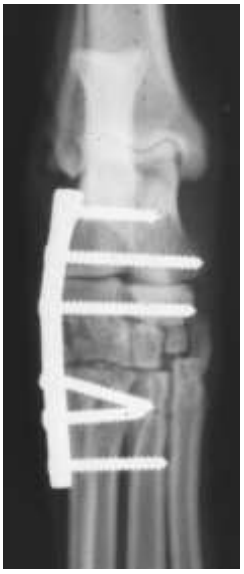
The procedure is performed under tourniquet following Esmarch bandage exsanguination of the distal limb. A plantarolateral approach is made to the calcaneoquartal joint and the superficial digital flexor tendon is protected medially. Any ligamentar debris and intra-articular fibrous tissue is removed, and the articular surfaces of the distal calcaneus and proximal T4 debrided to subchondral bone. A cancellous autograft may be used. Stabilisation of the arthrodesis can be achieved by a premeasured Steinmann pin and Figure of 8 tension band wire. Lag screw fixation without tension band wire support has also been described. The repair is supported in a cast or splint for 6 weeks until there is radiographic evidence of bone fusion.





Prognosis is good for return of normal limb function in the companion animal, but racing Greyhounds do not return to athletic performance.

Tarso-metatarsal arthrodesis



Tarso-metatarsal arthrodeses may be immobilised in a number of ways, but plate fixation on the lateral aspect of the hock provides rigid support. Removal of all the articular cartilage of the joint may be problematic.

External coaptation for several weeks is necessary.

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INVESTIGATION AND SURGICAL TREATMENT OF CERVICAL SWELLINGS

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There are a wide variety of causes of cervical swellings in dogs and cats including salivary mucocoeles, abscesses / granulomas and neoplastic masses amongst others. This lecture will discuss how to diagnose cervical swellings and highlight those conditions where surgical management is indicated.

Introduction

The incidence of cervical swellings in dogs and cats is unknown. Definitive diagnosis of the problem is required to guide therapy and for prognosis.

There is no known sex, breed or age predisposition, but the signalment of the patient may provide an indication of what the underlying problem may be e.g. salivary mucocoeles are often found in middle aged dogs and poodles and GSDs are over represented, younger sporting breeds are predisposed to trauma e.g. penetrating injuries and boxers, beagles and golden retrievers are reported to be overrepresented with regard to thyroid masses.

History

As might be expected, the discovery of a mass lesion by the owner will often prompt them to seek veterinary advice, and if the dog is groomed often it is the groomer who first finds such masses. There may be systemic signs associated with the underlying disease process such as pyrexia, lethargy and anorexia. On further questioning there may be evidence of neck pain, pain on opening the mouth, dysphagia or dysphonia and occasionally neurological deficits of the cranial nerves e.g. Horner's syndrome may be noticed.

Clinical examination

Although many patients will be stable on initial presentation, some may show signs of respiratory distress and it is important that these animals are triaged appropriately and receive the supportive care required before further investigation is performed.

If the patient is stable a full general physical examination is required to rule out other co-existing diseases, and to provide a picture of the animal's overall health.

If possible a preliminary oral examination should be performed with the animal conscious as it allows a more complete understanding of whether or not there is pain associated with opening the mouth. It may be necessary to delay this examination until the animal is sedated or preferably anaesthetised to allow full evaluation of the oropharynx safely. During the oropharyngeal examination pay particular attention to the presence of ptalysm or the presence of blood staining of the saliva; the ability of the animal to open its mouth without pain or restriction; the presence of halitosis and inspect the cheeks, gums, tongue, palate, tonsils and tonsillar crypts, larynx, lateral pharyngeal walls and pharyngeal arches carefully.

Examination of the cervical region should focus on the lymph nodes (mandibular in all cases and retropharyngeal if they are enlarged), mandibular and sublingual salivary glands, the larynx and trachea. The mass lesion should be assessed paying particular attention to the following details:

- Location and relationship to local structures including displacement;
- nature (soft, firm, fluctuant, ulcerated, pulsating (!));
- mobility (invasive / non-invasive)
- pain

The potential causes of cervical masses in dogs are shown in the table below.

Neoplastic	**Non-neoplastic**
Thyroid gland (adenoma, adenocarcinoma)	Congenital and developmental
Salivary gland (adenocarcinoma)	Metabolic conditions (tumoral calcinosis)
Fat (lipoma, liposarcoma)	Infectious and inflammatory conditions
Lymph node (lymphosarcoma, metastatic neoplasia)	Idiopathic conditions
Metastatic neoplasia (tonsillar squamous cell carcinoma etc.)	Iatrogenic conditions (seroma, haematoma)
Other soft tissue neoplasia (leiomyosarcoma, rhabdomyosarcoma, undifferentiated sarcoma etc.)	Traumatic conditions (bite wound, cervical expanding haematoma, oropharyngeal penetrating foreign body, subcutaneous emphysema etc.)
	Vascular conditions
	Miscellaneous conditions (salivary mucocoele)

Investigation

Depending on the results of the general physical examination and examination of the oropharynx and cervical region a blood sample should be collected for full biochemistry and haematology. An assessment of coagulation may also be appropriate at this time. If surgery of the ventral cervical region is a possibility then I prefer to draw a blood sample from a peripheral vein avoiding the possibility of haematoma formation at the surgical site that may complicate the surgical approach.

Both radiography and ultrasound examination of the head and neck will provide valuable information in the investigation of many cases of cervical swelling. In addition, thoracic radiographs may be required, either with respect to staging a suspected neoplastic process or providing information regarding the extent of the lesion. If there is a suspected foreign body remember that fragments of glass, metal and gravel need to be >1mm in size to be seen on a radiograph, while organic substances and plastic are usually radiolucent. Ultrasound examination is very useful in defining the nature of the mass (encapsulated or otherwise, fluid filled, solid tissue etc), assessing its relationship to local structures including the regional lymph nodes and the vascularity of the mass. Ultrasound may give an indication of the origin of the mass e.g. salivary gland, lymph node, thyroid gland etc. Foreign bodies may be identified or at least if foreign material is present within or near to the mass, distal acoustic shadowing may be present. Advanced imaging (CT; MRI), although not routinely available in a general practice setting at present provides excellent information in the investigation of penetrating oropharyngeal injuries, the staging of neoplasia and for surgical planning purposes etc.

Biopsy of the mass, either by fine needle aspiration or by incision biopsy may now be appropriate.

Surgical approach to the ventral cervical neck

The patient is anaesthetized and positioned in dorsal recumbency with a neck support. The front legs are drawn back and secured. A ventral midline skin incision is made. The sphincter colli and platysma muscle is then encountered and should be incised on midline. Underneath these muscles the strap muscles of the neck can be identified (sternohyoideus mm, sternothyroideus mm). The sternohyoideus mm. should be divided along the midline raphe using a push-cut technique. In this area you are likely to encounter an unpaired vein that may be cut (thyroidea ima vein present in about 70% of dogs). Now visible on the midline is the ventral trachea.

Laterally the following structures are present and should be considered during further dissection of the ventrolateral neck: oesophagus; external jugular vein; carotid sheath (common carotid artery; internal jugular vein; vagosympathetic trunk) and the recurrent laryngeal nerve. Dogs are not dependent on their jugular veins and both can be ligated, and since most dogs have a complete circle of Willis and good basilar/vertebral arteries, they can withstand unilateral and indeed bilateral carotid ligation. The same is not known for cats. Inadvertent unilateral ligation of a single carotid sheath will most likely result in Horner's syndrome as the major clinical sign. Damage to the recurrent laryngeal nerve will cause laryngeal paralysis. It is important to remember to preserve the segmental blood supply of the trachea.

Sialocoeles (salivary mucocoele)

Salivary tissue is abundant in the oral and pharyngeal cavities. There are four pairs of large, well-defined salivary glands that drain into the oral cavity in dogs: the parotid, mandibular, sublingual and zygomatic salivary glands.

A sialocoele (salivary mucocoele) is a subcutaneous or submucosal cavity created by and containing saliva. It forms following leakage of saliva from a disrupted salivary duct or gland. It is by far the most common salivary gland disease encountered in practice. The sublingual salivary duct is most frequently damaged but sialocoeles have been reported secondary to damage to all the salivary glands. Leaking saliva will typically follow the path of least resistance. The subcutaneous tissues of the intermandibular and cranial cervical region are the most common sites, with the tissue of the pharyngeal wall being the least common. Submucosal sialocoeles in the oral and oropharyngeal cavities are often referred to as ranulas and sublingual ranulas are commonest.

Sialocoeles are lined with inflammatory connective tissue and do not have an epithelial lining so they are not true cysts. They should resolve once the source of saliva is arrested and the cavity has been drained.

Most cases present with large, fluctuant swellings situated near the mandible or on the ventral neck. The masses are expected to be fluid filled, soft and non-painful. Dysphagia, oral bleeding and drooling can occur with the submucosal accumulation of saliva due to trauma during prehension and mastication. Upper respiratory tract obstruction may be encountered with pharyngeal mucocoeles due to occlusion of the common pharynx.

Physical presentation and FNAB are mainly used in the diagnosis of sialocoeles, as the gross and cytological appearance of sialocoele contents is very distinctive. Sialocoeles contain large volumes of honey coloured, mucinous saliva that forms long strings. Blood tingeing is also common particularly following aspiration in which case the contents can be very dark. However, in these cases the fluid will still form distinctive strings due to the high viscosity of the fluid and mucin can be identified microscopically.

As sialocoeles often shift to the most dependent point (ventral midline submandibular or ventral midline cervical), it can be difficult to establish which side they originate from. Sialography is the most accurate method of determining this but often if the patient is placed in dorsal recumbency the sialocoele will shift back to its original position. The opening of the mandibular duct can be difficult to identify as it sits on the ventral aspect of the mucosal fold on the sublingual caruncle.

Conservative management (aspiration) is considered to be ineffective as sialocoeles typically recur within a short period of time and infection is common following repeated aspiration. The definitive treatment is excision of the glandular tissue contributing to the build up of saliva but other techniques can also be used for selected cases.

Sialoadenectomy of the submandibular salivary gland complex (mandibular and sublingual salivary glands) is the treatment of choice for submandibular or cervical sialocoeles. There are two main surgical techniques described for excision of these glands through either a lateral or ventral approach.

Following surgery recurrence is uncommon occurring in less than 5% of cases.

Acute oropharyngeal penetrating injuries: 'pharyngeal stick injury'

Acute penetrating injuries and chronic abscess or sinus tract formation are the commonest groups of pharyngeal traumas in dogs and are also identified in the cat.

Fragmented stick is the commonest form of foreign body found but fishhooks, needles, bones and grass awns have also been identified. Larger breed dogs are over-represented possibly because stick injuries may occur when dogs carry or retrieve sticks and many witnessed cases are of this nature.

Retrospective studies show that most acute cases can be managed successfully, while some chronic cases may be very difficult to cure. Cases with perforations of the oesophagus have a worse prognosis, compared to cases with oropharyngeal lacerations alone.

There may be a history of chewing or chasing sticks or swallowing or chewing other foreign material. Common presenting signs are lethargy, dullness, pyrexia, hypersalivation, blood tinged saliva, dysphagia and oral or neck pain. Subcutaneous emphysema may be present at the time of presentation. Pharyngeal swelling may become life-threatening in some circumstances. The site of oropharyngeal penetration is rarely obvious on conscious oral examination. The commonest site of trauma is sublingual but tonsillar, palatine and dorsal or lateral pharyngeal wall injuries also occur frequently. Dorsal and caudal pharyngeal wall injuries can be very difficult to identify even endoscopically.

Often the diagnosis is suspected due either to historical information or clinical presentation. As previously mentioned, meaningful examination of the oropharyngeal region requires that the patient be anaesthetised. Oropharyngeal examination is performed with the patient in sternal recumbency. It is important to inspect the sublingual areas left and right of the frenulum; the tongue (base, left and right); the lateral pharyngeal walls and tonsillar crypts on both sides; the hard and soft palate and with the help of a laryngoscope the epiglottis is visualised with its attachments to the pharynx and the larynx. After the inspection of the glottis (including vocal cords and laryngeal cartilages), the patient is intubated. With the endotracheal tube in place, the larynx is depressed ventrally with the help of a laryngoscope and the pharynx is inspected completely. The caudal pharynx is inspected after rostral retraction of the soft palate. Retrograde nasopharyngoscopy with a flexible endoscope is indicated in cases with perforations of the soft or hard palate. The rostral oesophagus is inspected after ventral depression of the intubated larynx with a long-bladed laryngoscope or by using an endoscope. When perforations are not found at this time, complete cervical oesophageal endoscopy should be considered. When wood fragments are recognised within the soft tissues of a penetration tract retrograde withdrawal is not recommended because of the risk of fragmentation.

Radiographs of the thorax and cervical neck should be taken to look for the presence of free gas within the cervical tissue planes. It is unlikely that a foreign body will be identified if it is not radio-opaque, unless outlined by gas. Ultrasound can be useful in identifying foreign bodies and in my opinion this is an invaluable tool for investigation when performed by skilled operators. CT with or without intravenous contrast has been proven to be accurate in recognising wooden foreign bodies. Depending on the water content of the different layers of

the sticks, the foreign bodies show a variable attenuation pattern. CT images and three-dimensional reconstructions may aid in the planning of surgery in difficult cases. The use of MRI in dogs with chronic injuries has been evaluated in small number of cases and was helpful in the localisation of wooden foreign bodies. Other types of plant material (grass awns) were less apparent on MRI images.

In atypical cases, for example those cases where the diagnosis may be in doubt fine needle aspiration of masses may be of value, and rarely tru-cut biopsies.

I do not perform blood tests routinely, but rather I am guided by the clinical need of each individual.

Acute cases should be managed aggressively to control spreading cellulitis and to prevent the later formation of sinus tracts and abscesses that can lead to recurrent disease and damage to the vital structures of the neck. Conservative management at initial presentation has been identified as a possible contributing cause to the development of chronic disease. The general course of conservative management is that the patient responds to antibiotic and analgesic therapy but with relapse either immediately following withdrawal of therapy or within a short period of time afterwards.

The goal of the aggressive investigation and management of acute cases is to minimise the risk of abscess formation by removing foreign bodies early. As such, all tracts should be probed and explored. In acute cases in which there are identifiable indicators of pharyngeal injury e.g. emphysema without an obvious tract to follow, I explore the region surrounding the pharynx via a ventral midline incision (see above). To identify the penetration tract a sterile probe or rigid catheter may be inserted into the oropharyngeal laceration by an assistant. Using digital palpation of the neck, the surgeon identifies the probe and therefore also the penetration tract. The area of the penetration tract is explored by careful dissection around the muscular, nervous, and vascular structures, starting in the lower neck near the thoracic inlet, and working form unaffected (clean) towards affected (dirty) tissues in rostral direction. The areas rostral to the cricoid cartilage should be dissected with great care. The penetration tract (usually lateralised) is exposed and remaining foreign bodies are removed. The tract is gently flushed with saline. Wounds are closed over a Penrose or closed suction drain with only one point of exit per drain, in the lower neck area.

A bacteriology swab should be obtained from the cervical tissues following lavage and submitted, together with a piece of retrieved material, for aerobic and anaerobic culture.

Empirical antibiosis should be started, initially via and intravenous route (a second-generation cephalosporin or potentiated amoxicillin may be an appropriate choice), while awaiting culture results.

Most cases managed aggressively at initial presentation can be cured preventing the development of recurrence and the management chronic disease when the success rate following surgery is reduced

Canine thyroidectomy

Surgery has a major role to play in the management of thyroid tumours in dogs and in some cases may be curative. Frequently it is used with other adjunctive treatments such as external beam radiation and / or chemotherapy. Radio-iodine treatment for dogs is not currently available in the UK. External beam radiation and / or chemotherapy also provide treatment options for those dogs with lesions not amenable to surgery, where owners do not wish to proceed with surgery or when metastatic disease is present.

Thyroid hyperplasia and adenomata are usually not clinically detectable but are commonly found in dogs at post-mortem. Clinically detectable thyroid tumours in dogs are most often malignant (63-88 % of cases) and can be broadly classified as freely mobile or fixed. Ectopic thyroid tumours may be found in cranial to the larynx or in the cranial mediastinum. Malignant thyroid tumours may develop from either from the follicular cells (follicular carcinomas) or from the parafollicular C cells (medullary thyroid carcinomas). The metastatic rate is high with 33% of cases shown to have metastatic disease at the time of diagnosis, rising to 65-90% of cases through the course of the disease. These tumours commonly metastasise to the retropharyngeal and deep cervical lymph nodes and lungs as well as many other organs. Hyperthyroidism is found in approximately 10% of cases and most dogs with a thyroid tumour are euthyroid at the time of diagnosis. Hypothyroidism can develop and may be caused by lymphocytic thyroiditis.

Most dogs present with a palpable cervical mass. Compression or invasion of surrounding structures can cause other clinical symptoms such as coughing, respiratory distress, dysphagia, vomiting/regurgitation, anorexia, and facial swelling. Dysphonia and dyspnoea can be observed where the tumour has invaded the recurrent laryngeal nerve. Similarly Horner's syndrome may be encountered.

Canine thyroid tumours are highly vascular. Fine needle aspiration biopsies often contain excessive blood.

Freely movable thyroid tumours are best surgically removed without delay to prevent local invasive growth. The thyroid glands consist of two distinct lobes located lateral and ventral to the 5th-8th tracheal rings. The thyroid gland in dogs is 50 mm x 15 mm wide, but larger in immature and brachycephalic dogs and an isthmus may connect right and left thyroid glands. The parathyroid glands are salmon-coloured and distinct from the thyroid glands. On each side, the cranial parathyroid is external to the thyroid capsule while the caudal gland lies internal to the capsule and more medial. The cranial thyroid artery is a branch of the common carotid artery and provides the majority of arterial supply to dogs. The caudal thyroid artery is a branch of the brachiocephalic artery and unites with the cranial thyroid artery in the loose areolar tissue along the dorsomedial surface of the thyroid capsule and provides some arterial supply to dogs. The external parathyroid gland is vascularised by a branch of the cranial thyroid artery. The venous drainage is through the cranial and caudal thyroid veins. The thyroid nerve arises from the recurrent laryngeal nerve dorsomedial to thyroid glands.

