SECTION 1
PRESENTING COMPLAINTS

In this section, the common presenting complaints are listed alphabetically according to a stylised format.

- Each problem is defined, and the expected clinical signs listed, although not every case will show every sign.
- Causes for the problem are divided into 'common' and 'uncommon' to guide the reader, but are only the opinion of the authors, and may vary in different geographical locations.
- For each problem a logical diagnostic approach is suggested; any numbering indicates a suggested order for the investigations:
  - Clinical clues in the history.
  - Potential findings in the clinical examination.
  - Laboratory findings that aid the diagnosis.
  - Key results from imaging.
  - Special tests that may confirm the diagnosis

1.1 ABORTION

DEFINITION

The spontaneous expulsion of one or more fetuses before the end of full-term pregnancy, i.e. when the fetus is incapable of independent life.

RELATED CLINICAL SIGNS

- Abdominal pain
- Abnormal vulval discharge
- Fever
- Lethargy/depression
- Premature whelping is reported with live or dead pups or no live pups at term

COMMON CAUSES

Infectious

- Bacterial
  - *Brucella canis* in endemic countries; not endemic in UK
  - *Streptococcus* infection
- Viral: Canine herpesvirus-1 (CHV-1)

Non-infectious

- Congenital defects: various lethal defects
- Genetic causes: various lethal defects
- Maternal factors:
  - Illness
    - Diabetes mellitus (DM)
    - Eclampsia
    - Pregnancy toxaemia
  - Drugs
    - Corticosteroids
    - Griseofulvin
    - Itraconazole
    - Phenylephrine
    - Prolactin inhibitors
    - Prostaglandins
    - Progesterone-receptor blockers
  - Toxins: insecticides, plant toxins
  - Trauma
  - Hypoluteinization (low progesterone)
  - Advanced age
- Traumatic: dystocia

UNCOMMON CAUSES

Infectious

- Bacterial
  - *Escherichia coli*
  - *Campylobacter*
  - *Leptospira*
  - *Salmonella*
- Fungal
- Protozoal
  - *Leishmania*
  - *Neospora*
  - *Toxoplasma*
- Viral
  - Bluetongue virus
  - Canine adenovirus 1
  - Canine distemper virus
  - Canine parvovirus 1 (minute virus)

DIAGNOSTIC APPROACH

Clinical clues

Predisposition

- Advanced age
- Previous history of abortion
  - Assess for hypoluteinization by checking progesterone concentrations

History

- Abnormal vulval discharge
- Bitch whelps early with live or dead pups or no pups at term

Clinical examination

Visual inspection

- Often unremarkable

Notes on Canine Internal Medicine, Fourth Edition. Victoria L. Black, Kathryn F. Murphy, Jessie Rose Payne, and Edward J. Hall.

Physical examination
- Abdominal contractions and expulsion of fetus(es) in later pregnancy
- Vulval discharge: purulent, haemorrhagic, green, black, malodorous

Laboratory findings
Haematology
- May be normal
- HCT often low in pregnancy due to decreased plasma volume,
  e.g. 30–35% compared to 45–55%
- Mild mature neutrophilia common in pregnancy, may sometimes be more pronounced changes or bands

Serum biochemistry
- May be normal

Urinalysis
- May show evidence of inflammation with free catch or catheter samples

Imaging
Plain radiographs
- May show evidence of dystocia

Ultrasound
- May show evidence of fetal death

Special tests
- Examination of fetus post mortem
- Virus isolation/bacterial culture/PCR of fetus/placenta/vaginal secretions/milk

Tests of dam
- Serology ± PCR of dam for CHV-1, *B. canis*
- Serum progesterone to assess if sufficient to maintain pregnancy: should be > 2 ng/ml (6 nmol/l); if less than these values for > 48 hours suggests hypoluteinization but can be seen due to fetal death
- Thyroid hormone analysis: total T4/thyroid-stimulating hormone (TSH)

1.2 ALOPECIA

DEFINITION

Absence of hair from areas of skin that normally carry hairs, due either to a failure of production or to an increased loss of hair. Hypotrichosis refers to thinning of hair. Hair loss may be focal or diffuse, and symmetrical or non-symmetrical.

RELATED CLINICAL SIGNS

- Endocrinopathies are likely to cause concurrent systemic signs such as changes in drinking, eating, exercise tolerance and body weight
- Loss or absence of hair
- Self-traumatic lesions if pruritic skin disease

COMMON CAUSES

Primary follicular disease
Inherited abnormalities of follicular structure, ranging from absence of follicles that normally produce hair of a particular colour to complete absence of follicles, are uncommon except in specific breeds.

Secondary follicular disease
- Bacterial folliculitis/superficial pyoderma
- Demodectic mange
- Hyperadrenocorticism (HAC)
  - Iatrogenic
  - Pituitary- or adrenal-dependent
- Hypothyroidism
- Interdigital pyoderma
- *Malassezia* infection
- Seasonal flank alopecia (cyclic follicular dysplasia)

Self-trauma when pruritic
- Atopy
- Fleas and flea-allergic dermatitis
- Pyotraumatic dermatitis ('hot spot')
- Sarcoptic mange
- Secondary bacterial pyoderma

UNCOMMON CAUSES

Primary follicular disease

- Alopecia areata
- Alopecia mucinosa/follicular mucinosis: Shar pei
- (Auto)immune skin disease
  - Dermatomyositis
  - Erythema multiforme
  - Exfoliative cutaneous lupus erythematosus (ECLE)
  - Systemic lupus erythematosus
  - Pemphigus foliaceous
  - Sebaceous adenitis
- Catagen arrest: Weimaraners
- Colour mutant/dilution
- Congenital hypotrichosis/alopecia
- Follicular dysplasia
  - Black hair follicular dysplasia
  - Breed-specific follicular dysplasia
    - Loss of primary but retention of secondary hairs changing coat to reddish-brown: Siberian Husky and Alaskan Malamute
    - Flank and saddle region involved initially in red or black dogs: Dobermann
    - Flank and saddle hair loss: Airedale terrier, Boxer, English bulldog, Staffordshire terrier
    - Hair loss around face and over dorsum; cyclic initially but eventually permanent: Curly-coated Retriever, Irish Water spaniel, Portuguese Water dog
  - Medullary trichomalacia: German shepherd
  - Pili torti
  - Trichorrhexis nodosa
- Follicular lipidosis: Rottweiler
- Hairless breeds
- Pattern baldness
  - Caudal thigh: Greyhounds
  - Neck, trunk and thighs: Portuguese Water dog, American Water spaniel
  - Pinnal: Dachshund
  - Ventral and caudal: Boston Terrier, Chihuahua, Dachshund, Manchester Terrier, Whippet
- Pressure/traction alopecia: focal hair loss over bony prominences and sites of friction by collars, harnesses and coats
- Pseudopelade
- Subcorneal pustular dermatosis
- Trichoptilosis: Golden retriever

Secondary follicular disease

- Adrenal sex hormone imbalance/adrenal hyperplasia syndrome (alopecia X)
- Anagen defluvium
  - Cancer chemotherapy – greatest risk with curly- or wire-haired coats
  - Endocrinopathies
  - Infection
- Cicatricial (scar-related)
- Cold agglutinin disease
- Contact dermatitis
- Cutaneous vasculitis
- Dermatomyositis
- Dermatophytosis
- Epitheliotropic lymphoma including mycosis fungoides
- Follicular arrest
  - Post-clipping
  - Protein/calorie malnutrition
- Hepatocutaneous sydrome: more typically causes painful footpad hyperkeratosis
- Hyperoestrogenism
  - Adrenal gland disease
  - Excessive oestrogen administration (for urinary incontinence)
  - Excessive phytoestrogen ingestion (e.g. flaxseed)
  - Inadvertent exposure to human transdermal hormone replacement product
  - Ovarian remnant follicular cyst formation or malignant transformation
  - Testicular Sertoli cell tumour
- Hypo-oestrogenism
- Hyposomatotropism (pituitary dwarf)
- Hypotestosteronism
- Leishmaniosis
- Nutritional
  - Biotin deficiency
  - Vitamin A or E deficiency
- Radiation therapy
- Systemic lupus erythematosus (SLE)
- Telogen effluvium
  - Injection reaction
    - Epidural
    - Post-vaccinal
  - Post-partum
  - Pregnancy
  - "Stress"
- Thallium poisoning
- Vitamin A deficiency
- Zinc-responsive dermatosis

Self-mutilation
- Acral lick/flank sucking/neurodermatitis
- Cheyletiella
- Food allergy
- Lice
- *Trombicula* (Harvest mite)

DIAGNOSTIC APPROACH

1 Identification of infectious agents by sellotape strips, skin scrapes, hair plucks, bacterial and fungal cultures, and empirical treatment trials.
2 The presence or absence of pruritus narrows the differential diagnosis.
   If pruritic:
   - After ruling out infectious causes, trial therapy for bacterial pyoderma, fleas and possibly also for *Sarcoptes*, is acceptable
   - Intradermal skin testing is performed to identify atopic reactions
   - If all negative, an exclusion food trial is indicated

   If non-pruritic:
   - Consider endocrinopathy or breed-related problem
   - Skin biopsy is indicated if no cause is obvious

Clinical clues
- Pattern of hair loss and sites of self-mutilation can be informative
  q.v. Clinical examination
- Broken hairs on trichogram and ulcerated skin suggest self-trauma due to pruritus
- Concurrent systemic signs of polyuria/polydipsia (PU/PD), exercise intolerance, polyphagia or weight gain are suggestive of an endocrinopathy
- Non-symmetrical alopecia is suggestive of self-trauma or infection
- Presence of fleas or 'flea dirt' is diagnostic
- Repeated scratching and rubbing indicate pruritic causes
- Seasonality is suggestive of atopy, ectoparasites or seasonal flank alopecia
- Symmetrical alopecia is suggestive of an endocrinopathy
- Bilaterally symmetrical alopecia starting on the trunk is considered the hallmark of an endocrinopathy, but pruritic skin disease can also appear symmetrical.
- Evidence of pruritus:
  - Positive scratch reflex
  - Broken hairs, not hair loss

Predisposition
Breed predisposition may suggest primary follicular diseases:
- Canine hairless breeds: American Hairless Terrier, Argentine Pila, Chinese Crested, Mexican Hairless (Xoloitzcuintli), Peruvian Inca Orchid
- Colour-mutant alopecia: blue/fawn/red Dobermann, blue Chow Chow, blue Dachshund, blue Great Dane, blue Whippet, fawn Irish setter
- Follicular dysplasia: Curly-coated Retriever, Irish Water Spaniel, Portuguese Water dog
- Dermatomyositis: Collies

History
- Colour-mutant alopecia develops in young adults
- Congenital or hereditary hypotrichosis is usually evident from an early age
- Slow onset and bilateral truncal alopecia is suggestive of endocrinopathy
- Testicular mass, pendulous prepuce and attractiveness to other male dogs is suggestive of functional Sertoli cell tumour
- Other clinical signs (e.g. PU/PD, weight change) suggest a possible endocrinopathy

Clinical examination
Visual inspection
- Broken hairs if pruritic, otherwise hairs are absent
- Lesions secondary to self-trauma:
  erythema, excoriation, lichenification, hyperpigmentation
- Presence of fleas or 'flea dirt'
- Pustules, erythema, scaling in pyoderma

Physical examination
- Thickened skin in hypothyroidism
- Thinned skin in HAC
- Distribution of self-mutilation
  - Dorso-lumbar with flea-allergic dermatitis
  - Ear margins and elbows with sarcoptic mange

- Face, feet and ventrum with atopy
- Feet and ventrum with contact allergy
- Face, ears and feet with food allergy
- Face, ears, feet or multifocal with demodecosis
- Face, feet, mucocutaneous junctions with autoimmune skin disease

- Distribution of hair loss
  - Focal
    - Alopecia areata
    - Cicatricial
    - Demodecosis
    - Dermatophytosis
    - Injection reaction
    - Pattern baldness
    - Superficial pyoderma/bacterial folliculitis
  - Multifocal or diffuse but patchy
    - Colour dilution
    - Demodecosis
    - Dermatomyositis
    - Dermatophytosis
    - Epitheliotropic lymphosarcoma
    - Follicular dysplasia
    - Superficial pyoderma/bacterial folliculitis
  - Symmetrical, generalised, diffuse
    - Demodecosis
    - Dermatophytosis
    - Endocrinopathies
    - Superficial pyoderma/bacterial folliculitis
    - Telogen effluvium
    - Hair loss from the caudal trunk and thighs
    - Flea allergic dermatosis
    - Follicular dysplasia
    - Hyperoestrogenism
    - Pattern baldness of Greyhounds
  - Hair loss from the pinnae
    - Atopy
    - Cold agglutinin disease
    - Dermatophytosis
    - Demodectic mange
    - Otitis externa
    - Pemphigus erythematosus
    - Pemphigus foliaceous
    - Pinnal pattern baldness
    - Sarcoptic mange
    - Subcorneal pustular dermatosis
  - Hair loss from the feet
    - Atopy
    - Contact dermatitis
    - Demodectic mange
    - Pemphigus diseases
    - Interdigital pyoderma
    - Thallium poisoning

Laboratory findings

Haematology and serum biochemistry

- Unremarkable unless underlying endocrinopathy, e.g. hypercholesterolaemia in hypothyroidism, increased ALP activity in HAC

Dermatological investigations

- Bacterial and fungal cultures
- Hair plucks – *Demodex*, ringworm
- Hypercholesterolaemia and increased ALP in HAC
- Sellotape strips cytology: *Malassezia*
- Skin scrapes: *Sarcoptes*, *Demodex*

Imaging

- Usually unnecessary and unremarkable unless systemic signs (e.g. endocrinopathy)

Special tests

- Dynamic cortisol testing for HAC
- Exclusion diet trial
- Intradermal skin tests
- Sarcoptic mange antibody
- Therapeutic trial for sarcoptic mange
- Skin biopsy
- Thyroid function tests

1.3 ALTERED BEHAVIOUR

DEFINITION

A change in response to or interaction with the environment. Abnormal behaviour can be observed as a spectrum of consciousness (*q.v.* section 1.4) ranging from reduced response to even noxious stimuli to manic behaviour and hyperactivity. Animals may also demonstrate changes in sleep-wake cycles, social relationships, repetitive activities, or spatial disorientation.

Behavioural disorders and medical disorders causing behaviour change present a significant challenge due to the considerable overlap in presentations. Behavioural disorders can be considered *after* medical disorders have been excluded and are more common in younger animals presenting with increased reactivity or vigilance.

RELATED CLINICAL SIGNS

- Aggression
- Dullness and reduced responsiveness, *q.v.* section 1.4
- Excessive grooming
- Hyperactivity (excessive pacing, circling, altered sleep patterns)
- Inappropriate elimination
- Repetitive behaviours (fly catching, overgrooming, tail chasing, flank sucking)

COMMON CAUSES

Pain
- Ear disease
- Neuropathic
- Orthopaedic

Intracranial disorders
- Cognitive dysfunction
- Hydrocephalus
- Idiopathic epilepsy
- Inflammatory (meningoencephalitis of unknown origin)
- Neoplasia

Metabolic disorders
- Hepatic encephalopathy (portosystemic shunts, chronic hepatitis, acute hepatic failure)
- Hyperadrenocorticism
- Hypertension
- Hypothyroidism
- Toxins

Intestinal disorders
- Idiopathic chronic inflammatory enteropathy (CIE)/inflammatory bowel disease (IBD)
- Neoplasia

Urinary tract disorders
- Atopy
- Dermatological disorders
- Ectopic ureters
- Parasitic infection
- Urinary tract infection
- Urolithiasis

UNCOMMON CAUSES

Metabolic disorders
- Hyperthyroidism
- Phaeochromocytoma

Intracranial disorders
- Infectious disease, e.g. *Cryptococcus*, distemper, *Toxoplasma*, *Neospora*
- Storage disease

Dermatological disorders
- Bacterial hypersensitivity

Ocular disorders causing pain or impairing vision
- Cataracts
- Progressive retinal atrophy
- Uveitis

DIAGNOSTIC APPROACH

Thorough screening for medical disorders should be considered in young animals, or in patients with acute onset change. Environment, social interactions, exercise routine, and training history may influence likelihood of behavioural disorders.

Clinical clues
Predisposition
- Cognitive dysfunction and orthopaedic pain: older dogs
- Portosystemic shunt (PSS): young dogs and some breeds are predisposed (e.g. Yorkshire terriers, Miniature schnauzers, Pugs, Irish Wolfhounds)

- Some repetitive behaviours have breed associations:
  - Flank sucking: Dobermanns
  - Fly catching: CKCS
  - Hind-end checking: Miniature Schnauzers
  - Self-mutilation: Springer spaniels
  - Tail chasing: German shepherds
- Neuropathies can be heritable disorders, e.g. sensory neuropathy in Border collies

History
- Assess for risk of toxin exposure if acute onset change
- Circumstance of occurrence may increase suspicion of
  - pain disorders after rest or after exercise
  - metabolic disease, e.g. after eating may increase suspicion of a PSS

Clinical examination

Visual inspection
- Behaviour during consultation, or during circumstance of typical occurrence: video recordings of abnormal behaviour are encouraged
- Haircoat changes are suggestive of endocrine disorders
- In young dogs, examine for
  - disproportionate stunting, e.g. congenital hypothyroidism
  - domed cranium ± open fontanelle in hydrocephalus
  - stature, e.g. small size in EPI, PSS

Physical examination

Thorough examination for source of pain, with specific attention to musculoskeletal system
- Neurological and ophthalmic examination
- Rectal examination in cases of inappropriate elimination to assess for rectal, urethral, and prostatic disorders

Laboratory findings

Haematology
- May reveal microcytosis ± hypochromasia in PSS
- Mild, normocytic, normochromic, poorly-regenerative anaemia in hypothyroidism

Serum biochemistry
- Evidence of hepatic dysfunction (e.g. decreased albumin, cholesterol, urea, glucose, increased ammonia), including increased bile acids; increased liver enzyme activities not typical in PSS but detected in other hepatopathies
- Enteropathy may present with panhypoproteinaemia (hypoalbuminaemia and hypoglobulinaemia) ± low serum cholesterol
- Hypercholesterolaemia in hypothyroidism

Imaging

Plain radiographs
- In cases with excessive grooming may detect underlying musculoskeletal disease including degenerative joint disease or neoplasia

Ultrasound
- Abnormal vessel ± renomegaly may be observed in PSS
- Adrenal gland size in cases suspicious for hyperadrenocorticism
- Intestinal wall layering for enteropathy
- Liver size and echotexture when suspicious of hepatic dysfunction

Special tests

- Cobalamin and folate in cases with a suspicion of enteropathy, particularly relevant to cases of fly catching, inappropriate elimination, or pica
- Computed tomography (CT) angiogram to assess for PSS if not detected on ultrasound examination
- Low-dose dexamethasone suppression LDDS test (± ACTH stimulation test) to assess for hyperadrenocorticism
- Liver biopsy for hepatopathy
- Total thyroxine and TSH to assess for hypothyroidism
- Treatment trials
  - Non-steroidal anti-inflammatory drug (NSAID) or other analgesic trial for suspected pain
  - Antiepileptic therapy where partial seizures are a consideration
  - Exclusion diet trial where atopy or enteropathy is suspected

1.4 ALTERED CONSCIOUSNESS

DEFINITION

Consciousness is the state of arousal and ability to respond to the external environment. Diminished consciousness can be graded based upon lack of responsiveness to increasingly stimulatory external events. State of consciousness is an important component of the modified Glasgow Coma Scale (MGCS), useful prognostically and for monitoring purposes.

Consciousness is primarily determined by the ascending reticular activating system, an important set of neurons found within the brainstem. Disorders of the brainstem can result in dysfunction of these neurons, with a consequent reduced state of arousal of the individual. Diffuse forebrain dysfunction (cerebral disorders) may also result in diminished consciousness in some circumstances (*q.v.* modified Glasgow Coma Scale in section 5.8).

RELATED CLINICAL SIGNS

Grades of dysfunction

Depressed or obtunded

- Reduced responsiveness to external visual or auditory stimuli
  - Associated with brainstem, diffuse forebrain disorders, or systemic disease
  - Can have intermittent periods of more normal mentation

Stuporous

- Semi-comatose, responsive only to repeated noxious stimuli
  - Typically associated with brainstem disorders, including brain herniation

Comatose

- Unresponsive to repeated noxious stimuli
  - Typically associated with brainstem disorders, including brain herniation

Delirious

- Hyperactive with excessive or abnormal responses to external stimuli
  - Typically associated with forebrain disease

COMMON CAUSES

Congenital

- Hydrocephalus
- PSS

Inflammatory

- Meningoencephalitis of unknown origin (MUO)

Infectious

- Bacterial empyema, e.g. extension of otitis media
- Lungworm (*Angiostrongylus vasorum*)

Iatrogenic

- Head trauma
- Toxins or drugs, e.g. sedation or anaesthesia, hallucinogens

Metabolic

- Electrolyte disturbances, especially changes in osmolarity due to too-rapid correction of sodium disturbances
- Hepatic encephalopathy
- Hypoglycaemia
- Severe systemic disease, e.g. sepsis, congestive heart failure, etc.

Neoplastic

- Primary central nervous system (CNS) tumours: meningioma, glioma, lymphoma

Vascular

- Thromboembolic or haemorrhagic stroke

UNCOMMON CAUSES

Congenital

- Storage disease: signs may develop as dog ages

Infectious

- Distemper
- Protozoal: *Toxoplasma*, *Neospora*
- Rabies

Iatrogenic
- Complication of cerebrospinal fluid (CSF) sampling in dogs with increased intracranial pressure

Vascular
- Hyperviscosity, e.g. hyperglobulinaemia, hyperlipidaemia, primary erythrocytosis

DIAGNOSTIC APPROACH

Clinical clues
Predisposition
- Congenital disorders maybe suspected in young dogs, especially those with conformational changes
  - Disproportionate stunting, e.g. congenital hypothyroidism (*q.v.* section 1.45)
  - Domed cranium ± open fontanelle in hydrocephalus
  - Small stature in PSS
- Small purebred middle-aged dogs are predisposed to meningoencephalitis of unknown origin (e.g. Maltese terrier, Pug, West Highland White terrier [WHWT])

History
- Anti-parasitic prophylaxis may indicate lesser risk of *Angiostrongylus* infection
- Lifestyle may increase index of suspicion for infectious disease, e.g. raw-fed may increase suspicion of toxoplasmosis or exposure to toxins or risk of trauma

Clinical examination
Visual inspection
- Interaction with the environment, increasing external stimuli (to include visual and auditory tests)

Physical examination
- Assess for signs of systemic disease in cases of obtundation; mentation changes may be secondary to this, or, in multisystemic disease, may detect abnormalities on examination
- Heart rate and blood pressure (BP): a patient with bradycardia (pulse rate < 60 beats/minute) and hypertension (systolic BP > 160 mmHg) is at risk of increased intracranial pressure (Cushing's reflex)

Neurological examination
- Cranial nerve assessment is particularly important in stuporous and comatose animals given the high suspicion of a brainstem disorder
- Focus on assessment of forebrain (mentation, proprioception, menace response and response to nasal stimulation) and brainstem (proprioception and cranial nerves) functions
- MUO typically presents with a multifocal lesion localisation, whereas metabolic disease typically results in symmetrical deficits localising to the forebrain

Ophthalmic examination
- Papilloedema (optic disc swelling) is suggestive of increased intracranial pressure

Laboratory findings
Haematology
- Assess for signs of severe systemic disease (e.g. marked leukocyte changes supportive of an inflammatory focus)
- Increased PCV in primary erythrocytosis (typically > 68%)
- May reveal microcytosis ± hypochromasia in PSS

Serum biochemistry
- Evidence of hepatic dysfunction (low albumin, cholesterol, urea, glucose, increased ammonia) and increased bile acids
- Increased liver enzyme activities are not typical in PSS but are detected in other hepatopathies
- Hypoglycaemia
- Sodium concentrations: review previous results in hospitalised animals to assess for changes in sodium indicating too-rapid correction of marked hypernatraemia

Imaging
NB: Cross-sectional imaging (MRI) is often indicated for primary CNS diseases

Plain radiographs
- Screening in cases with a high suspicion of multisystemic disorders (e.g. assessing for neoplasia)

- Skull radiographs may be useful in cases of head trauma, but rarely in hydrocephalus

Ultrasound
- Assess for liver size and echotexture when suspicious of hepatic dysfunction. Renomegaly may also be observed in PSS.
- Screening in cases with a high suspicion of multisystemic disorders (e.g. assessing for neoplasia)

Special tests
- Assessment of haemostasis (platelet count, buccal mucosal bleeding time, prothrombin time [PT] and activated partial thromboplastin time [aPTT], thromboelastography) to assess for risk of hypo- or hypercoagulability where vascular disorder is suspected
- Baermann's technique, AngioDetect® or faecal smear to identify *Angiostrongylus* infection
- CSF analysis – cytology and biochemistry, PCR for *Toxoplasma* and *Neospora*
- EEG (electroencephalography) or BAER (brainstem auditory evoked response)
- MRI
- Response to mannitol or hypertonic saline in patients with suspected increased intracranial pressure

1.5 ANOREXIA/HYPOREXIA/INAPPETENCE

DEFINITION

A decline in food intake through loss of appetite whilst still physically able to eat
- Anorexia indicates a complete lack of food intake
- Dysorexia is a diminished, disordered or unnatural appetite (*q.v.* section 1.36)
- Hyporexia is a significant reduction in food intake
- Inappetence implies a decline in food intake through either a loss of appetite or a selective appetite
- Pseudo-anorexia is a condition where a dog wants to eat but is unwilling or physically unable to do so because of orthopaedic or neuromuscular dysfunction or pain associated with eating

RELATED CLINICAL SIGNS

- Pseudo-anorexia: the dog may try to eat but stop quickly because of pain or difficulty swallowing
- True anorexia: the dog will not attempt to eat, or may turn away as if nauseated

COMMON CAUSES

Anorexia/inappetence
- Diabetic ketoacidosis
- Drugs: many
- Fever
- Gastrointestinal (GI) disease
- Hepatic disease
- Hypercalcaemia
- Infectious/inflammatory diseases
- Neoplasia
- Pain
- Pancreatitis
- Uraemia

Pseudoanorexia
- Dental disease
  - Abscessed tooth root
  - Fractured tooth
- Oesophagitis
- Retrobulbar abscess/periorbital cellulitis
- Unpalatable diet

UNCOMMON CAUSES

True anorexia
- Anosmia, *q.v.* section 1.6
- Cardiac failure
- Drugs

- Hypoadrenocorticism
- Sialoadenitis
- Severe respiratory disease
  - Advanced neoplasia (primary or metastatic)
  - Diaphragmatic rupture
  - Pneumonia
  - Pleural effusion
- Neurological disease
  - Cerebral oedema
  - Hydrocephalus
  - Hypothalamic disease
    - Congenital cystic lesions
    - Infection/inflammation
    - Neoplasia
    - Trauma
- Psychological (food aversion)
  - Anxiety
  - Hospitalisation
  - Loss of companion
  - Social stress

Pseudo-anorexia

- Blindness
- Cranial nerve deficits
- Craniomandibular osteopathy
- Jaw fracture
- Oropharyngeal inflammation/ulceration
- Oropharyngeal neoplasia
- Osteomyelitis of jaw
- Temporal myositis
- Temporomandibular joint (TMJ) disease
  - Jaw locking
  - TMJ dysplasia or dislocation
- Tetanus
- Trigeminal neuritis/neuropraxia

DIAGNOSTIC APPROACH

Anorexia/hyporexia are common, non-specific findings with many potential causes.

1 Rule out dietary causes, drug administration, stressors.
2 Determine from history and direct observation whether the dog wants to eat or not and whether it is physically able to.
3 Pseudo-anorexia will be caused by a cranial nerve deficit or a lesion in the mouth, pharynx or oesophagus.
4 Examination of the mouth may require sedation or anaesthesia, and imaging of the skull.
5 Complete physical examination and relevant lab tests and imaging for causes of true anorexia.

Clinical clues

Predisposition

- Craniomandibular osteopathy in WHWT

History

- Coughing or other respiratory signs
- Does dog attempt to eat and then drop or regurgitate food, or does it avoid food?
- Nausea (e.g. signs of lip-smacking, retching, etc.) in metabolic or GI disease
- PU/PD in liver and renal disease
- Seizures if intracranial disease
- Stressful event leading to food aversion
- Traumatic event if jaw fracture
- Vomiting and/or diarrhoea with GI disease and hypoadrenocorticism
- Weight loss in excess of what is expected from not eating suggests increased energy usage, e.g. metabolic disease, neoplasia, etc.

Clinical examination

Visual inspection

- Depression
- Drooling saliva
- Dyspnoea if cardiac failure
- Open mouth if trigeminal neuropraxia or jaw locking
- Unilateral exophthalmos if retrobulbar abscess

Physical examination

- Abdominal masses palpable
- Abnormal lung and heart sounds
- Halitosis if oral disease
- Pain or resistance on opening mouth if myositis, foreign body (FB) or retrobulbar abscess
- *Risus sardonicus*, erect ears, muscle stiffness and spasms in tetanus

- Temporal muscle atrophy with myositis
- Trismus with myositis or TMJ disorder

Laboratory findings

Variable, often within normal limits (WNL)

Haematology

- Inflammatory leukogram with infection or inflammatory diseases

Biochemistry

- Azotaemia
  - Plus isosthenuria in renal failure
  - Pre-renal (hypersthenuria) in dehydration due to hypodipsia
- Hypercalcaemia most frequently associated with malignancy, especially lymphosarcoma and anal sac adenocarcinoma; primary hyperparathyroidism is rare
- Hyponatraemia / hyperkalaemia in hypoadrenocorticism
- Increased liver enzyme activities, bile acids ± bilirubin in hepatic diseases

Special tests

- Complete neurological examination
- Multiple depending on suspected condition

1.6 ANOSMIA

DEFINITION

The loss of the sense of smell. The most common reason for presentation is when an owner of a hunting dog perceives their dog has lost the ability to find game, or a detection dog is unable to find the substance it has been trained to seek. Even then, other signs of nasal disease are more likely to prompt presentation.

RELATED CLINICAL SIGNS

- Epistaxis, nasal discharge, sneezing
- Facial deformity
- Temporal muscle atrophy
- Xerostomia

COMMON CAUSES

- Diseases of the nasal cavity
  - Lymphoplasmacytic rhinitis
  - Neoplasia
  - Sino-nasal aspergillosis

UNCOMMON CAUSES

- Distemper
- Forebrain disease
- Sensory decline with age
- Sensory overload (temporary)
- Skull fracture(s)
- Trigeminal neuritis
- Traumatic/destructive lesions of the cribriform plate
  - Fungal infection
  - Neoplasia

DIAGNOSTIC APPROACH

It is difficult to assess olfaction but cranial nerve assessment, in particular nasal sensation, should be undertaken unless there is obvious nasal disease.

Clinical clues

Predisposition

- Sino-nasal aspergillosis in dolichocephalic dogs

History

- Sneezing, nasal discharge and epistaxis all indicate nasal disease
- Known traumatic event
- Neurological signs: depression, obtundation, seizures, visual deficits

Clinical examination

Visual inspection

- Epistaxis
- Facial deformity
- Nasal discharge

Physical examination
- Deformity of nasal bones
- Lack of air movement through nostrils
- Neurological deficits

Laboratory findings

Haematology
- Typically unremarkable unless significant blood loss due to epistaxis causing regenerative anaemia

Serum biochemistry
- Usually unremarkable unless significant blood loss due to epistaxis causing hypoproteinaemia

Urinalysis
- Unremarkable

Imaging
- Skull radiographs to look for fractures, turbinate destruction or expansile lesions
- CT is preferred as cribriform plate integrity can be assessed

Special tests
- *Aspergillus* serology not very sensitive but specific
- Rhinoscopy with nasal biopsy

1.7 ANURIA/OLIGURIA

DEFINITION

Anuria is defined as a failure of the kidneys to produce urine; oliguria refers to cases with inadequate urine production. IRIS guidelines for acute kidney injury (AKI) define this in the context of fluid volume responsiveness over a 6-hour period; with anuria defined as no urine produced and oliguria < 1 ml/kg/hour after intravenous fluid therapy to correct dehydration and hypovolaemia. These definitions are useful as part of decision making on whether renal replacement therapy is required.

