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SMALL ANIMAL NEUROLOGY

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VETERINARY PRACTICE

SMALL ANIMAL NEUROLOGY

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DEDICATION
To my parents

Commissioning Editor: Robert Edwards
Development Editor: Ewan Halley
Project Manager: Anitha Rajarathnam
Designer/Design Direction: Charles Gray
Illustrator: Deborah Maizels

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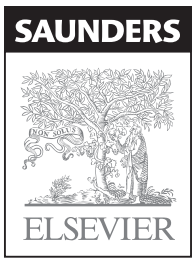
Series Editor: Fred Nind BVM&S, MRCVS

Susan N. Fitzmaurice

BVSc DipACVIM(Neurology) DipECVN MRCVS

With contributions by

Edward Friend BVetMed CertSAS MRCVS



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Introduction

Saunders Solutions in Veterinary Practice series is a new range of veterinary textbooks which will grow into a mini library over the next few years, covering all the main disciplines of companion animal practice.

Readers should realize that it is not the authors' intention to cover all that is known about each topic. As such the books in the *Solutions Series* are not standard reference works. Instead, they are intended to provide practical information on the more frequently encountered conditions in an easily accessible form based on real-life case studies. They cover that range of cases that fall between the boringly routine and the referral. The books will help practitioners with a particular interest in a topic or those preparing for a specialist qualification. The cases are arranged by presenting sign rather than by the underlying pathology, as this is how veterinary surgeons will see them in practice.

It is hoped that the books will also be of interest to veterinary students in the later parts of their course and to veterinary nurses.

Continuing professional development (CPD) is mandatory for many veterinarians and a recommended practice for others. The *Saunders Series* will provide a CPD resource which can be accessed economically, shared with colleagues and used anywhere. They will also provide busy veterinary practitioners with quick access to authoritative information on the diagnosis and treatment of interesting and challenging cases. The robust cover has been made resistant to some of the more gruesome contaminants found in a veterinary clinic because this is where we hope these books will be used.

Joyce Rodenhuis and Mary Seager were the inspiration for the Series. Robert Edwards has overseen their writing and production. The Series editor and the individual authors are grateful for their foresight in commissioning the Series and their unfailing support and guidance during their production.

NEUROLOGY

Neurological cases can be challenging, confusing and even downright frightening. In many cases we are dealing with one of the few organs in which severely compromised function is not compatible with life. It is easy to think that all neurological cases are hopeless. This book shows you that this is not always the case. For many patients, treatment is available which can provide an acceptable quality of life.

But before selecting that treatment a diagnosis must be established. Neurological diagnosis rests on the bedrock of a thorough and logical clinical neurological examination. All the electronic wizardry in the world will not give you a diagnosis without it. This book will guide you through that basic process, indicate the sort of extra help that electronic wizardry can sometimes provide and hopefully mean that those neurological cases are not so frightening after all.

Even for those cases that do end up being put to sleep, you will at least now know why you are doing it.

Fred Nind
Series Editor
2010

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Author's note

If you read no further, consider these three things:

1. Diagnosing the neurological disease is cheap and requires only observation and interpretation (i.e. there is no excuse for not trying).
2. Differentiating which disease is responsible for the neurological signs may be possible based on signalment and history (i.e. the answer may be in front of you).
3. Imaging of the nervous system can never replace the neurological examination and history in the management of cases (i.e. you cannot rely on technology).

Most people can't do the first, haven't checked the second, and put their faith in the third. These people are called clients. You know better.

The aim of this book is to make the recognition and interpretation of neurological signs easier for those who work in general practice. It is ordered in a way that I would normally conduct an investigation into a case. First, quiz the owners. Second, examine the patient. Then localize the lesion. Finally, if necessary, conduct further diagnostic tests. Neurologic diseases are only briefly described, as examples of the way clinical signs can present. Diagnostic testing is only briefly summarized, because if you have got that far in case management, you should be reading more detailed textbooks on radiology and clinical pathology. I have tried to limit the amount of neuroanatomy as it frightens most vets away from clinical neurology but it is difficult to throw away specific terminology and not run the risk of obscuring the message.

The vast proportion of a diagnosis comes from the history and clinical examination, so buy this book instead of an MRI machine.

Susan Fitzmaurice, 2010

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SECTION 1

THE NEUROLOGICAL CASE

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1 Taking a history

INTRODUCTION

Allowing the client to describe the problem without interruption is the fastest way to take a complete history. Admittedly, the information usually arrives in a disorganized order with extraneous detail or no detail at all. Points made will need clarification. Using a template to collect details aids the memory of the interviewer and assimilates the points of interest in a logical order.

- Never assume the client understands the medical jargon they use.
- Never assume the past diagnosis is correct.
- Many clients describe left and right orientation as they see it, facing the animal. Ask them to point out the side of the problem or ask if it is the same side as that currently visibly affected.

Gentle persistence is required. No one likes to be made to feel stupid, so be tactful when phrasing questions. Allow enough time, but do keep control of the situation. After being encouraged to recount the events, owners sometimes repeat the same story several times at which point it would be appropriate to gently deflect them with a new line of enquiry. Elicit information if it is not volunteered but avoid leading questions which limit observations to those suggested by the examiner. Listen to all parties in the exam room, including children. It is vital to get a description of what the client saw or heard rather than their interpretation.

Example: Dog presented for seizures:

Client: 'My dog fell down the stairs and hit its head.'

Vet: 'Did you see that happen?'

Client: 'No, but I heard a noise and found the dog on the floor thrashing about, hitting the wall with its limbs.'

COMPONENTS OF THE HISTORY

Signalment

The age, sex and breed of an animal. The incidence of many diseases is determined by the signalment.

Presenting complaint

The sign or group of signs which lead to the consultation and the reason for the referral. It may be a historical problem or one that is present at the time of the examination. Generally, the presenting complaint consists of the latest or most catastrophic problem to befall the animal. Chronic clinical disease may go unreported by the owner but must not be ignored by the veterinarian. Concurrent abnormalities affect clinical decisions, overall prognosis, financial outlay, as well as owner compliance.

Last normal

When the presenting complaint began. Clarify by asking if the animal was totally normal prior to that point in time.

History narrative

The description of the animal's abilities, disabilities, general health and daily routine. Clarify the extent of the animal's ability with specific questions:

Strength/gait/posture:

- Can he walk up/down stairs?
- Can he get into the car himself?
- Can he climb onto furniture by himself?
- Can he lift a hindlimb to urinate?
- Can he maintain a squatting position to defecate (without collapsing)?
- Can he keep up with you on a walk?

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Mental state:

- Is he house-trained? Has there been any urination/defecation in the house? Where? When?
- Does he respond to commands?
- Does he recognize/greet people/animals and have the interactions changed?
- Will he fetch specific toys if requested?
- Has his sleep pattern changed?
- How does he normally react at a veterinary practice?

CLINICAL TIP

Despite being specifically asked, owners sometimes will not mention that the animal has defecated or urinated in the house. Owners describe it as 'an accident' and continue to say the pet is house-trained. The vet thus misses an early sign of cerebral disease, polyuria, urinary or faecal incontinence or psychological disturbance. It is still worth asking if the animal is house-trained as it shows the animal was capable of learning and retaining information at some stage in its life.

Seizures:

- Pre-ictal
  - Is there any warning? Any triggers?
  - Description.
- Ictus
  - Time of day? Activity at or prior to the onset of seizures?
  - Single or multiple
  - Duration
  - Frequency
  - Description: physical movement, mental state, progression of event, each event similar or variable.
- Post-ictal
  - How does the seizure end? Description of activity/mental state.
  - Duration of recovery period, until the animal is back to its normal mental state and activity.

Progression of signs

- Improving, stabilized, worsening, waxing and waning/episodic/intermittent
- Response to rest/exercise/time of day/feeding.

Ask the owner to compare the animal's ability at the time of examination to that occurring at the commencement of signs for a better idea of the progression of the disease.

CLINICAL TIP

Sometimes a problem has been going on so long that owners accept it as normal or assume 'old age' is to blame.

Treatment

- What medications have been used? Start/finish dates. Compliance (check the amount dispensed and ask how much is left over)
- Serum levels at various anticonvulsant dose rates
- Response to physical rest. Clarify what the owner means by 'resting' the animal
- Side-effects noted: polyuria (PU), polydipsia (PD), polyphagia (PP), panting, sedation, hyperactivity, ataxia, vomiting, etc.
- Adverse reactions: dose-related/idiosyncratic
- Dietary supplements, homeopathy, alternative therapies.

Past history

- BIOP: been in the owner's possession. When did the owner acquire the dog? From whom?
- Any major illness/trauma?
- Has there been any previous occurrence of the same problem? Similar or different?

OTHER BODY SYSTEMS

It is very important to review the function of other body systems. Disease of other organ systems can produce signs suggestive of primary neurological disturbance (depression/lethargy, exercise intolerance, weakness, collapse, muscle spasm, pain).

Some owners regard this as an irrelevant diversion from the main problem so it can help to introduce these questions with a brief comment on the importance of an overview of their animal's general health.

Inquire about the occurrence of vomiting, diarrhoea, coughing, sneezing or any change in body weight. If

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vomiting is reported, request a description to determine if regurgitation is the real problem. Any alteration in thirst should be quantified and compared with past behaviour. Appetite change, ability to prehend and swallow food, and urination and defecation ability need to be questioned.

Daily routine

It is important to discover the role of the animal in the owner's lifestyle as this will influence treatment decisions and client expectations, e.g. working, agility, hunting, show dog vs. geriatric pet. An owner with a bad back living in an apartment block with no elevator is unable to carry a paralysed dog outside. A detailed inquisition of the owners' circumstances is not required but do remember to address future treatment requirements in your questioning.

- 'What does he do all day?'
- Exercise and feeding: when and how much?
- House-trained; urination/defecation habits
- Interactions with other humans/animals.

Travel

A travel history assesses the potential for vector-borne, parasitic and saprophytic infectious disease not found in the local area. Check for exposure to other animals in kennels.

Environment

Animals with chronic loss of vision may appear to develop sudden onset blindness when moved to a new location or if furniture is rearranged. Exchanging wall-to-wall carpet for floorboards removes a pet's ability to grip, and accentuates previously subclinical weakness and ataxia. Other animals in the household or kennels may be affected due to hereditary or environmental factors (e.g. shared food source).

Anything else?

This conversational gambit gives the client a chance to mention things they have only just remembered or may not have thought relevant when first recounting the history. It can be incredibly helpful.

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2 The neurological examination

Examination of nervous system function is achieved by observation, palpation and inducing reflexes and responses. The following section will explain the significance of the tests and how to perform them.

Each section of the examination is simply a part of the jigsaw (Fig. 2.1). The whole picture becomes clear once all the information is in place.

CLINICAL TIP

The more you practise, the easier it gets.

MENTAL STATE

Aim

To discover the degree of mental alertness and the quality of responsiveness.

Significance

Mental alertness is sustained by a network of neurons within the brainstem (ARAS: ascending reticular activating system) that project to the cerebrum. Damage to either alters alertness. Systemic illness commonly causes decreased alertness in the absence of primary structural neurological disease. Voluntary actions originate from the cerebrum and are influenced by the patterns of behaviour of that species and learnt behaviour imposed by domesticity; it is therefore important that the clinician is aware of normal species behaviour and the level of training (if any) of the animal. Inappropriate responsiveness to the environment indicates cerebral malfunction, or a psychological disturbance (Fig. 2.2).

A depressed mental state can alter responses. A response, used in a neurological sense, is an action which requires a degree of input from the cerebrum (*cf.* a reflex action). The menace response, proprioception and pain perception may be absent or inconsistently present with a decreased alertness.

Examination

Observe the animal. Notice its interaction with other animals and humans in the waiting room. Once in the examination room, remove any leads and allow the dog to wander around the examination room unrestricted unless there is a loss of balance, history of trauma, paralysis, or any possibility of aggression. This can be done while the history is being taken. Most dogs investigate new surroundings and then either rest, return to the owner for attention, or try and escape out of the examination room. If the latter occurs, ask the owner to restrain the dog. If the dog appears abnormally quiet, or is excessively timid, it is useful to walk the dog outdoors and see if its mental state changes. Ask the owner if the behaviour witnessed is the normal reaction of the animal at the veterinary practice or at home.

Most cats will refuse to exit their cat baskets. Once removed from this shelter, the normal cat usually seeks refuge under chairs, tables or behind waste bins, and refuses to move. Some normal cats do wander about the room and investigate and may or may not voluntarily return to the basket. It is a good idea to keep the cat confined until one's full attention can be given to observation of both the mental state and the gait. This is to prevent the cat from jumping until the balance and strength have been assessed. It is also because of the fact that many cats will walk a little initially and then refuse to budge.

Notice if, and how, the animal responds to its name, noise and visual stimuli. Does it walk about the room without taking note of distractions? Does it bump into objects, get stuck in narrow places and have problems reversing out, or turn a particular way when changing direction? Does it stop and appear to stare into space,

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Figure 2.1 Equipment required for the neurological examination.

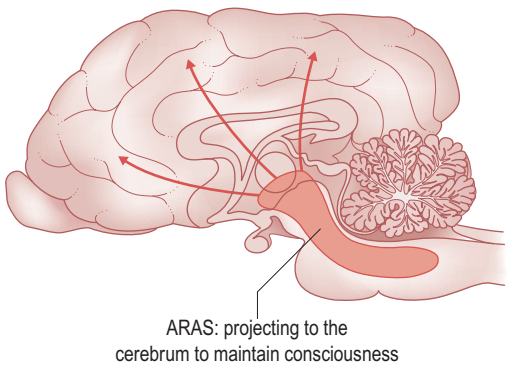


Figure 2.2 The ascending reticular activating system (ARAS).



Figure 2.3 Cat with an altered mental state.

at a wall or into a corner? None of these behaviours are normal (Fig. 2.3).

Assessment

Describe the abnormal mental state (see page 47) stating what is abnormal about the level of alertness or the appropriateness of the animal's response to its surroundings. This will act as a better comparison with subsequent examinations than a simple, bald statement of 'dull'. The clinician will need to decide if the evidence from the history and the examination supports a neurological cause of the altered mental state or not.

POSTURE

Aim

To describe the position of the limbs, head and trunk.

Significance

The position of body parts is notably influenced by balance, proprioception, coordination, strength and painful stimuli. The significance of an abnormality will only become obvious once the entire examination is complete.

Examination

Observe the animal while it sits, stands and moves about. For example, hindlimb weakness can manifest as an arched back with the hindlimbs positioned further cranially under the body (Fig. 2.4).

When sitting, the weak or paralysed hindlimbs may extend straight out between or amongst the forelimbs. The paretic animal is often slow to rise from a sitting position and the hindlimbs may not be fully extended



Figure 2.4 A myopathic Great Dane shifting the limb position to better support its body weight.

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Figure 2.5 Husky dog with paraparesis secondary to a chronic T3-L3 spinal cord lesion. Note the muscle atrophy.



Figure 2.6 Dobermann dog with cervical cord compression. Note the abduction of the left hindlimb.

when standing, or could be further apart than normal (abducted) (Fig. 2.5).

Watch for abduction of limbs which may slowly slide out to the side while the animal sits or stands. Note excessive flexion or extension of joints (Fig. 2.6).

Note the spontaneous movement of the neck. If the animal shakes its head or body, is the neck moved as vigorously as normal, or at all? Does the head turn when the animal changes direction or is the whole body, head-neck-trunk, moved as a unit? Is the head elevated into a normal position or is the spine held in one straight line? Restriction of neck movement is a sign of pain. Ventroflexion of the neck in cats is usually a sign of weakness. It may not be noticed if the cat is always lying down resting its head on the forepaws or ground. The neck may be lowered and the back may be arched in an effort to shift weight to the forelimbs in cases of hindlimb or back pain.



Figure 2.7 Cat with a right head tilt.

Watch for tremors of the trunk and the head at rest and during movement. Offer the animal something to smell and notice if there is any intention tremor of the head. Record the position of the head and if it is turned or tilted or both, and to which side (Fig. 2.7).

If the animal is recumbent, is it able to lift its head from the floor? And its forequarters? An animal in lateral recumbancy needs to lift its head before rising. If the neck is painful, or the animal is tetraparetic, it may not be able to rise unaided. Does it roll? Rolling is a sign of vestibular disease.

It is not necessary to perform the *righting reflex* by which an animal is held upside down off the ground by its pelvis. The normal response is for the animal to automatically assume a 45° angle to the horizontal with the forelimbs outstretched toward the ground. The sensory stimuli are from visual and vestibular input. If blindfolded, the normal animal will still assume the same posture. If vestibular dysfunction is present, the animal will curl up or writhe about.

Assessment

Description of abnormal posture (see pages 223, 224).

GAIT

Aim

To describe the animal's movement.

Significance

Movement is initiated by the upper motor neuron (UMN), a collective name for the nerve cell bodies within the cerebrum and brainstem and their axons which form the descending motor tracts (fasciculi) of the brainstem and spinal cord. The UMN initiates and maintains normal

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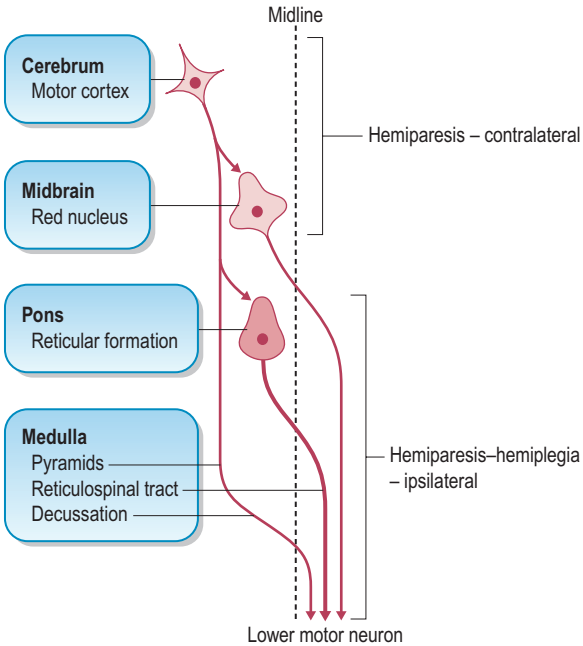


Figure 2.8 Pathways for voluntary control of movement.

movement and influences extensor muscle tone to support the body against gravity (Fig. 2.8).