The surgical approach is described above and the once the mass is identified it is carefully dissected from the surrounding structures. After the ligation of the caudal thyroid blood supply, mass dissection is performed from caudal to cranial. The carotid artery, jugular vein or vagosympathetic trunk may be incorporated in the tumour mass. The jugular vein, carotid artery, and vagosympathetic trunk can be sacrificed unilaterally with minimal postoperative morbidity. Finally, the cranial thyroid blood supply is ligated and divided to complete the tumour dissection. In cases of bilateral thyroid tumours an attempt is made to identify and spare one of the external parathyroid glands to prevent postoperative hypocalcaemia.

The long-term prognosis after surgical excision of a malignant thyroid tumour is guarded and depends on the histological malignancy grade and the presence of metastases. Dogs with small, freely moveable tumours treated with complete surgical excision have had median survival times of >3 years reported. In one study of dogs with fixed and invasive tumours treated with surgery and radiation therapy, median survival was 2 years. Interestingly 50% of the dogs in that study developed metastatic disease, but still survived 1-3 years. Radiation therapy alone can provide a good response.

Notes page

DIAGNOSIS AND MANAGEMENT OF DIAPHRAGMATIC HERNIAS

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The term hernia originates from Latin and means a rupture, while rupture also originates from Latin, rumpere to break.

The diaphragm is the major muscle of respiration. Diaphragmatic excursion and chest wall expansion increase the negative intrathoracic pressure required for inhalation. The sequelae to diaphragmatic defects and subsequent herniation of intra-abdominal contents are associated with significant morbidity and mortality.

Relevant anatomy

The diaphragm arises from four embryologic components: the septum transversum (central rendon), the two pleuroperitoneal folds, the cervical myotomes (dorsal and dorsolateral body wall), and the dorsal mesentery of the oesophagus (diaphragmatic crura). Failure of the development of the pleuroperitoneal folds, and subsequent muscular migration, results in congenital defects.

The diaphragm is a musculotendinous structure that separates the thoracic cavity from the abdominal cavity. It is composed of a central nonmuscular portion (central tendon) surrounded by a muscular portion. The muscular portion is divided into three separate areas: the pars costalis and pars lumbalis (both paired muscles) and the pars sternalis (unpaired muscle). The paired lumbar muscles form the diaphragmatic crura (R>L) and arise from the region of the third and fourth lumbar vertebrae. The pars costalis arising from the 8th to the 13th rib on each side interdigitates with the transversus abdominus muscle. Seen from the abdominal cavity each crus is a triangular muscular plate whose borders give rise to the tendinous portion. The thoracic side of the diaphragm is covered with parietal pleura, and the abdominal side with peritoneum.

Different structures traverse the diaphragm, including three distinct apertures that allow the aorta, oesophagus, and vena cava to pass. The aortic hiatus also allows passage to the thoracic duct and the azygous and hemiazygous veins. The arterial supply to the diaphragm derives from the right and left phrenic arteries arising from the paired phrenicoabdominal

arteries, the intercostal arteries, and the musculophrenic branches of the internal thoracic arteries.

The diaphragm receives its sole muscular neurologic impulse from the phrenic nerve (C5-7 in dogs and C4-6 in cats).

Definitions

In medical terms a hernia is a protrusion of an internal organ or tissue through a defect in the wall of the anatomical cavity in which it normally lies.

Hernias may be congenital or acquired, true or false. Congenital diaphragmatic hernias (CDH) occur because of embryologic defects in the diaphragm. Acquired diaphragmatic hernias (TDH) stem from all types of trauma, with blunt trauma accounting for the majority. True hernias develop through an existing or potential opening in the body wall that is pathologically enlarged or disrupted. False hernias occur through a rupture in the body wall.

Congenital pleuroperitoneal hernias are uncommon and many pups and kittens may die at or shortly after birth, although recently a few cases have been reported in the veterinary literature. Associated concerns with their presence in infants include pulmonary hypoplasia and pulmonary hypertension. In pups and kittens the veterinary surgeon should be alert for the presence of sternal abnormalities, cranial abdominal hernias and umbilical hernias in addition to both cardiac and pulmonary malformations and abnormalities. The most frequent places for such hernias to occur are the lumbocostal triangle (Bochdalek's foramen resulting in Bochdalek's diaphragmatic hernia) and the sterno-costal triangle (Morgagni's foramen, or Larrey's space resulting in Morgagni retrosternal diaphragmatic hernia).

Peritoneopericardial diaphragmatic hernia (PPDH) is the most common type of congenital diaphragmatic hernia: in this case lack of separation between the peritoneal and pleural spaces results in visceral organs herniating through Morgagni's foramen into the pericardial sac. In cases of congenital hernias, the herniated viscera often are located within a hernial sac in the pleural space, consisting of parietal diaphragmatic pleura and sometimes peritoneum.

By far, the most common cause of acquired diaphragmatic disorders is blunt trauma. Road traffic collisions are the leading cause of blunt diaphragmatic injury, whereas penetrating injuries can result from gunshot or stab wounds. The following theories have been postulated to explain the mechanism of rupture for blunt injuries:

- Shearing of a the stretched tissues;
- Avulsion of the diaphragm from its points of attachment;
- Sudden increase in the transdiaphragmatic pleuroperitoneal pressure gradient. The resting pressure differential between the pleural (-5 to -10 cm H_2O) and peritoneal (+2 to +10 cm H_2O) cavities can rise for a number of reasons including coughing and straining, without injury to the diaphragm. However the forces generated during blunt abdominal trauma may increase 100 fold over those encountered in those scenarios. The pressure differential is suggested to be exacerbated it the animal has an open glottis at the time of the blow.

Hiatus hernias are also classified as diaphragmatic hernias although they will not be considered further in these notes.

Diagnosis

Weimeraners and cocker spaniels, domestic longhair, Himalayan and Maine coon cats may all be at increased risk of PPDH. Patients with PPDH may also have co-existing abnormalities of the sternum and or cardiac defects. Polycystic kidneys have been reported in association with PPDH in cats.

There is frequently, but not always a history of trauma in TDH. In one study road traffic collisions were responsible 85% of the cases of DH reviewed with 15-25% of cases identified weeks after the traumatic incident.

Although signs of respiratory compromise might be expected this is not always the case. In chronic cases the clinical signs may be more vague such a weight loss, intermittent gastrointestinal signs e.g. vomiting etc. Auscultation of the thorax may reveal muffling of the heart sounds, borborygmi and loss of breath sounds.

Thoracic radiographs will often be sufficient to allow a confident diagnosis to be made. The presence of pleural effusion can complicate the diagnosis and repeating the radiograph after thoracocentesis may be of value in these cases. If the hernia is thought to be longstanding, thoracocentesis should be carried out with caution as in RARE cases removal of the fluid from the pleural space can precipitate re-expansion pulmonary oedema: if the patient starts to cough as fluid is withdrawn or the S_aO_2 drops unexpectedly the procedure should be stopped. Alternatively, ultrasonography can be used to confirm the integrity of the diaphragm with an accuracy of 93%. Positive contrast peritoneography may aid the diagnosis in some

cases (1.1ml/kg of water soluble iodinated contrast is used). The use of MRI and CT is described.

What is the cause of respiratory impairment following diaphragmatic hernia?

There are a number of common potential causes for respiratory compromise in the presence of diaphragmatic hernias (DH).

- Direct consequence of the presence of DH:
    - Loss of function of the normal respiratory mechanism (failure of diaphragmatic excursion);
    - Loss of functional residual capacity (mass effect from herniated organs and or pleural effusion / pneumothorax);
    - Atelectasis of the lung lobes.
- Associated injuries including:
    - Pulmonary contusions;
    - Rib fractures;
    - Flail chest;
    - Pneumothorax
    - Haemothorax.
- The effects of shock and pain.

Myocardial contusion is often present in the hours and days following trauma and cardiac output may decrease as a consequence. When myocardial injury is concomitant with impaired ventilation, tissue hypoxia can be more severe. Pain resulting from chest and abdominal contusion and accompanying injuries causes voluntary restriction of thoracic excursion and can therefore further compromise ventilatory capability.

How do I stabilise these patients?

Initial stabilisation of diaphragmatic hernia patients consists of medical therapy for shock and respiratory compromise. Close observation for signs of deterioration or failure to respond appropriately to medical therapy is paramount.

Cyanosis is a late sign of the need for oxygen, and any signs suggestive of hypoxia should be treated promptly to prevent this happening i.e., nasal flaring, dyspnoea, reduced mentation and signs of oxygen hunger such as abducted elbows, extended head and neck, and open-mouthed breathing. Oxygen supplementation must not induce undue stress that can result in a deterioration of the animal's condition. Patients that fail to respond to oxygen

supplementation may have severe ventilation perfusion mismatching as a consequence of atelectasis or pulmonary contusions.

Adequate volume replacement is essential. However, vascular support must be delivered with the knowledge that these patients often have concurrent pathology such as atelectasis and pulmonary contusions that can be exacerbated by injudicious fluid administration. A measured approach to volume resuscitation should be adopted rather than using the broad brush of recommended 'shock rate' fluids.

Timing of anaesthesia and surgery

Surgery is best performed after a period of patient stabilisation. Unfortunately, 10-15% of patients may die prior to surgery as a result of their injuries.

Historically, surgery was avoided where possible in the 24 hours following surgery due to an increased mortality rate. However, surgery is now recommended once the patient is stable to proceed to surgery and should not be delayed if a patient is deteriorating despite supportive care.

Some cases will require immediate surgical intervention because of the risk of acute decompensation. These include:

- Diaphragmatic hernia with intrathoracic gastric dilatation or GDV: if the stomach or proximal small intestines are herniated, the risk of pyloric outflow and cardial obstruction are high and cases may present with a tension gastrothorax as a result of GD or worse, GDV within the thoracic cavity. As normal a stomach tube may be passed in an attempt to relieve the pressure within the stomach.
- Rupture of the gastrointestinal tract;
- Strangulation of herniated organs / structures;
- Rupture of the biliary tract;
- Ongoing life threatening intrabdominal or intrathoracic haemorrhage;
- Tension pneumothorax secondary to lung or large airway injury.

Anaesthesia for diaphragmatic rupture

There are several key points to remember when anaesthetising patients with diaphragmatic defects, namely: minimise stress as stress increases oxygen requirements and exerts further strain on a system that is already compromised; aim for rapid induction and intubation to

gain early control of the airway; be prepared to provide ventilatory support from induction until recovery and remove residual air from the thorax before recovery.

It is easy to over-inflate the lungs when ventilating patients manually. If you consider that the tidal volume of a patient is calculated as weight (kg) x 10–15 ml/kg, and that the reservoir bag on a T piece is usually 500ml, it is easy to understand how we could over-inflate the lungs of a 4 kg cat by squeezing the bag too aggressively. This is dangerous in a normal animal and is even more so in an animal that is likely to have atelectasis due to DH. The aim is to use the minimal force possible to achieve a normal chest excursion. Frequently it is possible to adequately inflate the patient's lungs without completely closing the adjustable pressure limiting valve. One advantage of this technique is that (to a degree!) any excessive inflation pressure generated will be obtunded by escape of the anaesthetic gases through the partly open valve.

Obviously the effectiveness of the ventilation should be monitored and both capnography and pulse oximetry are useful in this regard.

Surgical approach

A ventral midline abdominal approach is used most commonly. The incision should extend from the xiphoid to a point no further cranial than the umbilicus. In rare cases a caudal median sternotomy or paracostal incision may be required to allow management of intrathoracic pathology.

Goals of surgery

- Identify the position of the hernia;
- Reduce hernia contents;
- Assess abdominal viscera for viability;
- Assess thoracic viscera for injury;
- Repair diaphragmatic defect: tension free repair of viable tissue;
- Remove air and fluid from the thorax.

Identify hernia

Most diaphragmatic tears are muscular and are located ventrally. The site of the hernia is usually easily identified if abdominal contents are herniated at the time of surgery. However, abdominal contents can spontaneously reduce and in any case there may be multiple points

of injury. The dorsal diaphragm and crura are difficult to access and this is the site where hernias are most likely to be missed.

Reduce herniated organs

The liver, small intestine and pancreas are most commonly prolapsed into the thoracic cavity when the diaphragm defect is on the right side, whereas the stomach, spleen, and small intestine prolapse on the left side. Although, gentle traction on the gastrointestinal tract is often all that is necessary to reduce it, care should be taken with soft parenchymal organs and in particular the liver. The liver may be congested and traction could be sufficient to cause capsular tears and subsequent haemorrhage. It is better to gently lift any herniated liver lobes: you may need to enlarge the defect to achieve this. When enlarging the defect remember the position of the aorta, post-hepatic cava and hepatic veins.

Non-reducible (incarcerated) hernias

Hernias become irreducible if the hernia ring is too small to allow the contents to be removed or if the hernia contents become incarcerated by adhesions to thoracic structures or the hernia ring.

Relative undersize of the hernial ring can occur in recent hernias where the hernia contents increase in size due to distension with gas or fluid (hollow organs) or through venous congestion. In chronic hernias the hernia ring may contract through fibrosis. Withdrawing fluid and / or gas from hollow organs may help or alternatively the hernial ring may be enlarged (remembering to avoid the caval foramen etc.!).

Intrathoracic adhesions between abdominal and thoracic organs of less than seven to 14 days duration are likely to be easily disrupted. Longer standing adhesions will be fibrous and will require dissection. Adhesions should be disrupted and dissected under direct visualisation. If they involve the caudal lung lobes or caudal thoracic structures, they will often be visualised and accessible through the abdominal incision. However, median sternotomy may be required. Partial resection of the involved organs may be necessary e.g. lung, liver lobe, spleen.

Assess viability of hernial contents

If there is concern over the viability of parenchymatous organs such as liver lobe or spleen, excision of diseased tissue is indicated. Ideally, when organs have been twisted on a vascular pedicle and require resection, resection should be performed without untwisting the pedicle to prevent the release of toxins and inflammatory mediators into the vascular system.

Liver lobectomies are often performed before the liver lobe is removed from the thoracic cavity.

Assess viability and integrity of thoracic contents

Flood the thoracic cavity with saline and ensure there are no points of leakage from the lungs. Remove the saline by suction prior to herniorrhaphy.

Thoracic drain placement (see below)

Thoracic drains are required if there is a risk of ongoing air or fluid accumulation and generally I would place one if the animal had pneumothorax at presentation, if there is concern over the integrity or viability of the thoracic organs or if there was a moderate or large pleural effusion at surgery. It is far better to place a thoracic drain at this stage and not use it than to have to place one postoperatively. Tubes should be placed under direct visualisation prior to closing the diaphragmatic defect.

Herniorrhaphy

Importantly, remember that you are trying to achieve an organ tight seal and not an air tight seal between the abdominal and thoracic cavities.

As for any herniorrhaphy, viable tissue should be sutured without tension to achieve the best repair. Careful handling of tissues is required to ensure that they are not compromised further. Recent diaphragmatic repairs are usually easy to reconstruct anatomically. Care must be taken to ensure that viable tissue is included in each suture bearing in mind that compromised tissues may not have started to necrose at the time of surgery. The margins of chronic hernias may have atrophied and the rolled edges be fixed in position by adhesions. Adhesions should be resected to allow anatomic reconstruction where possible. Congenital pleuroperitoneal hernias can present unique challenges as the defect is often very large.

Debridement of hernia rings is controversial. Advantages include the removal of non-viable tissue but disadvantages include the increase in size of defect that must be closed and the potential to exacerbate local tissue problems through trauma associated with debridement. Unless grossly necrotic tissues are present, debridement is probably not necessary and may in fact increase your chance of wound dehiscence.

Synthetic monofilament absorbable sutures are appropriate e.g. polydioxanone, but non-absorbable synthetic low-antigenicity suture material such as polypropylene may also be

used. However, diaphragmatic rents do not require long term support so nonabsorbable suture materials are generally not necessary.

A single row of simple interrupted or simple continuous sutures is appropriate. The defect should be sutured so that there is no tension across the site. Double rows of suture material do not add to the strength or stability of repair but do increase tissue handling and foreign materials at the surgery site so are contraindicated. I prefer to use continuous patterns to limit soft tissue irritation caused by suture knots but will use interrupted patterns if I am concerned over tissue viability. Use stay sutures and pre-place sutures before tying if using interrupted suture patterns to aid closure. Start with the radial component of complex tears and always work from the dorsal margin of the defect ventrally to make closure as easy as possible. If the hernia includes the caval foramen, take care not to restrict the caudal vena cava during herniorrhaphy.

If required, diaphragmatic advancement achieved by incising the diaphragm along its costal attachments and advancing it axially before suturing it in place, (8-ply) small intestinal submucosa (Vet Bio SIS), pericardial flaps, transversus abdominus muscular flaps and synthetic meshes e.g. polypropylene mesh may be used to augment the repair of the diaphragm where there has been tissue loss or contraction. The finished repair may be reinforced using an omental reinforcement or if necessary an omental pedicle flap.

Abdominal exploration

Briefly assess the abdominal contents to ensure that there are been no concurrent injury to organs that have not been herniated, or indeed other organs subsequent to trauma prior to routine closure of the abdominal wall.

Re-establish negative intrathoracic pressure

There are a number of methods that can be used to re-establish negative intrathoracic pressure:

- A thoracostomy tube can be placed in the 7th or 8th intercostal space under direct vision prior to repair of the defect. In recovery, the patient's position can be changed while attempting to aspirate air. The tube is removed when the patient has had a negative pressure for 6-12 hours.
- Transdiaphragmatic thoracocentesis. Following repair of the hernia a needle or intravenous catheter is placed through the diaphragm and into the thoracic cavity. Thoracic cavity air is evacuated of air using a three way tap and syringe.

- Feeding tube drainage. A 12-14 French feeding tube is brought into the peritoneal cavity through a paramedian stab incision in the cranioventral body wall. The tube is passed through the defect in the diaphragm just prior to its final closure: be sure that all fenestrations in the tube are beyond the diaphragm. The hernial repair is completed around the tube and the abdominal incision closed routinely. The thoracic cavity is evacuated of air using a three way tap and syringe. When negative pressure is obtained in all positions, the tube is gently pulled from the chest.
- Transthoracic needle thoracocentesis. This is performed after the procedure is complete.

The traditional technique of overinflating the lungs prior to final suture placement in order to re-inflate atelectatic areas of lung and to evacuate air from the thorax is contraindicated and probably contributed to the high mortality rates in the early reports of diaphragmatic hernia management. In this syndrome, increased permeability of the alveolar membrane leads to rapid pooling of fluid in the alveolar space and respiratory collapse. This is seen within a few hours of re-expansion and is usually progressive and fatal. The aetiology is uncertain and could relate to membrane injury secondary to endotoxaemia, hypoxia or reperfusion injury but what is clear is that its development is directly linked to rapid re-inflation and over-inflation of lung. It is far safer to slowly re-establish negative pressure in the thorax and to allow atelectatic areas of lung to re-inflate over time.