Failure to produce normal volumes of urine may also occur due to pre-renal causes (e.g. severe dehydration) and post-renal causes (e.g. bilateral ureteric obstruction, urethral obstruction, ruptured bladder). These are important exclusions before determining a renal cause for lack of urine production.

RELATED CLINICAL SIGNS

- Anuria is normally not the primary concern on presentation in patients; typically systemic causes of lack of glomerular filtration are the primary signs, including vomiting, anorexia, lethargy, or collapse related to potassium or central nervous system disturbances.
- Failure to produce urine due to post-renal causes is most commonly accompanied by stranguria and dysuria (*q.v.* section 1.19) except for ureteric causes, where abdominal pain and signs related to anuria may be the primary sign.

COMMON CAUSES

Infectious causes
- Leptospirosis
- Pyelonephritis

Toxins
- Ethylene glycol
- Grapes and raisins

Drugs
- NSAIDs

Vascular
- Severe hypotension or renal ischaemia, e.g. sepsis, multiple organ dysfunction, severe cardiac failure

Miscellaneous
- Cutaneous and renal glomerulovasculopathy (CRGV, 'Alabama rot')
- Hypercalcaemia

UNCOMMON CAUSES

Infectious
- *Babesia*
- *Leishmania*

Vascular
- Renal infarction

Miscellaneous
- Hyperviscosity, e.g. primary erythrocytosis, hyperglobulinaemia
- Myoglobinuria or haemoglobinuria

Drugs
- Radiographic contrast agents
- ACE inhibitors

Neoplasia
- Renal lymphoma likely bilateral if causing reduced glomerular filtration rate

DIAGNOSTIC APPROACH

1 Anuria or oliguria is usually detected in patients following identification of azotaemia on blood tests, or in systemically unwell patients where urine output is being monitored.
2 Exclude pre-renal causes by assessing hydration status, urinary specific gravity (if urine is available), and measuring urinary bladder size and body weight.
3 Response to intravenous fluid therapy (aim to correct hydration over a 12-hour period) can be monitored. Even with severe dehydration anuria is not anticipated.
4 Assess for post-renal causes by establishing history, passing a urinary catheter and renal imaging to assess bilateral ureteric obstruction (uncommon in dogs). Placement of a urinary catheter is also useful to enable ongoing urinary output monitoring.
5 Response to furosemide (furosemide stress test) following rehydration can be utilised to determine if functional renal tissue is intact. If the patient does not produce urine in spite of furosemide administration, prognosis for renal recovery without renal replacement therapy is considered guarded.

Clinical clues
Predisposition
- Patients that are severely systemically unwell for any reason (e.g. sepsis, pancreatitis) may be vulnerable to anuric AKI
- Renal amyloidosis: Shar pei

History
- Assess drinking patterns prior to presentation (polyuria and polydipsia may be present in patients with hypercalcaemia-induced renal injury, or acute on chronic kidney disease)
- Exposure to toxins or drugs (especially NSAIDs) inducing renal injury
- Risk factors for AKI
  - CRGV: woodland environment, seasonality
  - Infectious disease: vaccination status, seasonality, wet environment, and lifestyle for risk of leptospirosis

Clinical examination
Visual inspection
- Assess for hydration status (eye position)
- Assess for evidence of concurrent systemic disease (e.g. jaundice in leptospirosis or pancreatitis)

Physical examination
- Abdominal pain or pyrexia: may increase suspicion of pyelonephritis
- Hydration (skin tent, mucous membrane moisture, body weight if recent measurements are available) and volume status (heart rate, mucous membrane colour)
- Skin wounds in patients with suspicion of CGRV

Laboratory findings
Haematology
- Leukocytosis with neutrophilia and monocytosis and left shift (band neutrophils and toxic change) may increase suspicion of infectious causes
- Moderate-marked poorly regenerative anaemia implies a more chronic process (acute on chronic disease) in the absence of comorbidities

Serum biochemistry
- Azotaemia is detected in most oligo/anuric patients, so the degree of azotaemia is not prognostic in AKI. However, failure to produce urine in response to fluid therapy is
- Potassium should be monitored closely in anuric patients (q2 hours); intervention may be indicated
- Urinalysis is important in discriminating between pre-renal and renal azotaemia; sediment analysis may detect calcium oxalate monohydrate (ethylene glycol), and may detect evidence of tubular injury (glucosuria esp. in leptospirosis, proteinuria) or increase the suspicion of pyelonephritis (active sediment examination)
- Urine cytology submitted in an EDTA tube may be useful in cases with a high suspicion of pyelonephritis
- Venous blood gas in ethylene glycol toxicity will display metabolic acidosis with a high anion gap and hypocalcaemia

Imaging

Plain radiographs
- Excluding radio-opaque uroliths as a cause of bilateral ureteric obstruction (uncommon in dogs cf. cats)
- In patients with hypercalcaemia to screen for neoplastic causes

Ultrasound
- Assess renal perfusion and architecture (small and irregular associated with more chronic disease, pyelonephritis may be detected by renal architectural change and renal pelvis dilation (although not 100% sensitive)), AKI cases may display hyperechoic renal cortices and perirenal retroperitoneal fluid
- In patients with hypercalcaemia to screen for neoplastic causes

Special tests
- Infectious disease testing, e.g. microscopic agglutination test (MAT) and blood or urine PCR for leptospirosis
- Pyelocentesis for cytology and culture in cases with a high suspicion of pyelonephritis
- Skin biopsy for histology may increase suspicion of CRGV
- Toxin screen including ethylene glycol on urine or blood

1.8 ATAXIA

DEFINITION

Ataxia is defined as uncoordinated voluntary movement. This occurs when there is a dysfunction of the elements of the nervous system that are responsible for coordination. Disorders that can present with ataxia include cerebellar dysfunction, vestibular system dysfunction, and loss of proprioception (sensory).

RELATED CLINICAL SIGNS

Cerebellar ataxia
- Can also present with vestibular dysfunction as this involves the cerebellum (flocculonodular lobe)
- Hypermetria
- Intention tremor
- Wide-based stance

Vestibular ataxia
- Bilateral vestibular ataxia presents without a head tilt and falling to both sides, wide excursions of the head, and an uncoordinated gait
- Falling/leaning to one side
- Head tilt
- Nystagmus

Proprioceptive (sensory) ataxia
- Abnormal placement of limbs, e.g. crossing over legs when walking, or pacing (forelimb and hindlimb strides at the same time), knuckling
- May stumble, e.g. stepping over objects

COMMON CAUSES

Cerebellar ataxia
- Infectious – protozoal (*Toxoplasma*, *Neospora*), *Angiostrongylus vasorum*
- Inflammatory – meningoencephalitis of unknown origin
- Neoplasia
- Vascular – haemorrhagic or thromboembolic stroke

Vestibular ataxia
Peripheral vestibular
- Cranial polyneuropathy (possible link with hypothyroidism)
- Idiopathic (old dog vestibular)
- Neoplasia
- Otitis media

Central vestibular (brainstem or cerebellar)
- Cerebellar disorders as above
- Inflammatory – meningoencephalitis of unknown origin
- Neoplasia
- Trauma

Proprioceptive (sensory) ataxia
- Degenerative disease, e.g. canine degenerative myelopathy (CDM)
- Drug-induced
- Fibrocartilaginous embolism (FCE)
- Intervertebral disc disease
- Neoplasia

UNCOMMON CAUSES

Cerebellar ataxia
- Cerebellar abiotrophy
- Storage disease
- Trauma

Vestibular ataxia
- Central vestibular (brainstem or cerebellar)
- Cerebellar disorders as above
- Drug-induced, e.g. metronidazole toxicity
- Hyperviscosity: erythrocytosis, hyperglobulinaemia including multiple myeloma
- Peripheral vestibular
- Storage disease
- Trauma

Proprioceptive (sensory) ataxia
- Inflammatory – meningomyelitis

DIAGNOSTIC APPROACH

Clinical clues
Predisposition
- Canine degenerative myelopathy is present in numerous breeds; German shepherd dogs (GSDs) are predisposed
- Cerebellar abiotrophy has been described in more than 40 breeds of dog; there are neonatal (e.g. beagles), juvenile (e.g. Airedale terrier) and adult onset (Bernese mountain dog) disorders
- Greyhounds are predisposed to thromboembolic cerebellar events (ischaemic stroke)

History
- Duration and progression of signs may aid in index of suspicion (acute onset improving increases suspicion of vascular or drug-induced, gradual onset progression may increase suspicion of degenerative or neoplastic disease, acute onset rapid progression may increase suspicion of inflammatory or infectious disease)

Clinical examination
Visual inspection
- Mentation (*q.v.* sections 1.3, 1.4) is expected to be impaired with central brainstem vestibular disorders

Physical examination
- Thorough examination to assess for multisystemic disease

Neurological examination
- Cerebellar: ipsilateral loss of proprioception and menace response, contralateral head tilt and direction of fast phase of nystagmus (paradoxical vestibular)
- Proprioceptive ataxia: abnormal conscious and unconscious proprioception
- Vestibular: head tilt and nystagmus

- In brainstem disorders consciousness may be reduced and other cranial nerves may be affected, e.g facial nerve (blink reflex) and glossopharyngeal and vagal nerves (gag reflex)
- Peripheral disorders may detect concurrent facial nerve abnormalities and Horner's syndrome

Ophthalmic examination
- May be useful to assess for infiltrative central nervous system disease, e.g. may detect papilloedema

Laboratory findings

Haematology
- Uncommon to detect changes except thrombocytopenia in haemorrhagic stroke

Serum biochemistry
- May detect causes of increased risk of thromboembolism: hepatic disease, hyperadrenocorticism, protein-losing enteropathy (PLE), protein-losing nephropathy (PLN)

Urinalysis
- Assess for PLN in dogs with thromboembolic stroke

Imaging

Plain radiographs
- Screen for disseminated neoplasia in cases with a high suspicion

Ultrasound
- Screen for disseminated neoplasia in cases with a high suspicion

Special tests

- Coagulation parameters (PT and aPTT) to assess for haemorrhagic stroke
- CSF tap to assess for cytology and biochemistry (inflammatory) and infectious disease (e.g. *Toxoplasma* and *Neospora* PCR)
- Infectious disease testing
  - *Toxoplasma* and *Neospora* serology
  - Baermann technique or AngioDetect® for *Angiostrongylus*
- Otoscopy and myringotomy in cases with suspicion of otitis media
- MRI scan to assess for central nervous system causes of ataxia

1.9 BLEEDING

DEFINITION

Bleeding may be observed due to excessive bruising or haemorrhage following no or minor trauma (e.g. blood sampling, minor surgery, teething or exercise) or due to disorders of the bleeding tissue. Bleeding diathesis means a tendency to bleed or bruise easily.

Haematoma is red or discoloured skin that does not blanch under pressure and occurs due to the presence of blood under the skin. These lesions can be defined by size; petechiae refers to haematomas < 3 mm and ecchymosis refers to haematomas > 1 cm. Purpura is a bleed 3 mm to 1 cm diameter but is not a term commonly used in veterinary medicine.

RELATED CLINICAL SIGNS

As with any cause of bleeding, causes can be divided into disorders of the organ affected or related to a disorder of haemostasis (systemic disease).

COMMON CAUSES

Disorders of affected organ

- Neoplasia, in particular haemangiosarcoma in any location or leiomyosarcoma in gastrointestinal haemorrhage
- Trauma
- Vasculitis, e.g. adder bite evenomation

Systemic disease
- *Angiostrongylus vasorum* infection causes a mixed haemostatic disorder: pulmonary or CNS bleeding may occur due to coagulopathy or migration of larvae
- Primary haemostatic disorders
  - Thrombocytopenia: immune-mediated thrombocytopenia (IMTP), consumptive (disseminated intravascular coagulation, DIC)
- Secondary haemostatic disorders
  - Vitamin K deficiency due to anticoagulant rodenticide toxicity
- Tertiary haemostatic disorders
  - Hyperfibrinolysis following surgery or major trauma in sighthounds

UNCOMMON CAUSES

Disorders of affected organ
- FB e.g. nasal FB for epistaxis
- Infection, e.g. *Angiostrongylus vasorum* for pulmonary haemorrhage

Systemic disease
- Primary haemostatic disorders
  - Thrombocytopathia (hyperviscosity due to hyperglobulinaemia (e.g. neoplasia, *Leishmania*), inherited disorders)
  - Thrombocytopenia (*Ehrlichia*, bone marrow disease)
  - von Willebrand factor deficiency
- Secondary haemostatic disorders
  - Liver dysfunction, e.g. chronic hepatitis
  - Vitamin K deficiency (cholestasis, congenital)
- Vasculitis, e.g. sepsis, anaphylaxis

DIAGNOSTIC APPROACH

Clinical clues
Predisposition
- Breed predispositions to IMTP and immune-mediated haemolytic anaemia (IMHA), e.g. cocker spaniels
- Sighthounds appear to have a disorder of hyperfibrinolysis, observed as excessive haemorrhage after trauma or major surgery

History
- Concurrent haemorrhage elsewhere to increase suspicion of systemic disorder
- Risk factors of trauma, adder bite envenomation or FB
- Travel history increases index of suspicion of vector-borne diseases including *Dirofilaria*, *Ehrlichia* and *Leishmania* infections

Clinical examination
Visual inspection
- Observe for demeanour, and evidence of haemorrhage elsewhere

Physical examination
- Thorough examination for evidence of haemorrhage elsewhere: skin, especially of the ventral abdomen, and gums, rectal examination for melaena

Laboratory findings
Haematology
- Assess platelet count
- Coagulation times (PT and aPTT) to assess for secondary haemostatic disorders
- Eosinophilia may be present in parasitic disease

Serum biochemistry
- Useful to screen for systemic disease

Imaging
Plain radiographs and ultrasound
- Evidence of organ changes, e.g. mass lesions such as splenic mass in haemoabdomen, and for multisystemic disease

Special tests
- CT with contrast to screen for infection, neoplasia when appropriate
- Haemostatic disorders
  - Baermann technique or Angiodetect® for *Angiostrongylus vasorum*
  - Buccal mucosal bleeding time to assess for thrombocytopathia
  - Testing for *Leishmania* and *Ehrlichia* infection as appropriate

- There is currently no available test to detect hyperfibrinolysis; as a result sighthound breeds are often treated empirically ahead of surgery or following haemorrhage (most commonly using the anti-fibrinolytic tranexamic acid)
- Thromboelastography and rotational thromboelastometry to assess clotting *in vitro*
- Toxicology to detect rodenticide
- von Willebrand factor antigen levels

1.10 BLINDNESS

DEFINITION

Blindness can occur due to disorders associated with pre-retinal, retinal, optic nerve, or central nervous system lesions. Pre-retinal disorders result in a failure of light to reach the retina (*q.v.* section 2.7), retinal disorders result in a failure to detect light, optic nerve lesions result in a failure to transmit information from the retina to the cortical brain, and central nervous system lesions result in a failure to process visual information and therefore a loss of conscious perception of sight.

RELATED CLINICAL SIGNS

- Duration of the onset of blindness may have a significant impact on the dog's ability to cope and therefore the clinical signs; dogs with a slow progression may have adapted in familiar environments and signs may be more subtle than in those that suffer acute onset blindness.
- With some disorders dogs may present with signs of ocular pain including blepharospasm (squinting), rubbing eyes, or ocular discharge.
- Dogs with central nervous system disorders or systemic diseases as a cause of the blindness may present with concurrent neurological or systemic signs respectively.
- With sudden acquired retinal degeneration syndrome (SARDS) dogs also present with clinical signs more typically associated with HAC including polyuria, polydipsia, polyphagia, excessive panting with muscle wastage and pot-bellied appearance.

COMMON CAUSES

Pre-retinal

- Cataracts: congenital, diabetic
- Corneal opacity, e.g. chronic dry eye, *q.v.* section 2.7
- Hyphaema
  - Coagulopathy
  - Hypertension
  - Uveitis
- Uveitis
  - Immune-mediated
  - Neoplasia
  - Systemic disease

Retinal

- Ivermectin toxicity can cause retinal oedema with folds (vision loss is temporary and resolves in 2–10 days)
- Retinal detachment (systemic hypertension, infectious, neoplastic, cataracts, lens luxation)
- SARDS

Optic nerve

- Optic neuritis: inflammatory, infectious, neoplastic

Central nervous system

- Inflammatory (meningoencephalitis of unknown origin)
- Neoplasia
- Post-ictal

UNCOMMON CAUSES

Pre-retinal
- Corneal opacity
  - Chronic superficial keratitis
  - Pigmentary keratitis
- Hyphaema
  - Intraocular neoplasia
  - Trauma
- Uveitis
  - Pigmentary uveitis
  - Uveodermatologic syndrome

Retinal
- Progressive retinal atrophy
- Retinal detachment (congenital, liquefied vitreous, optic nerve colobomas)

Optic nerve
- Optic nerve neoplasia: to cause blindness would involve the optic chiasm
  - Meningioma
  - Glioma
  - Lymphoma

Central nervous system
- Anaesthesia-related complication
- Hypertensive encephalopathy
- Hyperviscosity: multiple myeloma, erythrocytosis

DIAGNOSTIC APPROACH

Clinical clues
Predisposition
- Pigmentary keratitis: Pugs
- Chronic superficial keratitis (pannus): GSDs
- Uveodermatologic syndrome: Akitas and Arctic breeds
- Pigmentary uveitis: Golden retrievers
- Progressive retinal atrophy: Border collies, Irish setters, poodles

History
- Nature of onset of signs may be useful in prioritising differential diagnoses
- Progressive retinal atrophy affects rods first, so the dog may initially lose sight in dim light
- Signs of concurrent systemic disease may increase index of suspicion, e.g. DM, SARDS, and neoplasia or infectious disease
- Specific questions to assess for other signs of central nervous system disorders may be useful, in particular signs of forebrain disease (mentation change, incoordination, seizures)

Clinical examination
Visual inspection
- Mentation is expected to be impaired with central disorders

Physical examination
- Blood pressure: hypertension may be detected in increased intracranial pressure, hypertensive retinopathy, and hypertensive encephalopathy
- Pupillary light reflex is highly valuable in lesion localization: this will be impaired in pre-retinal, retinal and optic nerve disorders with fixed mydriatic pupils, whereas central lesions will result in a normal pupillary light reflex
- Thorough examination to assess for multisystemic disease

Neurological examination
- Focus on assessment of forebrain, i.e. response to nasal stimulation, mentation, proprioception

Ophthalmic examination
- Full ophthalmic examination to assess for pre-retinal lesions (corneal, lens, iris, vitreous changes), retinal lesions (retinal detachment), or optic nerve head changes (optic neuritis)

Laboratory findings
Haematology
- Useful to screen for systemic disease

Serum biochemistry
- Useful to screen for systemic disease
- Dogs with SARDS may have increased ALP enzyme activity but interpret results of ACTH stimulation and LDDS tests with caution in

these patients as there is a risk of false positives

Imaging

Plain radiographs

- Screen for disseminated neoplasia in cases with a high suspicion

Ultrasound

- Screen for disseminated neoplasia in cases with a high suspicion

Special tests

- Electroretinogram (ERG)
- Ocular paracentesis may be useful in cases with uveitis
- Ocular ultrasound may be useful to assess for evidence of intraocular neoplasia, FB or trauma
- MRI and CSF in cases with optic neuritis or central blindness

1.11 CONSTIPATION

DEFINITION

Infrequent and difficult or absent defaecation, with abnormal retention of faeces in the large intestine.

Obstipation is prolonged, intractable constipation in which faeces have become so firm that defaecation is no longer possible, and which ultimately leads to secondary degeneration of colonic musculature.

RELATED CLINICAL SIGNS

- Anorexia, lethargy and vomiting
- Dyschezia (pain or difficulty in defaecation), *q.v.* section 1.46
- Failure to pass faeces or small, hard, dry faeces
- Haematochezia if intraluminal cause
- Paradoxical diarrhoea (scant liquid faeces passed around the constipated mass)
- Tenesmus (straining to defaecate), *q.v.* section 1.46

COMMON CAUSES

Anorectal pain

- Anal furunculosis
- Anal sac impaction, abscess, cellulitis

Dietary

- Foreign material, e.g. bones, hair
- Excessive or insufficient fibre
- Inadequate water intake

Drug-induced

- Anticholinergics
- Barium
- Kaolin-pectin
- Opioids

Environmental

- Dehydration
- Hospitalization
- Inadequate exercise

Extraluminal obstruction

- Healed pelvic fracture
- Prostatic enlargement – hypertrophy, abscess

Intraluminal obstruction

- Perineal hernia and rectal diverticulum
- Rectal tumour

Neuromuscular disease

- Lumbosacral disease

Orthopaedic disease (pain and failure to posture)

- Injury to pelvis, hip or pelvic limbs
- Spinal (range of lumbo-sacral diseases)

Water-electrolyte abnormalities

- Dehydration
- Hypercalcaemia
- Hypokalaemia

UNCOMMON CAUSES

Anorectal disease
- Anal or rectal stricture
- Anal sac adenocarcinoma
- Atresia ani
- Atresia coli
- Pseudocoprostasis (faecal impaction due to matted hair)

Drug-induced
- Aluminium hydroxide
- Antihistamines
- Barium sulphate
- Diphenoxylate
- Diuretics
- Iron preparations
- Loperamide
- Phenothiazines
- Sucralfate
- Verapamil

Extraluminal obstruction
- Pelvic collapse due to nutritional bone disease
- Prostatic neoplasia
- Prostatic or paraprostatic cyst
- Sublumbar lymphadenopathy

Intraluminal obstruction
- Benign stricture
- FB other than bone or hair

Metabolic
- Hyperparathyroidism
- Hypothyroidism

Neuromuscular disease
- Bilateral pelvic nerve damage
- Dysautonomia
- Hirschsprung's disease (congenital megacolon)

DIAGNOSTIC APPROACH

1 Confirm constipation by abdominal and digital rectal palpation, and imaging.

2 Identify underlying cause through history, physical examination and proctoscopy.

Clinical clues
Predisposition
- Benign prostatic hypertrophy in uncastrated older dogs
- Prostatic carcinoma more common in castrated dogs
- Sedentary dogs on low-fibre diet

History
- Dysuria with prostatic disease
- Old pelvic trauma

Clinical examination
Visual inspection
- Anal furunculosis
- Dyschezia
- Hindleg weakness or pain with lumbosacral disease
- Haematochezia if intraluminal cause
- Pseudocoprostasis
- Tenesmus

Physical examination
- Abdominal palpation
  - Enlarged palpable intra-abdominal prostate
  - Faecal material in colon
  - Paraprostatic cyst
  - Pelvic deformity
- Digital rectal palpation
  - Anal sac disease
  - Dry, impacted faeces
  - Paraprostatic cyst
  - Pelvic canal narrowing
  - Perineal hernia and rectal diverticulum
  - Prostatic enlargement
  - Rectal stricture
  - Rectal mass

Laboratory findings
Usually unremarkable
- Hypercalcaemia with anal sac adenocarcinoma or lymphoma
- Hypokalaemia causing colonic muscle weakness

Imaging

Plain radiographs
- Old pelvic fractures
- Prostatomegaly

Contrast radiographs (barium enema)
- Rectal stricture
- Colonic/rectal tumour

Ultrasound
- Colonic tumour
- Prostatic disease
  - Abscess
  - Benign prostatic hypertrophy
  - Cyst or paraprostatic cyst
  - Neoplasia

Special tests
- Thyroid function tests
- Proctoscopy

1.12 CORNEAL OPACITY

DEFINITION

Focal or diffuse abnormality of the cornea preventing the transmission of light.

NB: Owners may mistake lenticular nuclear sclerosis and cataracts for corneal disease.

RELATED CLINICAL SIGNS

- Blindness, *q.v.* section 1.10
- Diffuse or focal abnormalities of corneal colour
- Blepharospasm and epiphora if ulceration

COMMON CAUSES

White
- Chemosis = corneal oedema
  - Interstitial/infectious keratitis
  - Ulcerative keratitis
- Hypopyon = infection in anterior chamber, often due to FB penetration
- Lipid

Brown/black pigment
- Chronic corneal irritation: ectopic cilia, aberrant hairs or eyelashes
- Pannus

Red

q.v. section 1.40

UNCOMMON CAUSES

White
- Crystalline: calcium
- Fibrosis
- Glaucoma

Brown/black pigment
- Congenital endothelial pigmentation
- Dermoid
- Iris prolapse
- Neoplasia: limbal melanoma
- Persistent pupillary membrane
- Ruptured iris cyst
- Sequestrum

Blue
- Chemosis
  - Canine adenovirus (CAV-1, and rarely CAV-2 live vaccine)
  - Glaucoma

Red

q.v. section 1.40

DIAGNOSTIC APPROACH

- Examine cornea
- Fluorescein staining
- Palpebral and corneal reflexes
- *q.v.* section 1.40

Clinical clues

Predisposition

- Hyperlipidaemia: Miniature schnauzers
- Pannus: Australian Shepherd, Belgian Tervuren, Border collie, Greyhound, GSD, Siberian Husky

History

- Other signs of hyperlipidaemia
- Other signs of hypothyroidism
- Previous corneal ulceration

Clinical examination

Visual inspection

- Blepharospasm and/or epiphora if ulcerated
- Chemosis = corneal oedema
- Corneal ulcer
- Ocular discharge

Physical examination

- Opacity covering part or all of the cornea

Laboratory findings

- Hyperlipidaemia: hypertriglyceridaemia and/or hypercholesterolaemia with lipid deposit

Imaging

- Full ophthalmological examination

Special tests

- Fluorescein staining
- Palpebral and corneal reflexes
- Schirmer tear test

1.13 COUGHING

DEFINITION

A sudden expiratory effort in an attempt to try and clear excess secretion or foreign material from the lungs, bronchi or trachea resulting in a sudden, noisy expulsion of air from the lungs.

RELATED CLINICAL SIGNS

Associated signs

- Dyspnoea/tachypnoea, *q.v.* section 1.18
- Exercise intolerance/collapse, *q.v.* sections 1.22, 1.51
- Halitosis, *q.v.* section 1.29
- Nasal/ocular discharge, *q.v.* section 1.32

COMMON CAUSES

Allergic/immune-mediated

- Reverse sneeze, sometimes mistaken for coughing

Cardiovascular

- Cardiomegaly with left atrial enlargement and bronchial compression
- Left-sided failure causing pulmonary oedema (dyspnoea rather than coughing)

Environmental irritants

- Dust
- Passive smoking

Infectious/inflammatory

- Acute or chronic inflammatory disease anywhere from pharynx to pulmonary tissue can stimulate coughing
- Acute secondary to infectious agents, e.g. infectious tracheobronchitis (kennel cough)
- Bronchopneumonia
- Canine infectious respiratory disease (CIRD) complex: 'kennel cough'
- Chronic bronchitis
- Inhalation pneumonia, secondary to oesophageal disease or GORD (aerodigestive syndrome)

Parasitic

- *Angiostrongylus vasorum* – regional variation in prevalence in UK

Pleural effusion

- Very rarely causes coughing; dyspnoea is more important sign, *q.v.* section 2.21

Physical/traumatic
- Collapsing trachea
- Inhalation pneumonia, secondary to oesophageal disease
- Inhaled/ingested FB
- Laryngeal paralysis: coughing when drinking
- Pulmonary haemorrhage

UNCOMMON CAUSES

Allergic
- Asthma (much more common in cats than dogs)
- Eosinophilic inflammatory respiratory diseases (pulmonary infiltrate with eosinophils) e.g. eosinophilic bronchitis/bronchopneumonopathy/pneumonia

Cardiovascular
- Non-cardiogenic pulmonary oedema
- Pulmonary thromboemboli
- Pulmonary oedema (cardiac origin)

Environmental irritants
- Chemical fumes
- Smoke inhalation from fire
- Potassium bromide
- Talcum powder

Infectious/inflammatory
- Abscess rarely
- Bronchiectasis
- Chronic pulmonary fibrosis, especially in WHWT
- Distemper
- Fungal (not in UK)
  - Blastomycosis
  - Coccidioidomycosis
  - Histoplasmosis
- Granulomatous disease
- Hilar lymph node enlargement
- *Pneumocystis* pneumonia

Neoplastic
- Mediastinal
- Metastatic (more commonly causes dyspnoea than cough)
- Primary
  - Extrathoracic, e.g. rib/sternum/soft tissue
  - Laryngeal
  - Lymphosarcoma
  - Tracheal

Parasitic
- *Angiostrongylus vasorum*; regional variation in prevalence in UK
- *Dirofilaria immitis* (not currently in UK)
- *Oslerus osleri* (lungworm)
- Others, e.g. *Crenosoma vulpis, Eucoleous, Filaroides hirthii*, *Paragonimus*
- Visceral larval migrans

Physical/traumatic
- Iatrogenic secondary to inhalation of liquids or solids, e.g. force feeding, barium administration

DIAGNOSTIC APPROACH

1 Determine whether this is an upper or lower airway problem from signs and physical examination.
2 Rule out cardiac disease.
3 Rule out oesophageal disease.
4 Investigate airway disease by radiography, laboratory analysis and endoscopy.

CLINICAL CLUES

Predisposition
- Brachycephalics have obstructive upper airway disease
- Idiopathic megaoesophagus: Great Dane, GSD, Irish setter
- Kennelled pets are at risk of CIRD
- Large/giant breed dogs
  - Dilated cardiomyopathy
  - Laryngeal paralysis
  - Megaoesophagus
  - Pneumonia
- Local outbreak of coughing consistent with CIRD
- *Oslerus* infection: young dogs
- *Pneumocystis* pneumonia: Cavalier King Charles spaniel (CKCS)

- Small dogs of mid- to old age likely to have:
  - Chronic mitral valve disease: CKCS
  - Chronic obstructive lung disease, e.g. chronic bronchitis
  - Collapsing trachea
  - Pulmonary interstitial fibrosis: WHWT

History
- Associated dyspnoea
  - Obstruction, and alveolar and pleural space disease
- Duration of cough
  - Acute (< 2 weeks duration) likely "kennel cough"
  - Chronic (> 2 weeks duration)
- Environment: flare factors for cough
  - Access to intermediate hosts of parasites
  - Cough associated with walking on collar and lead: collapsing trachea
  - Owners who are heavy smokers causing passive smoking
  - Potential exposure to other parasites if the dog has been outside of UK
  - Seasonality for allergic airway disease
- Nature of cough
  - Acute coughing can often be assumed to be infectious in origin, until proven otherwise
  - Haemoptysis with coagulopathy, FB, neoplasia or trauma
  - Non-productive moist or dry suggests upper airway
  - Productive/non-productive moist or dry suggests pulmonary
    - Animal may swallow excessively after coughing if productive
  - Regurgitation (and inhalation) in primary oesophageal disease
  - Terminal retch or vomiting can be confused with GI disease
  - "Wheezy" coughs suggest airway inflammation
- Response to therapy
- Timing of the cough
  - Any association with eating food/drinking fluids suggests larynx or oesophagus

Clinical examination

Visual inspection
- Audible respiratory sounds
- Dyspnoea, *q.v.* section 1.18
- Halitosis; often a feature with inhaled FBs, *q.v.* section 1.29
- Ocular/nasal discharge with allergic or infectious conditions, *q.v.* section 1.32
- Respiratory pattern, e.g. any abdominal effort
- Tachypnoea

Physical examination
- Palpation
  - Cough elicited by tracheal pinch if upper airway
  - Larynx, trachea and thorax for abnormalities
- Auscultation
  - Airway noise
    - Inspiratory: upper airway
    - Expiratory: lower airway
  - Localise origin
    - Trachea: abnormal respiratory sound
    - Thorax: adventitious respiratory sounds, e.g. crackles and wheezes
  - Presence of murmur

Laboratory findings

- Eosinophilia may be associated with parasitic diseases and eosinophilic immune-mediated disorders
- Faecal examination may reveal the presence of parasitic larvae
- Increased serum globulins may be seen in certain inflammatory conditions, e.g. granulomatous disease
- Leukocytosis in pulmonary inflammatory disease
- Often unremarkable if upper airway disease

Imaging

Thoracic radiographs
- Assess heart size for cardiomegaly, especially left atrium
- Assess tracheal diameter throughout its length: can do dynamic assessment under fluoroscopy for tracheal collapse
- Assess lung patterns as bronchial, interstitial, alveolar or mixed
- Assess for presence of mediastinal abnormalities

- Inflated radiographs under general anaesthesia may give more information
- Right lateral and dorso-ventral (DV) (lungs) or ventro-dorsal (VD) (heart) projections should be performed

Special tests

- Assessment of vocal fold movement under a light plane of anaesthesia
- Baermann technique for lungworms including *Angiostrongylus*
- Blood gas analysis (if available) – arterial to assess oxygenation
- Bronchoscopy
  - Bronchoalveolar lavage (BAL) for cytological examination and culture
- Cardiovascular examination: ECG, echocardiography
- Fine needle lung aspiration/biopsy
- Knott's test and/or serology for *Dirofilaria immitis*
- Nuclear scintigraphic studies of ventilation-perfusion
- Thoracocentesis
- Ultrasound of the larynx to assess for laryngeal paralysis

1.14 DEAFNESS

DEFINITION

Deafness, or the inability to perceive sound, can be divided into conductive or sensorineural deafness. Conductive deafness results from disorders affecting the transmission of sound from the environment to the tympanic membrane and ossicles of the inner ear. Sensorineural deafness results from disorders affecting the cochlear system, eighth cranial nerve, or central nervous system (brainstem and forebrain).