Descending tracts

- Corticospinal – initiate movement
- Corticorubrospinal – initiate movement
- Reticulospinal – control tone and posture
- Vestibulospinal – control tone and posture.

Cerebral lesions usually have little effect on the strength and coordination of the gait as the corticospinal tracts are of minimal importance in the small animal.

The UMN terminates on interneurons within the spinal cord which have an excitatory or inhibitory effect on the lower motor neuron (LMN). The LMN is the collective name for the motor neurons and their axons which directly connect to muscle. The term LMN encompasses the motor neurons of the cranial nerves as well as the peripheral and spinal nerves, their neuromuscular junctions and the muscles themselves. It also applies to the autonomic nervous system although the LMNs in that case may be referred to as preganglionic or postganglionic.

UMN and LMN lesions both cause weakness. Weakness may be observed by a slowness to rise from sitting, inability to support the body weight or an inability to initiate and sustain movement. Weakness is a diminution of voluntary movement. Paralysis is an absence of voluntary movement.

Table 2.1 Characteristics of the motor supply

	UMN lesions	LMN lesions
Paresis or paralysis	Yes	Yes
Spinal reflexes	Normal to increased	Decreased or absent
Muscle tone	Normal to increased	Decreased or absent
Atrophy	Gradual (disuse)	Rapid (denervation)

UMN lesions are differentiated from LMN lesions by the quality of spinal reflexes and muscle tone. UMN lesions result in a loss of descending inhibition and a subsequent increase in muscle tone and spinal reflexes is observed. LMN lesions remove innervation to the muscles and a decrease in muscle tone and spinal reflexes is seen.

Observing the gait alone does not provide sufficient information for the clinician to assign the lesion to a specific part of the nervous system (i.e. LMN vs. UMN). Spinal reflexes must be tested and muscle tone must be assessed (Table 2.1).

It is also important to note that certain orthopaedic diseases can change the gait in a way that suggests neurological disease. Equally, lameness may result from certain neurological diseases. Thus, while observation is crucial, the clinician must actually touch the animal in order to differentiate orthopaedic from neurological causes of abnormal gait.

Strength is only one aspect of the gait. Balance and coordination change the direction and extent of limb movement. Cerebral lesions can result in circling, pacing, head pressing; movements which appear normal in execution but apparently purposeless in function, with a propulsive, relentless quality. The circling is towards the side of the cerebral lesion (Fig. 2.9).

Proprioception is the perception of the body in space. Position and movement of the head is chiefly detected by the vestibular apparatus in both inner ears (special proprioception). Receptors in muscles, tendons and joints project sensory information into the CNS (general proprioception). This is then transmitted to the cerebrum (conscious proprioception) and to the cerebellum (unconscious proprioception) by ascending pathways. Projections to the cerebellum enable it to regulate the gait. Cerebellar lesions do not cause weakness. Reduced

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Figure 2.9 A dog with a right cerebral lesion. Note the twisted lead, evidence of circling.



Figure 2.10 Observing the gait. Keep the dog between you and the owner.



Figure 2.11 Observing the gait. Grass obscures the view of paw movement.

or absent proprioception causes ataxia. Associated deficits found on examination help localize the lesion to the vestibular system, the cerebellum, the spinal cord, or the cerebrum. (Lesions of the special proprioception receptors are common. Lesions of the general proprioception receptors are rare.) Ataxia is a sensory phenomenon.

Examination

Observe the animal's ability to move. Normal animals are able to rise and walk unaided on floors with no grip (linoleum, floorboards). Weak and ataxic animals should ideally be examined on a non-slip surface. If the animal is paraparetic, support it under the abdomen so that any movement of the hindlimbs can be seen. If the animal is unable to rise, enlist assistance to hold the animal in a normal standing position and slowly move or coax it forward without releasing support. This is to determine if the animal is ambulatory tetraparetic (weak but able to walk unaided) or non-ambulatory tetraparetic (unable to walk without assistance). Sometimes paraparetic animals will not move the hindlimbs when a sling supports their abdomen. Remove the sling, and support the hindquarters with a hand under the inguinal area, or with a hand on either flank. Have an assistant coax the animal forward and watch for any hindlimb movement.

CLINICAL TIP

Do not confuse muscle tone with voluntary movement. UMN paralysis increases muscle tone and such an animal may be 'propped' up into an upright, standing, position. Rigor mortis also increases muscle tone, and there is no way that a dead animal could be said to possess voluntary movement.

Neurological deficits are noticeable when the animal is walking. Lameness shows up at faster gaits. Observe the gait from the side, front and from the rear. Squat down to observe the footfall of smaller dogs and cats (Figs 2.10, 2.11).

Walk the dog in small-radius circles: ask the owner to stay in the centre of the circle and lead the dog in a clockwise then anticlockwise direction. Watch for circumduction of the outer hindlimb; the limb on the periphery of the circle may swing out to the side in an arc when advanced. This indicates either a proprioceptive deficit, restricted movement of the joints, or increased muscle tone. If the dog will not cooperate by walking in tight circles, watch the limbs when the animal turns and changes direction in the examination room when off the lead.

Watch the dog step up and over any door sills or steps and note if the paws are dragged or scraped against the step.

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Allow cats to walk around the examination room. If they refuse to move, place them on the opposite side of the room to their owners or basket, which may encourage them to walk.

Assessment

The clinician must decide which, if any, of the limbs have abnormal movement. The head and trunk posture and movement should be commented upon. The animal's strength and coordination is then examined further with the postural reactions.

POSTURAL REACTIONS

Aim

- To assess the degree of voluntary motor function of each limb
- To assess the proprioceptive ability of each limb
- To detect deficits not noted when observing the gait
- To subjectively quantify the deficits.

Significance

Postural reactions are tests of strength and coordination. They distinguish the normal from the abnormal limb. Cerebral lesions can leave the gait unaffected, producing contralateral paresis and proprioceptive deficits only noted when performing postural reaction tests. Orthopaedic disease, myopathies and defects in neuromuscular transmission may leave an animal unwilling or unable to weight-bear on a limb, but the proprioception is left unaffected.

Examination

Hopping: The animal's weight is transferred to one limb which is then slowly pushed laterally, away from the centre of gravity.

Forelimbs: The animal is supported under the abdomen by one of the clinician's hands. The other hand grasps the antebrachium, flexing the limb off the ground. The animal is gently pushed laterally, away from the centre of gravity (Fig. 2.12).

The clinician notes any delay in initiating the hopping movement (a sign of weakness, or a conscious or unconscious proprioceptive deficit) and the degree of lateral movement of the tested limb (reduced by weakness, exaggerated with cerebellar disease). A weak limb may collapse, so the clinician must be ready to prevent the animal falling forward. If the animal cannot support weight on the forelimb being tested without collapsing, the clinician should support the forequarters with a hand under the sternum. Do not allow the owner to hold the head or the lead around the animal's neck as this removes the animal's weight from the forelimbs making it easier for the animal to hop (Fig. 2.13).



Figure 2.12 Good technique. Forelimb hopping. (Arrow indicates direction of movement.) Keep the animal in a normal position.

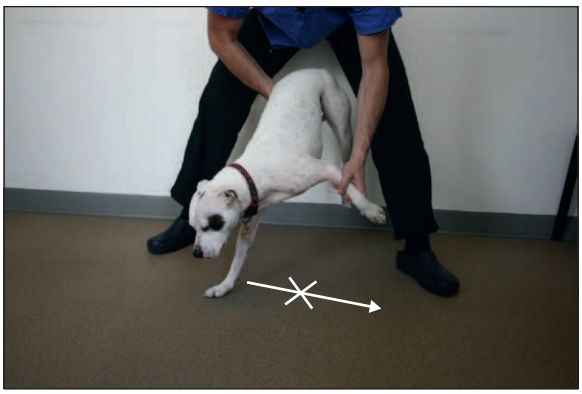


Figure 2.13 Forelimb hopping. Bad technique. The animal is lifted too high and is being pushed in a medial direction (see the arrow with the cross).

Hindlimbs: A similar technique is used. The body weight is supported by a hand under the animal's sternum; while the examiner's other hand holds one hindlimb in a flexed position. This is easier to perform with the animal's head facing the clinician. The point of supporting some or all of the animal's weight is to ensure that the limb being hopped is weight-bearing. It is very important, whether testing the fore- or hindlimbs, that the animal remains in as normal an anatomical posi-

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Figure 2.14 Hindlimb hopping. Keep the dog in a normal anatomic position. Push the animal in a lateral direction (arrow).



Figure 2.15 Hemiwalking. Push the dog laterally (arrow indicates the direction of movement).

tion as possible, with the spine as parallel to the ground as possible (Fig. 2.14).

Hemiwalking: This is a useful technique for larger, heavier dogs. The clinician stands on one side of the animal, lifts the ipsilateral fore- and hindlimb, and slowly pushes the animal laterally, away from the clinician (Fig. 2.15).

The animal hops on both limbs. This is a useful test for those animals that will not hop on the hindlimbs: normal dogs will sometimes extend the hindlimb and leave it to slide to the side when being pushed by the clinician. When hemiwalking, the animal has to hop or it will fall. This technique should be used with caution in the weak animal as the clinician is poorly positioned to prevent the animal from falling. Hemistanding describes the animal supporting its body weight using the limbs on one side of its body.

It is not physically possible to support the body weight of a giant breed dog while hopping it. This is overcome by leaving the forelimbs on the ground when hopping the hind, and vice versa when testing the forelimbs. Alternately, hemiwalking is used. Long-limbed sighthounds and short-limbed clinicians is another difficult combination which can be resolved with the same technique.

Assessment

When the body is pushed laterally and the shoulder is no longer vertically aligned with the paw, the limb hops laterally so that the paw is once again 'under' the shoulder and weight-bearing is achieved. An abnormal response is a delayed movement of the limb, an exaggerated movement of the limb (hypermetric and overreaches, the paw being placed too far laterally), no movement or collapse of the limb. Slow movement with a decreased range and collapse of the limb indicate weakness of the limb. A delayed onset of movement with preservation of strength is common with cerebellar lesions. A delayed onset with collapse of the limb indicates weakness.

Compare one forelimb with the other. Compare one hindlimb with the other. The normal hindlimb appears stiff and is placed slightly wider when hopped than would occur in a normal forelimb when hopped!

Strength is subjectively graded as:

- Absent: 0
- Decreased: 1+
- Normal: 2+

Wheelbarrowing assesses the forelimb gait. It is physically more difficult to perform than hopping. The clinician supports all the weight of the hindquarters by placing a hand under the animal's abdomen. The animal is then pushed forward. Cats almost uniformly refuse to cooperate and lie down which may be misinterpreted as forelimb weakness. It can be difficult to observe the movement of the forelimbs from one's position at the rear of the animal. It can be difficult controlling the direction the dog walks, and they may struggle when manipulated in this fashion. One way around this is for the clinician to place the other hand under the dog's head and dorsiflex the neck; this also removes visual clues and may highlight a proprioceptive deficit. It places

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an enormous strain on the clinician's back and the information obtained can be more reliably gained by hopping and proprioceptive testing.

CLINICAL TIP

It is vital that the clinician is able to apply postural testing in a consistent manner to a whole range of animals. Unreliable or difficult methods produce unreliable findings which the clinician cannot interpret.

Extensor postural thrust: The clinician stands behind the animal and with hands around the thorax, lifts the animal's forequarters and then the hind off the ground. The suspended animal is lowered to the ground, and its hindlimbs reach out and walk backwards. This is a useful alternative to hopping for testing hindlimb strength in cats. It is not routinely performed in dogs due to their size and weight. The normal response is for the hindlimbs to walk backwards, supporting weight to the same degree. A weak hindlimb is noted by it lagging behind or being dragged (Fig. 2.16).

Proprioceptive positioning ('knuckling'): Each limb, in turn, is tested by flexing the paw so that its dorsal surface becomes the weight-bearing surface. Normal animals will immediately turn the paw back to its natural position. An abnormal response is one in which the movement is delayed, does not occur, or replaces the paw too far forward, caudal, or lateral (abducted).

Technique is all important. Begin by supporting the animal's weight under the abdomen, when testing the hindlimbs, and under the sternum when testing the forelimbs. Weak or painful animals may have normal proprioception but be physically unable to support their body weight and correct the 'knuckled over' paw. Some normal animals will only correct the paw's position when the limb being tested is weight-bearing to some degree. The clinician slightly moves the animal, shifting its weight over to the tested limb to see if this is the case. Proprioceptive testing is a response and can therefore be affected by the animal's level of alertness. Cats do not tolerate 'knuckling' of the hindpaws and it is usually easier to test the hindlimb proprioception with placing responses (Fig. 2.17).

The paper slide test is harder to perform and less consistent results are obtained. The animal's paw is placed on a sheet of paper, which is then pulled to the side. The animal should return the displaced limb to a normal position. The problem is that a lot of normal dogs do not seem stimulated enough to return the limb to a

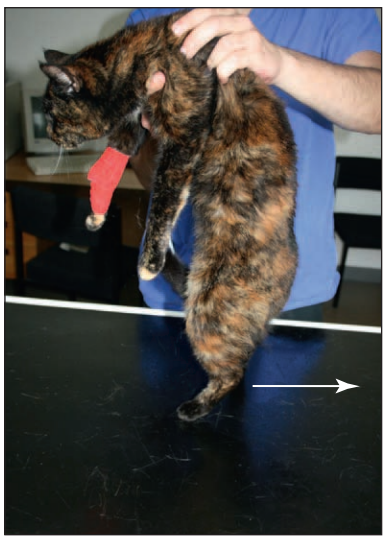


Figure 2.16 Lift the cat under the axillae and walk it backwards on a table top (arrow indicates direction of movement).



Figure 2.17 Proprioceptive testing. Once the paw is released by the clinician, the animal repositions the paw immediately.

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normal position, and thus allow the limb to remain on the paper sheet. There is some thought that the paper slide test assesses the proximal limb proprioceptors and the 'knuckling' tests the distal limb proprioceptors. This is irrelevant in the clinical setting. Proprioceptive deficits occur because of lesions in the sensory nerves, the ascending proprioceptive pathways, the sensory relay nuclei of the thalamus or the sensory cortex of the cerebrum.

Placing is another method of testing proprioception in smaller patients. The animal is held under the sternum and abdomen, picked up, and brought towards the edge of a table. The normal animal reaches for the table and places the forelimbs and hindlimbs on the table top without delay. Technique is very important. The animal must be brought towards the table at a speed which enables the animal time enough to move its own limbs. It must not be held so tightly against the clinician's body that it is unable to freely move its limbs. For this reason it is good practice to test the animal by holding it on either side. Small animals often get their hindlimbs caught in the clinician's lab coat pocket. *Visual placing* is the above technique performed without covering the animal's eyes. *Tactile placing* is the above technique performed with a hand over the animal's eyes (Fig. 2.18).

The animal soon learns to reach for the table, whether the eyes are covered or not. When performing tactile placing, approach the table at a slow, steady pace. Pausing in mid-space will induce the animal to reach for the table; this is a nice demonstration that the animal has learnt what to expect but it is not a measure of proprioception.

Proprioception is subjectively graded as:

- Absent: 0
- Decreased: 1+
- Normal: 2+

Abnormal wear of the claws and overlying fur: Proprioceptive deficits result in abnormal paw position when walking. Scraping, 'knuckling over' or dragging the dorsum of the paw against the ground flattens the dorsal surface of the middle two claws, wearing off the overlying fur. Examine all paws. This may not be noticeable if the dog is only walked on grass (Fig. 2.19).

PALPATION

Aim

To detect muscle asymmetry, atrophy, hypertrophy, muscle tone, joint laxity, or restricted movement, pain, masses, heat, cold, or swelling.

Significance

Pain and joint abnormalities affect the gait and posture. Atrophy occurs with disuse, denervation, secondary fibrosis of muscle, generalized weight loss, and in some



Figure 2.18 Visual placing. Note how the cat reaches for the table. Bandages alter the way animals move limbs. This cat was neurologically normal.



Figure 2.19 The dorsal surface of the claws has been worn by dragging them. The dog had a C1–5 cervical cord lesion.

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myopathies. Muscle enlargement may follow ischaemia, local inflammation, trauma, neoplasia, myotonia, and muscular dystrophy. The distal or proximal limb may be selectively involved.

Muscle tone is influenced by both the UMN and LMN. Judging the amount of muscle tone present, as assessed by palpation, contributes to the differentiation of LMN from UMN causes of paresis and paralysis.

Warmth of the extremities and pulse quality assesses limb perfusion.

Examination

Observation and palpation assess the symmetry of the left and right sides.

Head: The head is examined when the cranial nerve function is assessed. Ear canals should be palpated and smelt for evidence of otitis externa. Lymph nodes and tracheal sensitivity are checked as part of the physical exam.

Neck: Apply gradually increasing digital pressure to the dorsal spine and to the lateral spine to detect pain. A normal animal tolerates firm pressure. Hold the muzzle closed with one hand and slowly flex the neck laterally to either side. A normal animal is able to place its nose on its shoulder. An animal with neck pain refuses to move its neck. The clinician is able to feel increased neck tension or spasm and should not force the head laterally. Keep one hand on the neck while the head is dorsiflexed and then ventroflexed. The normal animal is able to look up at the ceiling and down at the floor without resisting neck movement. Do not perform this test in toy breed dogs with tetraparesis or neck pain as C1–C2 subluxation may be present. Fearful or excited normal dogs, or cats, may resist neck manipulation. Observe spontaneous movement of the neck; this is usually limited in range when neck pain is present.