Regardless of the method used, all patients are monitored carefully for at least the next 12-24 hours for signs of respiratory compromise. If signs of respiratory compromise arise e.g. dyspnoea, tachypnoea, etc., thoracocentesis of the right and left hemithorax should be performed to rule out unexpected pneumothorax or fluid accumulation, and certainly prior to attempting radiography.

Reduced abdominal domain

In cases of chronic disease the abdominal capacity (domain) may be reduced due to myofascial retraction. In this situation the abdominal muscles contract and can no longer stretch quickly back to their original length and fascial sheaths remodel. This may be complicated by any traumatic damage to the muscles incurred at the time of the original trauma. Consequently, direct closure of the surgical incision may be difficult or impossible after hernia reduction, or result in abdominal compartment syndrome.

Myofascial releasing incisions may be used to aid closure of the abdominal wound: this can be achieved by creating relaxing incisions of the external rectus sheath parallel to the

surgical incision through the linea alba. Alternatively, a staged approach to closure of the wound may be required or mesh support may be required.

Post-operative thoracic x-rays

It is good practice to radiograph the thorax following diaphragmatic herniorrhaphy to confirm the success of the procedure, the position of the thoracic drain if one has been used, and the severity of any remaining pneumothorax.

Recovery and complications

Most cases that survive surgery but die do so in the immediate postoperative period as the result of acute respiratory collapse. This may be secondary to re-expansion pulmonary injuries or pneumothorax due to previously undiagnosed lung injuries that become apparent as the lungs re-expand or due to thoracostomy tube complications. Patients need to be carefully monitored postoperatively to ensure that their respiratory status is not deteriorating and should continue to receive oxygen supplementation well into the recovery period. If there is any deterioration, diagnostic thoracocentesis and radiography early in the course of the problem to identify the cause is the safest option. Animals with empty abdomen syndrome may show signs of respiratory distress as a result of abdominal compartment syndrome. Ventricular arrhythmias are also common.

Less frequently, complications associated with the organs that have herniated are encountered. Gastrointestinal tract perforation and haemorrhage from splenectomy and partial hepatectomy are potential complications. Care must be taken to replace abdominal contents in a normal anatomical position to prevent vascular compromise and strangulation. The most frequently encountered problem of this nature is liver lobe torsion. This causes liver lobe congestion but more importantly can cause kinking of the caudal vena cava and abdominal venous congestion. Watch out for pancreatitis postoperatively as the pancreas may have been traumatised either at the time of the original injury or subsequently during reduction of the hernial contents.

What is the prognosis?

Reported mortality rates for TDH range from 12-48%: 10-15% of cases may die before being anaesthetised during the stabilisation and investigation period. Reported mortality rates for PPDH of 3.2-14 and 12.5% for cats and dogs have been reported.

In long-standing chronic hernias, the prognosis is guarded. Although these cases often present for elective repair of a well-tolerated hernia, the potential for significant intraoperative and postoperative complications increases making these cases surgical challenges.

What is diaphragmatic eventration?

Diaphragmatic eventration is the abnormal displacement of part or all of an otherwise intact diaphragm into the thoracic cavity. This occurs as a consequence of a thinning of the diaphragmatic musculature and may be a congenital or acquired abnormality. In human infants the congenital form of the condition is characterised by muscular paralysis, atrophy or aplasia. Acquired eventration in humans is usually as a consequence of trauma, inflammation or neoplastic invasion of the phrenic nerve.

In veterinary medicine the condition is seen most commonly in cats and is generally of no clinical significance but rather an incidental finding: it may however cause respiratory compromise. It is an important differential diagnosis for diaphragmatic herniation in cats but the uninterrupted continuity of the diaphragm differentiates eventration from herniation.

Diaphragmatic eventration is sometimes referred to as a peritoneocele: however in human medicine peritoneocele refers to a weakening in the floor of the abdominal cavity in the pelvic region.

FIRST AID OF WOUNDS AND FRACTURES

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Once life threatening problems are dealt with or identified, emergency management of wounds and fractures should be addressed. Emergency management should prevent any additional injury, minimise contamination and control systemic implication. Open wounds may be covered with a sterile dressing until the patient is stabilised. Many patients may be in pain from their injuries, so appropriate analgesia is important. Fractious patients may require sedation or general anaesthesia for wound evaluation to be performed.

Wounds

Emergency treatment of wounds can be carried out prior to definitive treatment later. Analgesia and effective restraint may be necessary, until the animal is stable enough for sedation or GA. Wounds can be classified by their cause and the type of tissue damage caused: incisional, abrasion, avulsion (or degloving), shearing, puncture or perforated, and burns. Regardless of the aetiology of the wound, the factor which has the single biggest impact of future healing is the presence of contamination and necrotic tissue.

Haemostasis

Bleeding should be controlled first. Apply direct pressure with sterile gauze swabs, or by bandaging. Pressure can be applied to brachial or femoral arteries if profuse arterial haemorrhage is present.

A form of tourniquet can be applied above the wound if it is on a limb. Narrow elastic tourniquets such as Penrose drains put significant pressure on neurovascular structures and should only be used for up to 5 minutes. Bands 5-10cm wide can be used for up to 30 minutes. Blood pressure cuffs can be placed proximal to the wound and inflated to 20-30cm H20 higher than arterial pressure- these can be left in place for up to 6 hours. Ultimately ligation may be needed for larger vessels, and the limb then relies on collateral circulation.

Control of Contamination

After adequate haemostasis the wound should be covered with a sterile dressing while preparing for lavage. Wearing sterile gloves and gown, the wound is packed with sterile gel

or soaked swabs and hair clipped from the wound outwards. The wound can then be lavaged with copious saline of Hartmanns. If gross contamination is present, use tap water followed by Hartmanns. Then cover the wound again with a sterile dressing.

Once the patient is stable a more thorough evaluation may be carried out. Appropriate chemical restraint may be required for examination. Diagnostic imaging may be used to check for foreign material, penetrating injuries, associated fractures, dislocations and tendon or ligament damage. A management plan should take into account the wound's location, size, damage to local structures and the amount of tissue loss.

Lavage reduces the number of bacteria present, and helps to loosen necrotic tissue and debris. Lavage solutions containing antibacterials or detergents should be avoided; they can cause cell damage, slow wound healing and may result in bacterial resistance.

The pressure for lavage solution needs to exceed the adhesive and cohesive forces of the contaminant, yet avoid pushing debris into the tissues and causing damage to vital tissues. The suggested force is 5–10 psi. In practice this can be achieved by using a bag of fluid with an 18–20 gauge needle fitted to the end of an attached giving set. The volume of lavage solution is equally important. For small, superficial wounds, 0.5–1 l is generally used; for larger wounds several litres of sterile lavage solution may be needed.

Any traumatic wound will require the debridement of devitalised tissues and foreign material in order to prevent infection and necrosis and to promote optimal wound healing. Debridement may be performed using a number of different methods.

Sharp debridement involves the use of a scalpel blade or scissors and may be carried out carefully in stages in order to preserve as much healthy tissue as possible. Subcutaneous tissue, fat, skin, fascia and muscle can generally be freely derided. Tendons, vessels, nerves and bone should be derided much more conservatively.

Mechanical debridement involves the use of dressings, irrigation or hydrosurgery. Wet to dry dressings are commonly used in veterinary practice but their use requires sedation or anaesthesia as removal is painful.

Autolytic debridement involves the use of wound dressings and solutions, e.g. hydrogels, and is not recommended in infected wounds.

Following debridement, a decision needs to be made about wound closure. Options include primary closure, delayed primary closure, secondary closure or secondary intention. If doubts exist over remaining contamination and necrotic tissue, a period of open wound management is indicated.

Antibiotics

Wounds correctly managed with lavage and debridement followed by closure do not routinely require antibiotics. However, deep wounds involving muscle, severe tissue damage, systemic infection or an immune-compromised patient are indications for broad spectrum intravenous antibiotics. A first generation cephalosporin or clavulanic acid potentiated amoxicillin are good first line choices.

Fractures

Definitive treatments of fractures, often requires a lengthy anaesthetic to get accurate radiographs and carry out surgery. Emergency management of the injury can improve the systemic condition of the animal, and help to achieve a reduction in complications when it comes to fixation.

Systemic consequences of fractures can be dramatic. Large amounts of blood can be lost from the circulation into the fracture site, especially in femoral, humeral and pelvic fractures- up to 30%. Cats with pelvic fractures will commonly be markedly anaemic a day or two after initial stabilisation.

Assessment

Obvious clinical signs such as deformity, swelling, crepitus, instability and pain will often be present. The most important factors to assess are the position of the fracture, its relationship to critical structures, whether it is open or closed. The extremities below the fracture can be assessed for the presence of pulses, pain sensation, and any oedema that indicates impaired venous and lymphatic return.

Blood supply to the distal limb- evidence of perfusion needs to be assessed, check warmth compared to other limbs, colour of pads/nail beds and capillary refill, palpable pulses, and Doppler ultrasound.

Neurological function- obviously if the limb is broken we cannot carry out a full neuro exam on the leg, but the most useful test is to evaluate the sensory function of the nerves supplying the distal limb. Pinching the skin of the digits can assess the radial and ulnar nerve

on the forelimb, and the sciatic and femoral on the hind limb. Absence of deep pain sensation carries a poor prognosis, although false negative results can occur if the animal is obtunded or moribund due to systemic disorders.

Classifying fractures further than this, in terms of number and position of fracture lines, requires careful radiography and is more appropriate to the ultimate fixation of the fracture than the actual emergency stabilisation of the animal.

Open fractures

Are fractures where the skin has been broken. Open fractures managed correctly on initial presentation have a much greater chance of functional recovery.

Open fractures are graded;

- Grade 1) Bone fragment penetrates the skin. The fragment usually pops back in again, so look for any small punctures
- Grade 2) Penetrating external wound causes a fracture- e.g. dog bite- bone not directly exposed
- Grade 3) High degree of tissue loss, high energy trauma, contamination

Open fractures should be treated as previously described for wounds- i.e. covered with a sterile dressing, then clipped, lavaged and dressed again.

Support dressings

Swelling, soft tissue damage, skin punctures and further blood loss can result from instability at fracture sites. In some cases the application of a suitable supportive padded dressing can minimise movement at the fracture site, preventing further damage and increasing patient comfort. Trying to apply support dressings above the elbow or above the stifle tends to result in the dressing slipping downwards and the extra weight making the fracture more painful due to a pendulum effect.

Fracture site	**Suitable dressing**
Radius and Ulna	Support dressing +/- splint
Tibia	Support dressing +/- splint
Carpus/tarsus and below	Support dressing
Femur	None, or full spica splint
Humerus	None, or full spica splint
Pelvis	None
Cervical Spine	Neck splint
Thoracolumbar spine	Back splint
Facial/maxilla/calvarium	None

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THE SAFE USE OF CYTOTOXIC DRUGS IN SMALL ANIMAL PRACTICE

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Cytotoxic drugs are commonly used in the treatment of veterinary patients diagnosed with cancer, within both specialist and private practice. Patients that may have been referred for treatment to a cancer specialist in the past can now be treated in their own general practice, whilst discussions with a specialist provide a suitable and optimal course of chemotherapy. Cytotoxic drugs are potentially hazardous for the people involved with their administration, and the patient receiving them, it is therefore imperative that as well as the veterinary surgeon (VS), the veterinary nurses (VN) and clients understand the potential hazards associated with these drugs and implement protocols for their safe use, following recommended guidelines already put in place by the Health and Safety Executive (HSE).

Cytotoxic drugs, also known as antineoplastic, anticancer, antitumour and cancer chemotherapy drugs are constructed using a wide range of chemical compounds and are used in the treatment of various types of cancer. By interfering with cell division they produce both cytotoxic and cytostatic effects and are therefore able to damage normal cells as well as the target neoplastic cells. Chemotherapy has become a conventional and frequently used cancer treatment modality and with clients becoming progressively better informed of the treatment options available, via access to websites and pet cancer forums, there is an increased pressure on VS to use cytotoxic drugs. The financial constraints are also less of a burden on clients opting for chemotherapy as a large number of clients now have their animals insured. The VS and VN must have an in-depth knowledge of the cytotoxic drugs they are handling as they are acknowledged to be an occupational hazard as stated within the Control of Substances Hazardous to Health Regulations (COSHH). The majority of cytotoxic drugs utilised, as well as being an irritant to the skin and mucous membranes have mutagenic, teratogenic or carcinogenic properties. Exposure to cytotoxic drugs can occur in various ways including: absorption through the skin, ingestion, inhalation and accidental inoculation. Risks of exposure, however, can be reduced to an absolute minimum by adhering to the rules and regulations set out by the Control of Substances Hazardous to Health Regulations 2002. Standard operating procedures (SOP), local rules and regular risk assessments should be implemented within individual practices.

Storage

Due to the risks involved when handling them, individual practice policies need to be established to protect not only the staff involved in the administration of cytotoxic drugs, but also their colleagues, the patients and the owners. Cytotoxic drugs requiring refrigeration should be kept in a designated fridge and if this is not possible they should be stored away from food in a clearly labeled area within the fridge, the refrigerator should be marked with cytotoxic warning signs externally. Other recommendations are to keep cytotoxic drugs in the main pharmacy area in a locked cupboard. On delivery cytotoxic drugs must be kept in their original sealed containers whilst being transported to their area of storage, where they should then be wiped down with alcohol or dilute bleach to deactivate and decontaminate any residual drugs on the bottles. Gloves should be worn by the handler at all times and standard operating protocols (SOP) should be written on the handling and storage of cytotoxic drugs.

Personal Protective Equipment and Drug Administration

Personal protective equipment (PPE) must be provided under the Personal Protective Equipment at Work Regulations Act 1992 by employers, and must be worn by employees. The following PPE should be worn: gloves, arm sleeves, gowns, eye protection and masks, with specific requirements for each. These include: gloves with increasing thickness from the cuff to the fingertips, arm-sleeves that are non-permeable, long sleeved gowns which are disposable and waterproof, eye protection in the form of goggles which need to fully cover the eyes, and respiratory masks as opposed to surgical masks which offer no respiratory protection. In the absence of specific chemotherapy gloves, double gloving with latex examination gloves should provide the handler with adequate protection and a non-absorbent gown should be worn. PPE should not only be worn by the person administering the compounds, but also the member of staff restraining the animal. Staff should take every precaution to minimise risks to themselves and colleagues whilst handling cytotoxic drugs and therefore all forms of PPE should be utilised. When preparing drugs for administration the use of a biological safety cabinet is recommended but the majority of smaller practices giving the occasional treatment will not have access to this. Preparing drugs without a safety cabinet is a health and safety issue, however if the occasional treatment is given it may be acceptable if certain protocols are followed. Drug preparation should be performed by trained staff in a designated area, PPE must be worn, preparation carried out on an absorbent mat, utilising ready-to-use formulations, never dispelling air from filled syringes, and using luer lock syringes are all examples of good practice. The use of closed systems such as Phaseal, which are needle free and reduce the risk of accidental inoculation and aerosolisation, using a room away from other people and animals and a no food or drink

policy within the chemotherapy administration room will reduce the risks to personnel. Pregnant and immunocompromised staff should not be involved in chemotherapy. A risk assessment should be developed to deal with the possibility of cytotoxic drug spillages and purpose made spill kits, as opposed to standard equipment found in the practice, should be used.

Drug Formulations

Cytotoxic drugs come in one of two forms, tablets or capsules for oral administration, and powders or solutions for injection. It is imperative for patient safety that staff understand the correct route of administration, as some drugs such as L-asparaginase will cause an acute and fatal anaphylaxis if given intravenously. Oral medications should never be crushed or broken and capsules never be opened, gloves should be worn during administration and they should be dispensed in their original container. Staff should thoroughly explain to clients the procedure of giving oral medications to their animal at home and the potential hazards involved. When administering intravenous cytotoxic drugs precautions must be taken to prevent extravasation and exposure to personnel. Adequate restraint of the patient, the use of large veins (for example the cephalic), the use of butterfly catheters for small volumes and conventional catheters for larger volumes, alternating legs between treatments, and aiming for a clean stick will reduce risks. The catheter should be firmly placed but not covered to allow monitoring of the site, and patency should be checked using sodium chloride as heparin can precipitate some drugs. The catheter should be flushed through with roughly 20mls of saline before being removed. There are a few drugs which are administered by the intramuscular and subcutaneous routes but their discussion is out with the scope of this lecture.

Adverse Effects

It has been reported that only one in four animals have adverse effects to chemotherapy with 5% of these patients requiring hospitalisation. Side effects are uncommon however some can have serious consequences for both the pet and the owner. Toxicity is the greatest treatment limiting factor when cytotoxic drugs are utilised and the most common side effects include: bone marrow suppression with resultant infection, gastrointestinal toxicity, extravascular injection of cytotoxic drugs (extravasation), and allergic reactions. Side effects can be further separated into those that require immediate attention (hypersensitivity and extravasation), and those which are delayed (neutropaenia and gastrointestinal effects). Staff should have knowledge of the side effects that may occur post discharge and be able to recognise and respond to immediate side effects. The drug which is most frequently associated with allergic reactions is doxorubicin. The clinical signs of anaphylaxis include:

pruritis, facial oedema, wheals, erythema, vomiting, restlessness and head shaking. Should an anaphylaxis occur administration of doxorubicin should cease and an anti-histamine should be given after which treatment can commence. It is recommended that if a patient has had a history of anaphylaxis during administration then an anti-histamine should be administered prior to treatment, prevention is better than cure. There are certain drugs which are classed as vesicants such as doxorubicin and vincristine, and will therefore cause serious damage to the surrounding tissue if they extravasate. Staff should have a sound knowledge of these and take every precaution to prevent this from occurring. Catheters should be placed cleanly the first time and the catheter site should be monitored constantly throughout administration. If extravasation should occur the infusion should be stopped, aspiration of any remaining drug from the catheter should be attempted and corticosteroids should then be administered systemically, locally and topically. Specific recommendations given for doxorubicin include: infiltrating the area with sodium bicarbonate, applying dimethylsulfoxide topically every 2 hours, and applying hydrocortisone cream and cold compresses. For vincristine, infiltration of the area with hyaluronidase and heat compressions is recommended. Other options include immediate surgical excision of the site and the use of x-shaped excisions through which suction is applied via a snakebite extractor kit until the drainage has stopped, this had been shown to be a successful treatment mechanism in humans that have had doxorubicin extravasation. Gastrointestinal effects are seen in a small number of patients post administration and the VN may be the first point of contact for the owner. The VN needs to know which questions should be asked to decipher whether or not the patient requires hospitalisation. The most common side-effects seen are weight loss, decreased appetite, diarrhoea and nausea. An anti-emetic, either cerenia or metoclopramide can be given pre treatment with oral medications as a follow up post treatment such as metoclopramide, cerenia or ondansetron. An appetite stimulant such as mirtazapine when weight loss and anorexia are evident can also be utilised. An animal with gastrointestinal signs should be seen when vomiting occurs more than 3 times in a 24 hour period, if the patient is incapable of retaining food or water without vomiting, anorexia of more than 2 days is present, and when there is watery or bloody diarrhoea, weakness, lethargy or depression.