Presbycusis is the progressive and irreversible bilateral degeneration of the cochlea and associated structures and results in age-related irreversible sensorineural deafness.

RELATED CLINICAL SIGNS

- Dogs may be difficult to rouse from sleep or startled easily.
- Dogs with otitis externa or media may display discomfort: head shaking, scratching, pain on opening mouth.
- In congenital deafness puppies may not be as responsive to cries of littermates during play.

COMMON CAUSES

Conductive

- Otitis externa
- Otitis media

Sensorineural

- Congenital deafness; pigment and non-pigment associated
- Otitis interna
- Ototoxicity: gentamicin, furosemide, or topical chlorhexidine
- Presbycusis: degenerative, occurring with old age

UNCOMMON CAUSES

Conductive

- Hereditary ear canal atresia

Sensorineural

- Adult-onset hereditary deafness, e.g. Collie
- Anaesthesia associated
- Trauma

DIAGNOSTIC APPROACH

Clinical clues

Predisposition

- Congenital sensorineural deafness most commonly associated with white or merle coat colour: Boxer, Dachshund, Jack Russell terrier
- Otitis externa: Cocker spaniel
- Secretory otitis media: CKCS

History
- Dogs with significant brainstem or forebrain disease are likely to have more pertinent neurological findings and history
- History of exposure to ototoxic drugs: aminoglycosides, loop diuretics, erythromycin, cisplatin and carboplatin
- Older dogs with gradual-onset progressive deafness are most likely to have presbycusis

Clinical examination

Visual inspection
- Assessment of behaviour, mentation, and evidence of ear discomfort
- Assessment for response to sounds in the consulting room; can be challenging due to detection of vibrations
- Presbycusis results in loss of detection of high-pitched sounds first

Physical examination
- Dogs with otitis media or interna may have pain on opening jaw

Neurological examination
- Assessment for concurrent neurological deficits

Otoscopic examination
- Full otoscopy, including assessment of the tympanic membrane

Laboratory findings

Haematology
- Typically unremarkable, may be useful to screen for systemic disease

Serum biochemistry
- Typically unremarkable, may be useful to screen for systemic disease

Imaging

Plain radiographs
- Radiography of the head may detect mineralisation of the ear canal, and opacity within the bullae

Special tests
- Brainstem-auditory evoked response (BAER)
- CT scan
- MRI and CSF

1.15 DIARRHOEA

DEFINITION

An increase in the water content of faeces, with a consequent increase in their fluidity and/or volume and/or frequency.

Diarrhoea can be:
- Acute (sudden onset, often self-limiting) or chronic (arbitrarily > 3 weeks duration) which may be continuous or intermittent
- Caused by primary GI disease (disorders of the small or large intestine or both), or secondary to disease elsewhere
- Self-limiting or life-threatening
- Due to alterations in one or a combination of:
  - the osmotic content of the stool
  - intestinal secretion
  - intestinal mucosal permeability
  - motility
- The consistency of diarrhoea can vary from soft with some retention of form, through 'cow-pat', to completely watery
- Small intestinal (SI) diarrhoea is characteristically large volume, not increased in frequency and watery, and may contain melaena if there is bleeding, *q.v.* section 1.31
- Large intestinal (LI) diarrhoea is characteristically small volume, mucoid and may contain fresh blood (*q.v.* section 1.26). Dogs may show urgency, increased frequency of defaecation and straining (*q.v.* section 1.46); they may continue to strain after defaecation

RELATED CLINICAL SIGNS

- Abdominal discomfort (*q.v.* section 2.17.1), excessive borborygmi, flatus, and halitosis (*q.v.* section 1.29) are non-specific signs

- Anorexia (*q.v.* section 1.5) in the presence of diarrhoea is generally an indication of serious disease
- Severe, chronic diarrhoea may be a protein-losing enteropathy with consequent hypoalbuminaemia and ascites/oedema
- Signs of dehydration, particularly in acute diarrhoea
- Vomiting can be associated with both SI and LI diseases

1.15.1 ACUTE DIARRHOEA

DEFINITION

Acute diarrhoea generally occurs abruptly in a previously healthy dog and is typically profuse but self-limiting and of short duration. It will have been present continuously for less than two weeks, or intermittently for less than four weeks. In severe cases it may be haemorrhagic and can be life-threatening. It is frequently associated with vomiting, and often involves the whole GI tract.

RELATED CLINICAL SIGNS

- Associated vomiting
- Borborygmi
- Dehydration
- Weight loss is not a feature

COMMON CAUSES

Primary GI disease

Dietary
- Dietary indiscretion
  - Excess intake
  - Inappropriate food due to scavenging
  - Sudden change in diet
- Food intolerance
- Food poisoning – contamination of food by
  - live bacteria or bacterial toxins
  - mycotoxins

Drug/toxin
- Antimicrobials
- NSAIDs

Infection – Bacterial
- Acute haemorrhagic diarrhoea syndrome (AHDS), previously termed haemorrhagic gastroenteritis (HGE)
- *Campylobacter jejuni*; *C. upsaliensis* is more prevalent and may be non-pathogenic
- *Clostridium perfringens*: type A toxin (netF) may be the cause of AHDS
- *E. coli*

Infection – Parasitic
- *Ancylostoma* hookworms (not in UK)
- *Giardia*
- *Trichuris* whipworms (uncommon in UK)

Infection – Viral
- Coronavirus (usually mild, but has been associated in UK with acute, profuse vomiting)
- Parvovirus

Obstructive (surgical)
- Intussusception

Secondary, non-GI disease
- Acute pancreatitis
- Hypoadrenocorticism: not common but important

UNCOMMON CAUSES

Primary GI disease

Dietary
- Food hypersensitivity (true food allergy)

Drug/toxin
- ACE inhibitors
- Anthelmintics
- Anti-arrhythmics
- Anti-cancer agents, especially methotrexate
- Blue-green algae
- Digoxin
- Heavy metals

- Laxatives
- Organophosphates
- Phenylpropanolamine

Infection – Bacterial
- *Bacillus piliformis*
- *Campylobacter coli*
- *Leptospira*
- *Salmonella*: more likely if fed raw meat
- *Shigella*
- *Yersinia*

Infection – Parasitic
- Ascarids and cestodes: infection is common, but is rarely a cause of diarrhoea
- *Cryptosporidium*
- *Cystoisospora:* rarely primary pathogen, except in puppies
- *Strongyloides stercoralis*
- *Uncinaria stenocephala* hookworms

Infection – Rickettsial
- Salmon-poisoning disease, *Neorickettsia helminthica* and *elokominica* (geographically limited to Pacific Northwest, United States)

Infection – Viral (often mild)
- Astrovirus
- Bocavirus
- Circovirus: more severe disease reported occasionally
- Norovirus
- Rotavirus

Obstructive (surgical)
- FB: typically results in lack of faecal output but a partial obstruction may cause diarrhoea
- Incarcerated bowel loop: internal or external hernia
- Intestinal volvulus
- Linear FB
- Mesenteric torsion

Secondary, non-GI disease
- Acute hepatitis
  - Adenovirus (CAV-1, infectious canine hepatitis) can also cause diarrhoea
  - Leptospirosis
- AKI
- Canine distemper
- Diabetic ketoacidosis
- Pyometra

DIAGNOSTIC APPROACH

1 Differentiate between primary (GI) and secondary (non-GI) disease by history, physical examination and laboratory findings.
2 If secondary GI disease is present, investigate and treat underlying cause
3 If primary GI disease is present, differentiate by history, physical examination, laboratory findings and imaging those that require only symptomatic support (e.g. anti-diarrhoeals, fluid therapy) from life-threatening causes that require either:
- Intensive medical/supportive therapy
  - AHDS/HGE
  - Leptospirosis
  - Parvovirosis
  - Salmonellosis

  or
- Surgical correction
  - FB
  - Incarcerated bowel loop (internal or external hernia)
  - Intestinal volvulus
  - Intussusception
  - Linear FB
  - Mesenteric torsion

Clinical clues
Predisposition
- Raw feeding: risk of *Campylobacter*, *Salmonella*
- Unsanitary environment
- Unvaccinated
  - Dogs with distemper usually have concurrent respiratory signs, and dogs with leptospirosis usually have hepatic and renal problems as well
- Young, not fully immunocompetent

History
- Access to toxins
- Acute onset of diarrhoea, etc.
- Contact with infected dogs
- Drug administration

- Other signs, e.g. vomiting
- Presence of blood in diarrhoea
- Scavenging
- Vaccination status

Clinical examination

Visual inspection

- Dull and depressed versus bright and alert indicates greater need for definitive diagnosis and intensive treatment
- Faecal staining of perineum

Physical examination

- Systemic signs
  - Pyrexia with infectious/inflammatory disease
  - Signs of dehydration
    - Depression
    - Dry mucous membranes
    - Skin tenting
    - Slow capillary refill
    - Sunken eyes
    - Tachycardia
- Oral examination
  - Jaundice with hepatobiliary disease
  - Linear FB under tongue
  - Oral ulcers from acute uraemia
- Palpation
  - Abdominal pain: *q.v.* section 2.17.1
    - Cranial abdominal pain in pancreatitis
  - Bunching of intestines with linear FB
  - Fluid or gas-filled bowel loops
  - FB
  - Mesenteric lymphadenopathy
  - Sausage-shaped mass consistent with intussusception
- Rectal examination
  - Abnormal faecal material
  - Advanced ileo-colic intussusception
  - Evidence of diarrhoea
  - Evidence of melaena or haematochezia
  - Rectal prolapse

Laboratory findings

Haematology

- Eosinophilia may be seen with parasitism
- Haemoconcentration indicative of dehydration (NB: check total protein)
- Leukopenia in parvovirus infection (~60% of cases) or overwhelming sepsis
- Leukocytosis ± left shift with infectious/inflammatory disease
- Marked haemoconcentration with normal serum proteins suggests AHDS/HGE
- Pre-regenerative anaemia with peracute disease

Serum biochemistry

Often unremarkable except for changes associated with dehydration, but helpful in ruling out non-GI diseases.

- Azotaemia may be pre-renal; check urine specific gravity (SG)
- Electrolyte abnormalities, especially hypokalaemia, inform choice of fluid therapy
- Hypoglycaemia sometimes seen in sepsis and in inadequate food uptake by puppies
- Non-GI diseases
  - Azotaemia in renal failure
  - Hyperkalaemia and hyponatraemia suggestive of hypoadrenocorticism
  - Increased liver enzyme activities secondary to intestinal inflammation, bacterial translocation, or because of primary hepatopathy
  - Lipase and canine pancreatic lipase (cPL) may be elevated in pancreatitis
- Serum proteins
  - Increased in dehydration
  - Decreased serum proteins may develop after rehydration in ulcerative/haemorrhagic diseases (AHDS/HGE, parvovirus)

Urinalysis

- Failure to concentrate urine fully is typical of hypoadrenocorticism
- Hypersthenuria in face of dehydration is appropriate
- Isosthenuria (SG 1.008–1.016) in face of dehydration is inappropriate, and indicates renal insufficiency

Faecal examination

- Bacterial culture may identify primary pathogen, and is indicated if there is haemorrhagic diarrhoea or pyrexia, but pathogenic bacteria can be found in the stool of healthy dogs
- Faecal cytology is of limited value. *Campylobacter*-like organisms and sporulating

Clostridia may be identified but are of uncertain significance
- Faecal SNAP® test or PCR for parvovirus
- Flotation tests to identify endoparasites
  - Baermann technique: *Strongyloides* larvae
  - Sodium nitrate, or formalin-ether: nematode and cestode ova
  - Sheather's sugar centrifugation: *Cryptosporidium*
  - Three zinc sulphate flotations 95% sensitive for the identification of *Giardia*
- SNAP® test for *Giardia*

Imaging

Radiographs
- Plain
  - FB
    - Free intra-peritoneal gas indicates perforated viscus
  - Intestinal distension with fluid or gas = ileus
    - Obstructive ileus with massively dilated stacked bowel loops
    - Physiological ileus is less severe and reflects inflammation or metabolic abnormalities
    - Mesenteric torsion causes distension of all intestinal loops
    - Strangulation causes dilation of one segment of intestine
    - Signs of obstruction
    - Fluid- or gas-filled, dilated bowel loop(s)
  - 'Gravel' sign
    - Soft tissue density
    - Intussusception
- Contrast
  - Often no value over plain films in acute diarrhoea
  - Radiolucent FB

Ultrasound
- FB obstruction
- Hepatic and pancreatic pathology
- Intussusception: transverse double concentric ring with hyperechoic centre, or > 5 layers in longitudinal view of bowel loop
- Mesenteric lymphadenopathy

Special tests
- Basal cortisol or ACTH stimulation test
- Assay for *Clostridium (Cl.) perfringens* and *Cl. difficile* toxins
- Endoscopic biopsy (rarely indicated in acute disease)
- Exploratory laparotomy
- Scanning electron microscopy of faeces for virus particles
- Serology – haemagglutination inhibition after parvovirus infection
- SNAP® cPL: a negative result helps rule out pancreatitis; a positive result requires further investigation, i.e. Spec cPL, imaging

1.15.2 CHRONIC DIARRHOEA

DEFINITION

Diarrhoea is defined as chronic if:
- It has been present continuously for a minimum of 3 weeks, and has not responded to symptomatic treatment
  or
- If there is a pattern of recurrent episodes occurring over a period of > 4 weeks

RELATED CLINICAL SIGNS

- Ascites (*q.v.* section 2.5) occurs if a protein-losing enteropathy (PLE) causes significant (< 15 g/l) hypoalbuminaemia
- Polyphagia and weight loss in the presence of diarrhoea are suggestive of malabsorption
- Weight loss is characteristic of SI disease, but dogs with chronic LI disease may also lose weight if the owner repeatedly withholds food

COMMON CAUSES

Primary GI disease

- Antibiotic-responsive diarrhoea (ARD), formerly small intestinal bacterial overgrowth (SIBO)
- Bacterial infection, but some potential pathogens can be present in the faeces of healthy dogs
- Food intolerance
- Giardiasis
- CIE/IBD
  - Histological types
    - Lympho-plasmacytic enteritis (LPE) and/or colitis
    - Eosinophilic gastroenteritis (EGE)
  - Treatment-response types
    - Antibiotic-responsive enteropathy (ARE)
    - Food-responsive enteropathy (FRE)
    - Non-responsive enteropathy (NRE)
    - Steroid-responsive enteropathy (SRE)/immunosupressant-responsive enteropathy (IRE)
- Irritable bowel syndrome (functional diarrhoea)

Secondary non-GI disease

- Exocrine pancreatic insufficiency (EPI)
- Portal hypertension; typically with associated ascites
  - Chronic liver disease

UNCOMMON CAUSES

Primary GI disease

- Alimentary lymphoma
- Chronic intussusception
- Food allergy
- Granulomatous (histiocytic ulcerative) colitis (almost exclusively Boxer and French bulldog)
- Histiocytic disease (diffuse, infiltrative)
- Histoplasmosis (geographically limited to the Ohio Valley in the United States)
- CIE/IBD
  - Granulomatous enteritis
  - Immunoproliferative small intestinal disease (IPSID)
- Intestinal adenocarcinoma
- Lymphangiectasia
- Parasitic
  - Hookworm
    - *Ancylostoma caninum*; not in UK
    - *Uncinaria stenocephala*
  - *Strongyloides stercoralis*
  - Whipworm: *Trichuris vulpis*
- Protothecosis
- Pythiosis (geographically limited to southern United States)
- Selective cobalamin malabsorption (Imerslund-Gräsbeck) *q.v.* section 5.7.1.2C
- Short-bowel syndrome

Secondary non-GI disease

- Chronic pancreatitis
- Gastrinoma (APUDoma)
- Hypoadrenocorticism
  - Classical, but acute presentation previously masked by symptomatic therapy
  - Atypical, i.e. normal electrolytes as only hypocortisolaemia
- Hyperthyroidism
  - Functional thyroid tumour
  - Raw feeding contaminated by exogenous thyroid tissue
- Portal hypertension
  - Cardiac tamponade
  - Portal vein thrombosis
  - Right-sided heart failure
- Renal disease

DIAGNOSTIC APPROACH

NB: Weigh and record weight for monitoring.

1. Rule out non-GI causes by history, physical examination and laboratory testing.
2. Rule out simple causes, such as diet-induced and parasitism from history and faecal examination, or empirical anti-parasiticide treatment.
3. Perform serum trypsin-like immunoreactivity (TLI) test to diagnose EPI *before* further investigations.
4. Anatomical localisation from history and faecal characteristics.

5 Suspect PLE from ascites due to hypoalbuminaemia.
6 Faecal culture is often unhelpful.
7 Cobalamin and folate to screen for malabsorption, *q.v.* sections 3.10, 3.15
8 Radiographs to screen for masses, partial obstruction, *q.v.* section 4.1.1
9 Ultrasound examination to examine bowel wall thickness and mucosal changes, and to identify masses
10 Endoscopic biopsy
11 Exploratory laparotomy and biopsy if endoscopy is unavailable or non-diagnostic, or if focal disease is found on imaging. Biopsy must always be performed, even if no gross abnormalities are found.

Clinical clues

Predisposition

- Clostridial enterotoxicosis in stressed (e.g. hospitalised) dogs
- EPI: GSD, Collie, Chow Chow, small terriers
- CIE/IBD
  - LPE: GSD, Shar pei, Soft-coated Wheaten terrier may also have concurrent PLN
  - EGE: GSD
  - Immunoproliferative small intestinal disease (IPSID): Basenji
- Granulomatous colitis: Boxer, French bulldog
- IPSID: Basenjis only
- Irritable bowel syndrome (IBS): toy breeds, working dogs
- Lymphangiectasia: Lundehund, Rottweiler, Yorkshire terrier
- Neoplasia in older dogs
- Selective cobalamin malabsorption (Imerslund-Gräsbeck syndrome): Australian shepherd, Beagle, Border collie, Giant schnauzer, Komondor

History

- Correlation of signs with specific food
- Drug administration
- Frequent mucoid diarrhoea ± haematochezia with tenesmus suggests colitis
- Full dietary history
- Melaena may indicate upper GI haemorrhage, and in association with diarrhoea suggests SI bleeding
- Mode of onset – abrupt versus gradual suggests infectious aetiology
- Other signs – vomiting, weight loss
- Previous surgery, especially intestinal resection
- Response to previous treatment

Clinical examination

Visual inspection

- Abdominal enlargement due to ascites or mass(es)
- Body condition score
- Demeanour
- Nature of faeces and style of defaecation help localise to SI or LI, but many are diffuse (mixed pattern diarrhoea)
- Pallor of mucous membranes due to blood loss anaemia
- Peripheral (ventral) oedema if severe hypoproteinaemia
- Poor-quality hair coat secondary to malabsorption
- Weight loss

Physical examination

- Palpation
  - Abdominal mass(es)
  - Abdominal pain
  - Ascites in PLE
  - Body condition score
  - Muscle condition score
  - Thickening of bowel loops suggests infiltration (IBD or lymphosarcoma)
- Rectal examination
  - Distal colonic mass or stricture
  - Evidence of melaena or haematochezia
  - Irregular mucosal texture due to inflammation or neoplasia

Laboratory findings

Haematology

- Eosinophilia sometimes seen in eosinophilic enteritis but many false positives and negatives. Consider parasitism or hypoadrenocorticism
- Haemoconcentration from intestinal fluid loss
- Leukocytosis (neutrophilia ± monocytosis) in severe inflammatory disease or perforation
- Lymphopenia with stress or lymphangiectasia

- Microcytic anaemia and thrombocytosis if chronic blood loss even if not visible
- Non-regenerative anaemia with chronic disease/malnutrition

Serum biochemistry
- Commonly unremarkable in primary, chronic GI disease
- Hyperkalaemia and hyponatraemia seen in hypoadrenocorticism and rarely in salmonellosis and whipworm colitis
- Hypocalcaemia in PLE
  - Total hypocalcaemia reflecting hypoalbuminaemia
  - Ionised hypocalcaemia reflecting malabsorption of calcium and vitamin D in PLE
- Hypocholesterolaemia is suggestive of malabsorption, especially lymphangiectasia
- Hypokalaemia of therapeutic importance
- Increased liver enzyme activities, secondary to intestinal inflammation
- Mild hypoproteinaemia if chronic GI bleeding
- Panhypoproteinaemia is typical in PLE, although severe inflammatory disease may cause raised globulins, e.g. IPSID in Basenji

Urinalysis
- Rule out proteinuria as the cause of hypoalbuminaemia

Faecal examination
- Faecal *Clostridium perfringens* and *Cl. difficile* toxins may give false positives
- Faecal culture may give false negatives and positives
- Faecal leukocytes on cytology indicate inflammation but are not specific
- Fungal culture and rectal cytology for histoplasmosis
- Occult blood test, but false positives are common
- Presence of undigested food is non-specific and unhelpful

Imaging

Radiographs
- Plain
  - To identify mass or FB or partial obstruction such as a chronic intussusception
- Contrast
  - 'Apple-core' sign with neoplasm causing partial obstruction
  - Barium follow-through studies generally unhelpful if just diarrhoea is present and plain radiographs are unremarkable
  - Barium enema is largely superseded by colonoscopy

Ultrasound
- Demonstrate free fluid or gas
- Identify masses, lymphadenopathy
- Measure bowel wall thickness

Special tests

- Basal cortisol + ACTH stimulation test if basal cortisol < 55 nmol/l
- Breath hydrogen for SIBO, but not readily available
- Canine CE-IBD test (Antech Diagnostics) – combination triple test for Anti-OmpC porins surface antigens, IgA antibodies, anti-canine calprotectin IgA antibodies and anti-gliadin IgA antibodies – not fully validated yet
- Exclusion diet trial
- Exploratory laparotomy and full-thickness biopsy
- Faecal alpha$_1$-protease inhibitor as a marker for a PLE (test only available in the United States)
- Faecal calprotectin as an inflammatory marker
- Faecal ELISA for *Giardia* antigen
- Folate and cobalamin to screen for SIBO, and infiltrative bowel disease
- Intestinal biopsy by endoscopy or laparotomy, *q.v.* section 5
  - Routine histology
  - Immunohistochemistry
  - PCR for antigen receptor rearrangements (PARR)
  - Histology-guided mass spectrometry (HGMS)
- Serum iron and total iron-binding capacity or serum ferritin to document iron deficiency
- Serum thyroxine, although hyperthyroidism in dogs is very rare
- Trypsin-like immunoreactivity (TLI)
- Vitamin D

1.16 DROOLING

DEFINITION

Dribbling of saliva from the mouth.

NB: Rabies should always be considered as a potential diagnosis in endemic areas before physical examination.

- Pseudoptyalism is drooling due to a failure to swallow normal amounts of saliva
- True ptyalism is increased production of saliva

RELATED CLINICAL SIGNS

- Drooling saliva from mouth
- Dysphagia
- Coughing, inappetence, etc. if non-pharyngeal disease
- Blood may be present with ulcerated oral lesions

COMMON CAUSES

Pseudoptyalism

- Anatomical abnormalities
  - Brachygnathism
  - Lip-fold deformities, especially in giant-breed dogs
- Oesophageal disease
  - Gastro-oesophageal reflux disease
  - Hiatal hernia
  - Obstruction: FB, stricture
  - Oesophagitis
- Oro-pharyngeal disease
  - Dental disease and malposition
  - Oral neoplasia
  - Oral ulcers
  - Oro-pharyngeal FB

Ptyalism

- Gingivitis
- Nausea, *q.v.* section 1.49
- Physiological
  - Anticipation of food (Pavlov reflex)
  - Increased ambient temperature
- Uraemia

UNCOMMON CAUSES

Pseudoptyalism

- Inability to close mouth
  - Botulism
  - Jaw fracture
  - Mandibular neuropraxia
    - trigeminal neuropraxia/neuritis
    - facial nerve paralysis
    - cranial nerve sheath tumours
  - Tetanus
- Masticatory myositis
- Myasthenia gravis
- Pharyngeal FB
- Tonsillar tumour: lymphoma, squamous cell carcinoma

Ptyalism

- Anaphylaxis
- Gastric tumour
- Infection
  - Candidiasis
  - Trench mouth
- Ingestion of toxic/irritant substances and plants
  - *Amanita*, Dumb Cane (*Dieffenbachia*), Philodendron, *Poinsettia*, toads, etc.
  - Caustics
  - Ivermectin
  - Metaldehyde
  - Metronidazole
  - Organophosphates
  - Trimethoprim-sulfa
- Neurological disease
  - Focal myasthenia gravis causing dysphagia and/or megaoesophagus
  - Seizure disorders
  - Vestibular dysfunction
- Rabies and pseudorabies (Aujeszky's disease)
- Salivary gland disease
  - Hypersialosis/sialoadenosis – phenobarbital responsive
  - Sialoadenitis
  - Salivary gland infarction
- Stomatitis
  - Eosinophilic stomatitis: CKCS

DIAGNOSTIC APPROACH

1 Distinguish oropharyngeal from oesophageal and gastric disease by the history
2 Examine oral cavity
3 Investigate for dysphagia, regurgitation and vomiting, *q.v.* sections 1.17, 1.41, 1.49

Clinical clues
- Concurrent dysphagia, regurgitation or vomiting
- Saliva staining of muzzle
- Blood-stained saliva if ulcerated

Clinical examination
Visual inspection
- Drooling saliva
- Lip-fold deformities

Physical examination
- Oral examination – may require sedation or GA
  - Dental diseases: tartar, gingivitis
  - FB
    - Solid FB between teeth, across hard palate or wedged in pharynx
    - Linear FB around base of tongue
  - Oral inflammation, masses or ulceration, *q.v.* section 5.1.1
  - Uraemic breath
- General examination as for regurgitation and vomiting, *q.v.* sections 1.41 and 1.49

Laboratory findings
Haematology
- Typically unremarkable

Serum biochemistry
- Commonly unremarkable in primary oropharyngeal disease
- Azotaemia if uraemic

Urinalysis
- Typically unremarkable except isosthenuria in uraemia

Imaging
Radiographs
- To identify dental diseases and bony involvement with infection or neoplasia

Special tests
- Head CT

1.17 DYSPHAGIA

DEFINITION

Difficulty or inability to prehend, chew or swallow food due to:
- Pain
- Physical failure to open and close the jaws and swallow:
  - Anatomical/physical
  - Functional/neuromuscular

RELATED CLINICAL SIGNS

- Coughing and/or nasal discharge if secondary inhalation
- Drooling (hypersalivation or failure to swallow saliva)
- Extension or lowering of head and neck
- Failure to prehend food, dropping food from mouth
- Halitosis if retained food
- Pain on opening mouth

COMMON CAUSES

Structural or functional diseases of the mouth and/or temporo-mandibular joint and/or pharynx:
- Dental and/or periodontal disease
- FB usually wedged between dental arcades
- Mandibular fracture/luxation
- Oral neoplasia including tonsillar squamous-cell carcinoma, *q.v.* section 2.16
- Stomatitis and oral ulceration
- Pharyngitis/tonsillitis

- Retropharyngeal abscess or lymphadenopathy
- Temporal myositis

Oesophageal disorders

- More typically causes regurgitation, *q.v.* section 1.41

UNCOMMON CAUSES

- Cleft palate
- Craniomandibular osteopathy
- Cricopharyngeal achalasia
- Linear FB under the tongue
- Lingual frenulum disorder – 'tongue tie'
- Mandibular osteomyelitis
- Neuromuscular disorders
  - Botulism
  - Hypothyroid neuropathy
  - Mandibular neuropraxia
  - Muscular dystrophy
  - Myasthenia gravis
  - Peripheral neuropathy
  - Polyradiculoneuritis/tick paralysis
  - Tetanus
  - Trigeminal neuritis or sensory neuropathy
- Nutritional secondary hyperparathyroidism ('rubber jaw')
- Rabies and Aujeszky's disease (pseudorabies)
- Salivary gland disease
  - Hypersialosis/sialoadenosis – phenobarbital responsive
  - Sialoadenitis
  - Salivary gland infarction
- Temporo-mandibular joint disease: dysplasia/jaw locking, fracture/dislocation
- Uraemic stomatitis

DIAGNOSTIC APPROACH

1 Distinguish between regurgitation and vomiting.
2 Confirm difficulty in swallowing by observation.
3 Distinguish morphological causes from functional causes by oropharyngeal inspection and imaging.

CLINICAL CLUES

Predisposition

- Craniomandibular osteopathy: WHWT
- Muscular dystrophy: Bouvier, CKCS
- Trauma in younger dogs
- Uraemia, neoplasia in older dogs

History

- Concurrent signs of renal failure (anorexia, PU/PD), if uraemic ulcers
- Mandibular neuropraxia may follow excessive chewing of bones, etc. and leads to failure to close jaw
- Potential exposure to rabies is important outside UK
- Swollen painful muscles precede atrophy in temporal myositis
- Trauma (road traffic accident, or fall from height) can cause oral fractures/luxations

Clinical examination

Visual inspection

- Observe patient eating to confirm dysphagia and assess jaw motion

Physical examination

NB: Rabies should always be considered as a potential diagnosis in endemic areas before physical examination.

- Auscultation
  - Chest for secondary inhalation pneumonia
  - Throat for upper airway obstruction
- Check extent of jaw tone, width of opening and presence of pain on opening mouth
- Check neurological function (jaw and tongue movements, gag reflex, trigeminal sensation) if no structural disease is obvious *before* sedation/GA
- Oral examination checking under tongue for linear FB complete examination may require sedation/GA
- Mandibular, retropharyngeal and cervical LNs if oral mass present
- Salivary glands for pain or swelling
- Temporal muscles for swelling or atrophy
- Tonsils may be hard if neoplastic

Laboratory findings

Haematology and serum biochemistry

- Creatine kinase activity increased in myositis
- Look for inflammatory and systemic diseases

Oral and pharyngeal sampling

- Swabs for culture rarely helpful
- Touch impressions, FNAs and biopsy for discrete lesions/masses and LNs

Imaging

Radiographs

- Plain
- Chest radiographs essential if oral mass present
- Head and neck radiographs only helpful for structural abnormalities
- Contrast
  - To assess swallowing function
  - Barium swallow, preferably with fluoroscopy, for functional dysphagia

Special tests

- Acetylcholine receptor antibody titre for myasthenia gravis
- CSF tap
- Electromyography (EMG) and muscle biopsy
- Thyroid function tests

1.18 DYSPNOEA/TACHYPNOEA

DEFINITION

Respiratory distress manifested as an inappropriate degree of breathing effort, reflected by changes in respiratory rate, rhythm and character.