Thoracic and lumbar spine: Place one hand under the animal's abdomen and with the other apply gradually increasing digital pressure to the dorsal spine to detect sites of pain. A normal animal tolerates firm pressure and does not collapse. If the animal resents palpation and struggles, wait until the animal calms down, and repeat the palpation to be sure that pain was the cause of the animal's reaction. Pressure on a painful thoracic or lumbar spine causes the abdominal wall to tense which can be felt by the examiner. It can be confused with abdominal pain. Acute pancreatitis is usually accompanied by vomiting (*cf*. acute spinal pain). Weak animals may flex the limbs and collapse when pressure is applied to the spine. Some normal animals sit (Fig. 2.20).

Lumbosacral junction (LSJ): There are several methods to detect pain at the LSJ. Apply direct pressure over the LS region. Extending one or both hips and applying pressure to the LS junction (*lordosis test*) can detect hip or LSJ pain. Extension or traction of the tail may cause pain. Palpation of the LS junction per rectum may cause pain. Pressure on LSJ while lifting the pubis may elicit pain.

Limbs: Palpate the limbs with the animal standing and in lateral recumbency. Palpate each muscle group and then press the long bones. Flex and extend the joints, feeling for crepitus, swelling, laxity or restriction of movement. A ruptured gastrocnemius tendon gives rise to a plantigrade stance: hold the stifle fixed in extension and gently press against the bottom of the paw: if the gastrocnemius tendon is intact the tarsus remains extended.

Assess muscle tone by the resistance to limb flexion: limb tone should be uniform between the fore and hindlimbs. Hit the muscle belly with a patellar hammer. A dimple develops in myotonia from sustained local muscle contraction.

Assessment

Muscle tone is assessed as absent (0), reduced (1+), normal (2+), increased (3+), greatly increased (4+).



Figure 2.20 Testing for spinal pain. Always palpate the abdominal wall at the same time.

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SPINAL REFLEXES

Aim

- To categorize the weakness or paralysis of a limb as either UMN or LMN in origin
- To localize the level of a spinal cord lesion
- To monitor progression of disease.

Significance

A reflex is composed of an afferent (sensory) arc and an efferent (motor) arc. An absent or decreased reflex indicates a lesion is present within the reflex arc. This is characteristic of LMN disease. UMN pathways supply inhibitory and excitatory influences to the LMN reflex arcs. In the normal animal, there is a net effect of descending inhibition of the spinal reflex arcs. When this inhibition is removed by a lesion of the UMN pathways, the reflexes increase. Knowledge of the reflex arcs enables the clinician to localize a lesion with greater accuracy (Fig. 2.21).

Examination

The animal is held down in lateral recumbency and the uppermost limbs are tested. Left and right sides are compared. Small dogs and cats are easier to examine if held in dorsal recumbency by the owner on the owner's lap. If increased voluntary muscle contraction from fear or excitement prevents the limb moving when reflexes are tested, try testing the lowermost limbs as they are often more relaxed than the uppermost limbs. If the lowermost limbs have a reduced reflex, be aware that this may have resulted from recumbency limiting limb movement.

If the dog cannot be placed in lateral recumbency without an unseemly tussle, test each limb while the animal is standing. The body weight must be supported so that the limb is non-weight-bearing during testing.

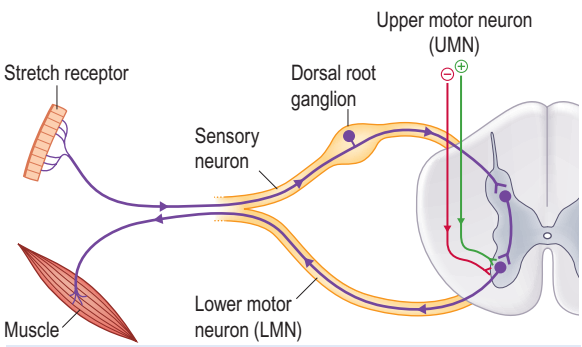


Figure 2.21 The stretch reflex.

Hindlimb

Patellar reflex

Afferent arc: femoral nerve (L4–6). Efferent arc: femoral nerve (L4–6).

Palpate the stifle. Check its range of movement. Ensure the stifle is at least partially flexed. Locate the patellar ligament. Strike the tendon directly with the hammer, or place your finger on the ligament and strike your finger. The patellar ligament is large and immobile enough to strike directly in most animals. Observe extension of the stifle (Fig. 2.22). It is present at birth but difficult to elicit under 4 weeks of age. It is one of the two most reliably evoked limb reflexes.

CLINICAL TIP

Locating the patella and its ligament prevents the embarrassment of repeatedly hitting the tibial tuberosity to no effect. This is a surprisingly common error.

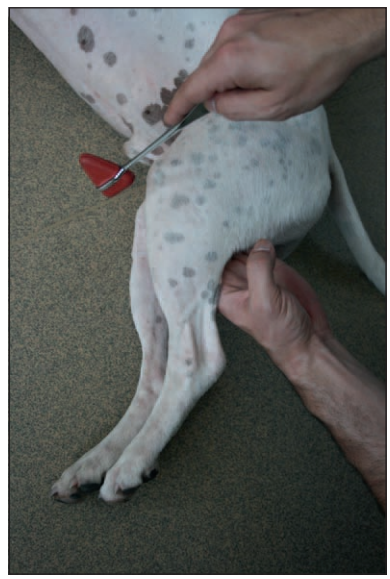


Figure 2.22 Strike the patella ligament and observe extension of the stifle.

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Cranial tibial reflex

Afferent arc: peroneal nerve (L6–S1). Efferent arc: peroneal nerve (L6–S1).

Support the limb under the stifle or medial tibia, allowing the tarsus to dangle. Strike the fleshy proximal portion of the cranial tibial muscle. Observe flexion of the tarsus. Peroneal nerve function is assessed during the withdrawal reflex by observing tarsal flexion.

CLINICAL TIP

A common error is to gently pat the limb with the reflex hammer. Apply as much force as used when clapping one's hands with moderate enthusiasm.

Gastrocnemius reflex

Afferent arc: tibial nerve (L7–S1). Efferent arc: tibial nerve (L7–S1).

Hold the metatarsus and with traction, extend the stifle and partially flex the tarsus. This tenses the gastrocnemius tendon. Strike the tendon. Observe contraction of the caudal thigh muscles. Striking the tendon can physically move the limb; do not confuse this for a reflex. This reflex can be difficult to elicit and is unreliably present. Tibial nerve function can also be assessed by observing normal extension of the tarsus.

CLINICAL TIP

Use a patellar hammer (reflex hammer, pleximeter) to elicit tendon reflexes. It is easier to deliver a uniform force (and it does not hurt as much as a pair of scissors when you need to strike your own finger).

Withdrawal (flexor) reflex

Afferent arc: depends on which dermatome is stimulated.

- Lateral digit: sciatic nerve (L6–S1)
- Medial digit: femoral nerve (L4–6).
- Efferent arc: sciatic nerve (L6–S1).

Pinch the lateral and medial digits in turn and observe flexion of the tarsus, the stifle and the hip. If this fails to occur, use hemostats to squeeze the nail bed, gradually increasing the pressure. Avoid damaging soft tissue. Do not jab at the paw with a needle; this is an unreliable stimulus. The withdrawal reflex is present at birth, with simultaneous extension of the contralateral limb (crossed extensor reflex) occurring up to 3 weeks of age (Fig. 2.23).



Figure 2.23 The withdrawal reflex. Note flexion of all joints.

CLINICAL TIP

If the hindlimb withdrawal reflex appears reduced in a paraplegic animal, compare it with the forelimb withdrawal reflex to judge what is normal for the animal.

Forelimb

Biceps reflex

Afferent arc: musculocutaneous nerve (C6–8). Efferent arc: musculocutaneous nerve (C6–8).

Palpate the biceps tendon insertion. Keeping the tendon under your finger, strike your finger with the hammer. Flexion of the elbow may or may not occur in the normal animal. Contraction of the biceps muscle can be seen in animals with smooth coats (Fig. 2.24). The musculocutaneous nerve function is more reliably assessed by the withdrawal reflex.

Triceps reflex

Afferent arc: radial nerve (C7–T2). Efferent arc: radial nerve (C7–T2).

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Figure 2.24 The biceps reflex.



Figure 2.25 The triceps reflex.

Palpate the triceps tendon in the partially flexed forelimb (Fig. 2.25). Steady it under your finger then strike your finger. Extension of the elbow may occur but it is often absent in normal animals. Radial nerve function is also assessed by observing normal elbow extension and weight-bearing.

CLINICAL TIP

If the reflex induces limb extension, ensure the limb is initially flexed otherwise the reflex action will not be seen.

Extensor carpi radialis reflex

Afferent arc: radial nerve (C7–T2). Efferent arc: radial nerve (C7–T2).

Support the forelimb medially at the elbow and strike the proximal part of the extensor carpi radialis muscle. Extension of the carpus is seen. (Physically pressing this muscle will also induce carpal extension.)

Withdrawal (flexor) reflex

Afferent arc: varies with the area stimulated. Efferent arc: musculocutaneous, axillary, radial, median, ulnar nerves.

Pinch the digit and observe flexion of the digits (C8–T2: median and ulnar nn), carpus (C8–T2: median and ulnar nn), elbow (C6–8; musculocutaneous n), and shoulder (C7–T1:axillary and radial nn). Present at birth, with simultaneous extension of the contralateral limb (crossed extensor reflex) occurring until 3 weeks of age.

The reflex withdrawal movement ONLY demonstrates the integrity of the reflex arc and does NOT indicate that the animal has perceived the stimulus that caused the reflex. Pain perception requires the noxious stimulus to be transmitted by ascending pathways to the brain. The integrity of the ascending sensory pathways is a prognostic sign in spinal cord disease.

MEMORIZE THIS

The presence of a withdrawal REFLEX does not signify intact pain PERCEPTION.

Other spinal reflexes

Perineal (anal) reflex

Afferent arc: pudendal nerve (S1–S3). Efferent arc: pudendal nerve (S1–S3).

Touching the left and right sides of the anal sphincter with a thermometer or closed forceps induces contraction of the anus (and flexion of the tail by the caudal nerves). The strength of anal sphincter contraction is judged by digital rectal exam. Anal tone in the animal preparing to defecate appears weak. A gaping anal sphincter showing mucosa is abnormal. Being able to rectal a conscious cat also indicates a weak anal reflex.

Anogenital reflex: Tactile stimulation of the anus and genitalia or caudal abdomen initiates urination and defecation until 3–4 weeks of age.

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Bulbocavernosus reflex: Squeezing the bulb of the penis or vulva will cause anal sphincter contraction and tail flexion. It tests the same reflex arc as the perineal reflex.

Cutaneous trunci (panniculus) reflex

Afferent arc: spinal nerves of the consecutive dermatomes of the lateral thoracic wall; ascending spinal cord pathways. Efferent arc: C8–T1 segments; lateral thoracic nerve; cutaneous trunci muscle.

Commence at the cranial edge of the ilial wings. Pinch the skin either side of the dorsal spine. Note that pinching one side induces bilateral contraction of the cutaneous trunci muscle (Fig. 2.26).

Afferent lesions occur within the spinal cord: pinching caudal to the spinal cord lesion does not produce the reflex contraction on either side. A '*cut-off*' point is established, with the reflex inducible cranial to that point and absent caudal to that point. The site of the spinal cord lesion lies a few cord segments cranial to the 'cut-off' point on the skin. This mismatch is due to the unequal growth of the spinal cord and the vertebrae.

Efferent lesions occur within the spinal cord at the C8–T1 cord segments, or within the nerve roots exiting these segments. The lesion may be unilateral in which case stimulation on either side of the spine will only produce a reflex contraction on the side opposite the lesion.

CLINICAL TIP

Horner's syndrome and an ipsilateral absence of the cutaneous trunci reflex often occur together secondary to avulsion of the caudal brachial plexus.

The reflex contraction of the cutaneous trunci muscle may be reduced in generalized LMN diseases. It may be difficult to see in obese dogs. It is present at birth.

CLINICAL TIP

The reflex is naturally absent cranial to the C8–T1 dermatomes (level of shoulder) and absent caudal to the wings of the ilium. Do not confuse this with a 'cut-off'.



Figure 2.26 A stimulus on one side will produce muscle contraction bilaterally (arrows, bilateral muscle contraction).

Scratch reflex

The stimulation of repetitive scratching of the lateral thorax or neck induces weak scratching movements of the ipsilateral hindlimb from 2–4 days old until 3–4 weeks of age in the normal neonate.

Extensor thrust reflex

Using gentle pressure, spread the digits. Extension of the limb should occur. This reflex can be difficult to assess. This is a different phenomenon to the extensor postural thrust response.

Assessment

Grading reflexes

Observe the force, speed and extent of flexion or extension after a single stimulus (Table 2.2).

Only the patellar reflex and the withdrawal reflexes are reliably elicited. Their absence signifies a lesion within the reflex arc (LMN disease) in either the sensory or motor arc. Sensory function is assessed further by checking pain perception and proprioception (Table 2.3).

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Table 2.2 Grading reflexes

Grade		Action
0	Absent, areflexic	No reflex movement
1+	Reduced, hyporeflexic	Slow, decreased, reduced movement
2+	Normal, normoreflexic	Brisk reflex action followed by relaxation
3+	Increased, hyperreflexic	Exaggerated, larger, brisker, reflex action
4+	Clonus, clonic	Increased, repetitive, sustained reflex action

Table 2.3 Variation in reflex function

Exceptions to the rule	Resolution
Neurologically normal dogs ≥10 years old may lack one or both patellar reflexes	Hypotonia, weakness, short-strided gait are also present in LMN disease
Decreased forelimb withdrawal reflex occurred in 31% of dogs with a C1-5 lesion	Imaging of both cervical areas, C1-5 and C6-T2

CLINICAL TIP

If the reflex is absent or reduced, first question your examination technique. Recheck. Alter your technique. Re-assess.

Spinal shock describes the flaccid paralysis and areflexia occurring in mammals following a complete spinal cord transaction. It may have resolved by the time the cat or dog is examined. An *inverted reflex* is one in which the movement elicited is opposite to that normally seen, e.g. flexion produced while testing the triceps reflex. It has not been assigned any diagnostic significance in the dog or cat.

Normal or increased reflexes indicate that the reflex arc is intact. Do not diagnose neurological disease on the basis of an increased spinal reflex in the absence of any other abnormality. Lesions severe enough to remove descending inhibition also affect the descending motor tracts and ascending proprioceptive pathways. UMN lesions are accompanied by normal, increased or clonic spinal reflexes. *Pseudo-hyperreflexia* occurs with a loss of antagonistic muscle tone, e.g. hyperreflexic patellar reflexes with a sciatic nerve lesion.

Evidence of UMN release

Crossed extensor reflex

Afferent arc: same as flexor reflex. Efferent arc: flexors of ipsilateral limb; extensors of contralateral limb.

Gentle pinching of a digit induces flexion of the stimulated limb and rapid reflex extension of the contralateral limb in animals. It is a sign of removal of inhibition of the reflex arc and is an abnormal finding if elicited in animals over 3 weeks old in lateral recumbency. Its presence or absence is not a prognostic sign. The reflex may be absent in normal kittens less than 3 weeks old.

A more noxious stimulation induces pain and causes the animal to struggle and voluntarily extend the contralateral limb.

Mass reflex is the exaggerated reaction which can occur when testing the withdrawal reflex or the perineal reflex in animals lacking descending UMN inhibition. Bilateral flexion of the hindlimbs, anal contraction and tail flexion, and possibly urine expulsion may occur.

Babinski's sign(s)

In normal adult humans scratching the lateral side of the sole from the heel to the toes then medially elicits flexion of the big toe. Dorsiflexion of the big toe with or without fanning of the other toes is called a Babinski's sign, and is abnormal in adults, indicating the presence of a corticospinal tract (UMN) lesion. Babinski's sign is normal in infants under three years of age and is widely depicted in various Medieval, Renaissance and Baroque paintings of Madonna and child.

Varying the stimulus by scratching, squeezing, stroking, percussing, and flexing the lower limb, gave scope for ego-aggrandisement and renaming of the sign, e.g. Chaddock, Scherzi, Oppenheim, Gordon, Schaefer, Strumpell. The abnormal clinical finding and its significance remains the same.

This reflex is not reliably present in small animals and is not required for localizing lesions. It is included here as almost every veterinarian has heard of it and tries to elicit it. Tonic extension of the big toe occurring in dystonic syndromes of humans is a sign of extrapyramidal disorders and is known as 'striatal toe'. Extrapyramidal refers to the location of descending tracts within the brainstem, and such pathways contribute the majority of motor tracts in small animals. Striatal toe is a tonic extension and Babinski's sign is an elicited action.

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Babinski's sign also refers to the paradoxical raising of the eyebrow during eye closure in cases of hemifacial spasm in humans.

PAIN PERCEPTION

Aim

To assess the integrity of pain pathways in the PNS and CNS.

Significance

Pain perception is a subjective assessment by the patient of a noxious stimulus. The patient's behavioural response to the stimulus is assessed by the clinician. Loss of pain perception in spinal cord disease indicates severe damage and a poor prognosis for recovery. An unconscious animal unresponsive to pain is said to be comatose.

Examination

Pinch the skin or squeeze the nail bed using a haemostat. Commence with gentle pressure. More force should be applied only if required to elicit a reaction. Pin pricks are unreliable stimuli and should not be used. Have a reliable assistant hold the head, or consider muzzling the animal. Some restraint of the patient is required but must not interfere with the interpretation of the behavioural response. Some animals turn to look at the source of pain, others vocalize or struggle to be released (Fig. 2.27).