Neutropaenia

Neutropaenia is a common side-effect of cytotoxic drugs, which usually manifests within 7 – 10 days post treatment. Patients that are mildly neutropaenic may not show any clinical signs of illness and treatment decisions should be made on the absolute neutrophil count and not from the total white blood cell count. It is important to remember that not all neutropaenic patients will present with pyrexia and some may have normal or decreased

temperatures but that this presentation does not rule out the presence of life-threatening infections. By gaining an accurate history from the client the VN can determine the likelihood of neutropaenia and should recommend that the patient be seen to establish the absolute white cell count. Patients with a neutrophil count of more than 1000/microlitre and no pyrexia can be managed as outpatients, whereas those with pyrexia or that are systemically ill should be hospitalised. These patients will require barrier nursing, antibiotic treatment, intravenous fluid therapy and a high level of nursing care, due to their inability to fight infection. Cyclophosphamide has been shown to cause a sterile haemorrhagic cystitis in both the canine and feline population. Patients will most likely be on protocols which incorporate prednisolone, which will be beneficial as it has diuretic and anti-inflammatory effects. The use of frusemide prior to treatment promotes urination and prevents the buildup of metabolites that irritate the bladder lining. The VN should be aware of the clinical signs of cystitis and advise the owner accordingly should this occur, for example ensuring the patient has an adequate intake of fluids.

Waste Disposal

The disposal of cytotoxic waste is an area with which all practice staff should be comfortable. Sharps should be disposed of in an impermeable container labeled for cytotoxic waste, and solid waste such as syringes and PPE should be placed in polythene bags before placement in cytotoxic bins. Vials that have less than 1ml of drugs left in them should be treated as solid waste and any expired drugs double wrapped and labeled before placement in a destruction of old pharmaceuticals (DOOP) bin. The kennels of patients receiving chemotherapy must be identified so that all members of staff can take the relevant precautions. Cytotoxic drugs can be excreted via patient's bodily fluids, for example, urine, faeces, vomit and saliva. It is important that veterinary staff know how to handle and dispose of excreta and that staff give the relevant information on this area to the client at discharge as most cytotoxic drugs are excreted unchanged. It has been documented in human patients receiving vincristine that approximately 12% of the drug is excreted in urine and up to 70% of the drug excreted in faeces. The use of routine PPE should be enough to protect both staff and clients from exposure via bodily fluids. Some drugs which are excreted in patient's urine may contain up to 90% of the active drug and it is therefore important that aerosolisation of this urine does not occur; therefore power hosing should be avoided. Clients must be made aware of the potential hazards of patient waste and the following instructions be given: faeces should be disposed of in plastic bags and urine spots should be diluted with water. Some drugs can be present in a patient's urine for up to 5 days post treatment and precautions should therefore be taken for this period of time at least. There are health and safety issues which must be addressed when cytotoxic drugs are being used due to the

potential hazards involved with their use. All staff involved with handling cytotoxic drugs should be adequately trained and provided with the appropriate PPE.

TREATMENT AND NURSING CARE OF ONCOLOGY PATIENTS – CASE STUDIES

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Feline Lymphoma

Lymphoma, also termed lymphosarcoma (LSA) is one of the most common cancers seen in companion animals, accounting for 83% of all haematopoietic tumours. It is responsible for almost one third of all feline malignancies and is frequently diagnosed in practice. The veterinary nurse (VN) should be aware of the cytotoxic drugs used in the treatment protocols of feline LSA and the potential side effects associated with them in order to provide patients with both safe and appropriate nursing care.

LSA is the malignant proliferation of lymphocytes, which are key components of the immune system. Feline LSA commonly presents itself in one of three identifiable forms, mediastinal, alimentary and multifocal, with the clinical presentation dependent on the area of involvement. The most commonly affected site is the gastrointestinal tract with clinical signs including decreased appetite and lethargy. Cytotoxic drugs are either used as a single treatment modality or as a multimodality treatment which is often necessary because LSA is a multisystem disease, meaning that surgery or targeted radiation alone are insufficient. Clients should be made aware of all the treatment options that are available to enable them, in conjunction with the veterinary surgeon (VS), to make an informed decision on the most suitable treatment protocol for their animal. Chemotherapy is utilised to increase the quality of a patient's life as well as their life expectancy, however cytotoxic drugs carry various side-effects and health and safety implications which must be considered. Feline LSA is classed as a haematopoietic tumour. LSA accounts for 90% of all haematopoietic malignancies diagnosed in the feline species. There are many factors which contribute to the development of LSA in cats, including feline leukaemia virus (FeLV), genetic components, exposure to carcinogens and other unknown factors. The VN should be aware of contributing factors as this is a common question from owners, and the VN can therefore provide them with accurate and relevant information. A link has also been described between immunosuppression and feline LSA, for example, cats that are positive to feline immunodeficiency virus (FIV) are at a higher risk of developing malignant lymphoma. The VN should take this into consideration when these patients are hospitalised to reduce the risk to other patients. The site at which the disease occurs can also be related to age and

FeLV. The development of FeLV vaccines has brought about a decrease in the number of new cases diagnosed, with a concurrent decrease in the number of FeLV related cases of LSA.

LSA can be classified using the National Cancer Institute working Formulation and the Revised European and American Lymphoma/World Health Organisation classification. LSA is not only classified by anatomical location but also by morphological (histological) criteria and immunophenotype. In developing a deeper understanding of the morphological classification and immunophenotyping of LSA the VN can be of more support to owners in discussing appropriate protocols for patients and will have a deeper understanding of the patient's condition. Histological or morphological grading is carried out by a pathologist and describes the microscopic appearance and level of infiltration of the tumour cells. In this way LSA can be classified as low-grade, intermediate-grade, and high-grade. Immunophenotyping is carried out by an external laboratory and classifies LSA as either B cell positive or T cell positive. B cell positive tumours are more widespread than T cell positive tumours which carry a poorer prognosis in the canine population. It has been argued that in comparison with canines, immunophenotyping in the feline population should not be considered prognostic. The prognosis for cats with feline LSA is dependent on several factors, which include histological grading (discussed above), and the stage of the disease.

Presentation and diagnosis

The patient was referred to the oncology department having being diagnosed with follicular lymphoma following biopsy of the left popliteal lymph node under general anaesthetic, at the referring practice. The histopathologist also reported that there was evidence of invasion into the surrounding tissues, that the tumour was advanced and it was likely that other peripheral lymph nodes were affected. Haematology had highlighted a severe lymphocytosis. On admission to the oncology department the patient had no abnormalities detected on physical examination, with the exception of the enlarged lymph nodes and dry skin on the foot pads and ears. The dry skin was most likely due to hormonal stimulation by the lymphoma which is called paraneoplastic syndrome. Her weight was stable and at 5.5kg appropriate for her size.

Staging

Staging describes the number and location of tumour cells and the presence or absence of systemic disease. Staging of cancer is carried out by the VS and is often confused with grading of tumours which is carried out by a pathologist. The VN needs to have an understanding of the correct terminology, which is crucial if they are to be involved in

discussing case work ups with clients and assisting the VS appropriately. Staging looks at the degree of the cancer by considering the location of the primary tumour, the size of the tumour, the number of tumours, and whether or not it has metastasised into the lymph nodes or other organs. The World Health Organisation (WHO) classification system has been modified from the human field and is widely utilised in the staging of veterinary cancer patients.

Staging is an important clinical process that allows us to determine the extent to which the disease has spread, and which will often dictate the treatment regime chosen. The general health of patients can be determined and secondary disease can be identified before chemotherapy treatment begins. Staging promotes effective treatment and minimises toxicity. Intra and post treatment restaging is often carried out to evaluate response to treatment and it is also utilised if there is a relapse or return of the disease prior to starting a rescue protocol. Staging tests include the following: physical examinations, complete blood counts, serum biochemistry panels, urine evaluation, lymph node evaluation, radiography, ultrasonograpy, computed tomography, magnetic resonance imaging and bone scintigraphy. Additional relevant information can be achieved with cerebrospinal fluid analysis and bone marrow biopsies.

The patient was staged prior to chemotherapy treatment, tests and results are listed below:

- Chest and nasal cavity radiographs - no sign of tumour spread
- Abdominal ultrasonography - marked enlargement of medial iliac lymph nodes and mild enlargement of cranial periaortic lymph nodes. Both liver and spleen appeared mottled with hyperechoic hepatic echogenicity: fine needle aspirates of liver and spleen were taken.
- Urinalysis - cystocentesis could not be performed as bladder only mildly full
- Biochemistry and haematology (performed at referring practice) – Blood smear in -house, adequate neutrophil count, further results pending
- Hepatic and splenic cytology, further staining requested on previous tissue samples to define tumour grade – No significant pathology detected on either splenic or hepatic samples. Tumour grade – Low grade follicular lymphoma.

Treatment protocol

Chemotherapy is the treatment of choice for patients with feline LSA and several protocols have been described and evaluated. The most common treatment protocols are either COP

or CHOP based. COP based protocols include the following drugs, cyclophosphamide, vincristine and prednisolone. In addition to these the CHOP based protocol incorporates the use of doxorubicin. With the exception of prednisolone these are classed as cytotoxic drugs. Studies have reported response rates of cats on COP-based protocols of 33 to 74.5% and for those on CHOP-based protocols response rates of 38 to 80%. By developing an understanding of the most common chemotherapy protocols that are used in the treatment of feline LSA the VN will gain knowledge of the specific drugs which are used and their potential side effects. This will enable the VN to monitor patients effectively post chemotherapy and provide them with the appropriate level of nursing care.

The patient was treated with a high dose COP protocol over a period of 5 months. The patient received intravenous cyclophosphamide and solu-medrone intramuscular as dosing with oral medications was unachievable. Sedation was necessary with medetomidine and butorphanol for intravenous treatments. Antiemetics were administered subcutaneously pre-treatment with frusemide given intravenously when cyclophosphamide was given. Haematology was checked 7 days after each chemotherapy treatment.

In-house protocol for COP

Cyclophosphamide

- Routes – IV and PO
- Premedication – frusemide, cerenia
- Duration – slow infusion over 15 minutes.
- Toxicities – neutropaenia and GI toxicity
- Special precautions – Sterile haemorrhagic cystitis can occur rarely. Irritant if extravasates.

Vincristine

- Routes – IV
- Premedication – cerenia
- Duration – bolus
- Toxicities – neutropaenia and GI toxicity (constipation in cats).

- Special precautions – Moderate tissue damage can occur if administered outside the vein.

The patient relapsed after receiving the COP protocol for 5 months presenting with lymphadenopathy and lymphocytosis. A rescue protocol using methotrexate was then utilised which was administered intramuscularly for 7 cycles with L-asparginase administered intermittently for 3 doses. The patient remained well on this protocol for a further 4 months before relapsing again. Chemosensitivity testing was utilised at this point, which facilitates the tumours response to specific chemotherapy agents. Several drugs were highlighted and the oncologist felt that mitoxantrone was the agent of choice from the list given. Mitoxantrone was given intravenously every 3 weeks and the patient was on their 6th cycle at the time of print and doing very well. When deviating from standard protocols advice should be sought from a board certified oncologist.

Feline Mammary Carcinoma

Tumours of the mammary glands are less common in feline patients than in canines or humans; however 85% of these tumours are malignant with adenocarcinomas being the most common type seen. Other types of benign and malignant tumours are rare. Typical presentation includes feline patients of ten to twelve years of age with both neutered or entire females being at risk. Studies have shown however that neutered patients are at a decreased risk. Particular breeds, such as the Siamese are also at an increased risk of developing the disease. Tumours can remain unnoticed until they are relatively large or have become ulcerated and the disease is frequently very advanced before treatment is sought. Multiple glands may be affected however single masses in one gland can also be seen. Feline mammary carcinoma is an invasive and rapidly spreading tumour and metastatic disease may be present in the draining lymph nodes and/or lungs.

Presentation and diagnosis

The patient was presented to the oncology department to discuss prognosis and treatment for a mammary squamous cell carcinoma diagnosed based on histopathology following mastectomy of the 4th left mammary gland. The owner reported that the patient was well in herself with no general signs of illness. Physical examination showed a markedly overweight cat (5.9kg BCS 8/9). The surgical mastectomy wound was completely healed and covered with normal skin however there was a deep subcutaneous nodular thickening present along the entire length of the wound. General physical examination was otherwise unremarkable.

Staging

The patient was staged prior to mastectomy, tests and results are listed below:

- Physical examination - unremarkable
- Biochemistry panel - no abnormalities detected
- Complete blood count - eosinophilia (differential diagnosis includes parasitism, hypersensitivity, paraneoplastic syndrome)
- Faecal analysis - no parasites or ova detected
- Urinalysis - no abnormalities detected
- Thoracic radiographs 3 views - no evidence of tumour spread
- Abdominal ultrasonography - no evidence of tumour spread in abdominal organs

Treatment and Outcome

Aggressive treatment of feline mammary carcinomas is recommended with surgical intervention in conjunction with chemotherapy due to the high metastatic potential of these types of tumours. Doxorubicin and carboplatin are the adjuvant chemotherapy drugs of choice. Radiation therapy can be used to reduce discomfort when the disease is end stage and palliative therapy is utilised.

The patient was admitted to the surgical department of the hospital and the remaining left mammary glands were excised en bloc with the scar and associated nodules present at the site of excision of the left 4th mammary gland. In addition the inguinal fat was excised and presumed to contain the inguinal lymph node. No grossly abnormal tissue was identified during the excision and there were no intraoperative complications encountered, the first chemotherapy treatment, carboplatin, was administered. A staged approach to complete mastectomy was undertaken as the surgeon was concerned that they would encounter significant wound tension had both the right and left mammary chains been resected at the same time. The patient was discharged on anti-inflammatories, antibiotics, opioids and anti-emetics. An appointment was booked at the referring practice for a wound check and complete blood count post chemotherapy. Histopathology results were received a few days post discharge with the following histological diagnosis, comedocarcinoma with lymph node metastasis, a form of mammary carcinoma in which plugs of necrotic malignant cells may be expressed from the ducts.

A second surgery to remove the right chain of mammary glands was performed three weeks after the first surgery and a second chemotherapy treatment (carboplatin) was administered.

Restaging was performed with no changes detected. Histology results from this surgery reported benign hyperplastic changes within the submitted mammary tissue.

Carboplatin

The patient received 6 doses of carboplatin intravenously at 3 weekly intervals under sedation. An anti-emetic was given pre-treatment and metacam was given daily at home by the owners. The patient's complete blood count was checked seven days after each treatment for neutropaenia and again every third week pre treatment to check for a delayed neutropaenia.

In-house protocol for Carboplatin

- Routes - intravenous
- Storage - refrigerate
- Premedications - Cerenia (maropitant)
- Duration - slow infusion over 30 minutes
- Toxicities - delayed neutropaenia and gastrointestinal toxicity (mainly inappetance/mild) reduced GFR.
- Special precautions - care in patients with reduced kidney function. Hypersensitivity after repeated injections. The patient was restaged 6 months post treatment and continues to be in remission.

Specific Nursing Considerations

As well as the nursing considerations already discussed there are specific aspects of care which can make the patients and clients treatment a more comfortable and stress free experience. Examples of these that I have found help are listed below:

- Try to make appointments that allow the patient's stay in the hospital to be as short as possible.
- Try to create as stress free an environment as possible for both long and short stay patients, for example, feline patients can be given cardboard boxes to hide in and canine patients offered treats post therapy.
- The VN can assist clients by preparing educational handouts of common side effects and include information on what situations can be dealt with at home and those which the client should be concerned about.

- Develop an understanding of the impact the diagnosis of cancer has on the patient's owner and extended family and be prepared to help owners through the grief and loss period. Always make sure that you have the time to spend with clients, which can be difficult in a busy veterinary environment, but crucial to support clients.
- Maintain a professional relationship with your clients at all times.
- Remember to look after yourself, VN working in the field of cancer will be faced with many ethical dilemmas and provide a huge support mechanism for both the patient, pet owners and colleagues. It is vital that the nurse does not neglect their own emotional needs and seeks help when necessary for example via professional counselling.

NURSING THE POISONED PATIENT

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Poisoned animals are frequently seen in practice. The patient may come into contact with the poison accidentally, or the poison may be administered to the animal maliciously. In many cases, the owners may be the cause of the problem, either by administering human medication to their pet, or by accidentally overdosing with a prescribed veterinary product. Due to the wide variety of toxins and their very varied effects on animals, recognising an intoxicated patient can be difficult; many intoxications give similar symptoms to other diseases.

If the owner has witnessed their animal ingesting a toxin, it is important they find any associated packaging. Get the owners to read out over the phone any printed information available such as the active ingredients and the concentration of the active ingredients. Try and get an estimate of the amount ingested or find out how much is missing from the packaging. This information, along with an estimate of the size of the animal, means you can start to calculate the likely dose the animal has consumed while the owner is on their way to the surgery. Finding out this information allows the clinical team to prepare for the patient's arrival, and have suitable equipment and medication to hand.

In many cases, patients may be presented to the veterinary hospital suffering from illness with no witnessed exposure to toxicants. Ultimately these patients must be treated symptomatically, with supportive care aimed at treating the affected systems and preventing further disease process. Even for known intoxications, an antidote is often not available, and the same goals exist: treat symptoms and patient, and facilitate elimination of the toxicant wherever possible.

Treatment

Treatment of the poisoned patients should concentrate on the following areas

1) Emergency stabilisation and supportive care
2) Reducing further absorption of the toxin
3) Using a specific antidote, if one exists.
4) Increasing elimination of the toxin
5) Treating effects of the toxin

Supportive care

What supportive care is required will depend on the condition of the patient at presentation. As with all emergency cases, assess airway, breathing and circulation (ABC) initially and address any problems that are highlighted. Common supportive care includes oxygen supplementation, intravenous fluids, and controlling body temperature - either active warming or cooling of the patient. Some patients will need to be sedated, or have anti-convulsants administered.