- Exertional, paroxysmal or continuous
- Orthopnoea indicates breathing difficulty whilst in a recumbent position
- Tachypnoea refers to an increased rate of breathing but does not necessarily indicate dyspnoea; it may be seen with exercise, fear or pain

RELATED CLINICAL SIGNS

- Coughing may indicate airway or pulmonary disease, *q.v.* section 1.13
- Cyanosis, *q.v.* section 2.6
- Exercise intolerance, *q.v.* section 1.22
- Increased expiratory effort: lower airway obstruction
- Increased inspiratory effort: upper airway obstruction
- Lethargy
- Open-mouth breathing
- Pallor suggests anaemia or haemorrhage

COMMON CAUSES

Haematological disorders

- Anaemia

Lower airway disorders

- Bronchial disease
  - Chronic bronchitis
  - Eosinophilic bronchopneumopathy
- Left atrial enlargement causing bronchial compression
- Lungworm/heartworm
  - *Angiostrongylus*
  - *Dirofilaria*: not in UK
- Tracheal collapse

Mediastinal disorders

- Neoplasia

Peritoneal cavity disorders

- Ascites (severe)
- Gastric dilatation-volvulus (GDV)
- Organomegaly
- Pregnancy

Pleural/body wall disorders

- Diaphragmatic rupture
- Pleural effusion
- Thoracic wall trauma/pneumothorax

Pulmonary parenchymal disorders
- Allergic/immune-mediated disease, e.g. eosinophilic pneumonopathy
- Bronchiectasis
- Metastatic neoplasia
- Pneumonia
  - Aspiration
  - Infectious
  - Parasitic
- Pulmonary oedema
  - Heart failure
  - Neurogenic
    - Asphyxiation due to airway obstruction
    - CNS trauma
  - Shock
  - Toxic
- Pulmonary thromboembolism (PTE): increasingly common but probably still under-recognised
  - DIC
  - HAC
  - Heartworm
  - Neoplasia
  - PLE
- Trauma causing bleeding disorder

Upper airway disorders
Brachycephalic obstructive airway syndrome (BOAS)
- Everted laryngeal saccules
- Laryngeal collapse
- Over-long soft palate
- Stenotic nares
- Tracheal hypoplasia

Laryngeal disease
- Laryngeal oedema
- Laryngeal paralysis

Tracheal disease
- Collapse
- Tracheal FB

UNCOMMON CAUSES

Haematological disorders
- Methaemoglobinaemia

Lower airway disorders
Extraluminal intrathoracic tracheal and/or bronchial compression
- Heart base mass
- Lymphadenopathy

Tracheal diseases affecting thoracic trachea
- As for cervical trachea below

Mediastinal disorders
- Mediastinal mass
- Mediastinitis
- Pneumomediastinum

Nasal cavity obstruction
- Neoplasia
- Rhinitis, *q.v.* sections 1.32, 5.10.1

Peritoneal cavity disorders
- Organomegaly/morbid obesity

Pleural/body wall disorders
- Congenital body wall disorder
- Peritoneo-pericardial diaphragmatic hernia (PPDH)
- Thoracic wall trauma/neoplasia/paralysis

Pulmonary parenchymal disorders
- Broncho-oesophageal fistula
- Distemper
- Lung lobe torsion with pleural effusion
- Lungworm
  - *Crenosoma vulpis*
  - *Eucoleus aerophilus (Capillaria aerophila)*
- Non-cardiogenic pulmonary oedema
- Paraquat poisoning
- Primary pulmonary neoplasia
- Smoke inhalation

Upper airway disorders
Cervical tracheal disease
- Neoplasia very rarely
  - Chondrosarcoma
  - Lymphoma
  - Osteosarcoma
  - Squamous cell carcinoma
- Tracheal parasites:
  - *Filaroides hirthi*
  - *Oslerus osleri*
- Trauma or stricture

Laryngeal disease
- Laryngeal neoplasia

Miscellaneous
- Central nervous system disorder
- Fear/pain
- Hyperthyroidism causing tachypnoea
- Metabolic acidosis
- Obesity hypoventilation (Pickwickian) syndrome
- Peripheral nerve, neuromuscular, muscular disorder

DIAGNOSTIC APPROACH

1 The over-riding concern is not to make dyspnoea worse by investigations.
2 Determine whether there is inspiratory or expiratory dyspnoea or airway noise.
3 Check for upper airway obstruction and treat.
4 Characterise intra-thoracic disease by imaging and investigate appropriately.
5 Discrimination between cardiogenic and non-cardiogenic causes of dyspnoea

- History: suspicion of upper airway obstruction, electrocution, seizures for non-cardiogenic oedema
- Physical examination: heart murmur, jugular pulsation for cardiogenic oedema
- Evaluation for cardiac disease: cardiac size and pulmonary vasculature on radiographs, point of care ultrasound scan or echocardiography

Clinical clues
Predisposition
- BOAS: brachycephalics
- Congenital disorders usually less than 1 year old
- Idiopathic pulmonary fibrosis: WHWT
- Laryngeal paralysis: old, large-breed dogs, especially retrievers, setters
- Metastatic pulmonary neoplasia: usually older animals, e.g. haemangiosarcoma, mammary, prostatic
- Tracheal collapse: small-breed, middle-aged to old dogs
- Tracheal hypoplasia: BOAS breeds

History
- Environment/geographical location, e.g. access to potentially infectious/toxic agents
- History of trauma recent or old: undiagnosed diaphragmatic rupture
- Regurgitation or vomiting and consequent aspiration, *q.v.* sections 1.41 and 1.49

Clinical examination
Visual inspection
- Abnormal discharge, deformities or lesions
- Cyanosis
- Oculo-nasal discharge
- Orthopnoea suggests effusion or heart failure
- Pattern of dyspnoea, e.g. inspiratory vs expiratory
- Rate and rhythm of respiration
- Restrictive vs obstructive

Physical examination
- Palpation
  - Abdomen: feels 'empty' with ruptured diaphragm
  - Compressibility of the cranial mediastinum in small dogs; loss of 'spring' can suggest a mass lesion
  - Larynx/trachea
  - Ribs for evidence of trauma, fractures, masses
  - Thoracic wall percussion for alteration in resonance, e.g. increased with pneumothorax, decreased with thoracic fluid or consolidated tissue
- Auscultation
  - Airway noise suggests fluid or solid obstruction
  - Cardiac auscultation for arrhythmias, murmurs, tachycardia
  - Change in voice if disease of larynx or affecting recurrent laryngeal nerve
  - Crackles suggest small airway disease
  - 'Fluid line' with effusions: increased breath sounds dorsally and muffled ventrally
  - Heart sounds may be muffled with pleural or pericardial fluid
  - Normal or abnormal breath sounds localise airway obstruction
  - Wheezes suggest large airway disease
- Percussion
  - Dull

  - Pleural or pericardial effusion
  - 'Fluid line' with effusions
  - Lung consolidation
- Hyper-resonant
  - Pneumothorax

LABORATORY FINDINGS

- Bacterial bronchopneumonia: may be leukocytosis, left shift and degenerate neutrophils
- Coagulation panel helpful if bleeding disorder, disseminated intravascular coagulation
- Dark, brown blood with Heinz body anaemia suggests methaemoglobinaemia
- Eosinophilia may suggest parasitic disease or eosinophilic disorder
- Often within normal limits

IMAGING

- Airway disease, *q.v.* section 4.3
- Fluoroscopy to investigate dynamic airway collapse
- Nasal lesions, see nasal discharge/sneezing, *q.v.* sections 1.32, 1.43
- Ultrasonography to identify pulmonary/mediastinal mass lesions ± ultrasound-guided aspirate/biopsy

SPECIAL TESTS

- Angiography
  - CT
  - Nuclear perfusion studies to investigate pulmonary vascular disease
- Blood gas analysis for pulmonary thromboembolism, plus tests of coagulation, metabolic disorders, and parenchymal diseases
- Bronchoscopy and BAL as for coughing if suspect airway ± pulmonary parenchymal disease
- Examination of laryngeal/pharyngeal region under anaesthesia
- Exploratory thoracotomy and lung biopsy
- Rhinoscopy, pharyngoscopy
- Specific tests for parasites
- Thoracocentesis and fluid analysis (cytology ± culture)

1.19 DYSURIA

DEFINITION

Dysuria (difficulty urinating) may be manifested as pollakiuria (frequently urinating small amounts) and/or stranguria (straining to urinate or pain on urination). Dysuria typically occurs with disorders of the lower urinary tract or its innervation.

RELATED CLINICAL SIGNS

Major signs

- Pollakiuria
  and/or
- Stranguria

Other potential signs

- Dyschezia
- Haematuria
- Nocturia
- Signs of systemic illness or neurological disease depending on the cause
- Urinary incontinence

COMMON CAUSES

Lower urinary tract disorders

- Bladder disorders
  - Bacterial cystitis
  - Neoplasia, e.g transitional (urothelial) cell carcinoma
  - Uroliths
- Urethral disorders
  - Neoplasia: transitional cell carcinoma of the bladder trigone
  - Uroliths
- Prostatic disorders
  - Prostatic carcinoma
  - Paraprostatic cysts, prostatitis, prostatic abscess in male entire dogs

Neurological causes
- Bladder atony
  - Secondary to lower motor neuron or upper motor neuron disorders
  - May occur due to urinary retention of any cause, and may impair recovery as it can be irreversible

UNCOMMON CAUSES

Lower urinary tract disorders
- Bladder disorders
  - Neoplasia: transitional cell carcinoma of the urethra
  - Polypoid cystitis
  - Sterile cystitis related to cyclophosphamide administration
- Urethral disorders
  - Proliferative urethritis
  - Urethral stricture
  - Transmissible venereal tumour
- Prostatic disorders
  - Benign prostatic hyperplasia: although common, it does not commonly cause dysuria
- Vaginal disorders
  - Neoplasia (leiomyoma, leiomyosarcoma)

Neurological causes
- Reflex dyssynergia
- Dysautonomia

Structural
- Perineal hernia with bladder retroflexion

DIAGNOSTIC APPROACH

Clinical clues
Predisposition
- Urinary incontinence, predisposing to bacterial cystitis is more likely with certain signalments
  - Urinary sphincter mechanism incompetence (USMI) can be detected in any dog, however most common in female neutered dogs
  - Young dogs, in particular Golden retrievers, increases the suspicion of ectopic ureter(s)
  - Neuter status influences risk of prostatic disorders
    - Benign prostatic hyperplasia in male entire dogs
    - Prostatic carcinoma in male neutered dogs

History
- Pollakiuria should be discriminated from polyuria (increased volume of urination)
- In dogs with bacterial cystitis an emphasis should be placed on establishing risk factors that may have predisposed to the development of infection:
  - Anatomic causes
    - Juvenile vaginitis in puppies
    - Hooded vulva in female dogs
    - Urinary incontinence: ectopic ureter, USMI
  - Immunosuppression
  - Presence of systemic disease (HAC, DM)
- Concurrent dyschezia increases suspicion of an intrapelvic mass lesion: enlarged prostate or lymph nodes

Clinical examination
Visual inspection
- Measuring bladder size (using ultrasonography) following voiding may help establish whether the dog is able to void their bladder; if it remains large, obstructive and neurological causes are more likely
- Observing urination patterns can be informative: dogs with reflex dyssynergia typically initiate urination as normal; however, the urinary stream abruptly stops due to lack of synchrony between bladder contraction and urethral sphincter relaxation: the same pattern can also be observed with urethral obstruction

Physical examination
- Passing a urinary catheter to assess for ease of passing aids assessment for urethral obstructive disorders: can be done in conscious male dogs but likely requires sedation or anaesthesia for female dogs
- Rectal examination to assess the urethra, and prostate in male dogs
- Thorough examination for risk factors for development of bacterial cystitis should be performed in patients, in particular assessing

conformation: examine for hooded vulva, perivulval inflammation or urinary pooling in female dogs or prostatomegaly in male dogs

Neurological examination
- Ability to express bladder is useful in dogs with concurrent neurological deficits
- Assessment for concurrent neurological deficits

Laboratory findings

Haematology
- Useful to screen for systemic disease, uncommon to detect abnormalities

Serum biochemistry
- Useful to screen for systemic disease, may detect azotaemia in dogs with post-renal causes (urethral obstruction) or renal causes (extension of bacterial cystitis to pyelonephritis)

Urinalysis
- Urine culture (ideally from cystocentesis sample, otherwise interpret positive culture with caution)
- Urine cytology to assess for neoplastic cells
- Urine sediment for evidence of urinary tract infection or neoplastic cells
- Urine specific gravity to assess for polydipsia and polyuria

Imaging

Radiographs

May need to do an enema first to fully assess urinary system for uroliths. Ensure that the whole length of urethra is included; move hindlimbs cranially to assess perineal urethra in males.
- Assess for radiopaque uroliths, prostate
- Contrast studies: retrograde urethrogram (plain radiographs or fluoroscopy) and intravenous excretory urogram

Ultrasound
- Assess kidneys, ureters, urinary bladder and prostate

Special tests
- CT with contrast for urinary system assessment: intravenous urogram with iodinated contrast improves sensitivity for ectopic ureter compared to plain radiography
- Cystoscopy to assess urethra, bladder, and sites of implantation of ureters
- MRI and CSF for dogs with neurolocalisation
- Prostatic wash and fine needle aspiration for cytology and culture
- Urinary BRAF mutation analysis: presence of BRAF mutation detected in ~80% of dogs with transitional cell carcinoma
- Urodynamic pressure profilometry

1.20 DYSTOCIA

DEFINITION

Difficulty with vaginal delivery of fetuses; can result in morbidity and mortality for fetuses, neonates and bitch.

RELATED CLINICAL SIGNS

- Abnormal vulval discharge
- Failure for labour to start or progress
- Maternal distress
- Protracted straining, non-productive
- Stillborn or weak puppies

COMMON AND UNCOMMON CAUSES

- Fetal dystocia
  - Malpositioned/postured
  - Oversized
- Fetal/maternal dystocia
  - Mismatch of birth canal to fetal size, e.g. brachycephalic/hydrocephalic breeds
- Maternal dystocia
  - Abdominal wall defects (hernia)
  - Birth canal defects (stricture)
  - Metabolic disease
  - Poor lubrication in pelvic/birth canal

- Primary: no myometrial contractions
- Secondary: uterine inertia
- Severe vulvar oedema
- Uterine torsion

DIAGNOSTIC APPROACH

Clinical clues

Predisposition

- Abdominal wall defects, e.g. hernia
- Brachycephalic and hydrocephalic breeds
- Metabolic derangements in bitch, e.g.
  - DM
  - Hypocalcaemia
  - Hypoglycaemia
  - Hypovolaemia
  - Pre-eclampsia
  - Pregnancy toxaemia
  - Sepsis/systemic inflammatory response syndrome (SIRS)
- Obesity of bitch
- Pelvic canal narrowing, e.g. after fracture
- Poor condition of bitch
- Prolonged gestation can result in fetal overgrowth, e.g. small litters
- Some breed lines
- Uterine inertia, e.g. large litters
- Vaginal canal abnormalities (stricture, vaginal hyperplasia, oedema)

History

- Difficulty with previous labour
- Failure for labour to start on expected date
- Prolonged time between stages of labour
  - Stage 1, increased frequency and strength myometrial contractions and progesterone and temperature drops, normally 12–24 hours
  - Stage 2, co-ordination of abdominal and myometrial contractions and delivery of fetus, normally 8 hours
  - Stage 3, expulsion of placenta, normally 12–24 hours
- Prolonged time between delivery fetuses (normally < 1 hour)
- Stillborn or weak puppies

Clinical examination

Visual inspection

- Can be normal
- Abnormal vulval discharge e.g. green, malodorous, haemorrhagic
- Maternal distress: agitation, trembling, hyperpnoeic
- Nesting behaviour
- Persistent straining

Physical examination

- Can be normal, i.e. normothermia, and slight hypothermia can be normal
- Fatigue/weak
- Fetus stuck in birth canal
- Moderate to severe pain
- Muscle tremors/tetany
- Retention of placenta
- Vomiting

Laboratory findings

Haematology

- Mildly decreased HCT normal at due date

Serum biochemistry

- Hypocalcaemia can lead to uterine inertia
- Hypoglycaemia can lead to uterine inertia
- Mildly decreased total protein at due date

Urinalysis

- Glucose or ketones can indicate DM or pregnancy toxaemia

Imaging

Plain radiographs

- Evaluate litter size
- Assess relative fetal size
- Assess for obstruction/malposition

Ultrasound/Doppler

- Evaluate fetal viability
  - Normal fetal heart rate > 200 bpm
  - Fetal distress results in bradycardia

Special tests

- Review gestational length: error in expected due date could mean a lack of labour is normal
- Vaginal examination: digital or via scope to assess for obstruction and whether cervix is dilated
- Uterine pressure monitoring (tocodynamometry), if available

1.21 EPISTAXIS

DEFINITION

Haemorrhage from the nose or nasopharynx. Typically this drips out of the rostral nares but it can also be swallowed, especially with more caudal lesions.

RELATED CLINICAL SIGNS

- Bleeding from rostral nares
- Haematemesis or melaena due, respectively, to vomiting or passage of swallowed blood may occur
- Nasal disorders may have concurrent nasal discharge (serous, mucopurulent), sneezing, nasal depigmentation/deformities, or pawing at face may be observed
- Systemic causes may have concurrent evidence of haemorrhage (petechiae, ecchymoses, melaena, haematuria); although primary haemostatic disorders are most likely to be responsible for epistaxis, secondary haemostatic disorders may also occasionally present with epistaxis.

COMMON CAUSES

Nasal disease

- Chronic rhinitis
- FB
- Infection
  - Oronasal fistula
  - Periapical tooth root abscess
  - Sino-nasal aspergillosis
- Neoplasia: carcinoma, sarcoma, lymphoma

Systemic disease

- *Angiostrongylus vasorum* infection causing a mixed haemostatic disorder
- Hypertension
- Primary haemostatic disorders
  - Thrombocytopenia: IMTP, bone marrow disease
- Secondary haemostatic disorders
  - Vitamin K deficiency (rodenticide toxicity)

UNCOMMON CAUSES

Nasal disease

- Infection
  - *Leishmania*
  - Osteomyelitis
  - Parasitic (*Linguatula serrata*)
- Neoplasia (leiomyoma)
- Trauma

Systemic disease

- Primary haemostatic disorders
  - Hyperviscosity due to erythrocytosis, hyperglobulinaemia in leishmaniosis or myeloma
  - Thrombocytopenia: consumptive (DIC), *Ehrlichia*
  - Thrombocytopathia: inherited, e.g. Glanzmann's thrombasthenia, Scott syndrome
  - von Willebrand factor deficiency (congenital)
- Secondary haemostatic disorders
  - Liver dysfunction (chronic hepatitis, toxicity e.g. xylitol)
  - Vitamin K deficiency (cholestasis, congenital)

DIAGNOSTIC APPROACH

Clinical clues

- Bilateral is more likely chronic idiopathic rhinitis, aspergillosis or bleeding disorder
- Unilateral is more likely a FB neoplasia, oronasal fistula or dental disease

Predisposition

- Breed predispositions to IMTP, e.g. Cocker spaniels
- Dolichocephalic dogs are predisposed to sino-nasal aspergillosis
- Glanzmann's thrombasthenia: Great Pyrenees, Otterhound
- Older dogs are predisposed to nasal tumours and dental disease
- Scott syndrome (inherited thrombocytopathia): GSD

History
- Assessing for presence of unilateral or bilateral nasal discharge is useful in prioritising differential diagnoses
  - Unilateral is more likely a FB neoplasia, oronasal fistula or dental disease
  - Bilateral is more likely chronic idiopathic rhinitis, aspergillosis or bleeding disorder)
- History of other bleeding sites may be useful, e.g. haematuria
- Presence of nasal stertor increases suspicion of nasal FB or tumour
- Recent dental extractions may increase suspicion of oronasal fistula
- Risk of exposure to anticoagulant rodenticide
- Travel history: increases index of suspicion of vector-borne diseases including *Ehrlichia* and *Leishmania* infection

Clinical examination
Visual inspection
- Assess for presence of sneezing (increases suspicion of nasal disease)

Physical examination
- Blood pressure to assess for hypertension
- Evidence of haemorrhage elsewhere, especially skin of the ventral abdomen, and gums, and rectal examination for melaena
- Nasal planum depigmentation most common in fungal disease
- Examination of head conformation including ocular retropulsion for signs of deformity suspicious for neoplasia
- Oral examination to assess for dental disease and oronasal fistula

Ophthalmic examination
- Hypertensive retinopathy indicating target organ damage

Laboratory findings
Haematology
- Coagulation times (PT and aPTT)
- Platelet count

Serum biochemistry
- Useful to screen for systemic disease, in particular evidence of hyperglobulinaemia

Imaging
Plain radiographs
- Nasal turbinate destruction: tumour or aspergillosis
- Soft tissue opacity: tumour most likely but can also occur with fluid accumulation
- Sinus hyperostosis: aspergillosis

Special tests
- *Aspergillus* serology; specific but poorly sensitive test, i.e. positive result supports infection but negative result does not exclude infection
- Baermann technique or Angiodetect® for *Angiostrongylus vasorum*
- Buccal mucosal bleeding time to assess for thrombocytopathia
- CT scan with contrast to assess for nasal diseases
- DNA test
  - Glanzmann's thrombasthenia
  - Scott syndrome
- Testing for *Leishmania* and *Ehrlichia* infection as appropriate
- von Willebrand factor antigen

1.22 EXERCISE INTOLERANCE

DEFINITION

Exercise intolerance is the decreased ability to perform physical exercise at the normally expected level or duration for dogs of that age, size and muscle mass.

RELATED CLINICAL SIGNS

- Muscle weakness/wasting
- Musculoskeletal pain
- Panting/tachypnoea

- Prolonged recovery from exercise
- Pyrexia
- Reduced stamina at exercise
- Reluctance to exercise

COMMON CAUSES

Cardiovascular disease
q.v. sections 1.18, 1.51, 5.2

Endocrine disease
- HAC
- Hypoadrenocorticism
- Hypothyroidism

Generalised weakness
- Anaemia
- Chronic inflammation/infection/wasting
- Drugs, e.g. anticonvulsants, antihistamines, diuretics, vasodilators, anti-arrhythmics
- Neoplasia
- Obesity
- Pyrexia

Metabolic disease
- Hypercalcaemia
- Hypo-/hyper-kalaemia, *q.v.* section 3.23
- Hypo-/hyper-glycaemia, *q.v.* section 3.17
- Hyponatraemia, *q.v.* section 3.24

Muscular disease
- Inherited myopathies

Neurological/spinal disease
- Cervical spondylomyelopathy (wobbler)
- Intervertebral disc protrusion
- Vestibular disease

Neuromuscular disease
- Myasthenia gravis

Respiratory disease
- *q.v.* sections 1.18, 1.51

Skeletal disease
- Cruciate ligament disease/rupture
- Degenerative joint disease

UNCOMMON CAUSES

Endocrine disease
- Diabetic ketoacidosis
- Phaeochromocytoma

Metabolic disease
- Acidosis
- Hepatic encephalopathy
- Malignant hyperthermia

Miscellaneous
- Nutritional deficiency
- Parasitism

Muscular disease
- Exercise-induced hyperthermia, *q.v.* section 2.25
- Muscular dystrophy
- Polymyositis
- Scottie cramp

Neurological/spinal disease
- Botulism
- Cerebellar disease
- Cervical myelopathy
- Discospondylitis
- Dyskinesia
- Fibrocartilaginous embolism
- Narcolepsy/cataplexy
- Neoplasia
- Peripheral polyneuropathies
- Spinal trauma

Skeletal disease
- Hypertrophic osteopathy (Marie's disease)
- Immune-mediated polyarthritis (IMPA)
- Panosteitis

DIAGNOSTIC APPROACH

1 Thorough history to discern any other clinical signs apart from exercise intolerance which itself is non-specific.
2 Complete physical examination covering all body systems.
3 Exercise test once cardiorespiratory diseases and lameness have been ruled out.

Clinical clues
q.v. sections 1.44, 1.51

Predisposition
- Centronuclear myopathy: Labrador retriever
- Cervical spondylomyelopathy: Dobermann
- Exercise-induced hyperthermia: Border collie
- Mitochondrial myopathy: Clumber and Sussex spaniels
- Scottie cramp: Scottish terrier

Clinical examination
Visual inspection
- Normal at rest, but dyspnoea/tachypnoea after exercise
- Gradual slowing down or ataxic on exercise

Physical examination
- Musculoskeletal pain
- Hyperthermia

Laboratory findings
- Elevated creatine phosphokinase (CPK)/aspartate aminotransferase (AST) in muscle disease e.g. polymyositis
  q.v. section 3.12

Imaging
q.v. sections 1.44, 1.51

Special tests
q.v. sections 1.44, 1.51
- DNA test for Labrador myopathy
- Malignant hyperthermia – exercise test will induce hyperlactacidaemia, hyperthermia, haemoconcentration mild respiratory alkalosis after a short period of exercise (*cf.* normal animals after long, strenuous exercise)
- Scottie cramp: administer serotonin antagonist to induce an episode in affected dogs, i.e. give 0.3 mg/kg orally of methysergide and exercise 2 hours later

1.23 FAECAL INCONTINENCE

DEFINITION

The involuntary passage of faecal material, due to an inability to retain faeces.

The cause can be
- Anatomical, as the result of reduced capacity or compliance of the rectum
- Neurological sphincter mechanism incontinence

RELATED CLINICAL SIGNS

- Dyschezia
- Haematochezia
- Involuntary passage of faeces
- Perineal staining
- Tenesmus
- Other signs of neurological deficits

COMMON CAUSES

Anal disease
- Anal furunculosis
- Associated with constipation
  - perineal hernia
  - anal sac disease

Neurogenic sphincter mechanism incontinence
- Lumbosacral disease
  - Canine degenerative myelopathy (CDM)
  - IVDD

UNCOMMON CAUSES

Neurogenic sphincter incontinence
- Congenital malformation
- Previous local surgery
  - Attempted perineal hernia repair
  - Damage to anal sphincter during anal sacculectomy
  - Rectal pull-through procedure

- Fibrocartilaginous embolism (FCE)
- Polyneuropathy
- Sacrocaudal dysgenesis (brachycephalic breeds)
- Spina bifida
- Spinal arachnoid cysts
- Spinal trauma

Neoplasia
- Perineal, rectal or colonic

Myopathy
- Polymyopathy

DIAGNOSTIC APPROACH

1 Distinguish inappropriate defaecation (e.g. improper house training) from true incontinence by observing defaecation.
2 Rule out rectal/anal disease by the presence of straining and physical findings on digital rectal examination.
3 Evaluate local (anal tone) and general neurological function if there is no obvious anatomical cause.

Clinical clues
Predisposition
- Lumbosacral disease and canine degenerative myelopathy (CDM) classically in GSDs, but now recognised in numerous breeds
- Perineal hernia in intact male dogs

History
- Chronic progressive with most neurological disease except FCE and trauma
- Tenesmus, dyschezia and haematochezia with local rectal/anal disease

Clinical examination
Visual inspection
- Other neurological deficits – tail carriage, hindleg ataxia, etc.
- Perineal masses, bulges

Physical examination
- Postural reflex testing
- Rectal examination to determine anal tone and local disease

Laboratory findings
- Usually unremarkable

Imaging
Plain radiographs
- Plain
- Vertebral abnormality

Contrast radiographs
- Myelography is often inadequate to image lumbosacral disease
- Epidurography and MRI scan are preferred for lumbosacral disease

Special tests
- Biopsy of any mass(es)
- Electromyography
- Genetic test for superoxide dismutase mutation in some cases of degenerative myelopathy
- Lumbar CSF collection

1.24 FLATULENCE/BORBORYGMI

DEFINITION

Flatulence is the accumulation of gas within the GI tract. Borborygmi are rumbling/gurgling sounds caused by GI contractions mixing and moving fluid and gas.

Some gas production in the GI tract is a normal physiological process, but excess production can be the result of GI disease or ingestion of non-absorbable fermentable substances.

- The gas may be:
  - Caused by bacterial fermentation
  - Gas diffusing from the blood
  - Swallowed air
  - The product of luminal chemical reactions (acid-alkali reactions)

- The presence of flatulence can be detected by finding abdominal distension or by auscultation
- Whilst eructation is the release of accumulated gas via the mouth, flatus is the release of gases via the anus. These are often odiferous because they contain volatile molecules such as ammonia, hydrogen sulfide, indoles, skatoles, volatile amines and short-chain fatty acids produced by bacterial metabolism.

RELATED CLINICAL SIGNS

- Abdominal discomfort
- Audible borborygmi
- Bloating
- Eructation (belching)
- Flatus

COMMON CAUSES

Aerophagia
- Dysphagia, *q.v.* section 1.17
- Dyspnoea, especially in BOAS, *q.v.* section 1.18
- Rapid/competitive eating

Diseases causing malabsorption and alterations in microbiome
- CIE/IBD
- Exocrine pancreatic insufficiency
- Intestinal dysbiosis/antibiotic-responsive diarrhoea/small intestinal bacterial overgrowth

Ingestion of non-absorbable substances
- Fermentable carbohydrates such as stachyose and raffinose found in soya and fermentable fibres

UNCOMMON CAUSES

- Administration of calcium carbonate to neutralise gastric acid or to treat hypocalcaemia, releasing carbon dioxide
- Diffusion of gas from the bloodstream
- Unimportant in dogs except in GDV
- Extreme exercise, causing aerophagia
- Ileus causing stasis of gut contents and secondary bacterial fermentation
- Ingestion of carbonated drinks
- Lactase deficiency: theoretical but never documented in dogs
- Lactulose

DIAGNOSTIC APPROACH

1 Obtain a full dietary history.
2 Auscultate and image chest if the dog is dyspnoeic.
3 Palpate and image the abdomen.
4 Investigate underlying causes of GI disease.

Clinical clues
The presence of audible borborygmi can be normal, especially in unfed dogs, with the sound mainly originating from gastric motility. As an isolated finding in an otherwise asymptomatic dog, increased borborygmi are not a concern. Excessive borborygmi in conjunction with flatus is abnormal, but the pattern of gut sounds is not pathognomonic for any specific disease.

Predisposition
- BOAS in brachycephalic dogs
- Chronic diarrhoea, *q.v.* section 1.15.2

History
- Audible gut sounds
- Concurrent diarrhoea
- Episodes of abdominal bloating and/or discomfort
- Repeated belching
- Odiferous flatus
- Soya-based food or unusual diet, e.g. beans, cabbage, lentils, Brussels sprouts

Clinical examination
Visual inspection
- Brachycephalic conformation
- Dyspnoea, tachypnoea
- Distended abdomen

Physical examination
- Abdominal distension or discomfort
- Borborygmi found on auscultation of abdomen
- Tympany on percussion in GD/GDV

Laboratory findings
- Usually unremarkable haematology and serum biochemistry except if significant primary GI disease
- Abnormal folate and/or cobalamin
- Low TLI in EPI

Imaging
Plain radiographs
- Alveolar pulmonary or pleural disease if dyspnoeic
- Gas-distended bowel loops

Ultrasound
- Excess intestinal gas

Special tests
- None

1.25 HAEMATEMESIS

DEFINITION

Vomiting of blood; *q.v.* section 1.49

The blood can be:
- Due to a generalised bleeding problem
  or
- Swallowed from oral, nasal or respiratory bleeding
  or
- From gastric and/or upper GI ulceration

RELATED CLINICAL SIGNS

- Epistaxis, bleeding oral lesion or coughing if swallowing pulmonary blood
- Generalised bleeding if coagulation disorder
- Vomiting of fresh or changed blood
  - Large volumes and very fresh blood will appear bright red
  - After a few minutes in gastric acid, blood is changed and the dog will vomit brown granular material ('coffee grounds') which owners may not recognise as blood

COMMON CAUSES

Endocrine
- Hypoadrenocorticism (not common but important)

Gastric ulceration
- Acute and chronic gastritis ± CIE/IBD
- Abrasive gastric FB
- Gastric carcinoma
- NSAID-induced ulceration, especially if in combination with steroids
- Portal hypertension in end-stage liver disease
- Uraemic gastritis

Generalised bleeding problem
q.v. section 1.9

GI disease
- Acute pancreatitis
- Parvovirus infection

Swallowed blood
- Epistaxis, *q.v.* section 1.21
- Oral bleeding

UNCOMMON CAUSES

- AHDS/HGE

Duodenal ulceration
- Neoplasia

Gastric ulceration
- Gastric leiomyoma/sarcoma
- Gastric lymphoma
- Gastrinoma
- High-dose steroids: dexamethasone more commonly than prednisolone
- Mallory-Weiss tear: a gastric mucosal tear due to violent vomiting
- Mast cell tumour

- Post-GDV
- Pythiosis (not in UK)

Shock
- Hypovolaemia
- Septic shock
- Neurogenic shock

Swallowed blood
- Following haemoptysis – bleeding lung lesion, usually carcinoma
- Ingestion of blood
- Oesophageal disease
  - Neoplasia
  - Severe oesophagitis
  - Trauma
    - Ingested FB
    - Stick injury

DIAGNOSTIC APPROACH

1 Distinguish vomiting of gastric blood from a generalised bleeding problem or swallowed blood.
2 Treat symptomatically but investigate by imaging and endoscopy, etc. if severe or not improving.