This test is usually performed at the end of the neurological examination as inflicting pain almost guarantees to lose the animal's cooperation.

Interpretation

The majority of clinical cases require the clinician to decide if pain perception is present or not. Do not obsess about the categories of superficial or deep pain. The bottom line is: Does the animal feel pain … or not? Note if a greater force or a lesser force than normal is required. A 'normal' force required to elicit pain is hard to quantify. Recall the digital pressure used to elicit a pulse: applying the same degree of pressure, or slightly more, to the nail bed with hemostats induces most calm animals to respond.

URINARY FUNCTION

Aim

- To assess the ability to void urine voluntarily
- To judge the degree of effort required to empty a bladder
- To estimate the degree of urine retention.



Figure 2.27 Pinching the toe to elicit pain. The animal struggles to escape.

Significance

Loss of bladder contraction results in urine retention. This may be secondary to a loss of afferent information from the bladder, signalling its fullness. Damage to the detrusor muscle from over-distension or infiltrative disease will also weaken bladder contractions. UMN spinal cord lesions result in paralysis of the bladder in which muscle tone may be palpated (unless secondary over-distension has occurred). LMN lesions of the spinal cord segments or nerves result in a flaccid paralysis of the bladder. The motor supply to the bladder's detrusor muscle is the parasympathetic pelvic nerve originating from S1–3 cord segments.

The urethral sphincter tone is increased with UMN lesions. Motor supply to the internal urethral sphincter composed of smooth muscle originates from the (sympathetic) hypogastric nerve and the L1–4 (dog) or L2–5 (cat) cord segments. The external sphincter, composed of skeletal muscle, receives motor innervation via the pudendal nerve originating from sacral cord segments S1–3. Loss of urethral sphincter contraction causes urinary leakage.

Examination

Palpate the bladder size and tone prior to and following voluntary urination. Observe the animal's ability to posture and initiate voiding, and the force and calibre

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of the urine stream. Do not assume that urine found in a patient's cage was voluntarily voided or that it signifies the bladder has been successfully emptied. Reflex action can cause an incomplete bladder contraction. Always palpate the bladder and express the residual urine.

Interpretation

A large flaccid bladder indicates a LMN lesion. Such a bladder may be easy to express from lack of both sympathetic and somatic innervation of the urinary sphincters. Depending on the level of the lesion, sympathetic innervation may persist, accounting for resistance to bladder expression. A distended tense bladder from a UMN lesion is typically difficult or impossible to manually express. It is also very difficult to express the bladder of normal male cats and dogs.

CRANIAL NERVES

Aim

To assess the motor and sensory functions of the cranial nerves (CNN) by observation, and by eliciting reflexes and responses.

Significance

The 12 cranial nerves enter or leave the brainstem passing through skull foraminae often in concert with each other (Table 2.4). CNN are labelled numerically from rostral to caudal. Skeletal and smooth muscle are innervated by cranial nerves: LMN lesions may therefore occur in generalized neuromuscular disease or dysautonomia. Cerebral lesions have a subtle effect on the contralateral CNN sensory or motor function and usually go unnoticed by the general practitioner.

Table 2.4 Mnemonic for cranial nerves

I	Olfactory	On
II	Optic	Old
III	Oculomotor	Olympus's
IV	Trochlear	Towering
V	Trigeminal	Top
VI	Abducent	A
VII	Facial	Fearless
VIII	Vestibulocochlear	Veterinarian
IX	Glossopharyngeal	Gained
X	Vagus	Virtue
XI	Accessory	And
XII	Hypoglossal	Honour

CN I: OLFACTORY NERVE

Aim

Rarely if ever tested.

Significance

The olfactory neurons run from the receptors in the nasal mucosa through the cribriform plate of the ethmoid bone to the olfactory bulbs. Olfactory receptors and nerves are purely sensory and mediate the sense of smell. Conscious perception of scent occurs within the pyriform lobes.

Examination

Strong scents such as ammonia may stimulate the trigeminal receptors (CN V) of the nasal mucosa which detect painful stimuli, rather than the olfactory receptors (CN I).

Interpretation

The animal is observed sniffing a food substance and is presumed to perceive the odour. Lack of a sense of smell is called anosmia.

CN II: OPTIC NERVE

Aim

To assess vision and pupil control.

Significance

The optic nerve is purely sensory, and transmits information to the occipital cortex for vision, and to the midbrain as the afferent arc of the pupillary light reflex (PLR). The afferent pathways for both phenomena are shared to a certain point. This fact aids localizing lesions: absence of both vision and the PLR indicate a lesion in the common pathways.

The ganglion cells of the retina form the optic nerve, the end of which is seen on retinal examination as the optic disc (Fig. 2.28). The optic nerve is myelinated by oligodendrocytes and surrounded by a subarachnoid space and meninges. It enters the skull via the optic canal, joining the brain rostral to the pituitary gland.

The majority of fibres (cat: 65%, dog 75%) in the optic nerves cross (decussate) to the opposite side to form the contralateral optic tract. The site of decussation is called the optic chiasm. The optic tract projects to the ipsilateral occipital cortex by way of the ipsilateral lateral geniculate nucleus and optic radiation. Thus, a complete optic nerve lesion will produce ipsilateral menace deficit

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Figure 2.28 Contrast agent outlining the optic nerve (arrow). A side-effect of myelography from cisterna magna injection.



Figure 2.30 The menace response. The contralateral eye is covered, and the menacing hand is not too close to the face.

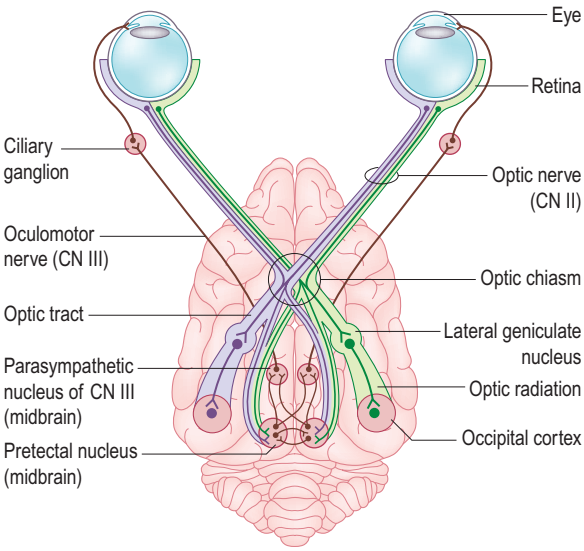


Figure 2.29 Vision and pupil constriction initially share a common pathway.

and a lesion in the optic tract or higher will produce a contralateral menace deficit (Fig. 2.29).

Examination

Obstacle course: Blindness is often first noted in a new environment so it may not be necessary to create an elaborate obstacle course in the examination room. Encourage the animal to walk around the room. Note any collisions with the furniture and on which side of the head they occurred. Do not misinterpret a loss of balance and falling into or against objects as blindness.

Visual following: A mentally alert visual animal may respond to the first cotton ball falling in front of its face and subsequently ignore similar efforts to gain its visual attention hence this response may be absent in normal animals.

Menace response: The examiner uses one hand to cover an eye, while the other hand is brought sharply towards the uncovered eye of the patient. Do not touch the vibrissae or stimulate the face with gusts of air, both of which stimulate the trigeminal nerve (CN V). The normal response to a menacing gesture is to blink (Fig. 2.30). It is not a reflex and requires the animal to pay attention. This may be gained by gently tapping the animal on the side of the head, and then repeating the menace response test. Systemically unwell animals may be too lethargic to respond appropriately.

It is a learnt response and is present in kittens and puppies by 10–12 weeks of age.

Interpretation

The menace response is defined as a blink immediately after a menacing gesture. An absent response is referred to as a *menace deficit* and may occur from a lesion in either the afferent or efferent pathways. It is not a reflex as the pathway requires cerebral integration and is affected by cerebral function (e.g. alertness). Facial

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Figure 2.31 Indirect ophthalmoscopy. This has the additional benefit of keeping the clinician's face away from the animal's jaws.

paralysis (CN VII) will prevent the eyelid closing but the eyeball may retract (CN VI). The menace response is still regarded as absent (0). Make a note in the records that the animal appears to be visual.

A unilateral cerebellar lesion may cause an ipsilateral menace deficit in the presence of normal vision and facial nerve function. A diffuse cerebellar lesion can produce a bilateral menace deficit.

The response is graded as absent (0), decreased (1+), or normal (2+).

Visual placing response: the normal animal reaches for the table.

Fundic examination: Indirect ophthalmoscopy (Fig. 2.31) allows the retina to be viewed as a whole, and has the benefit of not requiring placement of one's face adjacent to the patient's mouth. An assistant restrains the animal's head while the clinician shines light at the animal's eye and finds the bright yellow-green tapetal reflection. Resting one hand on the animal's head, an arm's length away, the clinician lowers the lens in front of the eye, illuminating the fundus. This is brought into focus by slightly moving the lens closer or further from the eye. A common mistake is to forget the focal distance of the hand-held lens and come too close to the eye.

Note any haemorrhage, detached retina, hyperreflective areas or hyporeflective areas. Vision will be affected before the PLR in retinal and optic nerve lesions.

The dazzle reflex: Strong light shone in the eye induces a reflex blink mediated by afferent input from the optic nerve to subcortical structures with the motor innervation supplied by the facial nerve (CN VII).

CN III: OCULOMOTOR NERVE

Aim

To assess pupil constriction and movement of the globe.

Significance

Afferent fibres leave the optic tract and pass to the ipsilateral pretectal nucleus in the midbrain. Most fibres then decussate to the contralateral parasympathetic nucleus of CN III also in the rostral midbrain. Preganglionic fibres exit the midbrain ventrally and pass to the ciliary ganglion in association with the CN III efferent neurons to the extraocular muscles. Postganglionic fibres, called short ciliary nerves, innervate the iris constrictors. The feline iris is supplied by lateral and medial short ciliary nerves. Loss of one of these nerves creates a D-shaped pupil; 5–8 short ciliary nerves innervate the canine iridial constrictors.

The somatic efferent branch of CN III innervates the dorsal rectus, ventral rectus, medial rectus, and ventral oblique extraocular muscles. These muscles have different roles in different positions of gaze. It also supplies the levator palpebrae muscles. The nerve cell bodies lie within the CN III nucleus in the rostral midbrain, in association with the parasympathetic nucleus of CN III. Both branches of the oculomotor nerve exit the brainstem ventrally and exit the skull through the orbital fissure to innervate intrinsic and extrinsic muscles of the eye.

Examination

Pupil size: Pupil size can be assessed simultaneously by standing approximately an arm's length away and viewing the face through an ophthalmoscope. The two tapetal reflections are compared. Examine the pupil size and reaction in ambient light and in darkness. Normal pupils should dilate equally in darkness (dark adaption). Always check for anatomic or mechanical problems of the iris, e.g. synechia, iris atrophy, subluxated lens and glaucoma as these affect pupil size, shape and symmetry (Figs 2.32, 2.33).

Interpretation

Anisocoria is always pathological. Sympathetic paralysis prevents the affected pupil from dilating fully in darkness but PLRs are normal.

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Arousal, fear or excitement increases the sympathetic output, dilating the pupils. Approaching sleep results in decreased sympathetic activity and removal of inhibition of the CN III parasympathetic nucleus, resulting in small pupils.

Pupillary light reflex (PLR): Using an ophthalmoscope, shine a strong light into one eye and observe that pupil constrict (Fig. 2.34). This is called a *direct PLR*.

The afferent arc is mediated by CN II and the efferent by the parasympathetic branch of CN III. The majority of afferent fibres decussating at the optic chiasm and then again after the pretectal nucleus explain the relative strength of the direct PLR. Shine the light in one eye and observe the pupil constriction in the contralateral pupil: this is called the *indirect PLR* and is normally less vigorous. *Pupillary escape* is the normal slight redilation of a normal pupil when under constant stimulation by light and is due to adaption of the retina.

CLINICAL TIP

The batteries in disposable flashlights (torches) weaken quite quickly creating an inadequate stimulus and inconsistent test results.

Swinging flashlight test: Shine a strong light into one eye for 2 seconds, then immediately transfer the light source to the other eye, and then back again (Fig. 2.35). The normal response is for both pupils to remain constricted regardless of which eye is being stimulated. When an afferent lesion is present, moving the light from the normal to the abnormal eye causes both pupils to dilate. The dilation of a previous constricted pupil, despite direct stimulation with light, is called the *Marcus Gunn sign*, or *Marcus Gunn pupil*. The degree of dilation is greater than that seen in the normal phenomenon of pupillary escape.

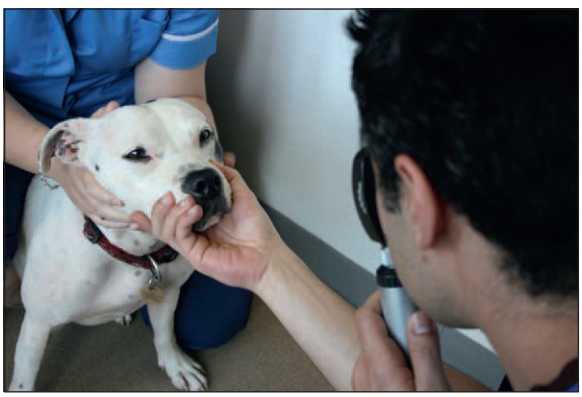


Figure 2.32 Viewing both pupils through a direct ophthalmoscope to assess symmetry.



Figure 2.34 Assessing pupil constriction.

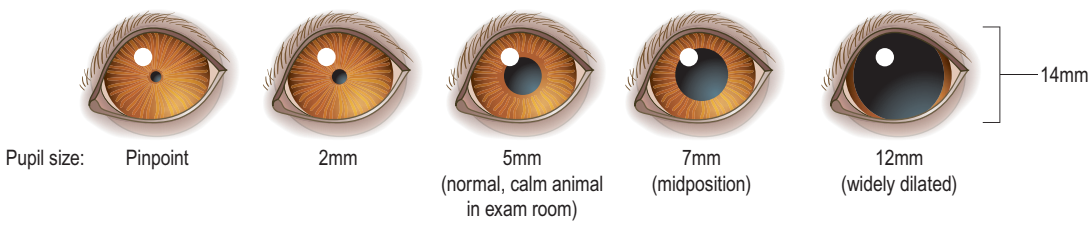


Figure 2.33 Description of pupil size.

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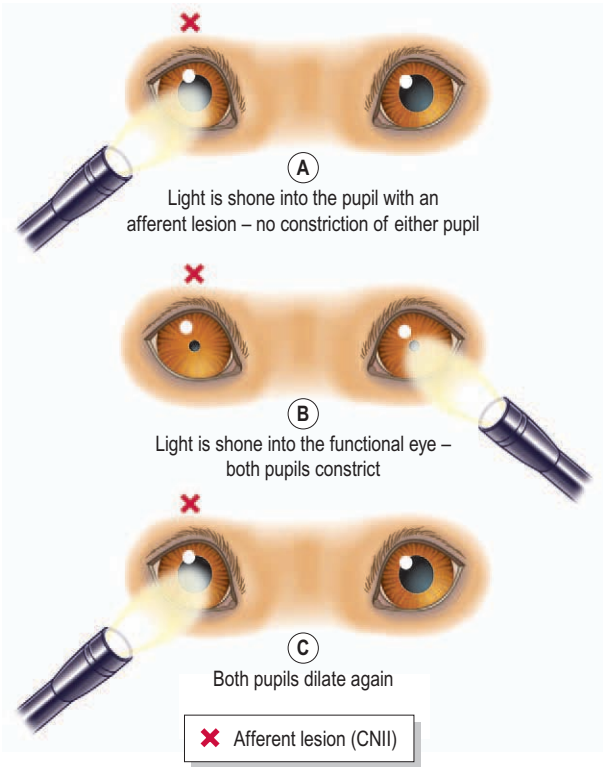


Figure 2.35 The swinging flashlight test (abnormal).

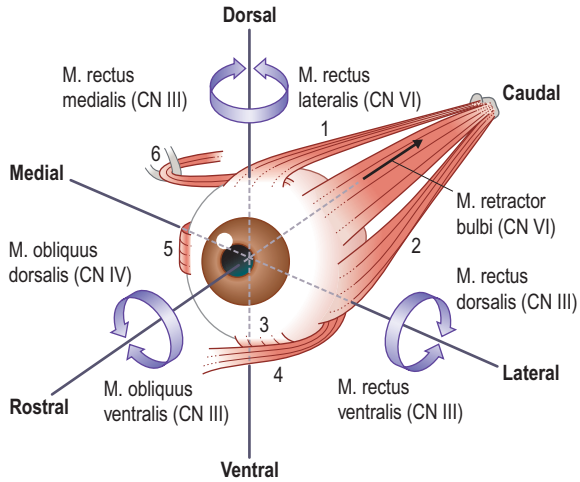


Figure 2.36 Extraocular muscle movement.



Figure 2.37 Support the head under the chin and observe the size and symmetry of facial structures.

Interpretation of PLR: Always interpret the results of the PLR with those of the menace response. The afferent pathways are initially shared. Draw the diagrams of both pathways and map the site of the lesion. Midbrain pathology will also affect the mental state and the gait.

Eye movement: Move the head from side to side and observe the eye movement. The detectors of head rotation are the semicircular canals of the vestibular apparatus. Sensory information enters the medulla via CN VIII, projecting rostrally via the medial longitudinal fasciculus (MLF) through the brainstem to the nuclei of CNN III, IV and VI. The efferent arc of this reflexive eye movement comprises CNN III, IV and VI, depending on the position of the head. The lateral rectus (CN VI) abducts the eyeball and the medial rectus (CN III) adducts. Normal animals have conjugate eye movements (Fig. 2.36).