Reducing Absorption

If the toxin has been absorbed through the skin, then clipping off the hair in that area, and washing the skin with mild soap or detergent will decrease absorption.

If the toxin has been ingested, then vomiting can be induced. Emesis should only be induced after considering a number of factors. Emesis is usually only effective if the substance has been consumed within the past 90 minutes, (though for some slowly digested toxins, emesis may still be effective after 2 or 3 hours). Emesis is contraindicated if the substance is caustic, or if aspiration of vomit is likely - such as in an animal with reduced levels of consciousness, an animal that is seizuring or is dyspnoeic.

Apomorphine is commonly used as an emetic in the dog, it is reliable and effective. Vomiting is usually seen within 5-10 minutes following subcutaneous injection. Apomorphine is contraindicated in cats, so xylazine injection is often used.

If emesis is ineffective, or contraindicated, then gastric lavage can be used as a means of gastric evacuation. Gastric lavage must be performed under general anaesthesia, with a cuffed endotracheal tube in place. A stomach tube is premeasured and marked, and then inserted into the stomach. Warm water, at a dose of 10ml per kg bodyweight, is then introduced into the stomach, and siphoned out again. The process is repeated until the water runs clear and no more stomach contents are removed.

Adsorbants such as activated charcoal are useful to reduce further absorption from the gut. They bind the toxin in the gut so it cannot be absorbed, and so passes through the body. Activated charcoal made from vegetable matter is considered the most effective, and is available as powder, tablets, granules or ready mixed as a suspension. Adsorbants orally after emesis or via a stomach tube after gastric lavage. Administering activated charcoal with dog food does have some reduction in its ability to absorb toxins, but the reduction in efficacy is unlikely to be clinically significant.

Antidotes

Some commonly encountered toxins have specific antidotes; these can be used as soon as they are available. It may not be practical to stock all antidotes, but it is sensible to know which antidotes exist.

Antidote	**Toxin**
Acetylcysteine ('Parvolex')	Paracetamol
Atropine	Organophosphates
Calcitonin	Vitamin D or calciferol
Antivenom	Snake bites
Ethanol **4-Methyl pyrazole (dogs only)**	Ethylene Glycol
Vitamin K1	Anticoagulant rodenticides
Methylene blue	Paracetamol, nitrates and chlorolates
Desferroxamine	Iron
Naloxone	Opiods
Penicillamine	Heavy metals

Recently attention has been directed to the use of intravenous lipid emulsions (IVLE) as a tool in managing intoxications with lipophilic drugs, known as "lipid rescue". Patients affected by toxins such as local anaesthetics (lidocaine, bupivicaine), permethrin and avermectin parasiticides (ivermectin, moxidectin etc) are potentially suitable for therapy with IVLE. The mode of action is uncertain at present, one possibility is that the lipid acts as a 'sink' for the lipophilic drugs, so keeping them away from their target receptors and preventing their effects. In cardiotoxic drugs the lipid may provide an energy source to the myocardium to increase performance. An initial bolus is given intravenously, followed by an infusion over 1-2 hours. Potential complications include the return of toxic signs as the lipid is metabolised and the toxin 'freed' again.

Increase Elimination

By encouraging the body to eliminate a toxin more quickly, we can reduce the risk of continued absorption. Laxatives can be given in addition to adsorbents. This speeds up gut transit times. Magnesium sulphate and sodium citrate are examples.

If the toxin or its metabolites are mainly excreted via the kidneys, then intravenous fluids and diuresis will increase elimination. Creating more alkaline urine with sodium bicarbonate can help with the excretion of weak acids such as ethylene glycol, via 'ion trapping'.

Peritoneal dialysis is indicated in some cases to aid elimination while also helping to manage consequences of toxicity such as acute renal failure.

Treat known likely effects of the toxin

In cases where the toxin is known, treatment can also be targeted to try and prevent the likely effects. For example, if the toxin is likely to cause gastric ulceration, administer drugs to reduce stomach acid production, and medication to speed the healing of any ulcers that may have already formed.

Ethylene Glycol

Ethylene glycol itself is not toxic, but it is rapidly metabolised after absorption from the gastro-intestinal tract; it is these metabolites that are harmful. Alcohol dehydrogenase is the enzyme in the liver that metabolises ethylene glycol into gycoaldehyde, which in turn forms glycolic acid. Glycolic acid leads to acidosis, and forms oxalate which causes renal damage. Cases are sometimes seen where ethylene glycol is maliciously mixed with food and left out for cats.

Clinical signs are often seen in 3 overlapping phases:

Phase 1 occurs 1 to 4 hours after ingestion, and ataxia, depression, vomiting, polyuria and polydypsia are seen.

Phase 2 occurs 4 to 6 hours after ingestion and coincides with the onset of metabolic acidosis caused by metabolites of ethylene glycol. Cardiopulmonary signs such as tachypnoea, tachycardia and pulmonary oedema can be seen. If large doses have been taken, signs may proceed to coma and death.

If the animal survives phase 2, they may go on to develop renal failure in Phase 3, 24 to 72 hours later, due to metabolites and reduced blood flow due to renal oedema.

Unfortunately many animals do not present until later on in the process, when the prognosis much less favourable. Diagnosis can be difficult as the symptoms can mimic other multi-system disease processes such as acute renal failure, gastroenteritis and pancreatitis. The

animal will often be azotaemic, but with a low blood calcium level, as oxalate binds to calcium to form calcium oxalate, so lowering blood calcium levels.

Treatment of early ethylene glycol toxicity cases relies on preventing toxic metabolites being formed, and speeding elimination of the unchanged ethylene glycol from the body. In cases where metabolism of the toxin is already advanced, treatment concentrates on managing acute renal failure, and addressing acidosis and hyperosmolarity issues.

In cases where ingestion has just been witnessed, gastric decontamination should be carried out and administration of an antidote initiated. Antidotes act as a preferred substrate for alcohol dehydrogenase, preventing metabolism of ethylene glycol and allowing it to be excreted unchanged.

Ethanol can be administered to symptomatic cases within 24 hours of ingestion, though it is most effective if given within a few hours, and the sooner the better. Azotaemic animals have already metabolised ethylene glycol so ethanol will have no effect. 4-Methylpyrazole (4-MP) also inhibits alcohol dehydrogenase, and is used in humans as an antidote. 4-MP has been used successfully in dogs; though it's poor availability and high cost mean use is not widespread.

If the animal presents with azotaemia, then ingestion was at least 24 hours ago, and emesis will be of no use. Fluid therapy will help increase the excretion of ethylene glycol and its metabolites. Fluid diuresis and diuretics are usually required to combat oliguria. Peritoneal dialysis is useful in helping to reduce blood levels of ethylene glycol, as well as urea, creatinine and potassium.

Grapes, Raisins, & Sultanas

Both fresh and dried grapes (Vitis vinifera) have been recognised as a cause of toxicity in dogs. The exact mechanism by which they have their effect is not known, but both red and white grapes, sultanas, raisins and currants have all been identified as causing acute renal failure in dogs. Possible causes suggested are tannins, mycotoxins, ocratoxin, polyphenolics or excessive vitamin D intake. As the mechanism of toxicity is poorly understood, no safe limits of ingestion are established, though it appears the effect on dogs is idiosyncratic, with reaction differing widely between individuals.

Early signs after ingestion are usually diarrhoea, lethargy and anorexia. 24 to 72 hours later, acute renal failure may develop.

Digestion of grapes or raisins is slow in dogs, so gastric decontamination should be carried out, followed by administering activated charcoal. To try and prevent renal failure, aggressive intravenous fluid therapy should be administered for at least 48 hours, whilst monitoring renal function.

Xylitol

Xylitol is a 5-carbon sugar alcohol, and is used as an artificial sweetener. It is commonly found in 'sugar-free' chewing gum and sweets, in tablets and medications, or as a sugar substitute in baking.

Xylitol increases insulin secretion in dogs, leading to a 2.5 to 7 fold increase in production. This seen commonly 30 -60 minutes after ingestion, though the effect can be delayed. The increase in insulin leads to hypoglycaemia, which becomes evident as vomiting, ataxia, tachycardia, seizures and coma. Ingestion of larger amounts can lead to acute liver damage. Treatment is by gastric decontamination, followed by blood glucose monitoring and dextrose supplemented intravenous fluids if required. Where larger amounts are ingested, liver protectants (e.g. S-adenosyl-L-methionine) are indicated, and cases that develop coagulopathy will require fresh frozen plasma.

Tremorgenic Mycotoxins

Mycotoxins are produced by fungal metabolism. Penitrem A. and Roquefortine are the two most commonly encountered tremorgenic mycotoxins produced by the fungi Penicillium, Aspergillis and Claviceps. Common sources include mouldy food, compost, silage, and cheese. Dogs are most commonly affected due to their less discriminate tastes when scavenging.

Mycotoxins are absorbed very rapidly, and clinical signs are usually seen within thirty minutes. They are lipophillic and cross the brain-blood barrier. Clinical signs are due to effects on neurotransmitter release in the CNS and peripheral nerves.

Early sings include vomiting, hyperaesthesia, muscle tremors, and rigidity. Signs may progress to include severe tremors, opisthotonos, seizures, nystagmus, and recumbency with paddling.

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DIABETES MELLITUS IN CATS

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Incidence and pathogenesis:

Diabetes mellitus (DM) is the second most common endocrinopathy in cats (following hyperthyroidism). It is caused by a multifactorial group of disorders that result in an absolute or relative lack of insulin. Its incidence in cats is ~1 in 200 veterinary patients in the UK (PetProtect), and is increasing, probably resulting from an increased incidence of obesity, reduced daily exercise (particularly in house cats) and an increased percentage of older cats. While it can occur in any age, sex or breed of cat, it is seen most frequently in older obese neutered male cats that take little exercise; with a peak incidence between 10 and 13 years of age. That said, recent data (PetProtect) shows that 50% of affected cats are of ≤ 7 years old, with the other 50% being ≥ 8 years of age. In Great Britain, Australia and New Zealand Burmese cats are predisposed to developing DM: with ~1 in 50 developing DM and the prevalence in some families reaching 1 in 10.

Classification of DM in cats (as with humans) is made by cause, not by whether or not they require exogenous insulin:

Type 1 – is to an **immune-mediated attack** on pancreatic islet beta cells. This appears to be very rare in cats.

Type 2 – has a complex multifactorial aetiology involving a combination of **peripheral resistance to the action of insulin, impaired insulin secretion**, environmental factors (e.g. diet, lack of exercise, urban living), islet amyloid deposition, genetic predisposition and chronic pancreatitis: 80-95% of cats with DM are type 2 (although many will also have concurrent complications as seen in secondary DM – see below). Diabetic cats are ~6 times less sensitive to insulin than normal cats, and male cats have lower insulin sensitivities and higher insulin concentrations than female cats. In cats, type 2 DM is often associated with the **accumulation of islet-specific amyloid polypeptide (IAPP)** which occurs in aggregates around pancreatic islets. While IAPP is co-secreted with insulin and accumulates

in normal cats as they age, it accumulates more extensively in cats with DM. The accumulation of IAPP acts as a barrier against insulin diffusion and antagonizes insulin action. **Obesity** is a significant risk factor for type 2 DM because it causes a reversible peripheral insulin resistance. **Genetics** also plays a role, particularly in Burmese cats. While cats with type 2 DM cats may initially be non-insulin dependent, progressive loss of islet cell function usually results in a need for exogenous insulin.

Secondary DM – can result from a number of different causes of **insulin antagonism**:

- Pancreatic pathology e.g. due to pancreatitis or pancreatic adenocarcinoma. **Pancreatitis** may be more common than previously thought as >50% of diabetic cats have evidence of past or current pancreatitis at necropsy. However, this does not mean that 50% of feline DM is caused by pancreatitis. Rather, it means that while pancreatitis can cause DM, unstable DM can also cause pancreatitis (due to the toxic effect of chronic hyperglycaemia). It can be very difficult to determine which came first. In cats, pancreatitis often occurs concurrently with inflammation of the liver and intestines, so called Triaditis. Where this occurs the presence of these other conditions can further complicate the clinical presentation, diagnosis and treatment of the DM. Chronic pancreatitis can eventually lead to significant loss of pancreatic function which can result in the development of DM and/or exocrine pancreatic insufficient (EPI). Pancreatic adenocarcinoma can also lead to progressive pancreatic dysfunction, and in one study was found in 19% of feline diabetics presented to referral institutions.
- Infection, most typically of the mouth (gingivostomatitis and/or periodontal disease), urinary tract, or skin.
- Underlying or concurrent disease e.g. **acromegaly, hyperadrenocortisim** and even **hyperthyroidism.**
- **Drug administration** e.g. corticosteroids, progestogens (megestrol acetate).

Diabetic remission ('transient DM'):

In 50-60% of cats with DM (up to 90% in some newer studies) the requirement for exogenous insulin may only be temporary. Cats with DM may lose the need for exogenous insulin, most typically within 1-4 months of first becoming diabetic. This **transient DM** may result from:

- Correction of **'glucose toxicity'** - prolonged hyperglycaemia causes impaired insulin secretion by islet beta cells and increased peripheral resistance. Exogenous insulin administration and the resulting reduction of hyperglycaemia can result in resolution

of this toxicity, at least initially.

- Reduction of obesity.
- Resolution of pancreatitis.
- Treatment of concurrent or underlying disease.
- Removal of diabetogenic drugs.

Remission is most likely to occur when newly diagnosed diabetics are treated aggressively with exogenous insulin and fed a high protein/low carbohydrate diet. The duration of remission can be variable, with an average of 18 months. Unfortunately, it is rare for relapsed cats to gain a second remission.

Clinical signs:

The history and clinical signs of DM in cats can be very subtle, and affected cats are often not presented for investigation until they become systemically ill. **The most consistent signs are polyuria, polydipsia, and polyphagia**. Because of the polyphagia some owners report their cats had **initial weight gain, followed by weight loss**. However, these early signs often go unnoticed by the owners, possibly because of free choice feeding and outdoor lifestyles. When urinary tract infections are present affected cats may present with signs of cystitis and/or renal failure. Unless ketoacidosis or concurrent disease is present, physical examination is usually largely unremarkable in the uncomplicated diabetic cat. The coat of many diabetics becomes ill kept and a pot-bellied appearance may result from hepatomegaly. Hind limb weakness and a plantigrade stance (due to diabetic neuropathy) are seen quite frequently, but cataracts occur rarely. Cats are frequently presented only when they become systemically ill with signs of anorexia, vomiting and/or diarrhoea, jaundice and depression. Cases of DM that result from chronic pancreatitis may have a history that includes episodes of depression, anorexia, vomiting, diarrhoea, and/or abdominal pain. In addition, since DM can arise secondary to chronic pancreatitis, and is seen once most of the pancreatic mass has been destroyed, it may also be accompanied by signs of EPI (i.e. a voracious appetite and large quantities of voluminous fatty faeces). Heart failure is common in diabetic cats, so the cat should be assessed for clinical signs consistent with congestive heart failure and/or cardiogenic shock. Interestingly, while ascites due to heart failure is generally uncommon in cats, it has been seen more frequently when heart disease occurs concurrently with DM, and may possibly relate to underlying acromegaly.

Diagnosis:

- **Diagnosis is based on documenting persistent fasting hyperglycaemia** (> 11

mmol/l) **and glucosuria in a cat with appropriate clinical signs (polyuria, polydipsia and polyphagia).** However, since stress-induced hyperglycaemia (often up to ~20 mmol/l, and occasionally higher) can result in glucose levels above the renal threshold (12-16 mmol/l) it can readily result in glucosuria. Because of this a single documentation of these findings is not diagnostic of DM. Allowing the cat to settle down and then re-testing it after a few hours may help to determine whether or not the hyperglycaemia is stress-induced. Alternately, the owner can be asked to test the cat's urine for the presence of glucose when it is at home. Interestingly, cats do not have to appear externally stressed for this to be an issue, in fact often the most stressed cats will be ones that do not struggle or vocalise. Interestingly, some authors are now talking about a pre-diabetic state in cats, diagnosing this at 9.7mmol/l for most cats, and 10.2mmol/l for Burmese cats.

- Assessing **serum fructosamine** concentrations can be useful as they give an indication of how raised the blood glucose levels have been during the preceding 1-2 weeks (which is a shorter period than in humans or dogs). However, some care is required as they can be raised where there has been prolonged stress hyperglycaemia; e.g. when a cat has been hospitalised. That said, prolonged stress-induced hyperglycaemia may occasionally require insulin therapy to return the cat to a euglycaemic state during or following resolution of the primary condition. Cats with acute onset DM, which is often associated with acute pancreatitis, may not have been diabetic long enough for the fructosamine level to rise. In addition, conditions that result in hypoalbuminaemia can give falsely reduced fructosamine levels, as can hyperthyroidism (because it incurs increased protein turnover). The combination of concurrent DM and hyperthyroidism can be particularly difficult to diagnose as the DM causes the thyroxin to be lower than expected and the hyperthyroidism causes the fructosamine to be lower than expected. Unfortunately, false positive and negative fructosamine results can occasionally be seen for no apparent reason.
- **Ketones** are found in the plasma and urine of cats with diabetic ketoacidosis (DKA). Thankfully, this condition is rarer in cats than dogs: 12-37% of cats are ketotic at the time of diagnosis, usually associated with concomitant disease, especially infection. This can be diagnosed using urine dip-sticks; and using plasma is more sensitive than using urine.
- **Other findings on clinical pathology** result from concurrent and complicating disease. Many cats with DM have mild to moderate increases in serum concentrations of cholesterol and liver enzymes. More severe changes, bilirubinaemia, acidaemia, uraemia, and electrolyte disorders usually indicate the

presence of complicated DM or DKA.

- It is important to look for urinary tract infections (UTIs); these are found in ~ 12% of cats with DM. Some of these will have a non-active urinary sediment (the lack of pyuria is believed to result from hyperglycaemia reducing neutrophil function) so it is essential to collect a sample by cystocentesis and **perform urine culture and sensitivity (C&S),** regardless of the sediment findings.
- **A thorough initial investigation** is always recommended in order to detect any concurrent disease(s) and so aid swift diabetic stabilisation. Ideally, this should involve performing a detailed physical examination (including body weight, body condition score, assessment of periodontal disease, and assessment of systemic blood pressure), assessing full haematology and serum biochemistry (including serum fructosamine and thyroxin levels), urine analysis (including C&S) and, where indicated, survey imaging and/or feline pancreatic lipase immunoreactivity (fPLI).