Clinical clues
Predisposition
- Gastric carcinoma more common in 7- to 10-year-old Belgian shepherds (Tervuren), Bull terriers, Chow Chows and Collies

History
- Anorexia and weight loss if severe gastric ulceration and especially if gastric carcinoma
- Diarrhoea and weight loss with gastrinoma
- Known administration of NSAIDs or steroids, or exposure to infection or toxins

Clinical examination
Visual inspection
- Bleeding at other sites if generalised coagulopathy
- Epistaxis or oral bleeding
- 'Prayer position' if cranial abdominal pain due to ulceration or pancreatitis

Physical examination
- Skin mass(es) which may be a mast cell tumour
- Palpation
  - Cranial abdominal pain in acute pancreatitis, deep gastric ulceration
  - Cranial abdominal mass

Laboratory findings
- Anaemia and hypoproteinaemia if severe bleeding
- Microcytic anaemia with thrombocytosis if chronic bleeding and iron deficiency
- Leukocytosis if pancreatitis or peritonitis is associated with incipient or early ulcer perforation
- Amylase/lipase is unreliably increased in acute pancreatitis

Imaging
- *q.v.* section 1.49

Special tests
- Buccal mucosal bleeding time, clotting profile
- Spec cPL for pancreatitis
- As for vomiting, *q.v.* section 1.49

1.26 HAEMATOCHEZIA

DEFINITION

The presence of fresh blood in faeces.
- Usually an indicator of LI or perianal disease
- Rarely seen with SI haemorrhage, and only if this is massive and/or the rate of intestinal transit is increased

RELATED CLINICAL SIGNS

- Abnormal stool shape/size if rectal mass
- Concurrent diarrhoea, often with mucus if colitis
- Normal stool consistency with blood on surface if focal lesion, e.g. rectal polyp
- Passage of bright red, fresh blood

COMMON CAUSES

Anal disease
- Anal sac infection
- Anal sac adenocarcinoma
- Perianal adenoma

Generalised bleeding disorder
q.v. section 1.9

Generalised GI disease
- AHDS/HGE
- Bacterial enteritis

Large intestinal disease
- Colitis
  - Idiopathic CIE/IBD
  - Food-responsive enteropathy
- Foreign material, e.g., bones
- Ileo-colic intussusception
- Rectal prolapse
- Rectal polyps
- Colonic or rectal neoplasia

UNCOMMON CAUSES

Large intestinal disease
- Acute neurological trauma causing colonic ulceration/perforation
- Infection; whipworms and hookworms are more uncommon in the UK but more common in subtropical and tropical regions, and in countries where routine deworming is not practised
  - Amoebiasis: not in UK
  - *Ancylostoma* hookworms (rare in UK)
  - Histoplasmosis (restricted geographically, i.e., not in UK)
  - Protothecosis
  - *Trichuris* whipworms
  - *Uncinaria* hookworms
- Blood vessel malformation (e.g. colonic angiodysplasia/vascular ectasia)
- Caecal inversion
- High-dose glucocorticoids
- Perianal carcinoma

DIAGNOSTIC APPROACH

1 Rule out generalised bleeding.
2 Localise problem to external anus or distal intestine.
3 Digital rectal examination.
4 Proctoscopy/colonoscopy.

Clinical clues
Predisposition
- Rectal prolapse is most common in young dogs with colitis

History
- Absence of diarrhoea helps rule out colitis
- Spotting of blood between bowel movements indicates anal disease or lesion close to anus

Clinical examination
Visual inspection
- Anal masses
- Bleeding at other sites if generalised
- Dyschezia if anal inflammation
- Protrusion of severe ileo-colic intussusception
- Rectal prolapse
- Tenesmus if inflammatory or neoplastic rectal disease

Physical examination
- Abdominal palpation
  - Colonic mass
- Digital rectal palpation
  - Anal sac disease
  - Rectal polyp or neoplasia

Laboratory findings
- Haematology and serum biochemistry usually unremarkable except for changes due to blood loss (anaemia, hypoproteinaemia)

Faecal examination
- Culture rarely helpful
- Parasitology for whipworms and hookworms

Imaging
- Sublumbar lymphadenopathy and/or periosteal reaction on lumbar vertebrae if metastasis from anal sac or rectal tumour
- Ultrasound examination limited by colonic gas

Special tests
- Barium enema (rarely performed)
- Proctoscopy/colonoscopy

1.27 HAEMATURIA AND DISCOLOURED URINE

DEFINITION

Haematuria may be observed as a gross change in urine colour or detected due to microscopic haematuria detected on dipstick or sediment analysis. Discolouration of urine can be caused by the presence of intact RBCs or free haemoglobin and other pigments including myoglobin.

RELATED CLINICAL SIGNS

- Haematuria or any change in urine colour may be the only clinical sign in some dogs.
- It may be in combination with signs of dysuria, *q.v.* section 1.19
- There may be concurrent evidence of systemic disease or bleeding diathesis. Some dogs with significant haemorrhage may even pass blood clots during their urine stream. As with any cause of bleeding, causes can be divided into disorders of the urinary tract or related to a disorder of haemostasis (systemic disease).
- Haematuria can be discriminated from haemoglobinuria (both will cause reddened urine) by sediment examination after allowing the urine to sediment, ideally, after centrifugation. Red blood cells will form a pellet whereas with haemoglobinuria the urine will remain tinged red with no pellet. A fresh sample should be examined as RBCs will lyse spontaneously in stored urine samples.

COMMON CAUSES – HAEMATURIA

Urinary tract disease
- Renal haemorrhage
  - Pyelonephritis
  - Trauma
- Bladder haemorrhage
  - Bacterial cystitis
  - Sampling method: cystocentesis sampling may result in mild bleeding into the sample
  - Trauma
  - Urolithiasis
- Urethral haemorrhage
  - Neoplasia (transitional cell carcinoma)
- Prostatic haemorrhage
  - Prostatic carcinoma (male neutered dogs)
  - Prostatitis (male entire dogs)
- Vaginal haemorrhage
  - Oestrus (female entire dogs)

Systemic disease
- Primary haemostatic disorders
  - Thrombocytopenia: IMTP
  - Thrombocytopathia: hyperviscosity due to hyperglobulinaemia, e.g. neoplasia, *Leishmania*
- *Angiostrongylus vasorum* infection causes a mixed haemostatic disorder

UNCOMMON CAUSES – HAEMATURIA

Urinary tract disease
- Renal haemorrhage
  - Idiopathic renal haemorrhage
  - Infarction

  - Neoplasia
  - Telangiectasia
  - Urolithiasis
- Ureteral haemorrhage
  - Neoplasia
  - Urolithiasis
- Bladder haemorrhage
  - Neoplasia (transitional cell carcinoma)
  - Polypoid cystitis
  - Sterile cystitis (cyclophosphamide)
- Urethral haemorrhage
  - Proliferative/granulomatous urethritis
  - Urolithiasis
- Vaginal haemorrhage
  - Subinvolution of placental sites (SIPs), metritis (post whelping female entire dogs)
  - Pyometra (female entire dogs)
  - Neoplasia (leiomyoma, leiomyosarcoma)

Systemic disease

- Primary haemostatic disorders
  - Thrombocytopenia: IMTP, *Ehrlichia*, consumptive (DIC), bone marrow disease
  - Thrombocytopathia (congenital, hyperviscosity due to erythrocytosis)
  - von Willebrand factor deficiency (congenital)
- Secondary haemostatic disorders (rare cause of haematuria, body cavity bleeds more likely)
  - Vitamin K deficiency (rodenticide toxicity)
  - Liver dysfunction (chronic hepatitis, toxicity e.g. xylitol)
  - Vitamin K deficiency (cholestasis, congenital)

COMMON CAUSES – DISCOLOURED URINE

Red or brown urine – see haematuria

- Haemoglobinuria also
  - IMHA (intravascular haemolysis)
  - Heat stroke

Dark yellow urine

- Bilirubinuria, *q.v.* section 2.13
- Concentrated urine

UNCOMMON CAUSES – DISCOLOURED URINE

Red or brown urine – also see haematuria

- Haemoglobinuria
  - Blood transfusion reaction (acute haemolytic)
  - Paracetamol (acetaminophen) toxicity
  - Snake envenomation
  - Splenic torsion
- Myoglobinuria
  - Rhabdomyolysis
  - Severe muscle trauma

Dark yellow urine

- Food dyes
- Drugs

DIAGNOSTIC APPROACH

Clinical clues

Predisposition

- Breed predispositions to IMHA and IMTP, e.g. cocker spaniels

History

- Concurrent haemorrhage elsewhere: increases index of suspicion of systemic disorder
- Signs of dysuria: increases index of suspicion of urinary tract disorder
- Travel history: increases index of suspicion of vector-borne diseases including *Ehrlichia* and *Leishmania* infections

Clinical examination

Visual inspection

- Observe urination – occurrence of haematuria may prioritise differential diagnoses but this must be interpreted in combination with other investigations
  - Haematuria at the beginning of the urinary stream implies genital tract or urethral disorders
  - Haematuria throughout the duration of the urinary stream occurs with many disorders (renal, ureteral, bladder, or systemic disorders, occasionally urethral and prostatic

disorders can also result in reflux of blood into the bladder)
- Haematuria during the terminal phase suggests a ventral bladder wall lesion and can also occur with intermittent renal haemorrhage (as the blood is pooled in the ventral bladder and expelled at the end)
- Haemorrhage occurring independent of urination implies a disorder of the distal urethra or genital tract

Physical examination
- Abdominal palpation in particular to assess for renal pain or asymmetry where possible
- Evidence of haemorrhage elsewhere: skin especially of the ventral abdomen, and gums, rectal examination for melaena
- Rectal examination to assess urethra, as well as prostate in males
- Vaginal examination in female dogs

Laboratory findings

Haematology
- Coagulation times (PT and aPTT) in secondary haemostatic disorders
- Evidence of haemolysis (spherocytes, ghost cells), and regenerative anaemia in IMHA
- Low platelet count in IMTP

Serum biochemistry
- Screen for systemic disease
  - Azotaemia may be detected in pyelonephritis (renal) or ureteric or urethral disorders (post renal)

Urinalysis
- Haematuria on voided urine samples but not cystocentesis samples suggests a urethral or prostatic (male)/vaginal (female) disorder
- Sediment examination
  - Active sediment in the presence of urinary tract infection (diagnosis of bacterial cystitis should prompt assessment for risk factors rather than symptomatic treatment alone)
  - Presence of crystals: not diagnostic of urolithiasis but increases index of suspicion
  - Neoplastic cells
  - RBCs (ideally fresh sample as red blood cells may lyse, especially in dilute or alkaline urine, resulting in a false impression of haemoglobinuria)
- Urine cytology: as above, but submission of an EDTA-preserved urine sample may complement routine sediment examination

Imaging

Plain radiographs
- Assess for radiopaque uroliths, prostate, ensure length of urethra included; may need to do an enema first to fully assess urinary system for uroliths
- Contrast studies retrograde urethrogram (plain radiographs or fluoroscopy) and intravenous excretory urogram

Ultrasound
- Assess kidneys, ureters, urinary bladder and prostate

Special tests

- For haemolysis
  - Coombs' test to aid diagnosis of suspected IMHA
- For haemostatic disorders
  - Buccal mucosal bleeding time for thrombocytopathia
  - von Willebrand factor antigen
  - Baermann technique or Angiodetect® for *Angiostrongylus vasorum*
  - Testing for *Leishmania* and *Ehrlichia* infection as appropriate
- For urinary tract disorders
  - CT scan with contrast
  - Prostatic sampling (wash or fine needle aspirate)
  - Urocystoscopy – assessment of vagina, urethra, prostatic urethra, bladder and implantation of ureters. In idiopathic renal haemorrhage this identifies the kidney responsible for the haemorrhage (ideally carried out during haemorrhagic event).

1.28 HAEMOPTYSIS

DEFINITION

Haemoptysis refers to coughing up blood or bloody sputum.

RELATED CLINICAL SIGNS

- Haemoptysis may be observed in dogs with signs of significant respiratory disease (coughing, tachypnoea, dyspnoea) or may occur in combination with evidence of a bleeding diathesis.
- Melaena may occur concurrently due to swallowed blood or concurrent gastrointestinal haemorrhage.
- As with any cause of bleeding, causes can be divided into disorders of the respiratory system or related to a disorder of haemostasis (systemic disease).

COMMON CAUSES

Respiratory tract disease

- Lower airway haemorrhage
  - *Angiostrongylus vasorum* (lungworm)
  - FB
  - Parenchymal lung disease
  - Neoplasia
    - Carcinoma
    - Haemangiosarcoma
- Pharyngeal and/or laryngeal haemorrhage
  - FB (stick injury)
  - Trauma
- Tracheal haemorrhage
  - FB
  - Trauma

Systemic disease

- Primary haemostatic disorders
  - Thrombocytopenia: consumptive (DIC)
- Secondary haemostatic disorders
  - Vitamin K deficiency: rodenticide toxicity

UNCOMMON CAUSES

Respiratory tract disease

- Lower airway haemorrhage
  - Infectious pneumonia: fungal, bacterial, protozoal, viral
  - Inflammatory disease: chronic bronchitis, eosinophilic bronchopneumopathy
  - Neoplasia: histiocytic sarcoma
- Pharyngeal and/or laryngeal haemorrhage
  - Neoplasia
  - Post-nasal drip: nasal or nasopharyngeal haemorrhage, *q.v.* section 1.21
- Tracheal haemorrhage
  - Neoplasia; chondrocarcinoma arising from tracheal ring
- Parenchymal lung disease
  - *Dirofilaria* (heartworm, increased suspicion in travelled dogs)
  - Leptospirosis (described in mainland European cases more commonly than elsewhere)
  - Pulmonary oedema can result in pink sputum (cardiogenic and non-cardiogenic)
  - Pulmonary thromboembolism

Systemic disease

- Primary haemostatic disorders
  - Thrombocytopenia: IMTP, *Ehrlichia*, bone marrow disease
  - Thrombocytopathia: hyperviscosity due to hyperglobulinaemia, e.g. neoplasia, *Leishmania*
- Secondary haemostatic disorders
  - Liver dysfunction: chronic hepatitis, toxicity e.g. xylitol
  - Vitamin K deficiency: cholestasis, congenital

DIAGNOSTIC APPROACH

Clinical clues

Predisposition

- Breed predispositions to immune-mediated thrombocytopenia (e.g. cocker spaniels)

History
- Concurrent haemorrhage elsewhere to increase suspicion of systemic disorder
- Coughing to increase suspicion of respiratory tract disease
- Risk factors for FB, e.g. throwing sticks or stick chewing
- Travel history increases index of suspicion of vector-borne diseases, including *Dirofilaria*, *Ehrlichia* and *Leishmania* infections

Clinical examination
Visual inspection
- Observe nature of cough if present
- Observe respiratory pattern to increase suspicion of upper, lower respiratory tract or parenchymal disorder; also may increase suspicion for presence of pleural space disease

Physical examination
- Thorough oral examination, and thoracic auscultation
- Evidence of haemorrhage: petechiation, ecchymoses elsewhere
  - Mucous membranes: gums, prepuce, vulva
  - Rectal examination for melaena
  - Skin, especially of the ventral abdomen

Laboratory findings
Haematology
- Assess platelet count
- Coagulation times (PT and aPTT) to assess for secondary haemostatic disorders
- Eosinophilia may be present in parasitic disease

Serum biochemistry
- Useful to screen for systemic disease

Imaging
Plain radiographs
- Assess for evidence of pulmonary changes

Ultrasound
- Point of care ultrasound to assess for pleural effusion ± ascites; may be detected in secondary haemostatic disorders, in particular rodenticide toxicity and neoplasia

Special tests
- Arterial blood gas analysis
- Bronchoscopy and BAL
- CT with contrast, preferably before bronchoscopy
- Haemostatic disorders
  - Buccal mucosal bleeding time to assess for thrombocytopathia
  - von Willebrand factor antigen levels
  - Baermann technique or Angiodetect® for *Angiostrongylus vasorum*
  - Testing for *Leishmania* and *Ehrlichia* infection as appropriate
  - Toxicology to detect rodenticide
- Leptospirosis serology and PCR
- Pulmonary thromboembolism can be challenging to diagnose: D-dimers, thromboelastography, CT with contrast may increase suspicion
- Screening for *Dirofilaria* (antigen testing e.g. SNAP 4DX® test), antibody testing, blood smear from marginal ear vein, or modified Knott's test)

1.29 HALITOSIS

DEFINITION

Halitosis is offensive, foul-smelling breath emanating from the mouth or nose. Oral diseases predispose to bacterial proliferation in necrotic tissue or retained food particles.

RELATED CLINICAL SIGNS

- Coughing if inhalation pneumonia
- Offensive breath
- Oral pain may indicate periodontal disease, inflammation or neoplasia
- Retained food associated with masses, ulcers
- Vomiting and/or diarrhoea if GI disease

COMMON CAUSES

- Poor oral hygiene

Abnormal ingestive behavior or pica
- Coprophagy, the ingestion of faecal matter
- Fetid food/scavenging
- High protein meals

Oral diseases
- Dental tartar or periodontal disease
- Food retention in lip folds, etc.
- Oral FB
- Oral neoplasia – beware a non-healing gingival lesion after tooth loss
- Stomatitis/pharyngitis

UNCOMMON CAUSES

- Open-mouth breathing

Abnormal ingestive behaviour or pica
- Aromatic foods: garlic, onion
- Chemical burns secondary to the ingestion of caustic substances
- Electrical burns secondary to chewing on electric cords

Oral diseases
- Cleft palate
- Jaw malocclusion
- Wet hair in bearded breeds
- Xerostomia

Oral contact with contaminated site
- Balanoposthitis
- Impacted anal sacs
- Infected/necrotic skin lesions, etc.
- Vaginitis

Remote causes producing malodorous exhalation
- Diabetic ketoacidosis
- GI disease
  - Gastritis
  - Intestinal obstruction
  - Malabsorption
  - Obstipation
- Liver disease
- Nasal disease
  - Chronic rhinitis/sinusitis
  - FB
  - Fungal infection: sino-nasal aspergillosis
  - Neoplasia
- Oesophageal disease: megaoesophagus with food retention
- Pulmonary disease
  - Abscessation
  - Aspiration pneumonia
  - Bacterial bronchopneumonia
  - Bronchiectasis
  - Broncho-oesophageal fistula
  - Inhaled FB
  - Necrotic tumour
- Uraemia

DIAGNOSTIC APPROACH

- History will help localise source of smell
- Biochemistry for metabolic disease including DKA and uraemia
- Investigations as for dysphagia (*q.v.* section 1.17) if an oral cause suspected, starting with oral examination

1.30 HEAD TILT

DEFINITION

Head tilt involves tilting of the head to either side of the body, away from its orientation with the trunk and limbs. The dog may appear to be trying to prevent itself from falling, or struggling to retain a balanced posture as cerebellar or vestibular disorders are responsible.

RELATED CLINICAL SIGNS

Head tilt should be discriminated from head turn and is most easily assessed by examining the position of the eyes and ears relative to one another when looking at the dog face on.

Cerebellar disorder
- Falling/leaning to one side
- Hypermetria
- Intention tremor
- Nystagmus
- Wide-based stance

Vestibular disorder
- Falling/leaning to one side
- Nystagmus

COMMON CAUSES

Cerebellar disorder
- Infectious
  - *Angiostrongylus vasorum*
  - Protozoal: *Toxoplasma, Neospora*
- Inflammatory: meningoencephalitis of unknown origin
- Neoplasia
- Vascular: haemorrhagic or thromboembolic stroke

Vestibular disorder
- Central vestibular (brainstem or cerebellar)
  - Cerebellar disorders as above
  - Inflammatory: meningoencephalitis of unknown origin
  - Neoplasia
  - Trauma
- Peripheral vestibular
  - Cranial polyneuropathy (possible link with hypothyroidism)
  - Idiopathic (old dog vestibular)
  - Otitis media
  - Neoplasia

UNCOMMON CAUSES

Cerebellar disorder
- Cerebellar abiotrophy
- Storage diseases
- Trauma

Vestibular disorder
- Peripheral vestibular
- Trauma
- Central vestibular (brainstem or cerebellar)
  - Cerebellar disorders as above
  - Drug-induced, e.g. metronidazole toxicity
  - Storage disease

DIAGNOSTIC APPROACH

Clinical clues
Predisposition
- Cerebellar abiotrophy has been described in more than 40 breeds of dog at different ages
  - Neonatal, e.g. beagles
  - Juvenile, e.g. Airedale terrier
  - Adult onset, e.g. Bernese mountain dog
- Greyhounds are predisposed to thromboembolic cerebellar events (ischaemic stroke)

History
- Duration and progression of signs may aid in index of suspicion
  - Acute onset but improving increases suspicion of vascular or drug-induced event
  - Acute onset with rapid progression may increase suspicion of inflammatory or infectious disease
  - Gradual, progressive onset may increase suspicion of degenerative or neoplastic disease

Clinical examination
Visual inspection
- Mentation is likely impaired with central brainstem vestibular disorders

Physical examination
- Thorough examination to assess for multisystemic disease

Neurological examination
- Cerebellar
  - Contralateral head tilt and direction of fast phase of nystagmus (paradoxical vestibular)
  - Ipsilateral loss of proprioception and menace response
- Vestibular
  - As above for cerebellar
  - With brainstem disorders consciousness may be reduced, and other cranial nerves may be affected (facial nerve – blink reflex, and glossopharyngeal and vagal nerves – gag reflex)

- With peripheral disorders may detect concurrent facial nerve abnormalities and Horner's syndrome

Ophthalmic examination
- May be useful to assess for infiltrative central nervous system disease, e.g. may detect papilloedema

Laboratory findings
Haematology
- Uncommon to detect changes, may identify thrombocytopenia in haemorrhagic stroke

Serum biochemistry
- Identify increased risks of thromboembolism: HAC, hepatic disease, PLE, PLN

Imaging
Plain radiographs
- Screen for disseminated neoplasia in cases with a high suspicion

Ultrasound
- Screen for disseminated neoplasia in cases with a high suspicion

Special tests
- Coagulation parameters (PT and aPTT) to assess for haemorrhagic stroke
- CSF tap to assess for cytology and proteins (inflammatory) or infectious (e.g. *Toxoplasma* and *Neospora* PCR)
- Infectious disease testing
  - Baermann technique or AngioDetect® for *Angiostrongylus*
  - *Toxoplasma* and *Neospora* serology
- Otoscopy and myringotomy in cases with suspicion of otitis media
- MRI to assess for CNS causes of head tilt

1.31 MELAENA

DEFINITION

Black, tarry faeces due to the presence of partially digested blood and or blood pigments, either from upper GI haemorrhage or swallowed blood.

RELATED CLINICAL SIGNS

- Dark, tarry faeces
  NB: Some medications (iron, metronidazole, tylosin, bismuth) can give faeces a dark colour, and the outside of an old faecal sample will become oxidized and become darker in appearance.
- Haematemesis
- Signs of hypovolaemia and/or anaemia if severe bleeding

COMMON CAUSES

Endocrine
- Hypoadrenocorticism (not common but important)

Generalised bleeding problem
q.v. section 1.9

Gastric neoplasia
- Gastric carcinoma
- Gastric leiomyoma/sarcoma

Gastric ulceration
- Abrasive gastric FB
- Acute and chronic gastritis
- NSAIDs
- Portal hypertension in end-stage liver disease
- Uraemic gastritis

Intestinal disease
- Inflammatory bowel disease

Intestinal neoplasia
- Adenocarcinoma
- Leiomyoma/sarcoma
- Lymphosarcoma

Swallowed blood
- Epistaxis
- Oral bleeding

UNCOMMON CAUSES

Gastric ulceration
- Gastrinoma (Zollinger-Ellison syndrome)
- Mast cell tumour

Intestinal disease
- Chronic intussusception
- GI ischaemia
  - Infarction
  - Mesenteric avulsion
  - Shock
  - Vascular malformation
    - Angiodysplasia/vascular ectasia
    - AV fistula
  - Volvulus
- Polyps
- Severe hookworm infestation (not in UK)

Pancreatic disease
- Severe acute pancreatitis

Swallowed blood
- Haemoptysis

Severe oesophageal disease
- Neoplasia
- Oesophagitis

DIAGNOSTIC APPROACH

1 Any cause of haematemesis is likely to produce melaena and the diagnostic approach is similar, *q.v.* section 1.25
2 The presence of diarrhoea in association with melaena suggests intestinal disease.

Clinical clues
Predisposition
- *q.v.* section 1.25

History
- Diarrhoea and weight loss with intestinal disease, gastrinoma
- Epistaxis, bleeding oral lesion or coughing if swallowed blood
- Haematemesis if gastric lesion

Clinical examination
Visual inspection
- Passage of dark, tarry stool
- Staining of perineal region

Physical examination
- *q.v.* section 1.25

Palpation
- Abdominal discomfort/pain
- Abdominal mass

Digital rectal examination
- Confirms melaena

Laboratory findings
- Haematology and serum biochemistry consistent with blood loss, *q.v.* section 1.25

Imaging
- *q.v.* section 1.49

Special tests
- Occult blood test
  - Indicates presence of haemoglobin in faeces
  - This test is aimed at detecting microscopic bleeding and is unnecessary if there is overt melaena
  - Melaena is only identifiable visually when > 1 ml/kg blood enters the GI lumen
  - It cross-reacts with all dietary blood, and so the dog must be on a meat-free or hydrolysed diet for at least 72 hours before the test can be interpreted

1.32 NASAL DISCHARGE

DEFINITION

Nasal discharge is typically expelled from the external nares, and can be serous, mucopurulent or haemorrhagic, or combinations of these.

RELATED CLINICAL SIGNS

In some cases of nasal disease (in particular those within the caudal nasal chambers), the discharge may be swallowed or inhaled through post-nasal drip; in these cases dogs may also have a cough.

- Concurrent nasal depigmentation/deformities
- Coughing due to post-nasal drip and aspiration of nasal discharge
- If epistaxis present, may also have haematemesis or melaena due to swallowed blood
- Pawing at face
- Stertor or snoring due to reduced nasal airflow

COMMON CAUSES

- Chronic idiopathic rhinitis
- FB
- Infection
  - Sino-nasal aspergillosis
  - Tooth root abscess
  - Secondary to regurgitation with concurrent aspiration pneumonia
- Neoplasia (carcinoma, sarcoma, lymphoma)

NB: If epistaxis, bleeding disorders should also be considered, *q.v.* section 1.21

UNCOMMON CAUSES

- Cleft palate
- Infection
  - Oronasal fistula
  - Osteomyelitis
  - Parasitic (*Linguatula serrata*)
- Neoplasia (leiomyoma)
- Trauma
- NB: Nasal disease can present secondary to reflux/regurgitation

DIAGNOSTIC APPROACH

The nature of the nasal discharge and its speed of onset suggest certain differential diagnoses but assessing for the presence of unilateral or bilateral nasal discharge is most useful in prioritising differential diagnoses.

Clinical clues

Predisposition

- Breed predisposition in sino-nasal aspergillosis suspected in Golden retrievers
- Dolichocephalic dogs are predisposed to sino-nasal aspergillosis
- Older dogs are predisposed to nasal tumours and dental disease

History

- Discharge
  - Bilateral: more likely chronic idiopathic rhinitis, aspergillosis or bleeding disorder
  - Unilateral: more likely FB neoplasia, oronasal fistula or dental disease
- Nasal stertor increases suspicion of nasal FB or tumour
- Recent dental extractions increase suspicion of oronasal fistula

Clinical examination

Visual inspection

- Presence of sneezing increases suspicion of nasal disease
- Stertor is suggestive of reduced nasal airflow

Physical examination

- Assess nasal airflow
  - Tissue or cotton wool to detect airflow at each nostril
  - Microscope slide and assess for condensation
  - Stethoscope and auscultate each side

- Examination of head conformation including ocular retropulsion for signs of deformity suspicious for neoplasia
- Examination of nasal planum for depigmentation (most common in fungal disease)
- Oral examination to assess for dental disease and oronasal fistulas

Laboratory findings

Haematology and serum biochemistry

- Useful to screen for systemic disease including coagulation profile, in particular for causes of epistaxis

Imaging

Plain radiographs

- Nasal turbinate destruction: tumour or aspergillosis
- Sinus hyperostosis (aspergillosis)
- Soft tissue opacity: tumour most likely but can also occur with fluid accumulation

Special tests

- *Aspergillus* serology: specific but poorly sensitive test, i.e. a positive result supports infection but a negative result does not exclude it
- CT scan with contrast to assess for nasal diseases
- Culture of nasal discharge is not useful; likely bacterial flora and positive *Aspergillus* culture is not diagnostic as it can be an environmental contaminant
- Nasal cytology (rarely useful) and tissue sample for histopathology
- Rhinoscopy to assess for nasal FB, tumours or aspergillosis

1.33 NYSTAGMUS

DEFINITION

Pathological nystagmus refers to inappropriate ocular movements; typically a slow phase in one direction; horizontal, vertical with a fast phase in the counter direction, or rotatory, and occurs due to a disorder of the vestibular system. This can be observed at rest (spontaneous nystagmus) or when the vestibular system is tested, e.g. putting the dog on its back (positional nystagmus).

Physiological nystagmus forms part of the oculocephalic reflex and is a normal response (e.g. when looking out of the window in a car). Blind dogs can have saccadic (quick, jerky) eye movements that do not reflect a vestibular disturbance.