Eye position: Face the animal and observe the position of the eye within the socket (Fig. 2.37).

The pupil sits in the middle of the palpebral fissure of normal animals. Deviation of the visual axis is called *strabismus.* When present in the normal head position, it is called *resting (spontaneous)* strabismus. Dorsiflex the neck and observe the eye position again. The eye position is best examined if the clinician is standing above the animal. Avoid retracting the skin of the forehead as this will elevate the eyelids, exposing more sclera, thus creating the illusion of ventral strabismus (Fig. 2.38).

Normal animals should gaze in the direction in which they are facing. CN III paralysis gives rise to a ventrolateral strabismus at rest. Convergent strabismus in which the visual axis is deviated medially is called esotropia. Outward turning of the eye, divergent strabismus, is called exotropia.

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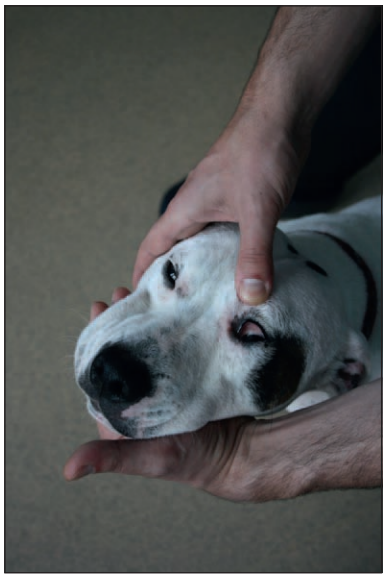


Figure 2.38 Bad technique. Note how the skin is retracted when restraining the head.

Positional (inducible) strabismus is that present with an alteration in head position (see page 236).

Upper eyelid position: Face the animal and compare the symmetry of the left and right palpebral fissures. Paresis or paralysis of the levator palpebrae muscle produces *ptosis*, decreasing the size of the palpebral fissure.

CN IV: TROCHLEAR NERVE

Aim

CN IV only has motor function. It plays a role in eyeball movement and position. Its limited function is difficult to assess.

Significance

The nucleus of CN IV lies within the caudal midbrain. The axons pass dorsally, decussate, and exit the brainstem dorsally. CN IV then passes ventrally and exits the floor of the skull via the orbital fissure to innervate the dorsal oblique muscle, an extraocular muscle. Contraction of the dorsal oblique rotates the dorsal aspect of the globe medially (intorts). Failure of this function is not noticed viewing the round canine pupil, but the slit-like vertically-oriented feline pupil may show strabismus with the dorsal aspect of the pupil rotated laterally.

Examination

Fundic examination may show rotation of retinal blood vessels.

CN V: TRIGEMINAL NERVE

Aim

To assess jaw strength, and the presence of sensation to the face.

Significance

CN V is composed of sensory and motor fibres. As the name suggests, there are three branches; ophthalmic (sensory to the eye, medial canthus and nostril), maxillary (sensory to the lateral canthus and nostril), and mandibular (motor to the muscles of mastication and sensory to the mandible). The motor nucleus of CN V lies within the pons. Its axons join with the sensory fibres of CN V, exit the pons, pass through the petrosal bone and exit the skull via the oval foramen. The sensory axons within all three branches of CN V unite and enter the skull via the oval foramen. Sensory impulses are transmitted to other cranial nerve nuclei to complete reflex arcs, and are also transmitted to the contralateral cerebral cortex. Unilateral cerebral lesions can reduce pain perception in the contralateral face; these deficits are subtle and may be overlooked. Unilateral lesions of the trigeminal nerve give rise to ipsilateral motor and sensory deficits. There are clinical situations in which either the motor or the sensory deficits occur alone.

Examination

Symmetry: Assess the symmetry of the temporalis and masseter muscles by observation and palpation of the head (Fig. 2.39).

The zygomatic arch and sagittal crest become prominent and *enophthalmos* occurs.

The position of the globe within the socket is easily assessed by viewing the animal's head from above: the curve of the cornea being more or less prominent. Swelling of the masticatory muscles pushes the eyes forward, causing *exophthalmos,* which is noticed as a bulging, prominent eye with sclera exposed on all sides of the iris (Figs 2.40, 2.41).

Jaw tone: Observe the resting position of the mandibles. If the mouth hangs open, push the lower jaw upwards and note if it passively drops once released. Open the mouth, testing the resistance to movement. If

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Figure 2.39 Masticatory muscle atrophy increasing the prominence of the zygomatic arch.



Figure 2.41 Exophthalmos secondary to masticatory muscle myositis.

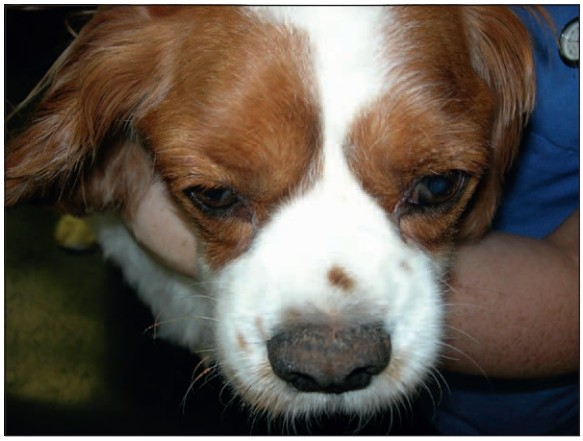


Figure 2.40 The right eye is enophthalmic, from right-sided Horner's syndrome.

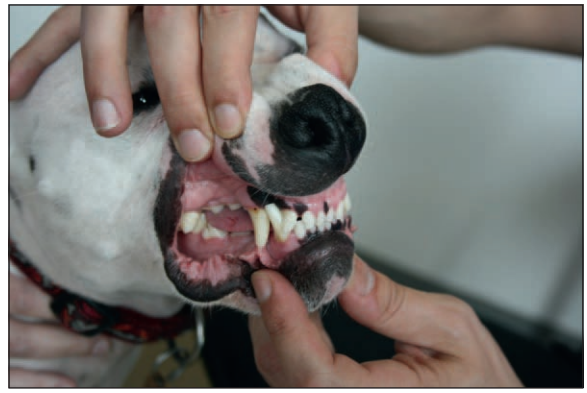


Figure 2.42 The jaw can be completely closed by the dog.

the animal is only aggressive towards strangers, ask the owner to open the mouth (Fig. 2.42).

To be time-efficient, this test can be followed by examination of the tongue, and the gag reflex.

Blink reflex: Touch the lateral canthus of the eye (CN V: sensation) and observe the reflex blink (CN VII: motor). The eyelids should fully close. Touch the medial canthus and observe the blink reflex. The blink is in reaction to touch rather than a menacing gesture (Fig. 2.43).

Facial sensation: This is also tested as part of the blink reflex (Fig. 2.44). Gently insert a pen or closed haemostats into each nostril in turn. The animal should pull its



Figure 2.43 The blink reflex: testing both the facial sensation and the ability to blink.

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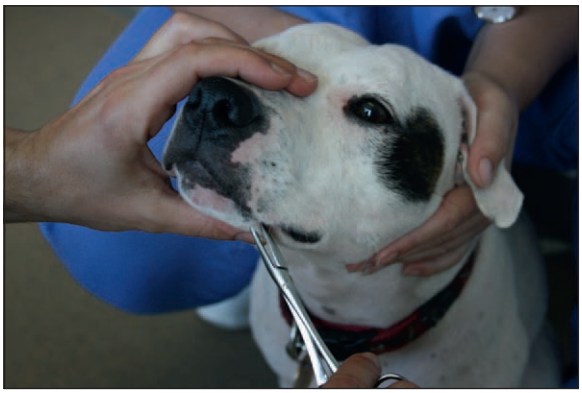


Figure 2.44 Testing facial sensation: the mandibular branch of the trigeminal nerve.

head away from the noxious stimulus. Touch or pinch the top lip (CN V) and watch for lip retraction (CN VII). Pinch the lower lip; the animal should pull its head away from the noxious stimulus.

CLINICAL TIP

Pinching and jabbing at a pet's face is guaranteed to shock the owners. Briefly explain what you are about to do and why.

Schirmer tear test: see CN VII.

Interpretation

An animal's behavioural response to pain is altered by its mental state. Reflexes may be reduced by lesions of the sensory or motor arcs. Lesions of CN V sensory fibres may give rise to abnormal sensations and scratching of one side of the face rather than the anaesthesia expected. Masticatory muscle has a different composition to appendicular muscle and may be specifically targeted by the immune system, therefore involvement of the masticatory muscles alone may not be due to a CN V lesion. Lesions of the CN V nucleus are accompanied by clinical deficits from surrounding structures: cerebellar peduncles, the ipsilateral ascending proprioceptive and descending motor tracts, and the ARAS.

CN VI: ABDUCENT NERVE

Aim

To assess the function of two extraocular muscles.

Significance

The abducent nerve contains motor fibres to the retractor bulbi and the lateral rectus muscles. Its nucleus lies within the rostral medulla. Axons exit the brainstem ventrally and leave the skull via the orbital fissure.

Examination

Globe retraction: Reflex retraction of the eye within the socket occurs in response to touch. The afferent arc is mediated by CN V (ophthalmic branch), and the efferent by CN VI. There are several ways of stimulating the afferent arc:

- Part the eyelids. Wait. The eyeball retracts as the cornea dries
- Part the eyelids. Blow on the cornea
- Part the eyelids. *Gently* touch the cornea.

The preferred method is to touch the eye through a closed eyelid. This technique is well-tolerated by the animal and does not traumatize the cornea. Gentle pressure stimulates globe retraction which, with practise, can be felt through the closed lid.

Eye position and movement: Paralysis of the lateral rectus can result in spontaneous medial strabismus and an inability to fully abduct the eyeball.

Interpretation

Globe retraction is difficult to assess in the presence of enophthalmos. Paralysis of function is due to LMN lesions (nerve or brainstem). Associated deficits localize the site.

CN VII: FACIAL NERVE

Aim

To assess movement of the muscles of facial expression and quantify tear production.

Significance

The facial nerve contains motor fibres to muscles of facial expression and parasympathetic fibres to nasal, salivary and lacrimal glands. Sensory fibres transmit information from the taste buds of the rostral 2/3 of the tongue. Facial nerve axons within the medulla are closely associated with the CN VI nucleus and the caudal cerebellar peduncle. CN VII lies adjacent to CN VIII when exiting the brainstem and passing into the internal acoustic meatus of the petrosal bone (Fig. 2.45).

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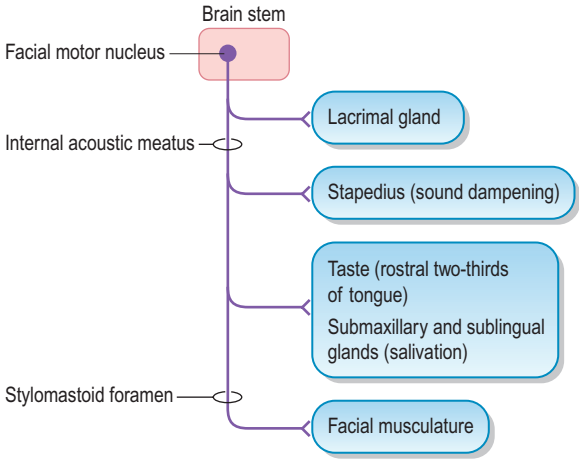


Figure 2.45 Pathway of the facial nerve.

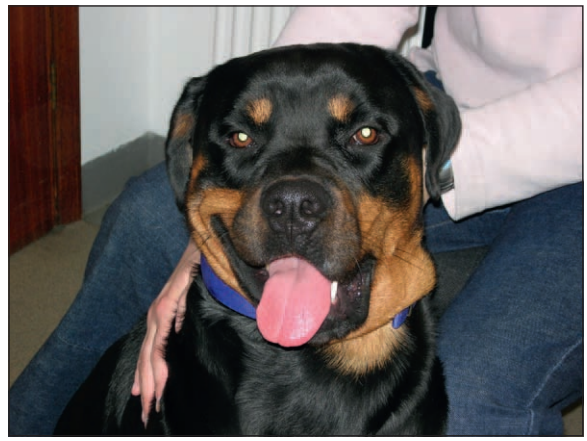


Figure 2.46 Left facial weakness. Note the more ventral position of the left upper and lower lips, the lack of retraction of the left oral commissure, and the slight deviation of the nose to the right.

Examination

Facial symmetry and movement: Observe facial symmetry by noting the position of the upper lips, lower lips, ears, nasal philtrum, and palpebral fissure. Lift the chin between the mandibles for a better view of lip asymmetry. Offer the animal an object to smell and note movement of both nostrils. Make a sudden noise and note symmetry of ear movement. Check the skin of the upper lip for bite wounds which occur when the lip is caught between the canines. Note the appearance of the cornea: it may look wet and shiny despite a reduced tear production (Fig. 2.46).

Blink reflex: The eyelids should close completely when stimulated by touch (CN V) or following a menacing gesture (menace response).

Lip retraction: The upper lip should retract when pinched with haemostats. This reflex requires sensation (CN V) and motor (CN VII) integrity.

Schirmer tear tests

Schirmer tear tests (Figs 2.47, 2.48) measure the aqueous tear film using a calibrated paper strip with an impregnated line of blue pigment. The basal level of tear production by the lacrimal gland (CN VII) is increased in response to stimulation (CN V: sensation; CN VII: motor).

Schirmer Tear Test I (STT I): measures both basal and reflex secretions. The rounded end of the STT strip is folded and placed under the lower lid just medial to the



Figure 2.47 Placement of the Schirmer tear test strip.



Figure 2.48 A normal STT I reading.

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lateral canthus. Tears wick up the strip, smearing the blue pigment as the liquid rises. The level of the blue dye is read at 60 seconds.

- Normal ≥15 mm
- Dry eye ≤10 mm
- Severe dry eye ≤5 mm.

False normal/increase: painful eye; artificial tears.
False decrease: atropine; anaesthetics; normal daily variation; variation between STT strip brand.

Schirmer Tear Test II (STT II): measures basal secretions. Administer a drop of topical anaesthetic to the eye. Dry the conjunctival sac with a sterile Q-tip (cotton bud). Place the STT strip under the lower lid just medial to the lateral canthus. Read the level of the blue dye at 60 seconds (Fig. 2.49).

- Normal ≥7 mm
- Dry eye ≤7 mm

Interpretation

LMN lesions cause weakness or paralysis of the effector organ. Skeletal muscle becomes weak, paralysed and flaccid. Glands cease secretion. Partial closure of the eyelids indicates weakness of the facial muscles. The lower eyelid may droop, widening the palpebral fissure. Flaccidity of the lips causes them to droop. Dependent ears are positioned further ventrally on the head.



Figure 2.49 Dry eye in a dog with a left trigeminal nerve lesion. Note the left temporalis muscle atrophy.

CN VIII: VESTIBULOCOCHLEAR NERVE

Aim

- To look for signs of balance disturbance (vestibular division)
- To distinguish between peripheral and central causes of vestibular derangement
- To subjectively assess hearing ability (cochlear division).

Significance

The vestibulocochlear nerve is purely sensory in function. The balance and hearing receptors develop in close association and are both found within the petrosal bone. Their axons join, leaving the petrosal bone together via the internal acoustic meatus, in close association with the facial nerve. The vestibular nerve enters the rostral medulla and its axons terminate, for the most part, in the vestibular nuclei. Some fibres travel directly to the cerebellum. The cochlear nerve enters the rostral medulla and terminates in the cochlear nuclei.

CN V, VII and VIII are anatomically close at the level of the brainstem.

Examination

Hearing: Making a noise and watching the animal's reaction can lose effectiveness if repeated or if the animal is fearful or excitable. Historical clues are more useful. Objective assessment of hearing is achieved with the brainstem audio-evoked response which tests the integrity of anatomical pathways.

Head position: a head tilt is commonly seen with vestibular dysfunction.

Nystagmus: *Spontaneous* jerk nystagmus is always an abnormal finding when the head is stationary. Record the direction of the fast, jerking, phase as horizontal, vertical, or rotatory, to the left, to the right, up, down, or changeable. Rotatory nystagmus is commonly confused with horizontal and vertical nystagmus. Distinguishing the direction is made easier by viewing a scleral or retinal blood vessel. If it rotates, the nystagmus is rotatory. Nystagmus may only be detected when performing a fundic exam; the significance is the same. Observe the eyes when the neck is flexed laterally, dorsally, and ventrally and when the head is stationary. If nystagmus appears in these new positions it is called *inducible or positional* nystagmus.

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Physiological nystagmus is a normal reflex generated by the vestibular receptors (sensory) and movement of the extraocular muscles innervated by CN III, IV, VI (motor). Turn the head horizontally for several cycles at a frequency of about 2 Hz and amplitude of approximately 45°. The fast phase of eye movement is in the direction of rotation. Try to prevent the owner from distracting the animal as visual fixation stops the fast jerking phase – but the smooth slow phase persists.

Positional (inducible) strabismus: Positional vertical strabismus may occur ipsilateral to the vestibular lesion. Normal animals maintain the iris within the centre of the palpebral fissure.

Interpretation

Subjective assessment of hearing cannot differentiate unilateral deafness from normal hearing.

Vertical nystagmus indicates central vestibular disease whether or not the fast phase is downwards or upwards. Nystagmus that changes direction also indicates a central vestibular lesion. Positional nystagmus can occur with either central or peripheral vestibular lesions.

CN IX: GLOSSOPHARYNGEAL NERVE

Aim

To test sensation (and movement) of the pharynx.