Treatment:

The treatment goals for feline DM have changed in recent years with the realisation of how common 'transient' DM is in this species. Whereas previously the goal of treatment was simply to control the clinical signs of the disease, the goals are now:

- Early detection
- Aggressive early treatment
- Aiming for the DM to be transient with only a temporary need for exogenous insulin

Treatment consists of various combinations of weight loss, increasing exercise, dietary modification, insulin administration, and/or oral hypoglycaemic agents.

- Initiating insulin treatment and dietary management as soon as possible after diagnosis will give the best chance of diabetic remission.
- Obese cats will benefit greatly from weight reduction.
- Increasing exercise, e.g. 10 minutes of daily play, can produce as much weight loss as calorie restriction. The amount of play should be the same each day.
- Oral hypoglycaemic agents may be successfully used in some uncomplicated diabetics once glucose toxicity has resolved following insulin therapy.
- To ensure long term compliance it is essential **to manage client expectations**. Owners should be informed that it is likely to take 2-3 months to fully stabilise their cat, and that insulin requirements may reduce after 3-4 months if the cat is treated appropriately.

Tips for optimising client compliance:

1. Provide enough information about the causes, diagnosis, treatment and prognosis of DM in cats at the initial consultation.
2. Explain expectations from the outset in terms of expected time for stabilisation, possible complications, possibility of fluctuating insulin requirements, need for home monitoring, etc.
3. Explain that the insulin requirements may be transient if the treatment is prompt and aggressive.
4. Allow clients enough time to discuss their concerns and ensure they understand the disease and what is involved. Many owners are daunted by the prospect of handling insulin and injecting their cat, so they need support until they are completely familiar with these procedures. Making use of nursing staff to provide client support is very helpful.
5. Be flexible with instructions regarding timing of feeding and insulin administration and be realistic in what the client can achieve – for example missing an insulin injection one day a week is better than not treating at all.

1. Dietary modification –

Cat with DM have difficulty assimilating the carbohydrates present in most commercial cat foods. **Recent studies have shown that diabetic cats have better glycaemic control and are more likely to be able to discontinue exogenous insulin when they are fed a diet that is high in protein and low in carbohydrate**. This is in contrast to the previously recommended high fibre diets. High fibre diets can help slow glucose absorption, but low carbohydrate, high protein diets appears to provide superior control. Where any carbohydrates are present in the diet they should be in the form of complex carbohydrates not simple sugars. *Most dry foods contain considerable quantities of carbohydrate,* whereas many canned foods contain relatively less. However, each food is different, so use caution in selecting the appropriate diet: always read the label.

The profile of the recommended diet may vary with the body condition of the cat (see below) and, in some cases, may be affected by concurrent illness. e.g. if chronic kidney disease is also present, a diet with lower protein and phosphorus content may need to be considered.

Non-obese diabetic cats: For the reasons described above these cats should be fed a diet that is **high in protein and low in carbohydrate**. See Table 1 (page 300) for profiles of commercial diets for diabetic cats and compare their protein and carbohydrate values. Good recommendations are *Nestlé Purina DM* and *Hills m/d.*

Obese diabetic cats will benefit from weight loss. However, this should be achieved very gradually, restricting calorie intake to no more than 75% of maintenance requirements and monitoring for changes in insulin requirement. Many obese diabetic cats respond well to feeding slightly lower quantities of **high protein and low carbohydrate diet**. However, where cats are severely overweight they may need to be fed a diet with restricted calories rather than simply decreasing the amount of the regular diet. This is because decreasing the amount of food fed also decreases the cat's intake of protein and vitamins. While this may only result in begging and stealing food it can, in extreme cases, result in significant deficiencies. A weight loss diet for a diabetic cat should still have a high amount of protein, which helps to prevent hepatic lipidosis, and it may help if it is supplemented with carnitine. In order to induce weight loss some of these diets contain an **increased amount of fibre** and are moderately fat restricted. Fibre provides a satiety factor, helping cats feel full with less calorie intake, as well as helping slow glucose absorption. While this works well in some cats, others find the high levels of fibre poorly palatable and many of these diets contain excessive amounts of carbohydrate. The increase in dietary fibre can also lead to management considerations in cats that use litter boxes as it increases the quantity of faeces produced. (Table 1 also contains profiles of feline diets designed to incur weight loss).

Diabetic cats benefit from being fed a well-balanced diet on a regular feeding schedule. Cats on once daily insulin are usually fed just before their morning insulin injection, then again in the early evening. Cats on twice daily insulin are usually fed just before both insulin injections. However, additional free-choice feeding is beneficial and suits many diabetic cats. In all cases, it is important to monitor the amount of food eaten on a daily basis.

Although not always practical, the ideal diet for most uncomplicated feline diabetics would therefore be high in protein and low in carbohydrate, it would be a wet/canned diet and it would be freely available all day, or at least given in multiple small meals.

2. Insulin –

There are a number of different types of insulin, and the choice is often based on personal preference. **Most cats with uncomplicated DM respond well to twice daily subcutaneous administration of Caninsulin™, lente (veterinary product recently discontinued in the UK), protamine zinc insulin (PZI) (veterinary product recently discontinued in the UK) or glargine insulin.** Typical actions are shown below but it is important to recognise that cats can respond very variably to exogenous insulin:

- **Lente –** peak 2-10h, duration of action 6-16h; ~all cats need twice daily injections.

- Caninsulin™ has a duration of action similar to lente, but should be started at a lower starting dose as it is more potent.
- **PZI** – peak effect at 3-12 h, duration of action 6-24h; most cats need twice daily injections, but 20-30% may cope with once daily.
- **Glargine** (Lantus™) – peak ~14 h, duration of action ~24h. Giving cats twice daily injections of glargine and feeding them on high protein/very low carbohydrate diets significantly increases the chance of gaining diabetic remission and therefore removing the need for long-term exogenous insulin administration. Although this is now the formulation of choice in many countries, particularly when aiming for rapid resolution of the DM, this is not licensed for use in cats in the UK so it can only be used after lente and/or PZI have failed. Detemir (Levemir™) is similar to glargine, and again not licensed for use in cats in the UK.
- The source of the insulin does not appear to matter too much in cats as anti-insulin antibodies do not cause many problems.
- ***Cats can be very unpredictable in their response to insulin administration and no one type of insulin or dosing regime will be suitable for all cats.***
- Twice daily dosing of an intermediate acting insulin (e.g. lenti or Caninsulin) is a good first choice. However, in some cats these insulins do not have a long enough duration of effect, so PZI or glargine are required. Some cats cannot absorb PZI very effectively: in these cats glargine may be necessary.
- Do not become overly concerned if an owner can not adhere to a strict dosing regime; it is better in 1 week to have 5 days of twice daily dosing and 2 days of once daily doing that for a cat to be euthanased because the owner is worried about being unable to inject twice daily every day.

Initiating insulin treatment:

- When starting treatment it is usually best to start at a low dose of insulin **(~0.25 IU insulin/kg/per injection [to a maximum of 3 IU/cat])**. As the dose may be small consider using 0.3ml syringes to assist in accurate dosing and/or using a less concentrated insulin (e.g. Caninsulin 40 iu/ml). Care must be taken to use the correct combination of insulin and syringe. Do not dilute insulin in order to achieve accurate dosing - this often damages the insulin and results in unpredictable results.
- The aim of therapy is to prevent the clinical signs of DM and, if possible, maintain blood glucose concentration between 5-14 mmol/l.
- On day 1 of insulin treatment check the blood glucose at the estimated nadir (i.e. the time of peak action of insulin, which is the lowest point of the curve e.g. 4-8 hours

after administration with lenti insulin) to ensure hypoglycaemia has not occurred. As long as blood glucose is ≥10 mmol/l continue on the same dose.

- Discharge the cat for 1 week with instructions for the owner on monitoring for signs of hypoglycaemia (see later for clinical signs). However, provided hypoglycaemia does not occur there should be no changes to the dose of insulin during this time. This is because it **takes 3-5 days for glucose homeostasis to adjust after starting or altering insulin doses.** During this time the owners may also be asked to monitor urine ketones at home (whether or not this is considered necessary depends on the individual cat, whether or not it has been ketotic before and whether or not it is systemically ill). The owners need to know to contact the veterinary practice if ketones become positive.
- To help reduce the risk of reduced absorption of insulin, owners should be trained to inject the insulin into a slightly different area of the cat's back each day.
- After a week the cat can be hospitalized for a 12-24 hour, every 2-4 hourly **blood glucose curve (BGC)**. If there is a significant response to insulin at this stage then around the expected nadir (e.g. 4-8 hours after injection) the testing interval should ideally be 1-2 hourly so the nadir can be accurately identified. However, before and after the nadir, 4 hourly is acceptable at this stage: if there is not a significant response to insulin then there is little value in testing more frequently than 4 hourly at this stage.
- At this stage the aim of a BGC is to see if there is any response to that dose of insulin and to ensure there are no periods of hypoglycaemia. Unfortunately, the nadir can be difficult to predict since there is a wide variation in the duration of action of the different insulins between different cats, so it needs to be determined for each individual.
- If there is no reduction in blood glucose then increase the insulin dose by a total of 0.5-1IU per injection (not per cat).
- Once the nadir blood glucose is 8-13 mmol/l consider performing a more complete BGC.
- *N.B. There is no value in performing a complete BGC if the cat is receiving a dose of insulin that is not having a significant effect on blood glucose concentrations since the nadir and duration of effect will be impossible to evaluate.*

Performing and interpreting BGCs:

- The aim of a BGC is to identify the blood glucose concentration at the nadir of the day, obtain a more precise time for the nadir, evaluate the duration of effect of the

insulin and ensure that a Somogyi over-swing (insulin-induced hyperglycaemic) is not occurring. Once these details have been identified, subsequent BGCs may only require every 4 hourly sampling, particularly if the cat is clinically stable.

- A BGC is performed by checking the cat's blood glucose concentration, and then giving it its usual breakfast and dose of insulin, then determining its blood glucose level every 1-2h during a 12-24h period. 12 hours may be sufficient if the blood glucose has increased back to its pre-insulin concentration after 12 hours, but if it has not the curve should be continued for 24 hours. If the level of the blood glucose at its nadir is too high then the dose of insulin may need to be increased. If the duration of action is too short then it may be necessary to change to a longer acting insulin (e.g. PZI or glargine), or switch from once daily to twice daily administration.
- Ideally, the blood glucose concentration should be maintained between 5-14 mmol/l.
- **Unfortunately, the use of BGCs can be very limited in cats that develop stress hyperglycaemia when hospitalized**.
- To reduce the risk of this occurring it is best to use peripheral ear veins rather than jugular or cephalic sampling
- A warm swab is held over the peripheral ear vein to help dilate the peripheral vessels.
- The edge of the ear is smeared with Vaseline to prevent the blood running into the hair coat.
- The ear is then held firmly and gently between four fingers, which act in pairs to raise the vein and prevent its movement.
- The vein can then be pieced using either a fine hypodermic needle or a lancet. Holding the vein still for a few seconds will allow a bleb of blood to form.
- The glucometer test strip or, where appropriate, the glucometer can then be applied directly to the bleb of blood.
- **It is meaningless to perform a BGC in a cat with stress hyperglycaemia. For these cats it is often possible to train their owners to check their cat's blood glucose level at home, and even to perform BGCs at home.**
- **BCGs can provide very useful information but studies have shown that BGCs in an individual cat will vary widely from day to day. It is therefore very important that major changes to treatment are not based on a single BGC. All BCGs need to be interpreted in conjunction with the cat's clinical status, fructosamine results and the environment in which the cat was situated when the curve was performed. The most important thing to consider is the *trend* of change, so comparing the current curve with previous BCG results is an**

important part of interpretation. Rather than thinking of a BGC over a 24 hour period, it is useful to think of a BGC over a week and aim to try and keep the BGC consistent from week to week (i.e. monitoring trends), but accepting daily fluctuations are a normal occurrence.

- After recommending a change the cat should be discharged on the new regime and the whole process repeated after a further 7 days. Only 1 change to the insulin regime should be made at a time.
- It is often recommended that BGCs should be performed every 1-2 weeks until the DM is stable. After this time they can be performed less frequently, and the insulin dosage can be adjusted in response to changes in clinical signs, daily water consumption and serum fructosamine concentrations.

Monitoring diabetic control:

There are 4 main aspects that need to be considered when monitoring longer term diabetic control:

1. Owner's observations regarding the presence and severity of ***clinical signs***. This is a vital part of monitoring and owners should be instructed to keep a diary recording the cat's:
    a. Body weight and body condition score
    b. Demeanour
    c. Appetite
    d. Thirst (just a subjective assessment by the owner is usually helpful)
    e. Coat condition
    f. Urination (weighing the litter tray regularly at the same time of day can give a useful subjective indication of changes in urination)
    g. At all times, but especially after altering the insulin dosage, the owners should be warned to look for signs of **hypoglycaemia** (a sudden desire to hide, excessive quietness, weakness, lethargy, shaking, ataxia, collapse and coma). Unfortunately, unlike dogs, cats rarely show polyphagia in response to hypoglycaemia. If the signs of hypoglycaemia occur the cat's gums should be rubbed with sugar water, jam or honey, and immediate veterinary attention should be sort.
    h. An instruction sheet should be issued to all owners with diabetic cats with the basic information that they need to know and the clinical signs that they need to monitor. More detailed information sheets are also useful to assist in educating the client about the disease and how it can be appropriately managed.

2. Serum ***fructosamine*** concentrations – can be performed monthly during the initial months of stabilization.
3. ***Serial BGCs*** – as explained above, caution needs to be taken when interpreting BGC results.
4. Monitoring for ***diabetic complications*** – this includes progression to DKA if the DM is not adequately controlled; periods of hypoglycaemia particularly if the cat's insulin requirements are variable; and conditions that may increase insulin requirements such as UTI, or result in variable insulin requirements such as pancreatitis.

In the longer term, once stabilized, diabetic cats should be reassessed at least every 3-6 months. At each check, the following should be performed as a minimum:

- Discussion with the owner – diabetic diary, any changes in clinical signs, etc
- Full physical examination
- Body weight, body condition score and calculate % weight change since the previous visit
- Serum fructosamine concentration

The following additional tests are also useful, where possible:

- Blood pressure measurement
- Full urine analysis including C&S and urine protein to creatinine ratio (UPC)
- Routine haematology
- Serum biochemistry

If the cat is free from clinical signs and the physical examination is unremarkable, then adequate glycaemic control is likely, and fructosamine concentration will assist in confirming this. If the fructosamine is low-normal, it may indicate that the cat is no longer diabetic. If clinical signs of persistent hyperglycaemia or episodes of hypoglycaemia are reported, or if there is evidence of weight loss, or other complications such as a peripheral neuropathy, then further diagnostics should be performed.

Periodic ***monitoring of urine for glucosuria and ketonuria*** in the home environment can also be useful for monitoring glycaemic control. It is not particularly helpful for owners to frequently measure urine glucose as this often results in owners making their own adjustments in insulin doses. However, it is useful in detecting the transient diabetic whose insulin requirements are reducing, as absence of glucosuria would make this likely. It is also useful for owners to periodically check for urine ketones, to try and detect developing DKA in

the early stages before the cat becomes too unwell.

Many diabetic cats will only be transiently diabetic so care needs to be taken to ensure detection of:

- Low blood glucose (<10mmol/l) prior to insulin administration
- Low/normal fructosamine concentration
- Persistent absence of glucosuria
- All the above may indicate resolution of exogenous insulin requirements

Suggested parameters for changing insulin dose and frequency are shown below, based on blood glucose measurement using lenti or PZI insulin in cats:

Blood glucose concentration (mmol/L)	**Recommendation**
Pre-insulin <10	With-hold insulin, check for diabetic remission
Pre-insulin 10-16	Total dose should be ≤ 1 IU/cat twice daily
Nadir <3	Reduce dose by 50%
Nadir 3-5	Reduce dose by 1 IU
Nadir 6-9	Keep dose the same
Nadir >10	Increase dose by 0.5-1 IU
Nadir ≤3 hours post insulin and/or glucose concentration returns to baseline within 8 hours	Change to longer acting insulin (PZI or glargine)
Nadir ≥8 hours post insulin	Reduce the dose but keep on twice daily or change to once daily

Problems with stabilization:

It is not uncommon for problems to be seen in the early stages of stabilisation. These are often related to the storage and administration of the insulin so they are usually quite easy to identify and remedy. Common problems include:

- Routine not adhered to e.g. insulin given at a different time each day
- Variable quantity or type of food being fed
- Ineffective insulin (out of date, incomplete mixing, poor storage [insulin will bind to the rubber stopper of the dispensing bottle if it is stored upside down or on its side, so causing loss of activity])
- Incorrect dosage because of syringe-type insulin-type mismatch (for this reason it is important to always use the correctly paired insulin and syringe)
- Poor injection technique

- Insulin overdose (leading to insulin-induced hyperglycaemia i.e. a Somogyi over-swing – see below)
- Out of date urine test strips

The first thing that should be done when there are problems with stabilisation is to ask the owner to demonstrate how they mix and inject the insulin in order to check that this is being done correctly. If these problems are eliminated as a cause of the poor stabilisation, then further investigations may be needed to locate the problem so that this can be remedied. In some cases it will be necessary to re-admit the cat for more detailed assessment which may include a 24 hour BGC.

Hypoglycaemia:

Hypoglycaemia can be a common complication of insulin treatment, arising for a number of reasons e.g. the insulin dose is increased too rapidly (particularly if stress hyperglycaemia is mistaken for poor glycaemic control), if a previous insulin resistance has resolved, or if a cat has reverted to a non-insulin dependant state.

It is important to be aware that hypoglycaemia can be difficult to recognise early in cats, as in contrast to dogs, cats do not always exhibit polyphagia when they become hypoglycaemic. The earliest sign that owners often note is that the cat hides more than usual. It then becomes quiet, weak and lethargic; it may shake, then become ataxic, collapse and eventually lapse into a coma.

If hypoglycaemia occurs, insulin should be discontinued until hyperglycaemia recurs and then re-instigated at half of the previous dose. If blood glucose levels are still low or normal when the cat is receiving 1 IU or less of insulin, then resolution of the insulin dependant state should be suspected.