RELATED CLINICAL SIGNS

Cerebellar disorders

- Falling/leaning to one side
- Head tilt
- Hypermetria
- Intention tremor
- Wide-based stance

Vestibular disorders

- Bilateral vestibular (more common in cats) presents without a head tilt but falling to both sides, wide excursions of the head, and an uncoordinated gait
- Falling/leaning to one side
- Head tilt

COMMON CAUSES

Cerebellar disorders

- Infectious:
  - *Angiostrongylus vasorum*
  - Protozoal (*Toxoplasma, Neospora*)
- Inflammatory: meningoencephalitis of unknown origin
- Vascular: haemorrhagic or thromboembolic stroke

Vestibular disorders

- Peripheral vestibular
  - Cranial polyneuropathy (possible link with hypothyroidism)
  - Idiopathic (old dog vestibular)
  - Otitis media
- Central vestibular (brainstem or cerebellar)
  - Cerebellar disorders as above

- Inflammatory: meningoencephalitis of unknown origin
- Neoplasia
- Trauma

UNCOMMON CAUSES

Cerebellar disorders
- Cerebellar abiotrophy
- Neoplasia
- Storage disease
- Trauma

Vestibular disorders
- Peripheral vestibular
  - Neoplasia
  - Trauma
- Central vestibular (brainstem or cerebellar)
  - Drug-induced, e.g. metronidazole toxicity
  - Cerebellar disorders as above
  - Storage disease
  - Congenital pendulous nystagmus (most common in Siamese cats but described in Belgian shepherds)

DIAGNOSTIC APPROACH

Clinical clues
Predisposition
- Cerebellar abiotrophy has been described in more than 40 breeds of dog at different ages
  - Neonatal, e.g. beagles
  - Juvenile, e.g. Airedale terrier
  - Adult onset, e.g. Bernese mountain dog
- Greyhounds are predisposed to thromboembolic cerebellar events (ischaemic stroke)

History
- Duration and progression of signs may increase index of suspicion
  - Acute onset but improving increases suspicion of vascular or drug-induced event
  - Acute onset with rapid progression may increase suspicion of inflammatory or infectious disease
  - Gradual progressive onset may increase suspicion of degenerative or neoplastic disease

Clinical examination
Visual inspection
- Mentation is likely impaired with central brainstem vestibular disorders

Physical examination
- Thorough examination to assess for multisystemic disease

Neurological examination
- Cerebellar
  - Ipsilateral loss of proprioception and menace response
  - Contralateral head tilt and direction of fast phase of nystagmus (paradoxical vestibular)
- Vestibular
  - As above for cerebellar, with brainstem disorders consciousness may be reduced, and other cranial nerves may be affected (facial nerve – blink reflex, and glossopharyngeal and vagal nerves – gag reflex)
  - With peripheral disorders may detect concurrent facial nerve abnormalities and Horner's syndrome

Ophthalmic examination
- May be useful to assess for infiltrative central nervous system disease, e.g. may detect papilloedema

Laboratory findings
Haematology
- Uncommon to detect changes
- Thrombocytopenia in haemorrhagic stroke

Serum biochemistry
- May detect causes of increased risk of thromboembolism (protein-losing nephropathy, protein-losing enteropathy, hepatic disease, hyperadrenocorticism)

Imaging
Plain radiographs
- Screen for disseminated neoplasia in cases with a high suspicion

Ultrasound
- Screen for disseminated neoplasia in cases with a high suspicion

Special tests

- Coagulation parameters (PT and aPTT) to assess for haemorrhagic stroke
- CSF tap to assess for cytology and proteins (inflammatory) or infectious (e.g. *Toxoplasma* and *Neospora* PCR)
- Infectious disease testing
  - Baermann technique or AngioDetect® for *Angiostrongylus*
  - *Toxoplasma* and *Neospora* serology
- Otoscopy and myringotomy in cases with suspicion of otitis media
- MRI to assess for CNS causes of head tilt

1.34 PARESIS/PARALYSIS

DEFINITION

Paresis is defined as weakness or partial loss of voluntary movement.

Paralysis (also known as -plegia) is the loss of voluntary movement and may be accompanied by a loss of sensory function.

Disorders may affect a single limb (monoparesis or monoparalysis), both pelvic limbs (paraparesis or paraplegia) or all four limbs (tetraparesis or tetraplegia).

RELATED CLINICAL SIGNS

- Weakness, incoordination or lack of voluntary movement in affected limbs
- There may also be a loss of sensation.
- Concurrent loss of urination, defaecation, and tail movement depending on localisation.

COMMON CAUSES

- Degenerative disease, e.g. canine degenerative myelopathy (CDM); previously termed canine degenerative radiculomyelopathy (CDRM)
- Fibrocartilaginous embolism (FCE)
- Intervertebral disc disease (IVDD)
- Neoplasia

UNCOMMON CAUSES

- Botulism
- Inflammatory: meningomyelitis
- Myasthenia gravis
- Nerve root tumour
- Polymyositis
- Polyradiculoneuritis (aka Coonhound paralysis)
- Protozoa: *Neospora*, *Toxoplasma*
- Tick paralysis (recognised in Australia, North America and South Africa but not in UK)

DIAGNOSTIC APPROACH

Clinical clues

- Duration and progression of signs will aid in index of suspicion
  - Acute onset but improving increases suspicion of vascular event
  - Acute onset and rapid progression may increase suspicion of inflammatory or infectious disease
  - Acute onset but stable may increase suspicion of FCE or IVDD
  - Gradual onset and progression may increase suspicion of degenerative or neoplastic disease
- Generalised weakness may be related to systemic disease, and must be excluded in these cases before assuming a neurological cause

Predisposition

- Dachshunds and many other breeds (e.g. Poodle, Cocker spaniel, Pekingese) are predisposed to IVDD
- CDM is present in numerous breeds: GSDs are predisposed and the condition has been linked to a mutation in the superoxide dismutase (SOD1) gene. Mutations in SOD1 have also been described in the Boxer, Chesapeake Bay Retriever, Pembroke Welsh Corgi, and Rhodesian Ridgeback.

History
- CDM is a slowly progressive condition affecting the hindlimbs and usually does not commence until at least 8 years of age
- Fibrocartilaginous embolism classically occurs acutely during vigorous exercise
- Monoparesis with marked muscle wastage and pain raises concerns of a nerve root tumour
- Neosporosis in young dogs affects the hindlimbs initially but can cause ascending paresis/paralysis

Clinical examination
Visual inspection
- Assess mentation and gait prior to examination

Physical examination
- Thorough examination to assess for multisystemic disease

Neurological examination
- FCE tends to causes lateralised signs and is normally not painful on examination
- IVDD can be localised as C1–C5, C6–T2, T3–L3, L4–S3 using spinal cord reflexes (limb and panniculus), proprioception, and assessment for spinal pain
- Polyradiculoneuritis typically causes an ascending (pelvic limbs affected first) paralysis, sensation is retained (dogs are unable to withdraw limbs but have a behavioural response to pedal withdrawal assessment), urination, defaecation and tail movement is usually preserved

Laboratory findings
Haematology and serum biochemistry
- Uncommon to detect changes
- CK and AST enzyme activities will be increased in polymyositis

Imaging
Plain radiographs
- Poorly sensitive and specific for CNS disease: contrast myelography in addition to radiography improves this
- May detect megaoesophagus in cases suspicious for myasthenia gravis; radiographs must be taken conscious as anaesthesia and sedation may cause false positives for megaoesophagus)

Ultrasound examination
- Screen for disseminated neoplasia in cases with a high suspicion

Special tests
- Acetylcholine receptor antibodies in cases suspicious for acquired myasthenia gravis
- CSF tap to assess for cytology and proteins (inflammatory) or infectious (e.g. *Toxoplasma* and *Neospora* PCR)
- Electrodiagnostics (EMG, nerve conduction velocities) in cases suspicious for peripheral nervous system, neuromuscular, or muscular disorders
- MRI scan (or CT ± contrast myelography) to assess for CNS causes of ataxia
- Muscle and nerve biopsies in cases suspicious for myositis or inflammatory/neoplastic neuropathy

1.35 PERINATAL DEATH

DEFINITION

Perinatal death is the sum of any stillborn puppies and puppies that die during the first week after birth, otherwise known as early neonatal mortality.

RELATED CLINICAL SIGNS

- Dead puppies < 1 week of age
- Stillborn puppies
- Vulval discharge
- Weak/fading puppies – failure to nurse, hypoglycaemic, weight loss, hypothermic

COMMON CAUSES

Infectious
- Canine herpes virus
- *Brucella canis/abortus*
- Mastitis
- Neonatal sepsis

Non-infectious
- Dystocia leading to fetal trauma
- Extended parturition, e.g. due to uterine inertia
- Fetal size/weight > 25% below average for breed reduces survival
- Large litters leading to pregnancy toxaemia
- Maternal neglect/illness

UNCOMMON CAUSES

Infectious
- Other bacterial infections: *Campylobacter*, *Salmonella*, enterotoxic *E. coli*, beta-haemolytic *Streptococci*, *Staphylococcus*, *Mycoplasma*, *Leptospira*
- Other viruses: parvovirus, rotavirus, coronavirus, distemper, canine adenovirus-1
- Parasitic/protozoal infections: *Cryptosporidium*, *Toxoplasma*, *Neospora*, *Leishmania*

Non-infectious
- Congenital anomalies/malformations, e.g. cleft palate
- Eclampsia
- Genetic causes
- Hypothyroidism in bitch
- Malnutrition/nutritional deficiency in bitch
- Maternal gestational DM

DIAGNOSTIC APPROACH

1 Review husbandry
2 Review previous breeding histry
3 Diagnostic testing – see special tests below

Clinical clues
Predisposition
- First litter
- Known genetic defects
- Large litters
- New dogs in kennels

History
- Problems with previous litters especially if in breeding kennels
  - Gastrointestinal signs
  - Respiratory signs
  - Other signs
- Vaccination status of bitch

Clinical examination
- Post mortem examination of dead puppies and placenta
- Vulval discharge

Laboratory findings
Haematology
- May indicate potential infection

Special tests
- Abortion PCR panel on placenta or fetal tissue
  - *Brucella canis*
  - *Campylobacter jejuni*
  - Canine distemper virus
  - Canine herpesvirus type 1 (CHV-1)
  - *Leptospira*
  - *Leishmania*
  - Minute virus (parvovirus type 1)
  - *Neospora*
  - *Salmonella*
  - *Toxoplasma*
- Culture (bacterial ± fungal) of placenta and fetal lung tissue
- Screening of bitch
  - Minimum data base
  - *Brucella* serology
  - Canine herpes virus serology
- Screening of stud dog
  - *Brucella* serology
  - Histopathology of placenta, liver, kidney, lung, heart and heart blood, brain, and any other lesional tissue(s)

1.36 POLYPHAGIA

DEFINITION

Eating in excess of normal caloric needs, either in response to a physiological or pathological increase in energy expenditure, or to a failure to absorb sufficient energy despite increased intake, or as an abnormal behaviour.

RELATED CLINICAL SIGNS

- Coprophagia (ingestion of faeces) and pica (bizarre intake) can be considered forms of polyphagia
- Increased appetite and even scavenging
- Weight gain or weight loss depending on underlying disease

COMMON CAUSES

Behavioural
- Boredom
- Competition
- Gluttony
- Highly palatable food
- Overfeeding

Drugs
- Anticonvulsants
- Glucocorticoids

Endocrine disease
- DM (non-ketotic)
- Hyperadrenocorticism (HAC)
- Hypothyroidism (eating in excess of requirement; not true increased intake)

GI disease
- EPI
- Intestinal malabsorption

Physiological
- Cold environment
- Increased exercise
- Lactation
- Poor-quality food
- Pregnancy

UNCOMMON CAUSES

Metabolic/endocrine disease
- Acromegaly
- Hyperthyroidism
  - Exogenous thyroid tissue contaminating raw food
  - Functional thyroid tumour (rare)
  - Iatrogenic
- Insulinoma (mild increase in appetite)
- SARDS

Drugs
- Amitraz
- Appetite stimulants
  - Capromorelin
  - Cyproheptadine
  - Diazepam (weak)
  - Mirtazapine
  - Progestagens

Neurological
- Psychogenic
- Destruction of satiety centre in hypothalamus (neoplasia, trauma)

Renal
- Protein-losing nephropathy without azotaemia may cause weight loss despite normal appetite; increased appetite is unusual

DIAGNOSTIC APPROACH

- Increases or decreases in weight in association with increased or decreased food intake can indicate the type of disease process
- If there is weight gain ± PU/PD, consider endocrinopathy
- If there is weight loss, first consider malabsorption, especially if diarrhoea is present

Clinical clues
Predisposition
- EPI and ARD/SIBO in GSDs
- Genetic mutation in Labradors: pro-opiomelanocortin (POMC) gene

History
- Weakness, lethargy and seizures
  - Insulinoma
- Weight gain
  - Drugs
  - HAC
  - Hypothyroidism
  - Insulinoma
  - Overeating
- Weight loss
  - Recent change in diet or physiological state
  - Ravenous appetite typical of EPI and benign causes of malabsorption
  - Coprophagia is seen most frequently in EPI and ARD/SIBO
  - Diarrhoea and weight loss
  - EPI and malabsorption
  - Physiological stress; exercise, lactation
  - Poor diet
- PU/PD
  - DM
  - HAC
  - Hyperthyroidism
    - Vomiting and diarrhoea, weight loss, tachycardia

Clinical examination

Visual inspection
- Emaciation if EPI or severe/prolonged malabsorption
- Pot belly and hair loss in HAC
- Overweight ± alopecia in endocrinopathy

Physical examination
- Normal if behavioural or physiological
- Weight gain or weight loss
- Abnormal body condition – loss of fat or lean muscle mass
- Hair loss in HAC
- Palpation
  - Ascites if severe hypoalbuminaemia (PLE, PLN)
  - Thin skin, pot-belly, secondary pyoderma and comedones in HAC
  - Thyroid mass if hyperthyroid

Laboratory findings
- Hypercholesterolaemia and high ALP activity in HAC
- Hyperglycaemia and glycosuria in diabetes mellitus
- Hypoalbuminaemia in PLE or PLN
- Hypoglycaemia in insulinoma
- Proteinuria in PLN

Imaging
- Adrenomegaly in HAC
- Altered hepatic echogenicity in endocrinopathy
- Hepatomegaly in endocrinopathy

Special tests
- Dynamic cortisol testing for HAC
- Folate /cobalamin for malabsorption
- Intestinal biopsies
- Serum insulin
- Serum T4
- cTLI test for EPI

1.37 POLYURIA/POLYDIPSIA (PU/PD)

DEFINITION

Polydipsia (PD) is a daily fluid intake of greater than 100 ml/kg/day.
Polyuria (PU) is a daily urine output of greater than 50 ml/kg/day.
Primary polyuria and secondary polydipsia is more common.

RELATED CLINICAL SIGNS

- Differentiate from urinary incontinence/ increased frequency of urination (pollakiuria)
- Excessive fluid intake or urine output
- Nocturia is inappropriate urination at night that usually reflects increased volume due to PU/PD
- Other signs depend on the cause of PU/PD
- PU/PD may be the only clinical sign

COMMON CAUSES

Primary polydipsia
- Hepatic encephalopathy

Primary polyuria
Osmotic diuresis
- Diabetes mellitus
- Diuretic administration
- Fluid administration

Renal insensitivity to antidiuretic hormone (ADH) = Nephrogenic diabetes insipidus (NDI)
- Secondary NDI
  - Glucocorticoid administration
  - HAC
  - Hypercalcaemia
    - Malignancy: lymphoma, anal sac adenocarcinoma, multiple myeloma
    - Primary hyperparathyroidism
  - Hypoadrenocorticism
  - Pyometra
  - Renal disease

UNCOMMON CAUSES

Primary polydipsia
- Acromegaly
- Diet: high sodium, low protein
- Exocrine pancreatic insufficiency/malabsorption (noted occasionally)
- Fever
- Neurological – lesion in the thirst centre of the hypothalamus
- Pain
- Psychogenic polydipsia

Primary polyuria
- Renal insensitivity to ADH
  - Primary NDI
  - Secondary NDI
    - Drugs
      - Diuretics
      - Phenobarbital
    - Hyperviscosity syndrome
    - Hypokalaemia
    - Jerky treats
    - Pyelonephritis
    - Renal medullary solute washout
- Osmotic diuresis
  - Dextrose infusion
  - Mannitol
  - Primary renal glucosuria
  - Fanconi's syndrome
  - Post-obstructive diuresis
- ADH deficiency = central diabetes insipidus (CDI)
  - Idiopathic
    - Congenital
    - Neoplastic
    - Trauma-induced

DIAGNOSTIC APPROACH

1 Confirm PU/PD by measuring water intake and urine SG, and distinguish from pollakiuria or incontinence.
2 Rule out pyometra by history, physical examination, laboratory results and imaging.
3 Serum biochemistry to rule out renal disease, hypercalcaemia and diabetes mellitus.
4 Compare glycosuria with blood glucose: hyperglycaemia indicates diabetes mellitus; glycosuria with euglycaemia indicates Fanconi's disease or jerky treat ingestion.
5 Dynamic cortisol testing for HAC.
6 Water deprivation test to distinguish psychogenic polydipsia from diabetes insipidus (*q.v.* section 5.3.1) only when safe, i.e. after ruling out the above.

Clinical clues
Predisposition
- CDI is generally reported in middle-aged animals
- Fanconi syndrome in young Basenjis
- Feeding of jerky treats, causing glycosuria
- HAC is most common in middle-aged dogs
- Hypercalcaemia is commonly reported as part of a paraneoplastic syndrome e.g. lymphoma, anal sac adenocarcinoma, multiple myeloma
- Primary NDI is a congenital disease with animals presenting at a young age
- Primary renal glycosuria reported in Norwegian elkhound, Shetland sheepdog, Schnauzer

- Pyometra reported in bitches 1–3 months post-oestrus

History
- Differentiate polyuria from urinary incontinence or pollakiuria
- Entire females – oestrus activity – when was the last season?
- Is it appropriate polydipsia?
  - Environmental changes in temperature
  - Change in diet from moist to dry will increase water intake
- Owner should quantify the polydipsia – difficult in multi-animal households, this must be done in normal environment without external stresses
- Other clinical signs noticed by the owner suggesting any organ involvement
- Polyphagia in HAC and EPI

Clinical examination

Visual inspection
- Behavioural/neurological abnormalities, e.g. hepatic encephalopathy, pituitary neoplasms
- Body condition
- Dermatological changes, e.g. alopecia with HAC
- Panting – HAC

Physical examination
- Eyes
  - Cataracts: diabetes mellitus
  - Jaundice: hepatic disease
  - Corneal lipidosis: HAC
  - Papilloedema: pituitary mass
  - Retinal vessel tortuosity: hyperviscosity syndrome
- External genitalia for discharge, e.g. pyometra
- Oral cavity: ulceration/stomatitis secondary to uraemia
- Skin: hair coat for thin skin, comedones, hair loss in HAC

Palpation
- Lymph nodes for enlargement, e.g. lymphosarcoma
- Anal sac, mammary glands, thyroid gland area (parathyroid tumour) for masses causing paraneoplastic hypercalcaemia
- Hepatomegaly – diabetes mellitus (DM), hyperadrenocorticism, some liver diseases
- Kidneys – not usually easy to palpate in the dog – if enlarged consider neoplasia, pyelonephritis, portosystemic shunt (PSS)
- Uterine enlargement: pyometra

Auscultation
- Bradycardia with hyperkalaemia secondary to renal failure or hypoadrenocorticism
- Tachycardia with toxaemia, dehydration

Laboratory findings

Haematology
- Normal neutrophils, eosinophilia, lymphocytosis in hypoadrenocorticism
- Neutrophilia, eosinopenia, lymphopenia: stress leukogram in hyperadrenocorticism
- Neutrophilia with left shift: pyometra, sometimes in pyelonephritis
- Non-regenerative anaemia: CKD, hypoadrenocorticism, hepatic disease
- Erythrocytosis: hyperviscosity syndrome (distinguish from dehydration-elevating PCV)

Serum biochemistry
- Alanine aminotransferase
  - Increased with liver disease, toxaemia, e.g. pyometra
- Albumin
  - Decreased with liver disease, nephrotic syndrome
  - Increased with dehydration
- Alkaline phosphatase
  - Increased with HAC, liver disease
- Calcium
  - Decreased with CKD, hypoalbuminaemia
  - Increased with malignancy, hypoadrenocorticism, primary hyperparathyroidism, vitamin D toxicosis, CKD (esp. juvenile nephropathy)
- Cholesterol
  - Increased with HAC, DM, liver disease, nephrotic syndrome
- Creatinine and urea
  - Increased with CKD severe dehydration, hypoadrenocorticism, hypercalcaemia
- Globulin
  - Increased with hyperviscosity syndrome, liver disease

- Glucose
  - Increased with DM, acromegaly, HAC
  - Normal in renal glycosuria, jerky treat ingestion
- Phosphate
  - Decreased with primary hyperparathyroidism, malignancy-associated hypercalcaemia
  - Increased with CKD vitamin D toxicosis, severe dehydration, hypoadrenocorticism
- Potassium
  - Decreased: post-obstructional diuresis, diuretic administration, DM
  - Increased: CKD hypoadrenocorticism, severe diabetic ketoacidosis
- Sodium and chloride
  - Decreased: hypoadrenocorticism, ketoacidotic diabetic (psychogenic polydipsia)
  - Increased: primary nephrogenic diabetes insipidus, central diabetes insipidus, dehydration
- Total bilirubin
  - Increased: liver disease
- Urea
  - Decreased: liver disease

Urinalysis

- Sediment analysis for urinary tract infection (UTI)
- Urine specific gravity
  - Greater than 1.030 the dog is not likely to have a concentrating defect, polydipsia is to replace non-renal losses
  - 1.008–1.012 is isosthenuria, i.e. the same osmolality as plasma which, if the dog is dehydrated, suggests severe impairment of renal concentrating ability
  - Less than 1.007 (hyposthenuria) implies tubular function is present as urine is being actively diluted in response to polydipsia
- Urine chemistry analysis, e.g. glucose, ketones, excessive bilirubin, protein
- Urine culture
- Urine protein:creatinine (UPC) ratio to quantify proteinuria

Imaging

Abdominal radiographs

- Abnormal soft tissue masses, e.g. enlarged adrenal glands, spleen, mesenteric/sublumbar lymph nodes
- Liver size
  - Decreased with chronic liver diseases, PSS (± renomegaly)
  - Increased with DM, HAC or infiltrative disease
- Renal size
  - Decreased with chronic interstitial nephritis, juvenile nephropathy
  - Increased with pyelonephritis, congenital PSS, neoplasia, amyloidosis
- Uterine enlargement suggests pyometra

Thoracic radiographs

- Mediastinal lymph node involvement if hypercalcaemic
- Ultrasonography
- Assess renal, hepatic, adrenal tissues, etc.

Special tests

- Amino acid quantification in urine for identification of tubular disease, e.g. Fanconi's syndrome
- Fractional excretion of electrolytes
- Bone marrow aspirate in hypercalcaemic cases with no identifying cause to look for multiple myeloma
- Measurement of plasma ADH – not readily available
- Modified water deprivation test – never perform in azotaemic animals. It is advisable to completely rule out HAC before performing this test.
- Parathyroid hormone (PTH) and PTH-related peptide (PTHrp) assays if hypercalcaemic to distinguish primary hyperparathyroidism from lymphoma
- Rule out HAC: urine cortisol:creatinine ratio, ACTH stimulation test.
  If suggestive of hyperadrenocorticism, perform further tests: low-dose dexamethasone suppression test, high-dose dexamethasone suppression test, endogenous ACTH
- Tissue biopsy, e.g. liver, kidney, lymph node
- Urine and plasma osmolality: osmolality is not affected by particle size unlike the SG

1.38 PREPUTIAL DISCHARGE

DEFINITION

Visible material dripping from the prepuce or causing the dog to frequently lick the area. The material may be haemorrhagic or purulent. Dripping of urine is a sign of incontinence, *q.v.* section 1.48.

RELATED CLINICAL SIGNS

- Abnormal liquid dripping from prepuce: white, yellow or green exudate, or serosanguinous fluid
- Dog licking prepuce, with hair staining
- Dysuria, *q.v.* section 1.19
- Malodour
- Pyrexia, lethargy and pain with prostatitis or prostatic abscess

COMMON CAUSES

Haemorrhagic

- Penile or preputial tumour
  - Haemangiosarcoma
- Prostatitis
- Prostatic neoplasia
- Urinary tract bleeding
  - Balanoposthitis
  - Bleeding disorder
  - Paraphimosis and associated trauma: inability to retract penis into prepuce
  - Trauma, e.g. breeding attempt
    - Fracture of os penis
    - Haematoma
    - Laceration

Purulent

- Balanoposthitis
  - Bacterial infection
  - Herpes virus
- Preputial FB e.g. grass awn
- Phimosis: inability to extrude penis from prepuce

UNCOMMON CAUSES

Haemorrhagic

- Penile or preputial tumour
  - MCT
  - Melanoma
  - Papilloma
  - Squamous cell carcinoma
  - Transmissible venereal tumour (TVT)
- Urinary tract bleeding
  - Pyelonephritis
  - Urethral prolapse
  - Urethritis
  - Urolithiasis

Purulent

- Cystitis and/or urethritis
- Penile or preputial tumours: as above
- Prostatitis or prostatic abscess
- Phimosis or paraphimosis
- Seminal fluid

DIAGNOSTIC APPROACH

1 Determine whether discharge is associated with urination
2 Examine penis; full examination requires sedation or general anaesthesia (GA)
3 Digital rectal examination
4 Laboratory analysis of discharge and urine
5 Imaging
6 Biopsy of any penile/preputial lesions

Clinical clues

Predisposition

- None

History

- Signs of dysuria
- Licking of prepuce

Clinical examination

Visual inspection

- Preputial discharge: haemorrhagic or purulent

Physical examination
- Exteriorisation of penis
- Digital rectal examination

Laboratory findings

Haematology
- Often unremarkable

Serum biochemistry
- Often unremarkable

Urinalysis
- UTI

Imaging

Plain radiographs
- Pelvis, including lateral view with legs drawn forward to image urethra

Contrast radiographs
- Retrograde urethrogram

Ultrasound examination
- Prostate

Special tests
- Bacterial culture of discharge
  - *Acinetobacter*
  - *Bacillus*
  - *Corynebacterium*
  - *E. coli*
  - *Klebsiella*
  - *Mycoplasma*
  - *Proteus*
  - *Pseudomonas*
  - *Staphylococcus*
  - *Streptococcus*
- Biopsy
- Coagulation profile
- Cytology of discharge, mass or preputial smear
  - Neoplastic cells: mast cell tumour (MCT), TVT
  - Pus: bacteria and neutrophils

1.39 PRURITUS

DEFINITION

Itching: the sensation that provokes the desire to scratch.

RELATED CLINICAL SIGNS

- Brown discolouration of coat if repeated licking
- Scratching and licking
- Visible signs are the end result of persistent scratching
  - Alopecia
  - Erythema
  - Excoriation
  - Lichenification

COMMON CAUSES

- Acral lick granuloma
- Anal sac impaction causing perianal rubbing
- Atopy (inhaled allergens)
- Demodectic mange
- Dermatophytosis
- Fleas and flea-allergic dermatitis
- *Malassezia* infection
- Pyoderma
- Sarcoptic mange
- Superficial pyoderma

UNCOMMON CAUSES

- Calcinosis cutis
- *Cheyletiella*
- Contact dermatitis
- Drug eruptions
- Food allergy
- Harvest mites
- Lice
- Pemphigus erythematosus and foliaceous
- Psychogenic: self-mutilation
- Sporotrichosis
- Syringomyelia causes phantom scratching, so probably not pruritus

- Tapeworm segments causing perianal irritation
- *Uncinaria* larval migration causing pedal licking
- Urticaria

DIAGNOSTIC APPROACH

1 Identification of infectious agents by sellotape strips, skin scrapes, hair plucks, and bacterial and fungal cultures.
2 After failing to identify infectious causes, trial therapy for bacterial pyoderma, fleas and possibly also for *Sarcoptes*, is acceptable.
3 Intradermal skin testing or serology is performed to identify atopic reactions.
4 If negative for atopy, an exclusion food trial is indicated.

Clinical clues

Predisposition
- Atopy in French bulldog, WHWT
- Demodecosis in short-haired breeds, especially Shar pei, Bull terrier
- Infectious diseases in young dogs

History
- Contact with infected animals
- Effectiveness of flea control
- Owner with pruritus is suggestive of fleas, *Sarcoptes*, *Cheyletiella*, dermatophytosis
- Phantom scratching in CKCS with syringomyelia is not true pruritus

Clinical examination

Visual inspection
- Pruritic animals will scratch spontaneously, or the scratch reflex may be stimulated
- Presence of fleas or flea dirt
- Self-traumatic lesions

Physical examination

Inspection
- Excoriations, erythema, alopecia and lichenification
- Lesions secondary to self-trauma: erythema, excoriation, lichenification, hyperpigmentation
- Pinnae are often particularly sensitive in *Sarcoptes* infection

Palpation
- Thickened skin in areas of repeated self-trauma

Distribution of lesions
- Symmetric lesions involving:
  - Lumbosacral areas and thighs suggests fleas
  - Ear margins and elbows with sarcoptic mange
  - Face, feet and ventrum with atopy
  - Feet and ventrum with contact allergy
  - Face, ears and feet with food allergy
  - Face, ears, feet or multifocal with demodecosis
  - Face, feet, mucocutaneous junctions with autoimmune skin disease

Laboratory findings

- Blood tests usually unremarkable
  - Eosinophilia may be present in allergic skin disease

Special tests

- Skin examination
  - Bacterial ± fungal cultures
  - Hair plucks: *Demodex*, ringworm/dermatophytosis
  - Sellotape strips: *Cheyletiella* and *Malassezia*
  - Skin scrapes: *Sarcoptes*, *Demodex*
- Exclusion diet trial
- Intradermal skin tests
- *In vitro* allergy testing: only reliable for atopy, not food allergy
- Skin biopsy

1.40 RED EYE (AND PINK EYE)

DEFINITION

Erythema of the directly visible parts of the eye.

RELATED CLINICAL SIGNS

- Blepharospasm
- Chemosis (corneal oedema)
- Epiphora; mucoid/mucopurulent discharge
- Miosis if painful, except mydriasis in glaucoma
- Periorbital pain in glaucoma
- Photophobia
- Reddening of some or all of the structures of the anterior eye

COMMON CAUSES

- Conjunctivitis and/or blepharitis
  - Allergic/atopy
  - Bacterial
  - Keratoconjunctivitis sicca
  - Trauma
- Corneal ulceration
  - Ectopic cilia/distichiasis
  - Environmental irritants: dust, smoke
  - Exposure keratitis
  - Eyelid abnormalities: ectropion, entropion
  - FB
  - Trauma
- Ectopic cilia/distichiasis
- Ectropion
- Entropion
- Hyphaema and subconjunctival haemorrhage
  - Coagulopathy
  - Trauma
- Prolapse of nictitans gland ('cherry eye')
- Uveitis

UNCOMMON CAUSES

- Albinism: iris lacks normal pigment
- Conjunctivitis
  - Arthropod bites
  - Chemical: acid, alkali, antiseptics, shampoos
  - Distemper
  - Plasma cell keratoconjunctivitis
- Dacryocystitis
- Episcleritis
  - Atopy
  - Nodular granulomatous episcleritis
- Glaucoma
- Hyphaema
  - Aberrant larva migrans (*Angiostrongylus*)
  - Envenomation
  - Lens luxation
  - Lymphoma
  - Neoplasia
  - Vasculitis
- Keratitis: bacterial, fungal
- Neoplasia
  - Haemangioma/haemangiosarcoma
  - Histiocytic disease
  - Mast cell tumour
  - Melanoma
  - Squamous cell carcinoma
- Neurological
  - Facial paralysis
  - Neurogenic keratoconjunctivitis sicca (KCS)
- Radiation
- *Thelazia* infection (only in imported dogs in the UK)
- Uveitis
  - Hyperviscosity syndrome
  - Hypertension
  - Immune-mediated
    - Idiopathic
    - Lens-induced, secondary to lens luxation
    - Pigmentary uveitis
    - Uveodermatological sydrome
    - Vaccine reaction: canine adenovirus
  - Metabolic
    - Hyperlipidaemia
    - Lens-induced in diabetes mellitus
    - Tyrosinaemia
  - Neoplasia: lymphoma, metastatic, primary
  - Scleritis
  - Systemic infection
    - Algal: *Prototheca*

- Bacterial: bacteraemia/septicaemia, *Bartonella, Borrelia, Brucella, Leptospira*
- Mycotic: aspergillosis, blastomycosis, coccidioidomycosis, cryptococcosis, histoplasmosis
- Parasitic: aberrant larval migration: *Angiostrongylus, Dirofilaria, Toxocara*
- Protozoan: *Hepatozoon*, *Leishmania*, *Toxoplasma*
- Rickettsial: *Ehrlichia*, Rocky Mountain spotted fever
- Viral: canine adenovirus 1

- Ulcerative keratitis

DIAGNOSTIC APPROACH

1 Physical examination to look for evidence of systemic disease.
2 Coagulation profile if hyphaema is present.
3 Laboratory investigation to rule in/out systemic disease: not necessary if local disease, particularly if only one eye is affected.
4 Full ophthalmological examination.