Significance

CN IX is a mixed nerve. It supplies motor innervation to salivary glands, the stylopharyngeus muscle (and possibly other pharyngeal muscles), and contains sensory fibres from the caudal tongue (taste buds), carotid sinus, and pharynx. The motor axons originate in the rostral medulla and exit the skull via the jugular foramen and tympanooccipital fissure. Sensory fibres pass through the same portals and enter the medulla, projecting into the solitary tract. From there, they either ascend rostrally to be consciously appreciated by the cerebrum, or project to other nuclei of the brainstem to create reflex arcs.

Examination

Gag reflex: Insert a finger, pen, or patellar hammer handle, into the caudal pharynx and touch one side, then the other. Observe pharyngeal contractions and elevation of the soft palate (gagging) (Fig. 2.50). CNN IX and X contribute to both the sensory arc and motor arc of this reflex.



Figure 2.50 Assess jaw tone, tongue symmetry, mucous membrane colour, odours, and the gag reflex.

This is usually combined with examination of the jaw tone and the tongue.

Interpretation

CN IX is closely associated with CN X and both are usually affected together in clinical disease. Neuromuscular disease commonly depresses pharyngeal motor function but not sensation: the animal may pull the head away without moving the pharynx. Many animals tolerate bilateral stimulation of the pharynx but normal animals do not display dysphagia.

CN X: VAGUS NERVE

Aim

To test sensation (and movement) of the pharynx, to assess laryngeal function.

Significance

Sensory and motor to pharynx and larynx.

Examination

Listen for inspiratory stridor.

Gag reflex: See earlier in this chapter.

Interpretation

See laryngeal paralysis, dysphagia, pages 152 & 158.

CN XI: ACCESSORY NERVE

Aim

Deficits are not recognized as a clinical problem.

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Significance

The internal branch arises from the nucleus ambiguous within the medulla, along with CN IX and CN X. The external branch arises from the ventral horn cells within C1–6 spinal cord segments and travels rostrally through the foramen magnum. The two branches unite within the skull forming CN XI, which then exits via the jugular foramen and tympano-occipital fissure along with CNN IX and X. The internal branch remains with CN X while the external branch splits off to innervate the trapezius, and parts of the sternocephalicus and brachiocephalicus muscles.

Examination

Palpation of the trapezius muscle.

Interpretation

A purely focal CN XI lesion would not cause forelimb weakness, nor result in dysphagia.

CN XII: HYPOGLOSSAL NERVE

Aim

To assess movement, size and strength of the tongue.

Significance

The hypoglossal nucleus lies within the caudal medulla. CN XII exits the skull via the hypoglossal canal and innervates the intrinsic and extrinsic tongue muscles.

Examination

If it is safe to do so, open the mouth and observe tongue movement and symmetry. Normal animals move the tongue when the mouth is open. Using a finger, attempt to retract the tongue and note the muscle tone. If the animal is aggressive, observe the mouth from a distance; the tongue should move during panting. The tongue can be palpated between the mandibles, which may give some idea of atrophy or hypertrophy.

Interpretation

LMN lesions lead to atrophy, flaccidity and weakness and this also applies to the tongue muscles. Enlargement of the tongue can occur in some myopathies. Uraemia may result in tongue necrosis.

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3 Localizing lesions

William of Occam (died circa 1350) pioneered the KISS ('Keep It Simple, Stupid!') approach to problem-solving with his philosophical principle that the fewest possible assumptions are to be made in explaining a thing. This principle is known as *Occam's razor* and is used in localizing neurological lesions.

The aim is to find the focus of the pathology. Thus all signs are assessed and judged if they can be ascribed to a single lesion or not.

Once this intellectual exercise is over, the clinician then prioritizes any tests required to diagnose the actual disease.

If the anatomical site of disease is not known, then testing cannot be performed in a logical, rational, cost-efficient and expedient manner (Table 3.1).

AETIOLOGY OF THE LESION

The history provides important information as to the nature of the underlying disease. It is wrong to assume that history-taking can be avoided by performing various blood tests and imaging techniques. In fact, that would be one of the most serious errors a clinician could make. Assessing the speed of onset, the rate of progression, either worsening or improving, and the duration of signs gives the first clues as to the diagnosis and hence the possible prognosis.

Pathological processes have typical patterns of behaviour and these can be simplistically demonstrated with graphs. These are guidelines only. Diseases have nuances and variations in clinical appearance. Textbooks only describe the common presentations (Fig. 3.1).

It is important to consider the nature of the underlying disease before planning any diagnostic tests. If all the possible underlying diseases (the differential diagnoses) are uniformly fatal within a short time span, it is only fair to the owner, who is funding the investigation, to inform them of this fact. Equally, if the suspected aetiology is one which will resolve without intervention within a short time frame, it is important to mention this to the owner, and consider using what could be termed 'benign neglect', i.e. wait and see. If the animal does not respond as expected within the timeframe expected, then further investigation is certainly justified.

Categories of disease

1. Malformation
2. Inflammation
3. Degeneration
4. Trauma
5. Tumour.

Some owners wish to know as soon as possible what is or is not going on. The clinician must be confident in assessing the benefit of investigation to the animal. The clinician must be able to explain to the owner the significance of abnormal results (or the lack of abnormalities) on diagnostic tests.

How to prioritize diagnostic tests

1. Utility
   - Will this test alter the treatment of the animal?
   - Will this test give more information about possible underlying pathologies than other diagnostic options?
2. Morbidity
   - What is the risk to the animal in performing the test? (How can this risk be decreased?)
   - What is the risk to the animal in NOT performing the test? (Is there another diagnostic method?)
   - Which is greater?
3. Interference
   - Will this procedure confound the results of future tests?
4. Cost
   - Can the owner afford to act upon the test results? (i.e. Will this test alter the treatment of the animal?).

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Table 3.1 The required thought process for diagnosis

Question	Answer
1. What is abnormal?	Take a history
2. What body system is responsible?	Examine the animal
3. Is it the nervous system?	Perform a neurological examination
4. Where is the lesion?	Interpret the exam findings
5. What could the lesion be?	Consider signalment, history, anatomic location
6. How best to diagnose?	Consider the anatomic location
7. How best to treat?	Consult textbooks and journals

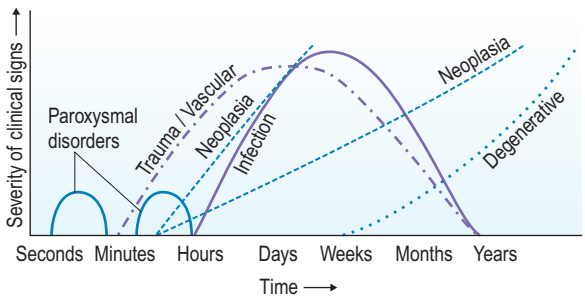


Figure 3.1 Temporal patterns of neurological disease.

Do not forget the basics ... co-existing disease

1. Neurologically affected animals may have disease elsewhere, e.g. arthritis of forelimbs as well as hindlimb ataxia. Physical examination is important.
2. Animals may have new, acute deficits and long-term unrelated signs, e.g. an acute balance loss from peripheral vestibular disease and residual proprioceptive deficits from an unresolved TL cord compression secondary to IVDD. History taking is important.
3. A neurological lesion may begin as a focus and expand to cause damage to surrounding structures, giving the appearance of multifocal disease, e.g. IV disc extrusion leading to myelomalacia. Repeat neurological examinations are important.

The accuracy of neurological diagnoses

Anatomic and aetiological diagnoses based solely on the history and examination are accurate in the majority of cases. In a study of human neurological patients, the errors in clinical diagnosis were attributed to incomplete or incorrect collection of historical and physical findings, insufficient knowledge of the spectrum of clinical manifestations associated with the patient's disease and incorrect analysis of the findings (poor diagnostic reasoning).

An incorrect initial assessment reduces the efficiency of patient care and delays initiation of appropriate treatment. Technology cannot be relied upon as a panacea for diagnostic difficulties as test results may be negative.

Interpretation of neurological signs

Apart from inadequate history-taking, the chief hurdles of the diagnostic steeplechase are an inability to examine the nervous system, and more commonly, an inability to interpret the findings of such an exam.

The trick of examination is to follow a routine, and train oneself to notice both the normal and abnormal.

Interpretation of findings requires a basic understanding of how the nervous system works. Happily, there are characteristics associated with each section of the nervous system (Boxes 3.1–3.9). Make a list of the abnormalities, apply 'Occam's razor', and see if the deficits can be assigned to one area of the nervous system (Fig. 3.2).

Box 3.1 TELENCEPHALON: cerebrum

- Altered alertness
- Altered behaviour
- Seizures
- Contralateral visual deficit
- Minimal gait change
- Compulsive walking, pacing, circling
- Contralateral proprioception deficits
- Contralateral paresis.

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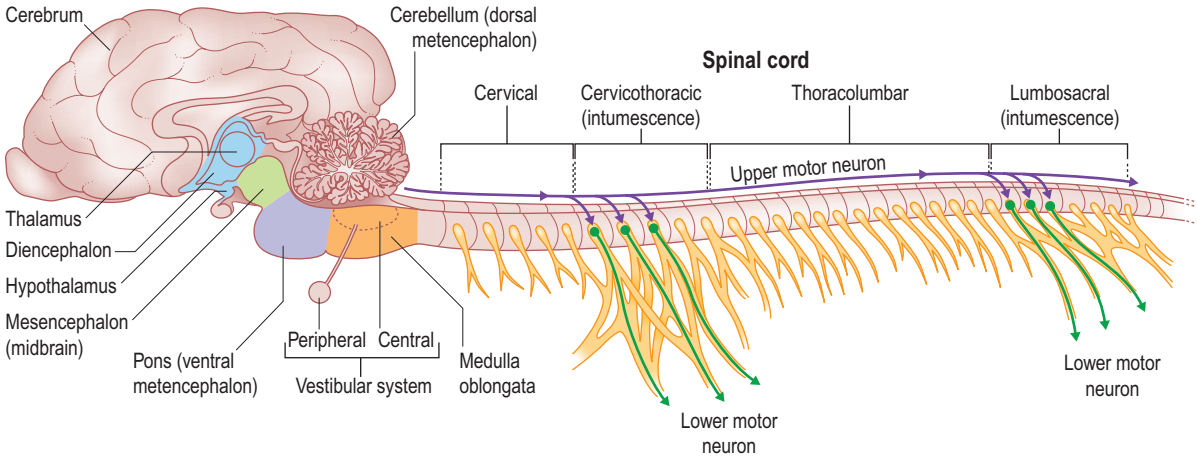


Figure 3.2 Divisions of the nervous system.

Box 3.2 DIENCEPHALON: thalamus, pituitary

- Altered alertness
- Altered behaviour
- Seizures
- Visual and PLR deficits
- Minimal gait change
- Compulsive walking, pacing, circling
- Contralateral proprioception deficits
- Contralateral paresis
- Altered temperature, appetite, thirst
- Endocrine dysfunction.

Box 3.3 MESENCEPHALON: midbrain

- Altered alertness
- PLR deficits
- CNN III , IV
- Major gait change; ataxia/paresis-paralysis
- Contralateral proprioception deficits
- Contralateral paresis.

Box 3.4 VENTRAL METENCEPHALON: pons

- Altered alertness
- CN V
- Major gait change: ataxia/paresis-paralysis
- Ipsilateral proprioception deficits
- Ipsilateral paresis.

Box 3.5 DORSAL METENCEPHALON: cerebellum

- Alert
- Ipsilateral menace deficit (not blind)
- Ipsilateral or contralateral pupil dilation
- ± vestibular signs
- Major gait change: ataxia, tremors
- Preservation of strength.

Box 3.6 MYELENCEPHALON: medulla oblongata

- ±Altered alertness
- CN VI–XII
- Major gait change: ataxia/paresis-paralysis
- Ipsilateral proprioception deficits
- Ipsilateral paresis.

Box 3.7 VESTIBULAR APPARATUS

- ± altered alertness
- Spontaneous nystagmus
- Altered posture: roll, fall, lean, head tilt
- Major gait change: ataxia
- ± ipsilateral proprioceptive deficits
- ± ipsilateral paresis/paralysis.

Box 3.8 SPINAL CORD: UMN

- Major gait change: ataxia/paresis-paralysis
- Ipsilateral weakness
- Ipsilateral proprioceptive deficits
- Normal/increased spinal reflexes
- Normal/increased muscle tone
- Minimal muscle atrophy
- ± absent sensation caudal to the lesion.

Box 3.9 LMN: ventral horn cell, ventral nerve root, nerve, neuromuscular junction, muscle

- Major gait change: paresis-paralysis
- Ipsilateral weakness
- ± Decreased spinal reflexes
- ± Decreased muscle tone
- Severe muscle atrophy.

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Table 3.2 Cranial nerve function

CNN		Components	Function
I	Olfactory	Sensory	Olfaction
II	Optic	Sensory	Vision
III	Oculomotor	Motor	Eyeball movement
			Raises dorsal eyelid
			Constricts pupil
IV	Trochlear	Motor	Eyeball movement
V	Trigeminal	Motor	Muscles of mastication
		Sensory	Face, eye, jaws, oral cavity
VI	Abducens	Motor	Eyeball movement
VII	Facial	Motor	Muscles of facial expression
			Tear and salivary glands
		Sensory	Taste
			Middle ear, blood vessels of the head, palate
VIII	Vestibulocochlear	Sensory	Balance
			Hearing
IX	Glossopharyngeal	Motor	Pharynx
			Salivary glands
		Sensory	Pharynx
			Carotid body and sinus
			Taste
X	Vagus	Motor	Pharynx, larynx, oesophagus, viscera
		Sensory	Pharynx, larynx, viscera
			Taste
			External ear canal
XI	Accessory	Motor	Trapezius, sternocephalicus, brachiocephalicus mm
XII	Hypoglossal	Motor	Tongue

See Table 3.2 for Cranial nerve function and Table 3.3 for Spinal reflexes.

Voluntary movement of the forelimb (Table 3.4)

The action of a muscle can vary with the limb position (Fig. 3.3).

Summary

- Radial nerve C7,8, T1,(2): extension of the forelimb
- Musculocutaneous nerve C6,7,8: flexion of the elbow
- Median and ulnar nerves C8, T1,(2): flexion of carpus and digits.

Voluntary movement of the hindlimb (Table 3.5)

Figure 3.4 shows the peripheral nerves to the hindlimb.

Summary

- Sciatic [L6,7, S1,(2)]: flexion of stifle, tarsus, digits and extension of hip, tarsus, digits
- Femoral [L4,5,6]: flexion of hip, and extension of stifle.

REFERRED PAIN

The phenomenon of tissue injury occurring in one region but appearing to originate from a distant site is called referred pain. The injured and healthy areas share segmental innervation. Referred pain from visceral to somatic structures is well-recognized in humans, probably due to the patient's language skills. It is rarely considered in veterinary medicine.

A common example of referred pain in small animals is the lameness originating from compression or infiltra-

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Table 3.3 Spinal reflexes

Reflex	Sensory nerve	Muscle	Peripheral nerve(s)	Cord segments
Biceps reflex	Musculocutaneous	Biceps brachii Brachialis	Musculocutaneous	C6,7,8
Triceps reflex	Radial	Triceps brachii	Radial	C7,8, T1,2
Extensor carpi radialis	Radial	Extensor carpi radialis	Radial	C7,8, T1,2
Flexor/ withdrawal reflex of the forelimb	Ulnar: lateral digit V Radial: dorsal digit III, IV Musculocutaneous: medial, 2 cm distal to elbow Axillary: lateral, 2 cm caudal to shoulder	Forelimb flexors	Axillary, musculocutaneous, median, ulnar, part of the radial n.	C6,7,8, T1,2
Patellar reflex	Femoral	Quadriceps femoris	Femoral	L4,5,6
Cranial tibial	Peroneal	Cranial tibial	Peroneal	L6,7, (S1)
Gastrocnemius	Tibial	Gastrocnemius	Tibial	(L6),7, S1
Flexor/ withdrawal reflex of the hindlimb	Lateral digit:sciatic Medial digit:femoral	Hip, stifle, tarsal flexors	Sciatic	L6,7, S1
Anal reflex	Pudendal	External anal sphincter	Pudendal	S1,2,3
Perineal reflex	Pudendal	Anal sphincter and tail flexors	Pudendal n., coccygeal n.	S1–3, Cd 1–n
Cutaneous trunci reflex	Segmental spinal nerves	Cutaneous trunci	Lateral thoracic n.	C8, T1

tion of a nerve root supplying that limb. Lateralized cervical disc extrusion and nerve root tumours are the most common diagnoses in the forelimbs. L7–S1 compression and discospondylitis are two common problems resulting in root signature lameness of the hindlimbs.

Cerebral tumours have presented as neck pain. It is thought to be a form of referred pain as meningitis was not a feature.

Visceral pain initiates a reflex spasm of the abdominal muscles called 'guarding'. Epaxial and abdominal muscles become rigid in an effort to limit motion during spinal pain. These protective mechanisms are not classed as referred pain.

Differentiating brainstem from spinal cord lesions (Fig. 3.5)

1. The brainstem consists of the medulla, pons, midbrain and thalamus.
2. The ascending reticular activating system (ARAS) runs from the brainstem to the cerebrum. If it is damaged, alertness is decreased.
3. Cranial nerves II to XII originate in the brainstem, and are numbered from rostral to caudal.
4. Lesions caudal to the red nucleus of the midbrain cause ipsilateral weakness and proprioceptive deficits.
5. Lesions rostral to the red nucleus cause contralateral weakness and proprioceptive deficits.
6. Sometimes it is difficult to distinguish between medulla oblongata and cervical cord lesions.

Localizing spinal cord lesions

1. Strength and proprioception are affected caudal to the lesion.
2. Postural reactions will distinguish the normal limbs from the abnormal limbs.
3. Presence or absence of spinal reflexes in the abnormal limbs indicates the segmental level of the lesion (Fig. 3.6):
   - Intact reflexes in paralysed limbs: lesion is rostral to those segments. UMN paralysis.
   - Absent reflexes in paralysed limbs: lesion is within those segments tested. LMN paralysis.
4. Some C1–5 cervical cord lesions cause more severe paresis in the forelimbs than the hindlimbs. This has been known as *central cord syndrome* but extradural lesions, such as IVD extrusion, are commonly implicated.