Somogyi Over-swing:

This describes a normal physiological response to hypoglycaemia induced by excessive insulin administration. This commonly occurs when insulin doses are increased too quickly with inadequate monitoring, or if the cat has very fluctuating insulin requirements. When blood glucose concentrations reduce to **< 3.5 mmol/l or when they fall very rapidly**, counter-regulatory hormones such as glucagon and adrenaline are secreted, resulting in a rebound hyperglycaemia and insulin resistance within a few hours. This hyperglycaemia persists for at least 24 hours in most cases, and can last for up to 72 hours, or occasionally even longer. Clinical signs of hypoglycaemia are rarely seen so the cat will present as insulin

resistant. The speed of reduction in blood glucose is often the trigger for a Somogyi over-swing, rather than significant hypoglycaemia, so clinical signs of hypoglycaemia may not be present.

Diagnosis is made by demonstrating hypoglycaemia or a rapid fall (e.g. >10mmol/l in 1 hour) in blood glucose. Unfortunately, it can be missed if blood samples are taken less frequently then every hour following insulin administration, and the subsequent rebound hyperglycaemia and insulin resistance can last for more than 24 hours, both of which tend to lead to a mis-diagnosis of insulin resistance. Serum fructosamine may also be elevated if rebound hyperglycaemia is prolonged. If there is a possibility of over-swing occurring it is advisable to reduce the insulin dose to 0.25 - 0.5 IU/kg for a few days and then reassess the cat's response. In the short term, hyperglycaemia is a "safer" state than insulin induced hypoglycaemia. If there are no improvements in the cat's clinical signs, and no reduction in its blood glucose concentration, then another cause of insulin resistance should be considered.

Fluctuating insulin requirements:

It is not unusual for some cats to have fluctuating insulin requirements, with uncontrolled DM one moment and then hypoglycaemia the next. The most common reason for this is the development of a concurrent disease that causes a mild insulin resistance that later resolves spontaneously, or waxes and wanes. Inflammatory diseases such as chronic pancreatitis are commonly associated with these fluctuating requirements. These cases can be extremely difficult to manage, and this is one situation where home blood glucose monitoring can be very useful.

Insulin resistance:

The majority of diabetic cats can be controlled with 1 IU/kg of insulin (per dose not per day). Insulin resistance is therefore generally defined as *insulin requirements exceeding 2 IU/kg.* It can be associated with:

- Recent weight gain - resulting in increased insulin resistance.
- Pancreatitis - this often results in very variable insulin requirements. Chronic pancreatitis can be difficult to diagnose and cats may show few clinical signs associated with it. Measurement of fPLI is the most sensitive test available for diagnosing pancreatitis, but even that is not 100% sensitive so a normal fPLI does not exclude pancreatitis. Achieving a diagnosis of pancreatitis is unlikely to alter the way a cat's DM is treated, and treatment for the pancreatitis itself is merely symptomatic, however it provides some explanation as to why a cat may have

extremely variable insulin requirements.

- Failure of insulin absorption (seen most commonly when the insulin is injected at the same site every day)
- Infection - most frequently gingivitis or UTI.
- Administration of diabetogenic drugs e.g. corticosteroids (dexamethosone is more diabetogenic than equipotent doses of prednisolone) or megoestrol acetate, particularly when given at high doses and/or long courses.
- Hyperthyroidism
- Acromegaly
- Hyperadrenocorticism
- Renal or hepatic insufficiency
- Anti-insulin antibodies (very rare in cats)
- Presence of certain types of tumour e.g. pancreatic adenocarcinoma

3. Oral hypoglycaemic agents –

These can act to increase insulin secretion, decrease peripheral insulin resistance, and/or decrease the absorption of glucose from the intestinal tract. They may successfully control some non-ketotic, uncomplicated diabetics, either temporarily or longer term, particularly when given in conjunction with dietary modification. Unfortunately, some of them, e.g. sulfonylureas, act by stimulating insulin secretion, so they can ultimately cause pancreatic islet cell exhaustion, and cause a non-insulin dependant diabetic to become insulin dependent.

There are several different types of drugs that have been shown to be at least somewhat effective in cats:

- **Sulfonylureas** e.g. glipizide (0.25-1.0 mg/kg PO q8-12h, adjust dose as needed); side effects include vomiting, anorexia, and hepatopathy. Periodic checks for serum biochemistry and haematology are recommended. It may take a few weeks of medicating to see the full effect of the drug.
- **Alpha-glucosidase inhibitors** e.g. acarbose (12.5-25 mg/cat PO with meals) can be useful in cats with diabetes and advanced chronic kidney disease that require a restricted protein diet which is consequently high in carbohydrates; side effects include flatulence, soft faeces and diarrhoea.
- **Transition metals** e.g. vanadium (0.2 mg/kg/day in food); side effects include anorexia, vomiting, diarrhoea and renal disease; chromium (200 ug/cat/day PO); side effects are as yet unknown. These compounds are thought to improve glucose

tolerance by increasing insulin sensitivity. The addition of chromium has been suggested to improve diabetic control in cats with DM and concurrent renal failure, where they need to be fed a renal diet rather than a high protein/low carbohydrate one.

Prognosis:

The prognosis of any cat with DM is very unpredictable. It depends on the owners' commitment, the compliance of the cat, the presence of concurrent and interacting disease, and the ease of glycaemic control. Many diabetic cats have an excellent quality of life. However, long-term the prognosis is generally guarded and when chronic pancreatitis is also present DM can be particularly difficult to control. The most common causes of death in diabetic cats appear to relate to pancreatitis, UTI's, renal disease and cardiac disease. On average, 50% of diabetic cats are dead within 12 to 17 months of diagnosis (particularly those with concurrent complicating diseases), with a median survival time varying from 13 to 29 months (Kraus and others 1997; Goossens and others 1998; Little and Gettinby 2008).

Prevention:

The risk of developing DM can be reduced by not allowing cats to become obese, by encouraging cats to exercise, by not breeding from Burmese lines that are known to be predisposed to DM and by not giving long courses of diabetogenic drugs. Routine screening of 'at risk' cats may allow early detection of DM, and aggressive treatment will increase the chance of gaining diabetic remission.

To improve early detection of feline DM:

- Perform routine urinalysis annually in all cats from 7 years of age.
- Consider an increased frequency of routine urinalysis (every 3-6 months) in those cats that are Burmese, indoor, obese, or receiving corticosteroid/progestogen treatment. Particular vigilance is required in cats where 2 or more risk factors are present.
- Utilise home urine sampling, using a non-absorbent litter (e.g. Katkor/Mikki litter/aquarium gravel in their litter tray).
- Add blood glucose measurement to pre-anaesthetic protocols in all cats over the age of 7 years.
- Care needs to be taken in interpretation of hyperglycaemia/glucosuria in samples collected in the practice because of the possibility of stress induced hyperglycaemia – following 'Cat Friendly Practice' principles* will help to minimise this and make

results easier to interpret.

- However, if all blood/urine glucose elevations are attributed to stress, early DM may be missed.
- Always follow-up up a high blood glucose concentration or glucosuria with a serum fructosamine measurement.
- Consider measuring serum fructosamine annually if a urine samples can not be obtained.
- Educate owners in observing their cats for early clinical signs of DM.
- Regular weighing (and calculation of percentage weight change) and body condition scoring to detect trends in changes of body weight or body condition.

*For more information see the Feline Advisory Bureau (FAB) Information Sheet on DM in cats and information on how to have a Cat Friendly Practice: www.fabcats.org

		PVD DM wet	PVD DM dry	Hill's m/d wet	Hill's m/d dry	RCW Diabetic dry
TYPICAL ANALYSIS	Unit	DRY MATTER				
Protein	%	55.6	54.1	52.8	51.1	46
Fat	%	23.1	17.1	19.4	22	12
Fibre	%	3.7	1.3	6	6	11.4
Carbohydrates	%	3.8	18.2	15.7	15	17.1
Declared M.E.	Kcal/Kg	4100	4100	4000	4200	3800

		PVD OM wet	PVD OM dry	Hill's r/d wet	Hill's r/d dry	Hill's w/d wet	Hill's w/d dry
TYPICAL ANALYSIS	Unit	DRY MATTER					
Protein	%	44.6	56.2	37.3	37.7	41.5	40
Fat	%	14.6	8.5	9.1	9.8	16.5	9.8
Fibre	%	10.2	5.6	15.4	15.1	10.6	9.2
Carbohydrates	%	23.2	22.4	31.5	32	24.2	35.3
Declared M.E.	Kcal/Kg	3200	3200	3100	3200	3800	3500

Table 1. Principle diets suitable for the management of feline diabetes mellitus in Europe. PVD = Purina Veterinary Diets, RCW = Royal Canin-Waltham, ME = metabolizable energy, kg – kilogram, DM = dry matter basis, NA = not available

It is recommended that non-obese cats with diabetes mellitus should be fed a diet that is high in protein and low in carbohydrate, without excessive dietary fat.
Diet Data January2009

References available from the author on request.

CLIENT INSTRUCTION SHEET FOR DIABETIC CATS

Name: **Date diabetes was diagnosed:**

Weight at time of diagnosis: **Estimated ideal weight:**

FEEDING

Diet: **Amount of food to be given:**

Frequency and timing of feeding:

INSULIN

Insulin type: **Dose:**

Storage instructions:

The insulin bottle should be kept upright in the door of the fridge. It is important that it is not frozen or left out at any stage since this will damage the insulin.

Administering the insulin:

When the insulin bottle is taken out of the fridge it should be rolled gently to mix the contents: it must not be shaken as this will damage the insulin. The correct amount of insulin should be drawn up in the appropriate syringe provided by your vet, and injected under the skin in the scruff of your cat's neck - use a slightly different site each day.

ADDITIONAL MEDICATION

- ……..

Monitoring your cat for signs of inadequate control of diabetes

- Please keep a diary of your cat, recording the following observations:
- Body weight – this should be recorded at least every 2 weeks if your cat is on a weight loss program. A subjective assessment of body condition should also be made regularly (graded 1 to 9: where 1 is skeletal and 9 is obese)
- Demeanour – e.g. bright and active, or lethargic, weak
- Appetite/ amount eaten daily
- Thirst/ amount drunk daily
- Coat condition
- Frequency/volume of urination
- Periodic urine ketones measurement using dipstick, as directed by your vet

Monitoring your cat for signs of insulin over dosage (hypoglycaemia)

Signs of hypoglycaemia (low blood sugar) can occur at any time but are most likely to occur at the time of maximal insulin action, so usually around 4-8 hours after giving insulin, depending on what type of insulin your cat is receiving. The lower the blood sugar levels go, and the more rapidly the levels drop, the more severe the signs that you notice will be:

- Wanting to hide, lethargy, weakness
- Hunger
- Disorientation, apparent blindness, shaking, wobbliness
- Collapse, seizures

The development of hypoglycaemia can be life threatening if left untreated so it is very important that early signs are recognised and treated.

What to do if you notice any of these signs?

If the signs are mild and the cat will eat, it should be offered food. This may be enough to relieve the signs. If signs are more severe or if the cat will not eat, glucose syrups, honey, jam or sugar water can be rubbed on your cat's gums. Your veterinary surgeon should then be contacted for further advice. If the signs have progressed to collapse or seizures an emergency vet should be contacted immediately.

Notes page

HYPERTHYROIDISM IN CATS

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Pathogenesis

Hyperthyroidism is one of the most common endocrinopathies of cats, affecting ~1 in 50 cats (Feldman and Nelson 2004; Peterson and Ward 2007). Ninety nine percent of cases result from benign nodular hyperplasia/adenoma (Peterson and others 1983; Peterson and Ferguson 1989), which results in the autonomous secretion of thyroxin (T4) and tri-iodothyronin (T3). These hormones produce a negative feedback affect on the pituitary gland, suppressing the release of thyroid-stimulating hormone (TSH), so any normal thyroid tissue atrophies. In 70% of the cats, both thyroid glands are affected (Peterson and others 1983; Peterson and Ferguson 1989; Feldman and Nelson 2004). In only 1% of cases is the disease caused by mild to moderately malignant thyroid carcinoma (Turrel and others 1988).

While the cause of the nodular hyperplasia/adenoma is unknown, it is believed to involve factors within the diet (possibly including iodine content, feeding canned food, frequent changes, food additives) (Tarttelin and others 1992; Kass and others 1999; Martin and others 2000), environmental causes (possibly associated with cat litter, toxins, pollution, goitrogens, exposure to allergens, exposure to flame retardant chemicals) (Scarlett and others 1988; Kass and others 1999), genetic mutation (Merryman and others 1999), and/or abnormal immune and/or hormonal responses.

Clinical signs

Hyperthyroidism is seen mainly in middle-aged to older cats; with the age at presentation ranging from four to 23 years (mean 13 years) (Peterson and others 1983; Thoday and Mooney 1992). However, it has occasionally been seen in younger cats (including one kitten of eight months old) (Gordon and others 2003). There is no sex or breed predisposition (Peterson and others 1983; Thoday and Mooney 1992; Martin and others 2000), although Siamese and Himalayan (Colour-point Persian) cats appear to be under-represented in some studies (Scarlett and others 1988; Kass and others 1999).

Most cats have a history of weight loss and polyphagia, usually occurring over several months (Thoday and Mooney 1992; Broussard and others 1995). In most cases clinical signs are insidious and progressive. Affected cats are frequently restless, potentially aggressive, and stop grooming. They have commonly been vomiting and/or had diarrhoea (the faeces often become bulky); some have polyuria/polydipsia (Peterson and others 1983; Thoday and Mooney 1992; Broussard and others 1995). Less common clinical signs include dyspnoea, seizures, or severe muscle weakness (the latter being due to hypokalaemic myopathy and/or low thiamine levels) (Nemzek and others 1994; Broussard and others 1995). The gastrointestinal signs may result from malabsorption and/or intestinal hypermotility, and may be associated with very low folate levels (which need to be treated for the diarrhoea to resolve). Polyuria/polydipsia may result from diuretic effects of T4, increased renal blood flow, associated renal insufficiency, or compulsive polydipsia. Associated cardiac hypertrophy may eventually result in congestive heart failure, with tachycardia, a gallop rhythm, systolic murmurs, dyspnoea, apathy, hind limb weakness due to aortic thromboemboli, or collapse (Liu and others 1984). Up to 85% of cats with hyperthyroidism may develop systemic hypertension. This may be detected as hypertensive retinopathy, including ocular haemorrhage (Stiles and others 1994), or cause clinical signs associated with cerebrovascular accidents, dementia, and/or renal failure. Poorly controlled cases can sometimes be presented with disorientation and signs of bilateral central vestibular disease (dilated pupils, lack of menace response, and neck ventroflexion), which is believed to result from a secondary thiamine deficiency (similar to thyrotoxicosis-associated Wernicke's encephalopathy in humans [Sechi 2008]). Approximately 10% of cats present with signs of inappetence, rather than polyphagia, and are often depressed and weak (so called apathetic hyperthyroidism) (Peterson and others 1983): this is frequently due to significant secondary or associated cardiac disease. Many hyperthyroid cats are presented for their routine vaccination with no owner complaints as their owner’s presume that the cat’s clinical signs are the normal result of ageing (Broussard and others 1995).

Diagnosis

Hyperthyroidism should be suspected when any older cat presents with weight loss, and especially when the weight loss is associated with a good appetite. However, inappetence should not rule out hyperthyroidism. Physical examination usually reveals rather poor body condition, an ill-kempt coat, and a thyroid nodule on either or both sides of the trachea in the ventral cervical region (80-90% of cases) (Peterson and others 1983; Thoday and Mooney 1992; Broussard and others 1995). Affected cats often have tachycardia, a gallop rhythm, and/or a systolic murmur. Cardiac effects result from a high output state, induced, in part, by a demand for increased tissue perfusion to meet the needs of increased tissue metabolism.

In addition, thyroid hormones can have a direct effect on cardiac muscle. Cardiovascular changes include left ventricular hypertrophy, left atrial and ventricular dilation, increased myocardial contractility, and decreased peripheral vascular resistance (Liu and others 1984). Hyperthyroid cats are often agitated, difficult to examine, and become easily stressed (Peterson and others 1983; Broussard and others 1995).

Clinical pathology almost always reveals raised liver enzymes (serum alanine transferase [ALT], alkaline phosphatase [ALP], and aspartate transferase [AST]) (Peterson and others 1983; Thoday and Mooney 1992; Broussard and others 1995). The hepatopathy may be secondary to a direct toxic effect of the thyroid hormones, hepatic lipidosis, malnutrition, or hepatic hypoxia resulting from cardiac failure (Thoday and Mooney 1992). The ALP may also be raised because of increased bone metabolism (Horney and others 1994; Foster and Thoday 2000). In some cases, serum glucose concentration may be increased, or azotaemia may be present (Peterson and others 1983). The latter may result from increased protein catabolism, reduced renal perfusion caused by associated cardiac insufficiency; renal damage induced by associated systemic hypertension, or be related to concomitant, but unrelated, chronic renal insufficiency. Hyperphosphataemia, hypocalcaemia, and secondary hyperparathyroidism may be detected, irrespective of the presence of renal insufficiency, possibly resulting from T4-mediated alterations in bone metabolism and increased phosphate absorption (Peterson and others 1983; Barber and Elliott 1996). Hypokalaemia may be present, or may develop apparently in response to the stress of the diagnostic investigations. When this occurs the resulting hypokalaemic myopathy may be seen as severe muscle weakness and neck ventroflexion, and the serum creatinine kinase concentration will increase (Nemzek and others 1994). Hypocobalinaemia is often present (Cook and others 2011).

Haematology may reveal erythrocytosis or, in very severe disease, mild anaemia. Leukocyte changes may include a mature neutrophilia, lymphopenia or lymphocytosis, eosinopenia or eosinophilia (Peterson and others 1983; Thoday and Mooney 1992). Unless there is concurrent renal insufficiency the urine is usually reasonably concentrated (Graves and others 1994; Thoday and Mooney 1992; Broussard and others 1995). Microalbuminuria may be present (Syme and Elliott 2003), and some cats develop significant proteinuria (i.e. urine protein to creatinine ratio >0.4), which is often associated with concurrent systemic hypertension (Syme et al 2006; Elliott and Syme 2006). Hyperthyroid cats are predisposed to bacterial urinary tract infections: 12-24% are affected (Mayer-Roenne and others 2007).

When investigating a cat for possible hyperthyroidism it is important to consider all possible

differential diagnoses and to look for evidence of multiple interacting diseases. This is because hyperthyroidism is seen most often in older cats, and this group of patients is often affected by more than one disorder. Diabetes mellitus, kidney disease, malassimilation syndromes (including inflammatory bowel disease, pancreatitis and/or exocrine pancreatic insufficiency, and early intestinal lymphoma), urinary tract infections, acromegaly, and hyperadrenocorticism are perhaps the most important differentials.