Clinical clues

Predisposition

- Cherry eye: Bulldogs
- Closed-angle glaucoma
- Exposure keratitis/ulceration: brachycephalics
- KCS in WHWT
- Pigmentary uveitis: Golden retriever
- Uveodermatological syndrome: Arctic breeds and Akita

History

- Known trauma
- Travel history for leishmaniasis

Clinical examination

Visual inspection

- Eyelid deformities
- Ocular discharge
- Erythema: diffuse or focal

Physical examination

- To look for evidence of systemic disease

Ophthalmic examination

- Erythema of conjunctival vessels primarily
  - Conjunctivitis
  - Corneal ulceration
  - Ectopic cilia
- Erythema of episcleral vessels primarily
  - Glaucoma – high intraocular pressure (IOP)
  - Uveitis – low IOP
- Focal erythema
  - Hyphaema
  - Masses

Laboratory findings

- Only indicated if systemic disease is suspected

Imaging

- Only indicated if systemic disease is suspected

Special tests

- Conjunctival cytology
  - Bacteria
  - Distemper inclusions
- Culture of conjunctival bacteria
- Direct and indirect ophthalmoscopy
- Fluorescein stain
- Slit lamp examination of anterior chamber
- Schirmer tear test
- Tonometry to measure intraocular pressure (IOP)

1.41 REGURGITATION

DEFINITION

Regurgitation is the expulsion of saliva and/or undigested food from the oesophagus out through the mouth.

It is exclusively a sign of oesophageal disease and is not part of the vomiting reflex. It is usually a passive process that predisposes to inhalation. However, it can be active, preceded by retching, if there is an acute obstruction (i.e. FB, recent stricture) causing oesophageal discomfort.

RELATED CLINICAL SIGNS

- Cachexia may develop in chronically malnourished cases
- Dog is usually keen to re-eat food unless there is pain on swallowing (e.g. oesophagitis)
- Pseudoptyalism occurs through inability to swallow saliva
- Secondary signs may develop from inhalation of regurgitated food
  - Coughing (inhalation)
  - Halitosis
  - Nasal discharge

COMMON CAUSES

Intra-luminal obstruction
- FB

Megaoesophagus
- Focal myasthenia gravis
- Idiopathic acquired
- Secondary megaoesophagus
  - Achalasia of lower oesophageal sphincter

Oesophagitis
- Gastric reflux
  - Acute and persistent vomiting
  - During anaesthesia
  - Hiatal hernia
  - Spontaneous reflux oesophagitis
- Ingestion
  - FB

UNCOMMON CAUSES

Extra-luminal obstruction
- Cranial mediastinal mass
- Peri-oesophageal abscess or mass
- Vascular ring anomaly, e.g., persistent right aortic arch

Intra-luminal obstruction
- Stricture
- Oesophagitis

Ingestive causes
- Caustics
- Hot liquids and food
- Irritants

Megaoesophagus
- Congenital idiopathic

Mural disease
- Diverticulum
- Gastro-oesophageal intussusception
- Primary neoplasia
  - Carcinoma
  - Leiomyoma/sarcoma
- Pythiosis
- *Spirocerca lupi* granuloma

Myopathies
- Dermatomyositis
- Dystrophin deficiency
- Polymyositis
- Systemic lupus erythematosus (SLE)
- Toxoplasmosis

Neuropathies/junctionopathies
- Bilateral vagal damage
- Botulism
- Brain stem disease
- Hydrocephalus
- Meningoencephalitis
- Dysautonomia: unlikely cause of oesophageal dysfunction, as striated muscle in dogs
- Generalised myasthenia
- Giant axonal neuropathy
- Peripheral neuropathy
  - Immune-mediated
  - Toxins: several outbreaks worldwide due to food contamination with unknown toxin
- Polyradiculoneuritis
- Tick paralysis

Toxins
- Anticholinesterase
- Acrylamide
- Botulism
- Food-related peripheral neuropathy
- Lead
- Thallium

Miscellaneous
- Distemper
- Glycogen storage disease Type II
- Hypoadrenocorticism
- Hypothyroidism; debatable whether a true cause or an association in older dogs
- Thymoma

DIAGNOSTIC APPROACH

1 Distinguish regurgitation from vomiting
2 Depending on clinical suspicion, investigate first by:
   - plain radiographs
   or
   - endoscopy
3. Fluoroscopic assessment of oesophageal motility, noting risk of inhaling barium
4. Special tests for causes of seondary megaoesophagus

Clinical clues
Predisposition
- Congenital megaoesophagus in Fox terrier, GSD, Irish setter
- Gastro-oesophageal intussusception in Shar pei breed
- Hiatal hernia seen in brachycephalics with BOAS, especially French bulldog and Pug, and in old dogs with laryngeal paralysis
- Idiopathic megaoesophagus is most common in GSD, Great Dane and Irish setter
- Persistent right aortic arch is most common in GSD and Irish setter and signs appear at weaning.
- Epidemic of megaoesophagus cases seen in countries where food has been contaminated by an unknown toxin causing a peripheral neuropathy

History
- It is essential to distinguish regurgitation from dysphagia and vomiting
  - Food prehension, chewing and swallowing are normal
  - Food is returned passively with no abdominal heave
  - Food may be tubular in shape and should not be acidic or contain bile unless there is also gastro-oesophageal reflux
- Onset of signs at weaning with congenital megaoesophagus, diverticulum or vascular ring anomaly
- Recent history of ingestion of caustics or of a GA (with presumed reflux) may precede oesophagitis or stricture
- Regurgitation may occur immediately after eating or hours later, depending on whether the oesophagus is inflamed or dilated

Clinical examination
Visual inspection
- Depressed and dyspnoeic if inhalation pneumonia is present
- Dilated oesophagus may occasionally be seen ballooning in the left cervical area
- May regurgitate and re-eat food
- Nasal discharge and coughing if inhalation present

Physical examination
- Auscultation
  - May hear food/liquid slopping in dilated oesophagus
  - Moist lung sounds if inhalation pneumonia
  - Normal heart sounds with persistent right aortic arch (PRAA)
- Gag reflex may be absent if pharynx is also affected
- Halitosis if retention of food in megaoesophagus or inhalation pneumonia

Laboratory findings
Haematology
- Possible inflammatory leukogram with inhalation pneumonia

Biochemistry
- Usually unremarkable, unless patient is dehydrated

Imaging
Plain radiographs of conscious dog
- Dilated gas-filled oesophagus in megaoesophagus

NB: Beware of over-interpretation of passive dilation under heavy sedation or GA

- Radio-dense FB
- Stricture not visible unless food accumulated proximally

Contrast radiographs: barium swallow after plain films
- Radiolucent FB
- Stricture
  NB: Risk of inhalation

Endoscopy
- To identify and potentially treat mural and intramural causes of regurgitation
  NB: Risk of inhalation

Special tests
- Acetylcholine receptor antibody titre for focal myasthenia gravis
- Anti-nuclear antibody for SLE
- Basal cortisol or ACTH stimulation test for hypoadrenocorticism
- Creatine kinase for polymyositis
- Fluoroscopy
- Manometry
- Oesophagoscopy
- Thyroid function tests

1.42 SEIZURES

DEFINITION

An epileptic seizure is defined as manifestation of excessive synchronous, usually self-limiting electrical activity of neurons in the brain. This results in transient, typically short episodes with convulsions or focal motor, autonomic or behavioural features and is due to abnormal excessive and/or synchronous epileptic neuronal activity in the brain.
Seizures can be focal or generalised in nature.
- Focal seizures are less common and present with lateralised or regional signs, e.g. facial twitching, hypersalivation, or behavioural change
- Generalised seizures are characterised by bilateral involvement and typically result in tonic-clonic movements, loss of consciousness, and often have autonomic signs, i.e. urination, defaecation, salivation

RELATED CLINICAL SIGNS

Epileptic seizures can have up to three phases; prodrome, ictal, and post-ictal stages.
1 The prodrome is not commonly observed in dogs and is detected as disrupted behavior predictive of a seizure.
2 The ictal stage is the seizure itself.
3 The post-ictal period occurs after the seizure and may last hours to days; during this time the dog may be disorientated and display abnormal behaviour such as aggression, compulsive walking, excessive drinking, eating, or vocalisation.

COMMON CAUSES

Extracranial causes
- Hypoglycaemia
  - Hepatic encephalopathy: PSS, chronic hepatitis
  - Hypoadrenocorticism
  - Insulinoma
  - Sepsis
  - Toxins, e.g. ivermectin, xylitol
  - Young toy-breed dogs

Intracranial causes
- Idiopathic
- Inflammatory: meningoencephalitis of unknown origin
- Neoplasia: meningioma, glioma
- Congenital: hydrocephalus

UNCOMMON CAUSES

Extracranial causes
- Electrolyte disturbances
  - Hypocalcaemia
  - Sodium derangements

- Hyperviscosity
  - Erythrocytosis
  - Hyperglobulinaemia
- Hypertensive encephalopathy
- Large/diffuse neoplasia
- Uraemia

Intracranial causes

- Degenerative, e.g. storage disease
- Infectious
  - *Angiostrongylus vasorum*
  - Bacterial empyema
  - Distemper
  - Fungal disease
  - *Toxoplasma* and *Neospora*
- Neoplasia
  - CNS lymphoma
  - Metastatic neoplasia
- Vascular: haemorrhagic or thromboembolic stroke

DIAGNOSTIC APPROACH

Clinical clues

Predisposition

- Idiopathic epilepsy is the most common intracranial cause of seizures in dogs aged 1–5 years
- Border collies and GSDs are predisposed to idiopathic epilepsy
- Middle-aged small- and toy-breed dogs are predisposed to meningoencephalitis of unknown origin

History

- Behaviour between seizures is important; dogs with idiopathic epilepsy would be expected to be normal after recovery from the post-ictal stage
- Duration and progression of neurological signs may aid in index of suspicion
  - Acute onset improving increases suspicion of vascular or toxin
  - Gradual onset progression may increase suspicion of degenerative or neoplastic disease
- Thorough history is important to discriminate between seizures, syncope, and movement disorders
  - Movement disorders may cause abnormal movements in all four limbs, but consciousness tends to be preserved
  - Syncope occurs more commonly during exercise or excitement and is normally characterised by brief, flaccid collapse with no post-ictal period

Clinical examination

Visual inspection

- Mentation may be impaired, additional signs of forebrain disease may be circling, head pressing, blindness and abnormal responsiveness

Physical examination

- Bradycardia and hypertension (Cushing's reflex) may be detected in dogs with increased intracranial pressure
- Thorough examination to assess for multisystemic disease

Neurological examination

- A full neurological examination should be performed
  - Normal between seizures if idiopathic epilepsy
  - Multifocal abnormalities are most suspicious for inflammatory, infectious or neoplastic disease
- The forebrain can be assessed with the menace response, proprioception, response to nasal stimulation, and observing mentation

Ophthalmic examination

- May be useful to assess for infiltrative central nervous system disease, e.g. may detect papilloedema

Laboratory findings

Haematology

- Uncommon to detect changes

Serum biochemistry

- Blood glucose to assess for hypoglycaemia as a cause (ideally close temporally to seizure)
- Electrolytes in particular calcium and sodium
- Evidence of hepatic dysfunction (low urea, albumin, cholesterol, glucose, increased bile acid stimulation test results and ammonia) or hepatic injury (increased enzyme activities)

Imaging

Plain radiographs

- Screen for disseminated neoplasia in cases with a high suspicion

Ultrasound

- Screen for disseminated neoplasia in cases with a high suspicion
- Assess for PSS; may be challenging to visualise

Special tests

- Fructosamine and serial fasted blood glucose measurement when suspicious of insulinoma
- CT angiogram to assess for portosystemic shunt and insulinoma
- MRI and CSF for cytology, proteins, and infectious disease PCR to assess for intracranial causes of seizures
- Toxicology in dogs with a high suspicion of toxin exposure
- Treatment trials with anti-epileptic drugs may be considered in cases where discrimination between a seizure and movement disorder is unclear and events are frequent

1.43 SNEEZING

DEFINITION

Classical sneezing is the forcible expulsion of air from the nose in an explosive spasmodic involuntary action resulting primarily from irritation of the nasal mucous membranes.

Reverse sneezing is forceful inspiration via the nose against a closed glottis, often occurring in paroxysms.

The differential diagnoses for sneezing and reverse sneezing are mostly shared, with the difference related to the location of the disorder, i.e. more caudal nasal disorders are more likely to present with reverse sneezing, but with a few exceptions noted below.

RELATED CLINICAL SIGNS

Sneezing is fairly simple for owners to recognise as it is very similar to sneezing in people. Reverse sneezing can be more distressing and harder to characterise, and can be mistaken for breathing difficulties by inexperienced owners.

- Concurrent nasal discharge: serous, mucopurulent, haemorrhagic or a mixture
- Coughing due to post-nasal drip and aspiration of nasal discharge
- Nasal depigmentation/deformities
- Pawing at face
- Stertor or snoring due to reduced nasal airflow

COMMON CAUSES

- Chronic idiopathic rhinitis
- Infection
  - Sino-nasal aspergillosis
  - Tooth root abscess
- Elongated soft palate causing reverse sneezing
- FB
- Neoplasia: carcinoma, sarcoma, lymphoma

UNCOMMON CAUSES

- Infection
  - Oronasal fistula
  - Osteomyelitis
  - Parasitic (*Linguatula serrata*, *Pneumonyssoides caninum*)
- Neoplasia (leiomyoma)
- Cleft palate
- Reverse sneezing may occur due to lower airway disease with irritation of the nasopharynx due to coughed-up secretions and with reflux

DIAGNOSTIC APPROACH

Clinical clues

Predisposition

- Breed predisposition in sino-nasal aspergillosis suspected in Golden retrievers

- Dolichocephalic dogs are predisposed to sino-nasal aspergillosis
- Older dogs are predisposed to nasal tumours and dental disease
- Reverse sneezing may occur in dogs with elongated soft palates, e.g. brachycephalic dogs, CKCS

Physical examination

- Assess nasal airflow
  - Tissue or cotton wool to detect airflow at each nostril
  - Microscope slide and assess for condensation
  - Stethoscope and auscultate each side
- Examination of head conformation including ocular retropulsion for signs of deformity suspicious for neoplasia
- Examination of nasal planum for depigmentation (most common in fungal disease)
- Oral examination to assess for dental disease and oronasal fistulas

Laboratory findings

Haematology and serum biochemistry

- Often unremarkable, but useful to screen for systemic disease

Imaging

Plain radiographs

- Nasal turbinate destruction: tumour or aspergillosis
- Soft tissue opacity: tumour is most likely but can also occur with fluid accumulation
- Sinus hyperostosis (*Aspergillus*)

Special tests

- *Aspergillus* serology: specific but poorly sensitive test, i.e. a positive result supports infection but a negative result does not exclude it
- CT scan with contrast to assess for nasal diseases
- Culture of nasal discharge is not useful; likely bacterial flora and positive *Aspergillus* culture is not diagnostic as it can be an environmental contaminant
- Nasal cytology (rarely useful) and tissue sample for histopathology
- Rhinoscopy to assess for nasal FB, tumours, or aspergillosis

1.44 STIFFNESS, JOINT SWELLING AND GENERALISED LAMENESS

DEFINITIONS

- Stiffness is a reduction in the range of joint movement and overall mobility.
- A joint swelling is enlargement of a synovial joint due to accumulation of fluid or soft tissue inflammation.
- Lameness is an abnormal gait resulting from partial loss of function or pain in a limb usually due to an orthopaedic problem.
- Generalised lameness affects two or more limbs and typically is related to systemic disease.
- Shifting lameness is when limbs are affected sequentially.

RELATED CLINICAL SIGNS

Stiffness, joint swelling, and generalised lameness may be identified together or in isolation.

- Abnormal or short-strided gait
- Change in mobility, e.g. reluctance to exercise, jump up/down onto furniture or climb/descend stairs
- Change in posture
- Joint swelling
- Tachycardia and tachypnoea may occur in the acute phase
- Weight shifting when standing
- Yelping or crying out, and sensitivity to touch due to pain

COMMON CAUSES

Spinal pain
- IVDD
- Steroid responsive meningitis arteritis (SRMA)

Musculoskeletal pain
- Cellulitis
- Degenerative joint disease (osteoarthritis)
- FB
- Fracture
- Immune-mediated polyarthritis (IMPA)
- Neoplasia: osteosarcoma
- Trauma

UNCOMMON CAUSES

Spinal pain
- Discospondylitis
- Nerve root tumour and other neoplasms of the spine

Musculoskeletal pain
- Aortic thromboembolism
- Metaphyseal osteopathy
- Myositis
- Osteomyelitis
- Panosteitis
- Septic joint

DIAGNOSTIC APPROACH

Clinical clues
Predisposition
- Breed predisposition
  - IVDD: Bassets, Dachshunds and, to a lesser extent, many other breeds, e.g. Cocker spaniel, Pekingese, Poodle
  - Metaphyseal osteopathy: Weimaraner
  - Osteosarcoma: large and giant breeds
  - SRMA: Beagle, Boxer, Whippet
- Juvenile dogs: SRMA, metaphyseal osteopathy and panosteitis
- Older dogs: neoplasia and degenerative joint disease

History
- Dogs with generalised lameness, in particular with inflammatory causes (IMPA), may present with signs of lethargy only; specific questions around normal behaviours may uncover more subtle signs of joint pain
- Lameness in multiple limbs is more difficult to detect than lameness in a single limb
- Spinal pain and abdominal pain can be difficult to discriminate; GI signs will aid this, i.e. in the absence of vomiting or diarrhoea, spinal pain would be considered more likely than abdominal pain
- Stiffness and significant lameness can be detected by owners, but joint swelling is not normally detected by owners

Clinical examination
Visual inspection
- Assess demeanour, ambulation, and navigating specific obstacles (e.g. stairs), and in some cases video recordings of the dog during episodes can be very useful
- Disorders involving multiple limbs can be more challenging than those affecting single limbs due to more subtle signs of lameness

Physical examination

During thorough examination pain may be localised; in some cases it may be important to pay attention for subtle signs of discomfort, e.g. lip licking
- Crepitus in degenerative joint disease
- Joint swelling and reduced range of movement
- Pyrexia is typically observed in steroid responsive meningitis arteritis, IMPA and metaphyseal osteopathy

Orthopaedic and neurological examinations
- To identify sites of pain and distinguish musculoskeletal causes of abnormal gait pain from neurological causes

Laboratory findings
Haematology and serum biochemistry
- Useful to screen for systemic inflammatory disease

Imaging
- Plain radiographs and abdominal ultrasound may be useful once pain is localised

Special tests
- Analgesia trial in the absence of suspicion of severe systemic disease
- Arthrocentesis cytology and culture for IMPA or septic joint
- C-reactive protein (CRP) may be increased in dogs with inflammatory disorders (SRMA, septic joint, IMPA)
- CSF cytology for SRMA
- Infectious disease screening in particular in travelled dogs: *Anaplasma*, *Ehrlichia*, *Leishmania*, and *Borrelia*
- Spec cPL can be increased in IVDD as well as pancreatitis, therefore may not aid in discriminating

1.45 STUNTING

DEFINITION

Stunting (retarded growth) is a reduced rate of growth resulting in a dog failing to attain the expected size, weight and/or height standards characteristic of a dog of the same age and breed.

RELATED CLINICAL SIGNS

- Proportionately small size or disproportionate stunting, i.e. normal body length but short limbs
- Signs of relevant organ dysfunction
  - Exercise intolerance
  - Neurological signs
  - Polyphagia
  - PU/PD
  - Vomiting and/or diarrhoea

COMMON CAUSES

- Congenital cardiac disease
- Chondrodystrophy
- Hepatic dysfunction
  - Congenital PSS
- Inadequate diet:
  - Underfeeding
  - Poor-quality food
- Malabsorption
  - Antibiotic-responsive diarrhoea (ARD or dysbiosis)
  - EPI
  - Food-responsive enteropathy
  - Parasitism: *Giardia*, hookworms
- Renal dysplasia

UNCOMMON CAUSES

- Achondroplasia and chondrodysplasia
- Congenital hypothyroidism
- Diabetes mellitus
- Endocarditis
- Food allergy
- Hepatic dysfunction
  - Glycogen storage disease
  - Lobular dissecting hepatitis
  - Portal vein hypoplasia: non-cirrhotic portal hypertension
- Hydrocephalus
- Hypoadrenocorticism
- Malabsorption
  - Chronic intussusception
  - CIE/IBD
- Oesophageal disease
  - Congenital megaoesophagus
  - Vascular ring anomaly
- Pituitary dwarfism
- Vitamin and mineral deficiencies
  - Vitamin A
  - Vitamin D
  - Vitamin B_{12}
    - Imerslund-Gräsbeck syndrome (selective cobalamin malabsorption)
    - Vegan diet
- Zinc deficiency

DIAGNOSTIC APPROACH

- Determine appetite
  - Anorexia suggestive of metabolic disease
  - Polyphagia suggestive of malabsorption or EPI
- Use other clinical signs to identify potential organ systems
- Serum biochemistry to identify metabolic causes
- Investigate specific organ systems

Clinical clues

Some breeds, such as Yorkshire terriers and poodles, have a range of normal sizes, but stunting can be recognised by comparison with unaffected littermates.

- Alopecia in pituitary dwarfism
- Chondrodystrophy is part of the normal breed standard in certain, short-legged breeds, and can cause type 1 IVDD
- Mental retardation can occur in untreated congenital hypothyroidism
- Disproportionate stunting is suggestive of chondrodysplasia or congenital hypothyroidism
- Dyspnoea/tachypnoea in inhalation pneumonia
- Intermittent forebrain signs due to hepatic encephalopathy or hydrocephalus
- Fibrous osteodystrophy (rubber jaw) in renal dysplasia

Predisposition

- ARD and EPI in GSDs
- Chondrodystrophy can be normal in several short-legged breeds: Dachshunds, Bassets
- Hydrocephalus in miniature and toy breeds: Chihuahua, Papillon, Yorkshire terrier
- Hydrocephalus in brachycephalics; Boston terrier, English bulldog, Pekingese
- Imerslund-Gräsbeck syndrome in Australian shepherd, Beagle, Border collie, Giant schnauzer, Komondor
- Inhalation pneumonia in brachycephalic breeds
- Pituitary dwarfism: GSD, Wolfdog (Czechoslovakian, Saarloos), Spitz, Miniature Pinscher, and Karelian Bear Dog
- Renal dysplasia in a wide range of breeds: Alaskan Malamute, Bedlington terrier, Boxer, Bulldog, Chow Chow, Cocker Spaniel, Collie, Dobermann, Finnish Harrier, Golden and Labrador retriever, Great Dane, Irish Wolfhound, Keeshond, King Charles spaniel, Lhasa Apso, Miniature schnauzer, Norwegian Elkhound, Rhodesian Ridgeback, Rottweiler, Samoyed, Shih Tzu, Soft-coated Wheaten Terrier and Standard Poodle

History

- Failure to grow
- Signs associated with relevant organ system
  - GI signs and polyphagia in malabsorption
  - PU/PD in DM, PSS, renal dysplasia

Clinical examination

Visual inspection

- Abnormal behaviour and obtundation in hepatic encephalopathy or hydrocephalus
- Shortened legs in chondrodystrophy
- Small size: proportionate or disproportionate?

Physical examination

- Domed skull and/or open fontanelle in hydrocephalus
- Small size
- Underweight

Laboratory findings

- Abnormalities associated with relevant organ system, e.g. azotaemia with renal dysplasia
- Hyperglycaemia and glycosuria if diabetes mellitus

Imaging

Abnormalities associated with relevant organ system

Radiographs

- Poor bone mineralization
- Pneumonia
- Shortened limb bones
- Small kidneys

Ultrasound

- Abnormal kidneys with renal dysplasia
- PSS ± renomegaly

Special tests
- Cobalamin for B_{12} deficiency
- DNA test for Imerslund-Gräsbeck syndrome
- DNA test for pituitary dwarfism
- Dynamic bile acids for PSS
- IGF for pituitary dwarfism (growth hormone assay not commercially available)
- TLI for EPI

1.46 TENESMUS AND DYSCHEZIA

DEFINITION

- Tenesmus is straining to defaecate or urinate.
- Dyschezia denotes pain or difficulty in defaecation and is often associated with tenesmus in distal LI and/or perianal disease.
- Faecal tenesmus is straining to pass faecal material.
- Urinary tenesmus is straining to pass urine, *q.v.* section 1.19

RELATED CLINICAL SIGNS

Primary signs
- Signs of pain
  - Circling repeatedly before attempting defaecation
  - Stopping straining abruptly and/or crying out during attempted defaecation
- Tenesmus: straining to pass faecal material or after defaecation
  - Straining after defaecation if there is colono-rectal inflammation or mass
  - Straining before passing stool and may ultimately pass small volume feces or liquid if constipated

Associated signs
- Anorexia and vomiting if severely constipated
- Blood and mucus mixed in faeces if colitis
- Blood on surface of faeces if focal bleeding lesion, e.g. polyp
- Distorted faecal shape if rectal mass/stricture present

COMMON CAUSES

- All causes of constipation, *q.v.* section 1.11
- Anal sac
  - Abscess/perineal cellulitis
  - Anal sacculitis
  - Impaction
- Colorectal disease
  - With diarrhoea
    - Acute colitis
    - Chronic colitis
  - Without passing stool
    - Colitis
    - Constipation
- Pelvic fracture
- Perineal disease
  - Anal furunculosis
  - Perineal hernia and rectal diverticulum
- Benign prostatic hypertrophy
- Spinal cord injury

UNCOMMON CAUSES

- Anorectal stricture
  - Inflammatory
  - Non-accidental injury
  - Post-surgical
- Anal sac adenocarcinoma
- Colorectal disease
  - With fresh blood and/or faecal deformation (indented or ribbon-shape)
    - Rectal polyp
    - Rectal tumour
- Idiopathic megacolon
- Paraprostatic cyst

- Pseudocoprostasis
- Prostatic carcinoma
- Rectal FB
- Vulval mass

DIAGNOSTIC APPROACH

1 Distinguish between urinary and faecal tenesmus by history and observation.
2 Examine perineum and perform rectal examination if faecal.
3 Look for underlying cause of constipation.

Clinical clues

Predisposition
- Anal sac adenocarcinoma: middle-aged female dogs
- Perineal hernia: male entire dogs

History
- Confirm whether dyschezia or dysuria is cause of tenesmus by observation and physical examination

Clinical examination

Visual inspection
- Observe defaecation and urination to distinguish which is associated with straining
- May strain and cry out, stop, walk around and try again
- Pseudocoprostasis visible

Physical examination

- Abdominal palpation
  - Distended abdomen
  - Enlarged, firm colon if constipated
  - Full bladder if obstructed
  - Small bladder if cystitis/urethritis
- Anal furunculosis
- Perineal swelling

Rectal examination
- Anal sac disease
- Constipation
- Pain
- Perineal hernia
- Prostatomegaly
- Rectal mass
- Stricture: benign strictures are rare, neoplasia more likely
- Vulval mass

Laboratory findings
- Haematology and serum biochemistry usually unremarkable

Imaging

Plain radiographs
- Extent of colonic impaction
- Identifies:
  - Abnormal faecal material (e.g. bones) if constipated
  - Pelvic bone and some spinal lesions
  - Pelvic mass
  - Prostatic enlargement
  - Sublumbar lymphadenopathy

Ultrasound
- Colonic masses
- Prostatic enlargement

Special tests

- Barium enema
- Colonoscopy
- Advanced imaging: myelogram, CT, MRI

1.47 TREMORS

DEFINITION

Tremors are involuntary, repetitive, rhythmic, muscular oscillations. They can be generalised or affect single body parts.

RELATED CLINICAL SIGNS

- Circumstance of occurrence is useful; intention tremors are those that occur during voluntary movement, only when standing (orthostatic tremor), and idiopathic head tremors can be terminated by distracting the dog.

- In addition to tremors depending on aetiology
  - Ataxia
  - Generalised seizures
  - Vestibular dysfunction
- Marked sustained tremors may cause hyperthermia due to sustained muscle contractions.

COMMON CAUSES

Cerebellar disease
- Corticosteroid responsive tremor syndrome (formerly known as white hairy shaker syndrome)
- Meningoencephalitis of unknown origin

Idiopathic
- Idiopathic head tremor
- Senile-related tremor

Metabolic disease
- Hepatic encephalopathy
- Non-specific
  - Anxiety
  - Pain
- Toxins
  - Tremorgenic mycotoxin
  - Chocolate toxicity

UNCOMMON CAUSES

Cerebellar disease
- Abiotrophy
- Congenital (hypoplastic)
- Infectious, e.g. *Toxoplasma* or *Neospora* infection
- Neoplasia
- Storage disease
- Vascular: thromboembolic or haemorrhagic

Miscellaneous
- Distemper
- HAC-induced myoclonus
- Hypoaldosteronism
- Hypocalcaemia
  - Eclampsia
  - Hypoadrenocorticism
- Primary orthostatic tremor
- Toxin induced
  - Metaldehyde

DIAGNOSTIC APPROACH

Clinical clues
Predisposition
- Corticosteroid responsive tremor syndrome: small toy-breed types
- Idiopathic head tremor: Boxers, English bulldogs and Dobermanns
- Nursing bitches at risk of hypocalcaemia
- Orthostatic tremor: large-breed dogs
- Older dogs, especially small-breed dogs, predisposed to senile tremor

History
- Circumstance of tremor
  - If localised consider cerebellar disorders
  - Voluntary movement more likely intention tremor
- Risk of exposure to toxins, including mouldy food contaminated by tremorgenic mycotoxin
- Systemic disease or pain if behavioural causes of tremor are suspected

Clinical examination
Visual inspection
- Assess demeanour, ambulation, and navigating specific obstacles, e.g. stairs, etc.
- In some cases video recordings of the dog during episodes can be very useful

Physical examination
- Thorough examination for signs of systemic disease or pain
- Full neurological examination

Laboratory findings
Haematology and serum biochemistry

Useful to screen for systemic inflammatory disease
- Evidence of hepatic dysfunction and/or injury: albumin, cholesterol, glucose, bilirubin, liver enzyme activities, and a bile acid stimulation test
- Hypoglycaemia: insulinoma
- Hypocalcaemia: eclampsia

Imaging
- Unremarkable in most cases

Special tests
- ACTH stimulation test for hypoadrenocorticism
- Analgesia trial in the absence of suspicion of severe systemic disease
- LDDS test for HAC where clinical signs are present
- MRI and CSF tap cytology for cerebellar disorders

1.48 URINARY INCONTINENCE

DEFINITION

Urinary incontinence is the loss of voluntary control of urination with consequential unconscious urinary leaking. This must be discriminated from nocturia (urinating overnight) or inappropriate urination (e.g. behavioural or during excitement or apprehension).

Urinary incontinence may occur due to a failure to store urine (storage disorders), or due to an inability to completely void the bladder during conscious urination and resultant urinary leaking (overflow incontinence).

Urinary incontinence may present as constant leaking, or occurrence may be related to specific circumstances (lying down, or during activity).