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Table 3.4 Forelimb movement, muscle, nerve and cord segments

Movement	Muscle	Nerve	Cord segments
Advance the forelimb; elevate, draw forward, pull limb and shoulder forward or backward	Trapezius Omotransversarius Rhomboideus Serratus ventralis Cleidomastoideus Superficial pectorals	Accessory CN XI Cervical spinal nerves	Cervical spinal cord
	Supraspinatus	Suprascapularis	(C5),6,7
Shoulder flexion	Deltoideus Teres major Teres minor Subscapularis Infraspinatus	Axillary	(C6),7,8
Shoulder extension	Deep pectorals	Caudal pectorals, C8 T1	
	Supraspinatus Infraspinatus	Suprascapularis	(C5),6,7
	Subscapularis	Subscapularis	C6,7
Elbow flexion	Biceps brachii Brachialis Coracobrachialis	Musculocutaneous	C6,7,8
	Ext. carpi radialis	Radial	C7,8, T1,(2)
	Pronator teres	Median	C8, T1,(2)
Elbow extension	Triceps brachii Anconeus	Radial	C7,8, T1,(2)
Carpal and digital flexion	Flexor carpi radialis Superficial digital flexor	Median	C8, T1,(2)
	Flexor carpi ulnaris	Ulnar	
	Deep digital flexor Muscles between flexor tendons	Median/ulnar	C8, T1,(2)
	Palmar interosseous mm	Ulnar	
Carpal extension	Extensor carpi radialis Extensor carpi ulnaris	Radial	C7,8, T1,(2)
Digit extension	Common digital extensor Lateral digital extensor Ext. pollicis longus et indicis proprius	Radial	C7,8, T1,(2)

Cauda equina syndrome

Cauda equina (horse's tail) describes the appearance of nerve roots travelling caudally within the spinal canal before exiting through the appropriate intervertebral foramina. Cauda equina syndrome describes the clinical signs resulting from lesions in this anatomic area.

Sciatic nerve roots

- Decreased withdrawal reflex (particularly hock flexion)
- Pseudo-hyperreflexia of the patellar reflex
- Weight-bearing on the dorsal surface of the paw
- Atrophy of gluteal and caudal thigh muscle groups
- Decreased sensation of associated dermatomes.

Sacral nerve roots

- Reduced/absent anal and urinary sphincter tone and perineal reflex
- Atonic bladder
- Reduced/absent skin sensation of the perineum.

Caudal (coccygeal) nerve roots

- Reduced/absent tail tone, movement and sensation.

FOCAL LESIONS WITH MULTIFOCAL SIGNS

Skull

See Table 3.6 for multiple CNN deficits.

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Table 3.5 Hindlimb movement, muscle, nerve and cord segments

Movement	Muscle	Nerve	Cord segments
Hip flexion	Iliopsoas	Femoral	L4,5,6
	Tensor facia lata		
Hip extension	Superficial, middle, deep gluteals	Caudal gluteal	L7, (S1,2)
	Biceps femoris	Cranial gluteal	L6,7, S1
	Semimembranosus, semitendinosus		
Hip adduction	External obturator,	Obturator	(L4),5,6
	Pectineus, adductor		
	Gracilis, sartorius		
Hip abduction	Middle gluteal	Cranial gluteal	L6,7, S1
	Deep gluteal		
Outward rotation of the hip	Internal obturator	Sciatic	L6,7, S1,(2)
	Gemelli		
	Quadratus femoris		
Stifle flexion	Biceps femoris	Sciatic	L6,7, S1,(2)
	Semitendinosus		
	Popliteus	Tibial	
	Gastrocnemius		
	Superficial digital flexor		
Stifle extension	Quadriceps femoris	Femoral	L4,5,6
	(Tensor fascia lata)	(Cranial gluteal)	(L6,7, S1)
Tarsal flexion and digital extension	Cranial tibial, peroneus longus, long digital extensor, lateral digital extensor, extensor hallicus longus, peroneus brevis	Peroneal	L6,7
Tarsal extension	Gastrocnemius, superficial and deep digital flexors	Tibial	L7, S1
	(Biceps femoris, semitendinosus, gracilis)	(Sciatic)	[L6,7, S1,(2)]
Digital flexors	Superficial and deep digital flexors	Tibial	L7, S1
Plantar surface of hindpaw	Interosseous mm	Tibial	L7, S1

Cavernous sinus syndrome (CSS)

This is an uncommon neurological presentation but one which is similar to retrobulbar disease due to the effects on the intrinsic and extrinsic ocular muscles. It can occur unilaterally or bilaterally and in both cases, neoplasia is the most common cause. In humans, bilateral CSS was more frequently associated with vascular, infectious, and non-infectious inflammatory diseases (Fig. 3.7).

Ventricular system

Blockage of CSF flow leads to dilation of ventricular compartments rostral to the obstruction. Compression of adjacent brain tissue creates signs distant from the original focus. The brainstem lesion may subsequently develop cerebral signs due to dilation of the lateral ventricles (Table 3.7).

Herniation patterns

When compensatory mechanisms fail to cope with the increasing intracranial pressure, brain tissue shifts position within the skull. The brain may herniate under the falx cerebri, under the tentorium cerebelli, or caudally through the foramen magnum. Changes in breathing pattern, posture, or pupil size and reactivity are noted. Compression of the oculomotor nerve or midbrain results in mydriasis which is unresponsive to light.

The lesion may initially present as cerebral disease but subsequent pressure on the brainstem produces a dramatic change in gait, posture or alertness. Equally, an

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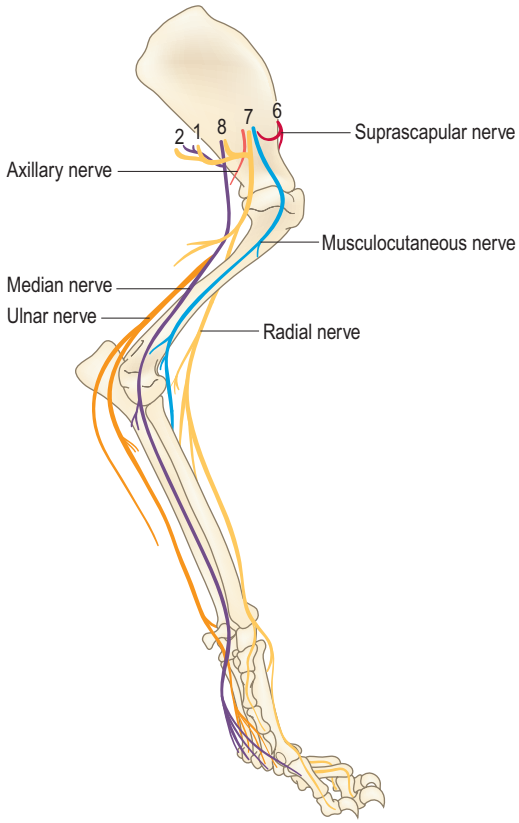


Figure 3.3 Peripheral nerves to the forelimb.

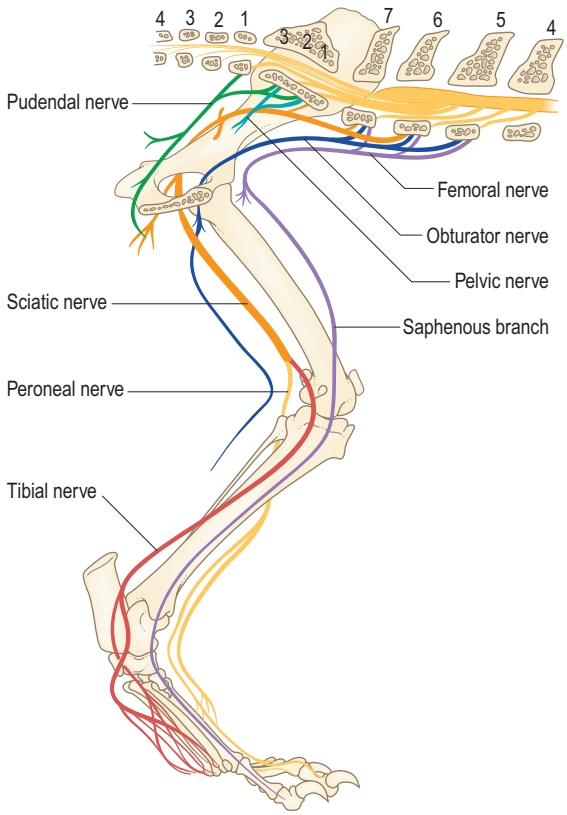


Figure 3.4 Peripheral nerves to the hindlimb.

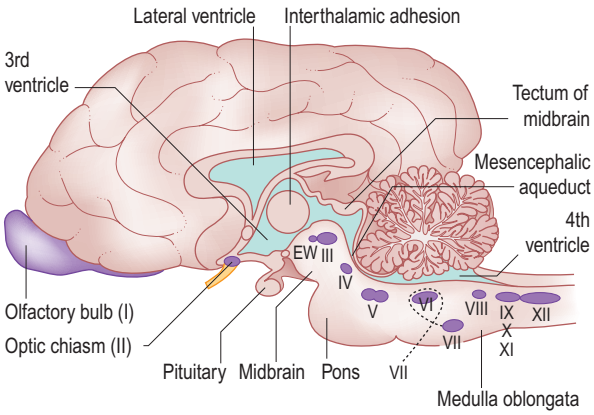
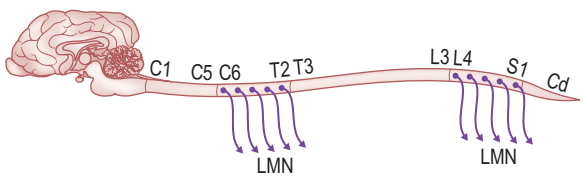


Figure 3.5 Cranial nerve nuclei within the brainstem. EW, Edinger–Westphal (parasympathetic nucleus of CN III).



	C1–C5	C6–T2	T3–L3	L4–S1	Cd
Forelimb	UMN	LMN	Normal	Normal	Normal
Hindlimb	UMN	UMN	UMN	LMN	Normal
Bladder	UMN	UMN	UMN	LMN	LMN

Figure 3.6 Signs associated with spinal cord lesions at different segmental levels. UMN, upper motor neuron; LMN, lower motor neuron.

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Table 3.6 Multiple CNN deficits

Site	Affected cranial nerves	Other signs
Orbital fissure	III, IV, V(1), VI	Horner's syndrome
Cavernous sinus	III, IV, V(1,2), VI	Horner's syndrome, seizures, neurotrophic keratitis
Orbital apex (retrobulbar)	II, III, IV, V(1), VI	Horner's syndrome (exophthalmos, restriction of eye movement)
Pituitary/sella turcica	II, III	Mental state, seizures, behaviour change
Cerebellopontine angle	V, VI, VII, VIII	Ataxia; paresis, cerebellar signs
Jugular foramen	IX, X, XI	Ataxia, paresis
Tympanic bulla	VII	Horner's syndrome, inner ear (cochlear; vestibular); meningitis; chronic otitis externa
Skull base syndrome	CN V–XII	Horner's syndrome, ± pain from mass eroding/invading bone or soft tissue, or from meningeal involvement

CN V(1): ophthalmic branch, V(2): maxillary branch, V(3): mandibular branch.

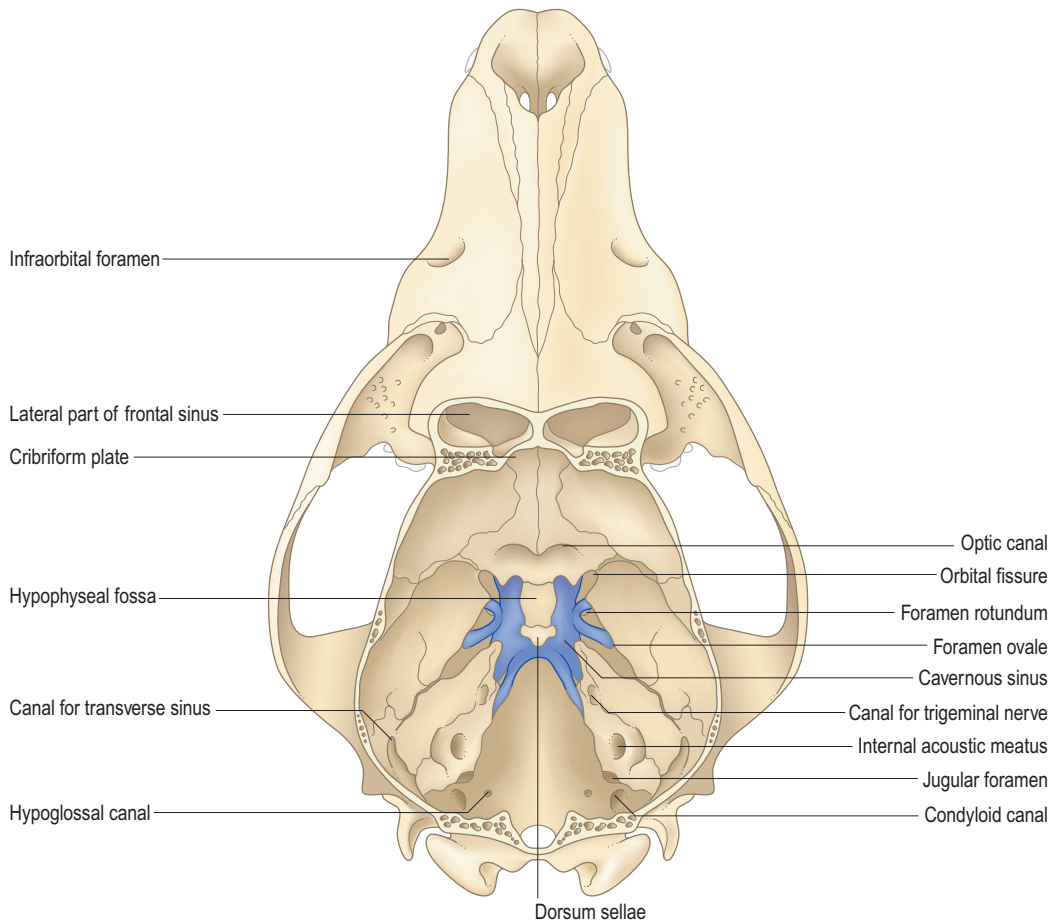


Figure 3.7 Foramen of the skull and the cavernous sinus.

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Table 3.7 Relationship of the ventricular lumen with divisions of the CNS

Major component	Subdivisions	Associated CNN	Derivatives	Lumen
Prosencephalon	Telencephalon	CN I	Cerebral cortex Basal nuclei Limbic system Rhinencephalon	Lateral ventricles Rostral part of third ventricle
	Diencephalon	CN II	Thalamus Hypothalamus	Most of the third ventricle
Mesencephalon	–	CN III, IV	Midbrain tectum (corpora quadrigemina) and tegmentum (cerebral peduncles)	Mesencephalic aqueduct
Rhombencephalon	Metencephalon-dorsal Metencephalon-ventral	CN V	Cerebellum Pons	Fourth ventricle
	Myelencephalon	CN VI–XII	Medulla oblongata	Fourth ventricle
Remainder of neural tube	Spinal cord	–	Spinal cord	Central canal

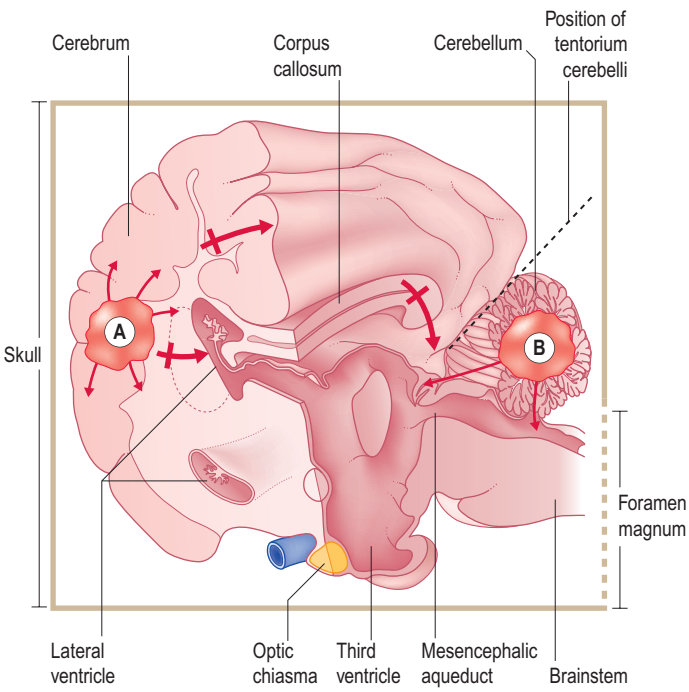


Figure 3.8 Herniation of the brain. (A) Cerebral hemisphere mass causing a lateral deviation of the falx cerebri (midline shift) and compression of the lateral ventricle (↔ arrows) with herniation under the tentorium cerebelli (↔ arrows); (B) Subtentorial mass compressing the brainstem, causing rostral herniation under the tentorium or caudal herniation under the tentorum or caudal herniation through the foramen magnum.

expanding cerebellar lesion can increase pressure within the caudal fossa and compress the brainstem (Fig. 3.8).

Multifocal neurological disease

Lesions in anatomically separate areas of the nervous system are referred to as multifocal. The disease processes commonly responsible for this scenario are:

1. Trauma
2. Inflammation, e.g. infectious or non-infectious inflammatory disease
3. Vascular, e.g. vasculitis, haemorrhage
4. Degeneration, e.g. lysosomal storage disease, mitochondrial disease.