A full cardiac investigation is recommended prior to considering treatment options for the hyperthyroidism, especially surgery. Thoracic radiography may reveal cardiomegaly, pulmonary oedema or pleural effusion (Peterson and others 1983). Electrocardiography commonly reveals abnormalities, including sinus tachycardia, increased R-wave amplitude, atrial and ventricular arrhythmias and intraventricular conduction disturbances (Peterson and others 1982, 1983; Moise and Dietze 1986; Broussard and others 1995). Abnormalities are also seen frequently on echocardiography. These may include hypertrophy of the left ventricular free wall and interventricular septum, increased left atrial diameter at end diastole, and hyperdynamic wall motion (Moise and Dietze 1986; Bond and others 1988).

A definitive diagnosis of hyperthyroidism is based on detecting elevated serum concentrations of total T4 (and possibly T3) (Peterson and others 1983, 2001; Thoday and Mooney 1992). Measurement of T3 alone is not usually recommended, as it is less sensitive than T4 (Peterson and others 1987). Unfortunately, some cats with hyperthyroidism also have a T4 concentration that is within the normal range. This may be due to early or mild hyperthyroidism, daily variations in T4 concentrations, or the concurrent presence of severe systemic illness causing a reduction in T4 (euthyroid sick syndrome) (Peterson and Gamble 1990; Thoday and Mooney 1992; McLoughlin and others 1993; Mooney and others 1996a; Peterson and others 2001).

If hyperthyroidism is suspected despite a high normal T4 concentration:

- *Retest the cat*:
- Retest the cat, either immediately, or in a few weeks time (Peterson and Gamble 1990). Assessing free T4, as well as total T4, may help in confirming the presence of hyperthyroidism (Mooney and others 1996b; Peterson and others 2001).
- *T3 suppression test*:
- Protocol: Collect a blood sample, give 25mg of T3 orally every eight hours for seven doses, then collect a blood sample two to four hours after the seventh dose (i.e. on day three). An increase in T3 concentration confirms successful medication.

Suppression of the T4 concentration (below 50% of baseline, <1.5 ug/dl [<20 nmol/l] does not occur in hyperthyroid cats (Peterson and others 1990; Refsal and others 1991). This is a useful test at ruling out but hyperthyroidism, but cannot reliably be used to confirm hyperthyroidism (Peterson and others 1990; Refsal and others 1991). In addition, unless the cat is hospitalized, relies on the owner being able to reliably administer the T3.

- *Thyrotropin-releasing hormone (TRH) stimulation test*:
- Protocol: Collect a blood sample; give 0.1 mg/kg TRH IV, and then collect a second blood sample four hours later. Assess both samples for serum T4 concentration. Stimulation to greater than 50% does not occur in hyperthyroid cats. Side effects of TRH include transient salivation, vomiting, tachypnoea, and defecation (Sparkes and others 1991; Peterson and others 1994). While this test is good at detecting mild or early hyperthyroidism (Peterson and others 1994), it is not so good at detecting hyperthyroidism in a cat with severe concurrent disease and euthyroid sick syndrome (Tomsa and others 2001).
- *Thyroid-stimulating hormone (TSH) response test:*
- This test is not recommended in the diagnosis of hyperthyroidism as it cannot reliably differentiate between normal cats and cats with mild hyperthyroidism (Peterson and Ferguson 1989; Mooney and others 1996b; Peterson and others 1990). In addition, TSH is very difficult to obtain.
- *Nuclear isotope scanning:*
- This technique can be used to detect hyperactive thyroid tissue, to determine whether one or both thyroid glands are overactive, and if there is any ectopic thyroid tissue (Peterson and others 1983). The procedure is relatively safe and simple to perform, but requires sedation and access to a licensed facility.
- *Trial course of anti-thyroid therapy:*
- Administering a trial course of anti-thyroid therapy (see below), for approximately 30 days, and observing for changes in clinical signs, can help in trying to decide whether or not a cat is clinically hyperthyroid. However, there is a potential risk of side effects, and doing so requires monitoring of haematology and serum biochemistry.

Treatment

It is essential that renal function is assessed prior to considering possible treatment options. This is because resolution of the hyperthyroid state is associated with an increase in blood urea nitrogen (BUN) and creatinine concentration, and a decrease in glomerular filtration rate (GFR) and effective renal blood flow. Because of this, some cats without prior evidence

of renal insufficiency, or with only mild renal impairment, develop signs of uraemia following treatment for hyperthyroidism (Graves and others 1994; Adams and others 1997). Studies have shown that 17-49% of treated hyperthyroid cats develop azotaemia within 6 months of hyperthyroid treatment, and this is more likely to occur in cats with subnormal thyroxin concentration post-treatment (Williams and others 2010a and b). In order to ascertain what effect resolving the hyperthyroid state will have on any particular cat it is recommended that all cats receive initial medical therapy with methimazole or carbimazole, or dietary therapy (Hill's y/d), prior to considering radiotherapy or surgery (Graves and others 1994; Adams and others 1997). Cats that do show significant uraemia or develop renal failure following radiotherapy or surgery should be given levothyroxin to maintain a euthyroid or mild hyperthyroid state (Graves and others 1994; Adams and others 1997).

Hyperthyroidism can be treated medically, surgically, with radioiodine (I^{131}) or with diet. Prior to deciding which treatment to use the cat should be assessed for concurrent disease, especially renal disease, systemic hypertension and heart disease, all of which occur commonly in association with hyperthyroidism. The interplay between systemic blood pressure and renal function is complex. While systemic hypertension is detrimental to kidney function, a sudden fall in blood pressure (e.g. associated with a sudden fall in T4) can exacerbate renal dysfunction by causing a sudden fall in renal blood flow. Changes in T4 need to be made gradually so there are no sudden changes in renal blood pressure. By maintaining renal blood pressure, hyperthyroidism can mask low-grade renal insufficiency. It is essential to check serum urea and creatinine concentrations and urine specific gravity prior to inducing irreversible reduction of T4 (i.e. by thyroidectomy or I^{131} treatment). A short course of medical therapy or dietary therapy may reveal the presence of masked renal insufficiency. If hypothyroidism occurs following irreversible treatment of hyperthyroidism, and renal function suffers, this can be reduced by treating with synthetic thyroxin.

- *Medical therapy* tends to be given to stabilize the cat prior to surgical treatment, to check for masked kidney disease prior to thyroidectomy or I^{131} treatment, or when neither I^{131} or surgery are possible.

Methimazole and carbimazole block T3 and T4 synthesis. It takes 1-3 weeks before a significant decrease in T4 concentrations occur after beginning treatment.

Carbimazole is broken down to methimazole in vivo. Unfortunately, bioavailability and volume of distribution of methimazole is highly variable between cats.

Dose for both is 2.5-5.0 mg PO every 8-24 hours initially, reducing to every 12-24 hours. If the cat has concurrent renal insufficiency, start with a low dose and monitor renal values as the dose is gradually increased. Preliminary studies with topical transdermal applications show promise.

When cat and owner compliance is good, the successful response rate is approximately 85% with medical treatment.

Poor compliance results from:

- The need for frequent medication.
- The need for frequent blood samples to look for possible side effects. Blood dyscrasias occur in 2-10% of cats and include eosinophilia, lymphocytosis, leukopenia, thrombocytopenia, and/or agranulocytosis, hepatopathy, jaundice, cutaneous reactions (typically pruritus of the head and neck), bleeding tendencies or, very occasionally, myasthenia gravis, or immune-mediated haemolytic anaemia (IMHA).
- Frequent side effects. Up to approximately 20% of cats develop anorexia, vomiting or lethargy. Mild side effects may resolve despite continued treatment.

Other medical therapies include:
Propranolol (□-adrenoceptor blocking agent) may be added to reduce tachycardia, arrhythmias, and hypertension (2.5-5.0 mg/cat PO every 8-12 hours).
Stable iodine helps to decrease T3 and T4 synthesis and reduce thyroid gland vascularity, but the effect can be transient and inconsistent. Give potassium iodide 30-100mg/cat/day PO for 10-14 days prior to surgery using 100g potassium iodide/100ml solution, or potassium iodate ~20mg/cat every 12 hours PO.
Calcium or sodium ipodate is a radiopaque iodine agent that reduces T3 concentrations. Its effect can be transient, and it may be difficult to obtain (15 mg/kg PO every 12 hours).

- *Surgical thyroidectomy.* The success depends on the stability of the patient, the expertise of the surgeon (a bilateral thyroidectomy is usually performed), and the expertise of the anaesthetist (e.g. do not give atropine).

Successful response rate is > 95%. Ectopic overactive thyroid tissue is a cause of failure, as it is present in ~20% of cats with hyperthyroidism that are referred for I^{131} therapy, and this is missed at surgery.

Reduce the risks of surgery by making the cat euthyroid prior to surgery (see *medical therapy* above).

Surgical risks include anaesthetic risks in older patients (often with concurrent renal ± cardiac disease), iatrogenic damage to parathyroid tissue leading to transient or permanent hypocalcaemia, or to the local nerves leading to laryngeal paralysis or Horner's syndrome.

- *Radioiodine (I^{131})* is taken up by and destroys the overactive thyroid tissue (including ectopic tissue), but spares the normal tissue.

Successful response rate is > 95%, but it may take a few weeks, or occasionally months, for the normal tissue to recover function.
Availability of facilities and length of stay in hospital varies from 2 days to 4 weeks depending on country and state, as it often depends on the interpretation of radiation safety laws.
Side effects are few and include transient dysphagia or dysphonia, or permanent hypothyroidism (~2%).

- *Dietary therapy* currently consists of Hills y/d. This diet is low in iodine; without dietary iodine the cats cannot make T3 or T4 and signs of hyperthyroidism abate will the diet is fed (exclusively). Preliminary studies look promising, but larger studies are needed.

Prognosis

Without treatment, cats with hyperthyroidism will usually die of concurrent kidney heart or liver disease, or systemic hypertension. With treatment, prognosis varies from good to guarded, dependent on the presence of heart disease, kidney disease, and systemic hypertension, whether or not any damage has become permanent prior to treatment of the hyperthyroidism, and which treatment options are available.

References

Adams WH, Daniel GB, Lrgendre AM. Investigation of the effects of hyperthyroidism on renal function in the cat. Can J Vet Res 61: 53-56, 1997

Barber PJ and Elliott J. Study of calcium homeostasis in feline hyperthyroidism. JSAP 37: 575-582, 1996

Bond BR, Fox PR, Peterson ME, Skavaril RV. Echocardiographic findings in 103 cats with hyperthyroidism. JAVMA 192(11): 1546-1549, 1988

Broussard JD, Peterson ME, Fox PR. Changes in clinical and laboratory findings in cats with hyperthyroidism from 1983 to 1993. JAVMA 206: 302-305, 1995

Cook AK, Suchodoloski JS, Steiner JM, Robertson JE. The prevalence of hypocobalinaemia in cats with spontaneous hyperthyroidism. JSAP 52: 101-106, 2011

Elliott J, Syme HM (2006) Editorial: Proteinuria in chronic renal failure in cats – prognostic marker or therapeutic target? JVet Int Med 20, 1052-1053

Feldman EC, Nelson RW. Feline hyperthyroidism (thyrotoxicosis). In: Canine and Feline Endocrinology and Reproduction, 3rd edition. Editors EC Feldman and RW Nelson. St. Louis: WB Saunders; 2004. p152-218.

Foster DJ, Thoday KL. Tissue sources of serum alkaline phosphatase in 34 hyperthyroid cats: a qualitative and quantitative study. Res Vet Sci 68: 89-94, 2000

Gordon JM, Ehrart EJ, Sisson DD. Juvenile hyperthyroidism in a cat. JAAHA 39(1): 67-71, 2003

Graves TK, Olivier NB, Nachreiner RF, Kruger JM, Walshaw R, Stickle RL. Changes in renal function associated with treatment of hyperthyroidism in cats. Am J Vet Res 55(12): 1745-1749, 1994

Horney BS, Farmer AJ, Honor DJ, et al. Agarose gel electrophoresis of alkaline phosphatase isoenzyme in the serum of hyperthyroid cats. Vet Clin Path 23: 98-102, 1994

Kass PH, Paterson ME, Levy J, James K, Becker DV, Cowgill LD. Evaluation of environmental, nutritional, and host factors in cats with hyperthyroidism. JVIM 13: 323-329, 1999

Liu S, Peterson ME, Fox PR. Hypertrophic cardiomyopathy and hyperthyroidism in the cat. JAVMA 185(1): 52-57, 1984

McLoughlin MA, DiBartola SP, Birchard SJ, Day DG. Influence of systemic nonthyroidal illness on serum concentration of thyroxine in hyperthyroid cats. JAAHA 29: 227-234, 1993

Martin KM, Rossing MA, Ryland LM et al. Evaluation of dietary and environmental risk factors for hyperthyroidism in cats. JAVMA 217: 853-856, 2000

Mayer-Ronne B, Goldstein RE, Erb HN. Urinary tract infections in cats with hyperthyroidism, diabetes mellitus and chronic kidney disease. JFMS, 9(2): 124-32, 2006

Merryman JI, Buckles EL, Bowers G, et al. Overexpression of c-Ras in hyperplasia and adenomas of the feline thyroid gland: an immunohistopathological ananlysis of 34 cases. Vet Path 36: 117-124, 1999

Moise NS, Dietze AE. Echocardiographic, electrocardiographic, and radiographic detection of cardiomegaly in hyperthyroid cats. Am J Vet Res 47(7): 1487-1494, 1986

Mooney CT, Little CJL, Macrae AW. Effects of illness not associated with the thyroid gland on serum total and free thyroxine concentration in cats. JAVMA 208: 2004-2008, 1996

Mooney CT, Thoday KL,Doxey DL. Serum thyroxine and triiodothyronine responses of hyperthyroid cats to thyrotropin. Am J Vet Res 57(7): 987-991, 1996b

Nemzek JA, Kruger JM, Walshaw R, et al. Acute onset of hypokalemia and muscular weakness in four hyperthyroid cats. JAVMA 205: 65-68, 1994

Peterson ME,Gamble DA. Effect of nonthyroidal illness on serum thyroxine concentrations in cats: 494 cases (1988). JAVMA 197: 1203-1208, 1990

Peterson ME, Ferguson DC. Thyroid disease. In: Textbook of veterinary internal medicine. Ed. SJ Ettinger. WB Saunders, Philadelphia, pp1654-1667, 1989

Peterson ME, Ward CR. Etiopathologic findings of hyperthyroidism in cats. Vet Clin North Am, 37: 633-645, 2007

Peterson ME, Keene B, Ferguson DC, Pipers FS. Electrocardiographic findings in 45 cats with hyperthyroidism. JAVMA 180(8): 934-937, 1982

Peterson ME, Kintzer PP, Cavanagh PG, Fox PR, Ferguson DC, Johnson GF,Becker DV. Feline hyperthyroidism: Pretreatment clinical and laboratory evaluation of 131 cases. JAVMA 183(1): 103-110, 1983

Peterson ME, Graves TK,Cavanagh I. Serum thyroid hormone concentrations fluctuate in cats with hyperthyroidism. JVIM 1(3): 142-146, 1987
Peterson ME, Graves TK,Gamble DA. Triiodothyronine (T3) suppression test: An aid in the diagnosis of mild hyperthyroidism in cats. JVIM 4(5): 233-238, 1990a

Peterson ME, Broussard JD,Gamble DA. Use of the thyrotropin releasing hormone stimulation test to diagnose mild hyperthyroidism in cats. JVIM 8(4): 279-286, 1994

Peterson ME, Melian C,Nichols R. Measurement of serum concentrations of free thyroxine, total thyroxine and total triiodothyronine in cats with hyperthyroidism and cats with nonthyroidal disease. JAVMA 218(4): 529-536, 2001

Refsal KR, et al. Use of the triiodothyronine suppression test for diagnosis of

hyperthyroidism in ill cats that have serum concentration of iodothyronines within normal range. JAVMA 199: 1594-1601, 1991

Scarlett JM, Moise NS,Rayl J. Feline hyperthyroidism: a descriptive and case-control study. Prev Vet Med 6: 295-309, 1988

Sechi G. (2008) Thyrotoxicosis-associated Wernicke's encephalopathy. J Gen Intern Med.;23(6):897.
Sparkes AH, Jones BR, Gruffydd-Jones TJ, Walker MJ. Thyroid function in the cat: assessment by the TRH response test and the thyrotrophin stimulation test. JSAP 32: 59-63, 1991

Stiles J, Polzin DJ,Bistner SI. The prevalence of retinopathy in cats with systemic hypertension and chronic renal failure or hyperthyroidism. JAAHA 30: 564-572, 1994

Syme HM,Elliott J. Prevalence and significance of proteinuria in cats with hyperthyroidism. BSAVA Congress Proceedings, pp533, 2003

Syme HM, Markwell PJ, Pfeiffer D, Elliott J (2006) Survival of cats with naturally occurring chronic renal failure is related to severity of proteinuria. JVet Int Med 20, 528-535

Tarttelin MF, Johnson LA, Cooke RR, et al. Serum free thyroxine levels respond inversely to changes in levels of dietary iodine in the domestic cat. N Zealand Vet J 40: 66-68, 1992

Thoday KL,Mooney CT. Historical, clinical and laboratory features of 126 hyperthyroid cats. Vet Rec 131: 257-264, 1992
Tomsa K, Glaus TM, Kacl GM, Pospischil A,Reusch CE. Thyrotropin-releasing hormone stimulation test to assess thyroid function in severely sick cats. JVIM 15: 89-93, 2001

Turrel JM, Feldman EC, Nelson RW, Cain GR. Thyroid carcinoma causing hyperthyroidism in cats: 14 cases (1981-1986). JAVMA 193(3): 359-364, 1988

Williams TL, Peak KP, Brodbelt D, Elliott J, Syme HM. Survival and the Development of Azotemia after Treatment of Hyperthyroid Cats. JVIM, 2010: 24: 863-869

Williams TL, Elliott J, Syme HM. Association of iatrogenic hypothyroidism with azotemia and reduced survival time in cats treated for hyperthyroidism. JVIM, 2010: 24: 1086-1092

Williams TL, Elliott J, Syme HM. Restoration of euthyroidism in medically treated hyperthyroid cats with iatrogenic hypothyroidism improves renal function. JVIM 2012; 26(3): 753-754 (Abstract EN-1).