RELATED CLINICAL SIGNS

- Neurological disease depending on the cause
- Systemic illness
- Urinary tract infection: haematuria, pollakiuria, pungent urine

COMMON CAUSES

Storage disorders
- Detrusor hyperreflexia/instability (urge incontinence)
  - Calculi
  - Infection: urethritis
  - Neoplasia
  - Polyps
- Ectopic ureter
- Urethral sphincter mechanism incompetence (USMI)
- Urinary tract infection

Overflow incontinence
- Partial obstruction
  - Neoplasia
  - Prostatic disease (abscess, paraprostatic cyst, benign prostatic hyperplasia, neoplasia)
  - Urethritis
  - Urolithiasis
- Neurogenic (brainstem or spinal cord disorders)

UNCOMMON CAUSES

Storage disorders
- Persistent urachus
- Ureterocoele
- Urethrovaginal or urethrorectal fistula

Overflow incontinence
- Detrusor atony
- Detrusor urethral dyssynergy
- Dysautonomia

DIAGNOSTIC APPROACH

Clinical clues
Predisposition
- Neuter status influences risk of prostatic disorders
  - Neutered dogs are most likely to have carcinoma
  - Entire dogs are most likely to have benign prostatic hyperplasia, abscess, or paraprostatic cysts
- USMI can be detected in any dog, however most common in female neutered dogs
- Young dogs, in particular Golden retrievers, increase suspicion of ectopic ureter

History
- Assess for polyuria and polydipsia
- Concurrent dyschezia increases suspicion of intrapelvic mass lesion (enlarged prostate or lymph nodes)
- In dogs with bacterial cystitis an emphasis should be placed on establishing risk factors that may have predisposed to the development of infection:
  - Anatomical causes (juvenile vaginitis in puppies, hooded vulva in female dogs, urinary incontinence (e.g. ectopic ureter, USMI)
  - Presence of systemic disease (hyperadrenocorticism, diabetes mellitus)
  - Immunosuppression
- Urination in inappropriate places whilst the dog is conscious and aware is likely behavioural unless there is a UTI or severe PU/PD

Clinical examination

Visual inspection
- Measuring bladder size (using ultrasonography) following voiding may help establish whether the dog is able to void their bladder (if this remains large, obstructive and neurological causes are more likely)
- Urination pattern
  - Dogs with reflex dyssynergia or urethral obstruction typically try to initiate urination as normal; however, any urinary stream stops abruptly due to lack of synchrony between bladder contraction and urethral sphincter relaxation or obstruction

Physical examination
- Conformation risk factors for development of bacterial cystitis
  - Hooded vulva, perivulval inflammation or urinary pooling in female dogs
  - Prostatomegaly in male dogs
- Rectal examination to assess the urethra, and prostate in male dogs
- Passing a urinary catheter (can be done in conscious male dogs but likely requires sedation or anaesthesia for female dogs), to assess for ease of passing aids assessment for urethral obstructive disorders

Neurological examination
- Assessment for concurrent neurological deficits
- Ability to express bladder useful in dogs with concurrent neurological deficits

Laboratory findings

Haematology
- Typically unremarkable; it may be useful to screen for systemic disease

Serum biochemistry
- Useful to screen for systemic disease, may detect azotaemia in dogs with post-renal causes (urethral obstruction) or renal causes (extension of bacterial cystitis to pyelonephritis)

Urinalysis
- Cytology to assess for neoplastic cells
- Culture: ideally from cystocentesis sample, otherwise interpret positive culture with caution
- Sediment for evidence of urinary tract infection (pyuria) or neoplastic cells
- Urine specific gravity to assess for polydipsia and polyuria

Imaging

Plain radiographs
- Assess for radiopaque uroliths, prostate, ensure length of urethra included, may need to do an enema first to fully assess urinary system for uroliths
- Contrast studies retrograde urethrogram (plain radiographs or fluoroscopy) and intravenous excretory urogram
- Bladder may be intrapelvic in urinary sphincter mechanism incompetence

Ultrasound
- Assess kidneys, ureters, urinary bladder and prostate

Special tests
- CT with contrast for urinary system assessment (improves sensitivity for ectopic ureter compared to plain radiography)

- Cystoscopy to assess urethra, bladder, and implantation of ureters
- MRI and CSF for dogs with neurolocalisation
- Prostatic wash and fine needle aspiration for cytology and culture
- Urinary BRAF: assess urine for presence of BRAF mutation detected in a high proportion of dogs with transitional cell carcinoma
- Urodynamic pressure profilometry

1.49 VOMITING

DEFINITION

Vomiting (emesis) is a reflex act characterised by forceful expulsion of gastric ± small intestinal contents from the stomach, and coordinated by the vomiting centre in the medulla.

The multiple afferents to the vomiting centre mean vomiting can be caused by primary GI disease or disease elsewhere in the dog. Vomiting can be acute (sudden onset, often self-limiting) or chronic (arbitrarily > 3 weeks' duration)

RELATED CLINICAL SIGNS

- Changes in appetite and weight loss
- Expulsion of gastric contents:
  - Digested or undigested food
  - Bile and mucus
  - Blood, *q.v.* section 1.25
- Preceded by nausea
  - Frequent swallowing/gulping/retching
  - Hypersalivation, *q.v.* section 1.16
  - Lip-licking
  - Restlessness/anxiety
- Repeated contractions of abdominal wall
- Signs of dehydration
- Tremors if ingested tremorgenic mycotoxin, *q.v.* section 1.47

COMMON CAUSES

Acute vomiting

Primary GI disease

- Acute haemorrhagic diarrhoea syndrome (AHDS)/haemorrhagic gastroenteritis (HGE), *q.v.* section 1.15.1
- Dietary indiscretion
  - Adverse reaction to a natural component in food, e.g. histamine
  - Change in diet
  - Scavenging: garbage intoxication, mycotoxins
  - Over-indulgence
- Gastric foreign body
- Intestinal obstruction: FB, intussusception
- Parvovirus infection
- Gastric dilatation-volvulus – ineffective attempts to vomit

Secondary, non-GI disease

- Acute pancreatitis
- AKI and post-renal obstruction
- Diabetic ketoacidosis
- Drugs: chemotherapy agents, digoxin, morphine, NSAIDs
- Hypoadrenocorticism: not common, but too important to be overlooked
- Pyometra
- Toxins, e.g. ethylene glycol, mycotoxins, plant alkaloids
- Vestibular disease, motion sickness

Chronic vomiting

Primary GI disease

- Chronic gastritis
  - Enterogastric reflux (bilious vomiting syndrome)
  - *Helicobacter*
  - Idiopathic
- Gastric ulceration
  - NSAIDs
- Inflammatory bowel disease/chronic inflammatory enteropathy

Secondary, non-GI disease

- Chronic pancreatitis
- Hypercalcaemia
- Uraemia

UNCOMMON CAUSES

Acute vomiting

Primary GI disease

- Distemper
- Intestinal volvulus
- Peritonitis
- Psychogenic

Secondary, non-GI disease

- Acute hepatitis
- Anaphylaxis
- Diaphragmatic rupture
- Heat stroke
- Leptospirosis
- Prostatitis
- Septicaemia/endotoxaemia
- Sialoadenitis/salivary gland infarction

Chronic vomiting

Primary GI disease

- Chronic hypertrophic pylorogastropathy (CHPG)/gastric antral mucosal hypertrophy
- Gastric ulceration
  - Gastric carcinoma
  - Gastrinoma
  - Gastric mast cell tumour: gastric
- Gastroparesis
  - Visceral myopathy: leiomyositis
  - Visceral neuropathy
  - Mesenteric ganglionitis
- Hiatal hernia: can cause regurgitation and/or vomiting
- Obstipation
- *Physaloptera* (not in UK)
- Pyloric stenosis
- Pythiosis (not in UK)

Secondary, non-GI disease

- Acute and chronic hepatitis
- Cholecystitis
- Dysautonomia
- Gall bladder mucocoele
- Hepatoencephalopathy
- Histamine release from remote MCT
- Hyperthyroidism
- Hypertriglyceridaemia
- Hypocalcaemia
- Intracranial disease
  - Encephalitis
  - Head trauma
  - Meningitis
  - Raised intracranial pressure
    - Hydrocephalus
    - Space-occupying lesion: cyst, neoplasia
- Salmon-poisoning (not in UK)
- Sialoadenosis: phenobarbitone-responsive
- Swallowed blood from oral bleeding or epistaxis

DIAGNOSTIC APPROACH

1 Distinguish vomiting from regurgitation by clinical clues below.
2 Rule out secondary causes of vomiting by history, physical exam and minimum laboratory database.
3 Treat acute vomiting symptomatically unless surgical disease is suspected.
4 Investigate chronic vomiting by laboratory tests, imaging and endoscopy.

CLINICAL CLUES

Predisposition

- Gastric carcinoma: 7- to 10-year-old Collies, Belgian shepherds and Bull terriers
- Intussusception is more common in immature dogs
- Parvovirus infection is more likely in young, unvaccinated dogs
- Pyloric stenosis is seen most often in brachycephalic dogs and soon after weaning
- Pyometra and diabetes mellitus are more common in middle-aged to older unspayed females
- Salivary gland infarction in Jack Russell terriers
- Scavenging is more common in Labradors
- Tremors associated with mycotoxin ingestion

History

- Access to garbage, or history of roaming
- Lethargy and weight loss in hypoadrenocorticism
- Metoestrus phase for pyometra
- Systemic illness if secondary vomiting, e.g. PU/PD in diabetes mellitus

- Timing of vomition: more frequent and sooner after feeding the more acute and nearer to the stomach the cause

Clinical examination

Visual inspection

- Distinguish vomiting from regurgitation by presence of prodromal nausea and abdominal contractions/heaves
- Inspect content of vomit: bile, partially digested food, worms, blood
- 'Prayer position' with cranial abdominal pain, *q.v.* section 2.17.1

Physical examination

- Assess degree of dehydration
- Borborygmi: absence of gut sounds indicates ileus and possible peritonitis
- Bradycardia if hypoadrenocorticism
- Enlarged salivary glands in sialoadenosis/sialoadenitis/infarction
- Jaundice if hepatopathy
- Pale mucous membranes and tachycardia if bleeding gastric ulceration
- Palpation
  - Abdominal masses
  - FB
  - Pain if pancreatitis, peritonitis or gastric ulceration

Laboratory findings

Haematology

- Inflammatory leukogram if pyometra, pancreatitis, peritonitis or deep gastric ulceration
- Neutropenia if parvovirus infection or acute peritonitis (bowel perforation)
- PCV dramatically increased in AHDS/HGE
- PCV increased in dehydration

Serum biochemistry

- Amylase and lipase unreliably increased in pancreatitis
- Azotaemia and isosthenuria in uraemia
- Electrolyte disturbances if prolonged vomiting
- Hyperglycaemia and glycosuria if diabetes mellitus
- Hyponatraemia/hyperkalaemia in hypoadrenocorticism

Imaging

Radiographs

Plain

- Abdominal fluid (peritonitis)
- Abdominal mass
- Abnormalities of other organs
- Free abdominal gas (GI perforation)
- Obstructive GI gas pattern
- Pyometra
- Radiopaque FB

Contrast

- Low yield
- Only indicated if plain films are unremarkable and supportive lab studies are non-diagnostic

Ultrasound

- Abnormalities of other organs
- Abdominal masses
- FB
- Gastric tumour
- Intussusception

Special tests

- ACTH stimulation test
- Gastroscopy is only indicated in chronic vomiting after ruling out systemic disease, and for FB removal
- Exploratory laparotomy
- GI biopsy
- Spec cPL

1.50 VULVAL DISCHARGE

DEFINITION

Increased volume of mucoid discharge or change in nature of discharge, e.g. purulent or haemorrhagic.

RELATED CLINICAL SIGNS

- Attractive to other males
- Excessive licking of vulva
- Hair staining in perineum

- Increased volume mucoid discharge or purulent or haemorrhagic discharge
- Pollakiuria/discomfort on urination
- Staining of bed/carpet/hair in perineum
- Scooting
- ± PU/PD
- ± Incontinence
- ± Pruritus
- ± Systemic illness

COMMON CAUSES

- Abortion/miscarriage
- Endometritis/pyometritis
- Ovarian remnant syndrome
- Pseudopregnancy
- Reproductive cycle, e.g. proestrus/oestrus
- Vulvovaginitis

UNCOMMON CAUSES

- Congenital/structural anomalies
  - Vestibulo/vaginal stenosis
  - Septal bands
  - (Pseudo)hermaphroditism
- Stump pyometritis
- Sub-involution of placental sites (SIPS) post-partum
- Vaginal FB
- Vaginal/uterine neoplasia
- Secondary to systemic disease e.g. DM, HAC
- Secondary to infection e.g. *Brucella canis*, herpes virus, transmissible venereal tumour

DIAGNOSTIC APPROACH

The reproductive status of the bitch (intact, nulliparous, post-partum, multiparous or neutered) will rule in/out many of the potential diagnoses.

Clinical clues

Predisposition

- Atrophic or juvenile vulva with excessive skin folds and perivulvar dermatitis
- Exposure to hormone products can increase discharge
- Haemorrhagic discharge more suggestive of neoplasia, systemic bleeding disorder, proestrus or SIPS
- Overweight dogs with urinary incontinence and excessive skin folds and urine scalding
- Vulval discharge seen in intact and especially breeding bitches, more than neutered females

History

- Accompanying signs, e.g. attractive to males, pain, pollakiuria
- Details of recent oestrus in entire females
- In neutered females review if any signs of oestrus present, in case of ovarian remnant
- Malodorous purulent/sanguinous discharge typically associated with bacterial infection
- May be systemically well; lethargy/inappetence/pyrexia with systemic disease
- Other clinical signs suggestive of systemic disease or venereal disease
- PU/PD in DM and HAC
- Sterile inflammation is normally mucoid and non-malodourous
- Vaginitis is more common in spayed females

Clinical examination

Visual inspection

- Excessive licking/grooming
- Nature of vulval discharge: colour, smell, volume
- Staining of perivulval hair
- Swelling of vulva

Physical examination

- Assess vulval conformation, e.g. hooded vulva
- Inflamed vulval membranes
- Inflamed, moist, erythematous perivulvar skin
- Vulval discharge

Laboratory findings

Haematology

- Anaemia post-partum or with SIPS
- Leukocytosis ± left shift ± toxic neutrophils with infections such as pyometra
- Leukopenia in some cases of pyometra

Serum biochemistry

- Unremarkable unless systemic illness

Urinalysis
- Identify concurrent urinary tract infection

Imaging
Plain radiographs
- Evidence of enlarged uterus with pyometra

Ultrasound
- Evidence of changes to the uterus
- Evaluate for ovarian disease: cysts/neoplasia/remnant
- Evaluate for concurrent urinary tract disease

Special tests
- *Brucella canis* serology
- Coagulation profile if bleeding for evidence of systemic bleeding disorder
- Ovarian remnant investigations
  - Anti-mullerian hormone
  - Laparoscopy/laparotomy
  - Progesterone
  - Vaginal cytology
- Progesterone testing for stage of oestrus
- Retrograde vagino-urethrogram
- Vaginal investigations for nodules, masses, FB, strictures
  - Biopsy
  - Culture for aerobic and *Mycoplasma* culture: normally mixed flora present
  - Cytology: assess stage of cycle or evidence of ovarian remnant
  - Digital rectal palpation
  - Vaginoscopy and cystoscopy

1.51 WEAKNESS, COLLAPSE AND SYNCOPE

DEFINITIONS

Weakness is a state of lacking strength, firmness, or vigour.
- Can be due to:
  - lassitude/fatigue, which is a lack of energy or
  - generalised muscle weakness; asthenia is a true reduction in muscle tone
- Collapse is a transient or persistent loss of postural tone in one of more limbs
- Syncope is a collapse due to cardiac disease and is caused by deprivation of energy substrates, either oxygen or glucose, which briefly impairs cerebral metabolism
  - Episodes are usually transient with either flaccid muscles or opisthotonus, but other causes of collapse can be more sustained
  - Most commonly results from impaired cerebral blood flow

RELATED CLINICAL SIGNS

Weakness
- Cachexia
- Generalised muscle weakness

Collapse
- Muscle flaccidity
- Transient loss of consciousness

Signs related to underlying disease
- Altered appetite
- Altered defaecation
- Congestive heart failure
- Cough
- Cyanosis
- Dysphagia
- Dyspnoea/tachypnoea
- Lameness
- PU/PD
- Stertor
- Vomiting
- Weight loss or gain

COMMON CAUSES

Weakness is a non-specific sign and the causes are too numerous to list. Causes of collapse are listed.

Cardiovascular disease
Acquired disease
- Dilated cardiomyopathy

- Arrhythmogenic right ventricular cardiomyopathy

Arrhythmia
- Bradyarrhythmia
  - Mobitz type 2 second degree or third-degree atrioventricular (AV) block
  - Sick-sinus syndrome
- Tachyarrhythmia
  - Supraventricular tachycardia
  - Ventricular tachycardia

Congenital disease
- Aortic stenosis
- Pulmonic stenosis

Pericardial disease
- Pericardial effusion
  - Idiopathic haemorrhage
  - Right auricular haemangiosarcoma

Vascular
- Vasovagal syncope
  (aka reflex-mediated syncope, neurocardiogenic syncope, situational syncope)
  - Combination of bradycardia and vasodilation
  - Triggered by excitement, cough, emesis, urinating, defaecating, etc.

Endocrine/metabolic
- Hypoadrenocorticism
- Hypoglycaemia

Haematological
- Acute blood loss anaemia (e.g. ruptured splenic mass)
- Immune mediated haemolytic anaemia
- Erythrocytosis increasing blood viscosity

Neurological
- Vestibular disease

Orthopaedic disease
- Fracture
- Osteoarthritis

Respiratory tract disease
- Hypoxia secondary to:
  - *Angiostrongylus vasorum*
  - BOAS
  - Pleural effusions
  - Pulmonary fibrosis
  - Tracheal collapse

UNCOMMON CAUSES

Cardiovascular disease

Acquired disease
- Myxomatous mitral valve disease after major chordae tendinae rupture

Congenital disease
- Right to left shunting disorders (patent ductus arteriosus [PDA], ventricular septal defect [VSD])

Pericardial disease
- Effusive constrictive pericarditis
- Heart base tumour/chemodectoma
- Mesothelioma

Vascular
- Carotid sinus hypersensitivity
  e.g. brachycephalic dogs, carotid sinus inflammation/neoplasia
- Iliac thrombosis

Endocrine/metabolic
- Hyperkalaemia
- Hypokalaemia
- Hypocalcaemia
- Hyponatraemia/hypernatraemia: only if rapid changes in sodium
- Hypothyroidism
- Phaeochromocytoma

Iatrogenic
- Drug administration, e.g. phenothiazines, drugs with effects on systemic BP

Neurological
- Exercise-induced collapse
- Increased intra-cranial pressure (e.g. space occupying lesion)
- Movement disorders
- Narcolepsy/cataplexy

Other
- Myasthenia gravis causes weakness
- Pain
- Pyrexia

Respiratory tract disease
- Non-cardiogenic pulmonary oedema
- Pulmonary thromboembolism
- Tracheal/bronchial FB

DIAGNOSTIC APPROACH

1 History – collapse episodes should be documented and recorded where possible
2 Confirm the presence or absence of respiratory signs.
3 Rule out orthopaedic disease with physical examination.
4 Rule out haematological and metabolic disease by laboratory testing.
5 Investigate suspected cardiac disease.

Clinical clues
- Distinguish syncope from seizures
  - Does the dog go flaccid or develop opisthotonus (syncope) or have tonic movements (seizure)?
  - Is there a rapid recovery (syncope) or a prolonged post-ictal period (seizure)?
  - Is the dog normal between episodes?
  - Is the dog otherwise unwell?

Predisposition
- Auricular haemangiosarcoma and pericardial haemorrhage: GSD
- Aortic stenosis: Boxer, Golden retriever, Newfoundland
- Arrhythmogenic right ventricular cardiomyopathy: Boxer
- Dilated cardiomyopathy (DCM): large- and giant-breed dogs
- Heart-based tumours: Boxer
- Hypoxia and vasovagal syncope: brachycephalic dogs with BOAS
- Idiopathic pericardial haemorrhage: giant-breed dogs, Golden retriever
- Pulmonary fibrosis: WHWT
- Sick sinus syndrome: Schnauzer and WHWT
- Tracheal collapse: Yorkshire terriers

History
- Characterisation of type of collapse
  - Association with
    - Exercise or excitement: cardiovascular, respiratory
    - Feeding or starvation: endocrine, metabolic
    - Rest or waking: neurological
  - Duration of event
  - Altered behaviour before or after episode – neurological
  - Mucous membrane colour during episode
    - Cyanosis: cardiovascular, respiratory
    - Pallor: cardiovascular, respiratory, haematological
  - Frequency of episodes
    - Continuous weakness: endocrine/metabolic
    - Episodic collapse: cardiovascular, respiratory
  - Behaviour during collapse
    - Flaccid or opisthotonus ± urination: cardiovascular syncope
    - Tonic-clonic movements, hypersalivation, urination, defaecation: neurological
- Differentiate from seizures; this is important and sometimes difficult
- Drug/chemical/parasite exposure
- Familial history of congenital heart disease, seizures
- Other clinical signs reflect underlying disease, e.g. polydipsia, altered appetite, dysphagia, vomiting, polyuria, incontinence, altered defaecation, respiratory signs, weight loss or gain suggestive of concurrent disease

Clinical examination
Visual inspection
- Brachycephalic conformation
- Gait abnormalities
- Respiratory pattern
- Weight loss

Physical examination
- Mucous membrane colour and capillary refill time
- Neurological system including reflexes, etc.
- Skin for any evidence of endocrinopathies

Auscultation
- Assess for any pulse deficit/arrhythmia

- Abnormal percussion suggesting fluid/consolidation in thoracic cavity
- Abnormal respiratory noise/effort
- Cardiac evaluation for murmurs, etc.

Palpation
- Abdominal palpation for evidence of ascites, masses
- Femoral arteries for pulse
- Lymph nodes
- Musculoskeletal system
- Peripheral pulse volume and rhythm

Laboratory findings

Haematology
- To assess for anaemia or erythrocytosis

NB: In acute blood loss the PCV may remain normal for 12–24 hours
- Check total proteins in conjunction

Serum biochemistry
- To assess in particular:
  - Glucose
  - Electrolytes/ions (sodium, potassium, calcium, magnesium)
  - Muscle enzyme activities (CPK, AST) increased by muscle disease and prolonged seizure
  - Blood urea
  - Liver enzyme activities (ALT, ALP)
  - Proteins

Urinalysis
- Proteinuria predisposing to hypercoagulable state leading to thromboembolism

Imaging

Plain radiographs
- Inspiratory films to assess cardiac size and lung fields
- Expiratory films or fluoroscopy to assess dynamic airway collapse
- Skeletal radiographs may be indicated by the results of physical examination

Ultrasound
- Abdomen
  - Adrenal glands
  - Free fluid
  - Masses
  - Urinary obstruction
- Thorax
  - B lines
  - Free fluid or air
- Echocardiography

Special tests
- Acetylcholine receptor antibody titres or edrophonium response test if myasthenia gravis suspected
- ACTH stimulation to investigate hypoadrenocorticism
- Baermann technique or Angiodetect® for *Angiostrongylus vasorum*
- Blood gas analysis: resting, pre- and post-exercise
- Blood glucose
  - High normal/increased insulin in face of hypoglycaemia is diagnostic for insulinoma
  - Hypoglycaemia with xylitol toxicity
- CT for more detailed assessment of lungs and abdominal structures; contrast can help identify abnormalities
- ECG: in clinic and Holter monitor or event recorder
- Echocardiography with bubble study in suspected cardiac disease
- Electromyography (EMG), nerve conduction velocities, and muscle and nerve biopsies in investigation of neuromuscular disease
- Exercise testing
- Liver function tests, e.g. bile acids
- MRI and CSF analysis for suspected neurological causes of syncope or true seizures
- Respiratory tract investigation e.g. bronchoscopy and BAL
- Serum lactate: resting, pre- and post-exercise
- Thyroid function tests to investigate hypothyroidism

1.52 WEIGHT GAIN/OBESITY

DEFINITION

Weight gain occurs through the accumulation of fat, muscle mass or fluid, or by the growth of large masses.
Obesity is an abnormal accumulation of body fat and develops when caloric intake is increased and/or energy use is decreased.

- Any increase > 15% over ideal body weight is considered overweight
- Obesity is usually defined as an increase in body fat such that body weight is 20% greater than ideal
- Obesity predisposes to significant health risks
  - Cardiovascular disease
  - Diabetes mellitus
  - Orthopaedic disease: cruciate ligament disease, degenerative joint disease and osteoarthritis, IVDD

RELATED CLINICAL SIGNS

- Increased appetite
- Increased body mass
  - Ascites
  - Increased fat deposition
  - Large mass
- Lethargy/exercise intolerance

COMMON CAUSES

- Decreased exercise
  - Inactive lifestyle
  - Limited by orthopaedic (especially osteoarthritis) or cardiorespiratory disease (especially BOAS)
- Dietary
  - High-calorie diets
  - Overeating/overfeeding
- Drugs
  - Corticosteroids
  - Phenobarbital
  - Progestagens
- Genetic predisposition: pro-opiomelanocortin (POMC) mutation
- Hepatosplenomegaly
- HAC
- Hypogonadism, especially in bitches after ovariohysterectomy
- Hypothyroidism
- Pregnancy

UNCOMMON CAUSES

- Acromegaly
- Increased abdominal organ size
- Insulinoma
- Large mass (e.g. lipoma, haemangiopericytoma, granulosa cell tumour)
- Peripheral oedema/ascites
- Pyometra

DIAGNOSTIC APPROACH

1 Record weight and body condition score.
2 If an increase in body weight is noted which cannot be reduced by decreasing dietary intake or if there are concurrent clinical signs, a pathological cause should be sought.
3 If overfeeding can be ruled out, and there is no obvious fluid accumulation or mass, an endocrinopathy is most likely.

Clinical clues

Predisposition

- Acromegaly in dogs in metoestrus or treated with progesterone
- Genetic predisposition in Labradors and Flat-coat retrievers with POMC mutation
- HAC in middle-aged to older dogs
- Hypogonadism after neutering
- Hypothyroidism
- Insulinoma in middle-aged to older bitches
- Osteoarthritis in older dogs

History

- Administration of progesterone or recent oestrus in acromegaly
- Increased appetite in HAC, diabetes mellitus, insulinoma

- Lethargy in hypothyroidism
- PU/PD in HAC
- Recent mating in pregnancy
- Recent oestrus before pyometra

Clinical examination

Visual inspection
- Alopecia in hypothyroidism and HAC
- Obesity

Physical examination
- Abdominal mass if neoplasia
- Ascites
- Enlarged uterus or foetuses in pregnancy
- Increased subcutaneous fat and thickened skin in hypothyroidism
- Increased appetite in HAC, insulinoma
- Intra-abdominal fat accumulation and a pot belly characteristic of HAC
- Subcutaneous oedema
- Thin skin in HAC
- Tubular structure if pyometra
- Weakness and/or seizures in hypoglycaemia caused by insulinoma

Laboratory findings

Haematology
- Inflammatory leukogram sometimes in pyometra
- Stress leukogram in HAC

Serum biochemistry
- Hyperglycaemia and glycosuria in diabetes mellitus
- Hypoalbuminaemia if ascites/oedema
- Increased ALP activity in HAC

Imaging
- Adrenal mass(es)
- Hepatosplenomegaly
- Occult thoracic and abdominal masses
- Pregnancy and pyometra

Special tests
- DNA test for POMC mutation not yet available commercially
- Dynamic cortisol testing
- Raised serum insulin in face of hypoglycaemia
- Increased IGF-1 in acromegaly
- Thyroid function tests

1.53 WEIGHT LOSS

DEFINITION

A dog will lose weight when its energy use or loss exceeds its energy intake and can, therefore, occur with increased use or loss, or with decreased intake or absorption, or with both. A loss of more than 10% of body weight is considered significant.

RELATED CLINICAL SIGNS

- Reduced body condition
  - Cachexia denotes extreme weight loss resulting from metabolic derangements with associated weakness and depression, and often cannot be corrected by increasing dietary energy intake alone
  - Emaciation is severe weight loss and equates to approximately > 20% loss of body weight when bony prominences become noticeable
- Loss of muscle mass will occur in cachexia, but loss in the absence of loss of fat suggests inflammatory myopathies or muscular dystrophy
- Other signs may be associated with the primary cause,
  e.g. diarrhoea in malabsorption, dyspnoea in cardiac failure, pyrexia, etc.

COMMON CAUSES

Normal to increased appetite,
q.v. section 1.36
Physiological
- Exercise
- Lactation

- Planned weight loss programme
- Pregnancy (weight gain later)

Pathological

- Malabsorption
  - ARD/SIBO
  - EPI
  - Mild IBD/CIE
- Non-ketotic DM
- Regurgitation, *q.v.* section 1.41
- Vomiting, *q.v.* section 1.49

Decreased appetite

Any condition causing anorexia, q.v. section 1.5

- Chronic kidney disease
- Fever
- Dysphagia and regurgitation: tries to eat but cannot swallow, *q.v.* section 1.41
- Heart failure (cardiac cachexia)
- Hypoadrenocorticism
- Ketoacidotic diabetes mellitus
- Malabsorption
  - Severe CIE/IBD
- Liver disease
- Neoplasia (cancer cachexia)
- Pain

UNCOMMON CAUSES

Normal to increased appetite

- Chronic blood loss
- Cold environment
- Hyperthyroidism
  - Dietary: raw food containing thyroid tissue
  - Iatrogenic
  - Rare functional tumour
- Fanconi syndrome
- Malabsorption
  - Bile salt malabsorption
  - Lymphangiectasia
  - Short bowel syndrome
- Poor quality food
- Protein-losing nephropathy
- Systemic fungal infections, especially histoplasmosis
- Temporal myositis
- TMJ disease
- Underfeeding
  - Accidental lack of food
  - Errors when calculating energy requirements
  - Malicious withholding of food
- Widespread skin burns

Decreased appetite

- Malabsorption
  - Alimentary lymphoma
- Oral disease
  - Oral neoplasia
  - Severe dental disease
  - TMJ dysplasia
  - Temporal muscle myositis
- Severe intestinal parasitism
- Severe pyoderma

DIAGNOSTIC APPROACH

1 Record weight, body condition score and muscle score.
2 The differential diagnoses can be subdivided by whether the dog wants to eat or has a reduced or absent appetite.
3 Weight loss simply due to lack of food intake causes an expected weight loss of ~2% body weight per week.
4 Weight loss in excess of 2% body weight per week indicates increased energy loss or usage.
5 Other signs may help localise the organ system involved and direct investigations.

Clinical clues

Predisposition

- Congestive heart failure (CHF) in small breeds (especially CKCS) with valvular endocardiosis, and large breeds with DCM
- EPI in Collies, GSDs
- Internal malignancies most commonly in Boxer, Flat coat retriever, Bernese mountain dog
- Malabsorption through lymphangiectasia in Lundehund, Rottweiler, Yorkshire terrier
- PLE ± PLN in Soft-coated wheaten terrier

History

- Abnormal faeces in malabsorption and EPI

- Cough/dyspnoea/ascites in CHF
- Inadequate intake identified in history
- Increased appetite in malabsorption and EPI

Clinical examination

Visual inspection

- Bony prominences and ribs obvious when emaciated
- Jaundice if liver disease

Physical examination

- Neurological examination
- Palpable goitre with functional thyroid mass
- Palpable mass if intra-abdominal neoplasia

Laboratory findings

Haematology

- Anaemia suggests chronic blood loss
- Eosinophilia suggests parasitism
- Leukocytosis suggests inflammation or infection

Serum biochemistry

- Azotaemia if renal disease
- Glycosuria and hyperglycaemia if diabetes mellitus
- Hypoproteinaemia suggests PLE, PLN or liver disease
- Increased liver enzyme activities ± hyperbilirubinaemia if liver disease
- Proteinuria if PLN

Faecal examination for parasites

Imaging

- CHF
- Occult thoracic and abdominal masses
- To identify effusions

Special tests

- Bronchoscopy
- Cardiac evaluation
  - Cardiac biomarkers
  - Electrocardiography
  - Echocardiography
  - Holter monitoring
- Calcium status
  - Ionised calcium
  - Parathyroid hormone
  - Parathyroid hormone-related protein
  - Vitamin D
- Endocrine testing
  - Fructosamine
  - Basal cortisol ± ACTH stimulation testing
  - Total thyroxine ± free thyroxine (free T4)
- Intestinal evaluation
  - Folate and cobalamin
  - Endoscopic or surgical intestinal biopsy for malabsorption
  - Serum TLI for EPI
- Neuromuscular evaluation
  - Acetylcholine receptor antibodies
  - Cerebrospinal fluid analysis
  - Muscle and nerve biopsy
  - *Toxoplasma* and *Neospora* serology
  - 2M antibody for masticatory myositis
- Nutritional evaluation of the diet
- Pancreatic testing
  - DGGR lipase – not pancreas-specific
  - Spec cPL
  - TLI
- Renal evaluation
  - Glomerular filtration rate
    - Exogenous creatinine clearance
    - Iohexol clearance
  - Symmetric dimethylarginine (SDMA)
  - Urine protein:creatinine ratio
- Therapeutic trials
  - Antiparasitic medication
  - Exclusion diet