Animals may present with a focal lesion which expands and creates further neurological deficits, e.g. brain herniation, myelomalacia. Certain anatomical sites create a variety of deficits which may initially appear as if separate regions are individually affected, e.g. a generalized neuromuscular disease causing cranial nerve lesions and appendicular weakness; dysautonomia; a lesion in the region of the cerebellopontine angle causing paradoxical vestibular disease, hemiparesis, facial nerve and trigeminal nerve paralysis.

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SECTION 2

ALTERATION IN MENTAL STATE

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4 Alteration in mental state – an introduction

INTRODUCTION

This is the most common reason for animals to be presented to the veterinarian. Owners remark that the animal is 'not himself', 'not right'. When pressed for details, the next observation is just as non-specific: lethargy, depression. This should be no surprise; many diseases result in a less reactive mental state, drowsiness or apparent indifference to stimuli.

Alertness is promoted by the ascending reticular activating system (ARAS), a network of neurons projecting from the brainstem to the cerebral cortex. Damage to the ARAS alters the level of consciousness.

CLINICAL TIP

Not all lethargic animals have brain disease.

Presenting signs

Some animals are described as having a 'far-off' gaze, or staring, vacant expression. This may signal a depressed state of alertness. Mydriasis, blindness, a lack of blinking, and a rigid neck posture can create the same impression. Deafness and exercise intolerance will alter an animal's responsiveness to commands.

What is consciousness?

Consciousness is the state of awareness of the self and the environment. The former is anthropomorphized by owners, while the latter is used by veterinarians to judge the level of mental responsiveness. This state of awareness relies on the animal being alert.

Consciousness is graded by the decreasing level of function from alert to comatose. The term semi-comatose has been used in texts, but as coma is defined as a total lack of responsiveness to stimuli, using the prefix 'semi-' can confuse communication.

Describing the mental state

1. Alert
   - Conscious; capable of responding to sensory stimuli. A more clinically useful statement would be to describe what the animal is responsive to and if that response is normal or appropriate.
2. Obtunded/depressed/lethargic
   - A mild to moderate decreased interaction with or interest in the environment
   - Responds slowly to verbal stimuli
   - Increased hours of sleep.
3. Stupor
   - Unresponsive unless roused by a noxious stimulus
   - Appears to be in a deep sleep to which it returns once the stimulus has ceased.
4. Coma
   - Complete unresponsiveness to any stimuli. Unrousable
   - Graded according to a scale for monitoring progression of causative disease.

Delirium: In the human this is characterized by disorientation, fear, irritability, agitation and misperception of sensory stimuli which may be episodic and interspersed with lucid intervals. It results from a generalized impairment of brain function and occurs in metabolic or toxic disorders or multifocal cerebral disease. It may precede or follow stupor/coma.

Dementia: This is a persistent insidious reduction in mental awareness or understanding in an alert patient. The term is generally applied, in humans, to those with a chronic problem with little chance of improvement, e.g. degeneration, trauma, tumour.

Localizing the lesion

Stupor and coma result from one or both of the following:

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- Diffuse, bilateral cerebral impairment
- Failure of the rostral brainstem ARAS within the mid-pons, extending rostrally to the thalamus.

CLINICAL TIP

Stupor and coma usually result from sub-acute or acute brain disease

A lesion in the compact brainstem damages a larger proportion of the ARAS with a more profound effect on consciousness than would the same sized lesion in the cerebrum which only picks off a small fraction of the now-dispersed fibres.

CLINICAL TIP

It is more common for coma to result from brainstem lesions than from cerebral disease

Five observations aid in specifying the area of dysfunction:

1. State of consciousness
2. Pattern of breathing
3. Size and reactivity of the pupils
4. Eye movements and oculovestibular responses
5. Skeletal muscle motor responses.

RESPIRATORY PATTERN

Normal respiration

Respiration maintains normal oxygenation and acid–base balance. Abnormalities can alter mental state, exercise ability and voice production.

Inspiratory respiratory muscles

- Diaphragm: accounts for most of thoracic volume change during quiet inspiration. Innervated by the phrenic nerve which originates from cord segments and nerve roots C5–7, found at vertebral level C4–C6
- External intercostal muscles elevate the lower ribs. Innervated by segmental spinal nerves.

Either muscle set alone can provide adequate ventilation at rest. Most of the work of breathing is moving the elastic tissues of the thoracic wall and lungs.

Expiratory respiratory muscles

- Used in forced expiration. The internal intercostals muscles and the abdominal muscles contract the rib cage caudally, decreasing intrathoracic volume. Intra-abdominal pressure is increased, pushing the diaphragm cranially.

Stimulus for respiration

Spontaneous respiration is produced by rhythmic discharge of motor neurons in the medulla. It is stimulated by changes in arterial blood and CSF/brain ECF concentrations of CO_2 or H+ and O_2.

These chemical changes are detected by the aortic bodies, each a collection of specialized cells near the arch of the aorta, and the carotid bodies, found associated with the carotid bifurcation bilaterally. Chemoreceptor cells are also present in the medulla.

Increased cellular metabolism raises the CO_2 concentration and hence H+ concentration, which in turn stimulates respiration via the chemoreceptors, particularly those of the medulla.

Acidosis from ketone bodies will stimulate respiration causing hyperventilation.

Loss of HCl from vomiting decreases ventilation, allowing CO_2 (and H+) to increase.

Hypoxia (low PaO_2 or decreased O_2 delivery in vascular stasis) also increases ventilation by stimulating the carotid and aortic bodies.

Afferents from the carotid body travel to the medulla via CN IX, and those from the aortic bodies, via CN X.

Autonomic motor pathways from the medulla descend to innervate respiratory muscles. Voluntary control of respiration is mediated by the cerebrum and descending pathways bypass the medulla and innervate respiratory muscles.

The pneumotaxic centre in the pons and afferent signals from the vagal nerves during lung expansion or deflation modify the spontaneous discharges from the medulla.

Movement of joints stimulates respiration. This is employed during anaesthesia to combat breath holding during induction. Gently stretching and flexing the limbs can stimulate inspiration.

Abnormal respiration

Figure 4.1 shows the respiratory patterns.

Mechanical hypoventilation

Hypoventilation occurs with skeletal muscle weakness caused by lower motor neuron (LMN) disease, e.g. poly-

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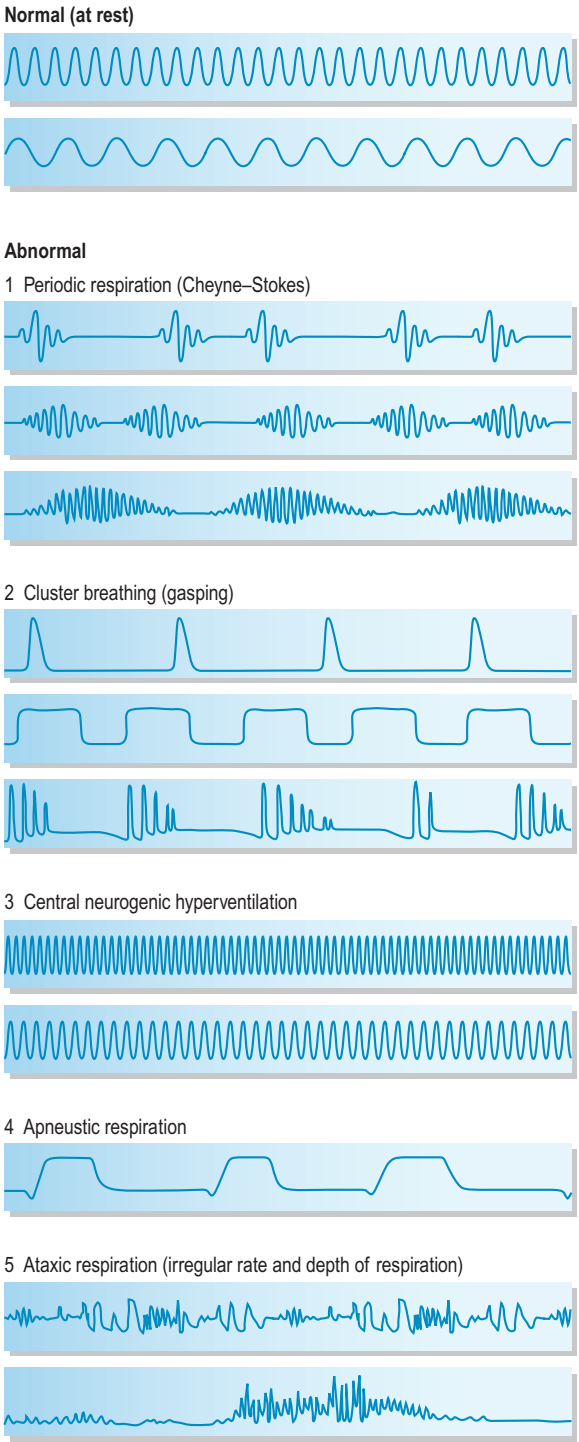


Figure 4.1 Respiratory patterns.

radiculoneuritis, botulism, or upper motor neuron (UMN) disease such as a cervical disc extrusion. The animal is physically unable to ventilate itself and the thoracic wall movement is either minimal or absent. Preservation of diaphragmatic movement with loss of intercostal and abdominal muscle function can produce paradoxical ventilation in which the thorax retracts on inspiration and expands during expiration.

Dogs unable to expand the thorax will be unable to bark or cough.

Cervical cord lesions cranial to spinal cord segments C5–7 will weaken the diaphragm and intercostal muscles; severe lesions, such as those obliterating the pain pathways, will produce respiratory paralysis and death.

The degree of hypoventilation is judged by the arterial partial pressure of CO_2 ($PaCO_2$).

Periodic respiration (Cheyne–Stokes respiration)

Brain damage can produce hypersensitivity to arterial PCO_2. Hyperventilation ensues, lowering the arterial PCO_2. Apnoea results. Hypoxia then stimulates the carotid and aortic chemoreceptors and respiration recommences. As endogenous CO_2 builds up the pattern repeats.

The hyperventilation phase lasts longer than the apnoea. Regular respiration occurs in a smooth crescendo ending in a smooth decrescendo.

Periodic breathing with a shorter, less regular cycle develops with severe increases in ICP and the resultant ischemia of the brainstem (*cluster breathing or gasping*).

It occurs with a lesion anywhere from cerebral hemispheres to upper pons. It may also occur in sleep, hypoxia, uraemia and severe congestive heart failure.

Hyperventilation with brainstem injury

Sustained rapid, regular and fairly deep hyperventilation occurs with rostral brainstem lesions. It also follows pulmonary complications of brain damage which cause systemic hypoxia or metabolic acidosis: aspiration, congestion, infection or neurogenic pulmonary oedema.

Apneustic breathing

Ventilation pauses at full inspiration and again at end-expiration. It occurs with pontine lesions.

Ataxic breathing

An unpredictable pattern of deep and shallow breathes occurring randomly with irregular pauses. It is seen with

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lesions of the respiratory centres of the (central) medulla, e.g. trauma, cerebellar herniation or haemorrhage.

PUPIL SIZE AND REACTIVITY

Table 4.1 indicates pupil size and pupil light response (PLR). Figure 4.2 shows pupil size in the comatose patient.

EYE MOVEMENT AND OCULAR–VESTIBULAR INTERACTION

The neurons contributing to the generation of nystagmus are intertwined with those brainstem neurons subserving consciousness.

The vestibulo-ocular reflex (VOR) is a compensatory mechanism designed to keep images stabilized on the retina when the head rotates. Rotation of the head to the left, for example, results in the eyes slowly moving to the right. The sensory input to the reflex comes from the semicircular canals, part of the vestibular apparatus. The term VOR is applied to the reflex generated by rotating the head relative to the body and also by the simultaneous rotation of the head and body as one item. Sensory input from the joint and muscle receptors in the

Table 4.1 Pupil size and pupil light response

Pupil size	Light response	Interpretation
Unilaterally dilation	Slow or absent	CN III compression secondary to tentorial herniation
Unilateral dilation	Slow	Ipsilateral or contralateral cerebellum
Bilaterally dilation	Slow or absent	Inadequate brain perfusion Bilateral CN III paralysis Midbrain
Bilateral mid-position	Absent	Rostral brainstem or medulla
Unilaterally dilated or equal	Cross-reactive (Marcus Gunn*)	Optic nerve injury Extensive retinal pathology
Bilaterally constricted (pinpoint)	May be difficult to determine (reactive in metabolic disease)	Opiates, Metabolic encephalopathy Cerebrocortical disease Pontine lesions
Unilaterally constricted	Intact	Injured sympathetic pathway

*Marcus Gunn swinging light test to look for a relative afferent pupil defect.

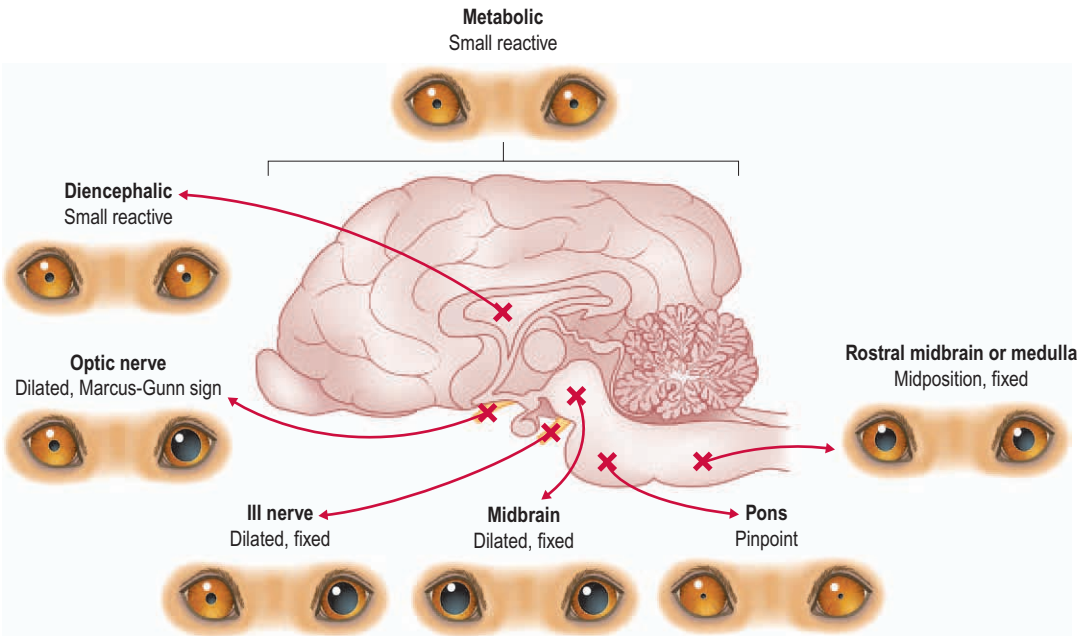


Figure 4.2 Pupil size in the comatose patient.

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neck (*cervico-ocular reflex*) contributes very little to the VOR at the usual frequencies of rotation used in clinical examination. There is negligible contribution from the vestibular otoliths which react to linear acceleration.

The motor function is performed by the extraocular muscles. The afferent (CN VIII nuclei) and efferent arcs (CNN III, IV, VI) are connected by the medial longitudinal fasciculus (MLF) within the brainstem. An intact reflex signifies that a long length of brainstem, from medulla to midbrain, retains some function. The reflex is abolished by a lesion anywhere along this pathway. Associated deficits aid localization. In the comatose animal, the lesion is usually destruction of brainstem tracts and hence the prognosis is grave.

The oculocephalic reflex is synonymous with the VOR induced by passive rotation of the head, keeping the body still. The *doll's head manoeuvre* is used to describe the same method of stimulating the VOR to produce the compensatory slow phase eye movement but it was originally used to refer to the elevation of the eyelids and upward rotation of the eyes generated by ventroflexing the neck. To avoid confusion, VOR is the preferred term.

Physiological nystagmus is a normal phenomenon induced by rotation of the head. The slow, vestibular component, the VOR, moves the eyes in the direction opposite to the rotation. As rotation continues, a fast jerk, or saccade, in the direction of rotation then takes place, aligning the eyes 'straight ahead'. A nystagmus is thus formed by the slow drift phase opposite rotation, and the fast jerk in the direction of rotation. The re-aligning quick phase requires a structurally intact pontine or mesencephalic reticular formation and descending supratentorial influences. Absence of physiological nystagmus has the same implications as loss of the VOR.

SKELETAL MOTOR RESPONSE

Abnormal postures in the comatose animal can indicate the level of neurological damage and hence aid in monitoring and prognosis. These postures may be present at rest or assumed when the animal is physically stimulated.

Decorticate posturing (flexor posturing)

- Described in comatose humans with the arms flexed over chest and the legs extended
- Caused by lesions above the red nucleus
- Indicates severe brain damage.



Figure 4.3 Opisthotonus.

Decerebrate posturing (extensor posturing)

- Extension of all limbs, opisthotonus
- More severe than decorticate posturing
- Caused by brainstem damage below the red nucleus (mid-collicular), e.g. midbrain and cerebellum lesions.

Opisthotonos (Fig. 4.3)

- Muscular spasm causing dorsiflexion of head, neck and spine
- Accompanied by extension of forelimbs ± hindlimbs
- Occurs when inhibition of extensor muscles is removed, e.g. midbrain lesion (removal of descending inhibition), tetanus and strychnine toxicity (prevention of local spinal inhibition).

Decerebellate rigidity

- Opisthotonus with thoracic limb extension, flexion of pelvic limbs (hips) up under the body, or pelvic limbs rigidly extended
- Acute cerebellar lesion.

Adversive syndrome

- Leaning, head turning, propulsive circling, or ocular deviation toward the side of the rostral thalamic lesion.

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5 Head trauma

INITIAL PRESENTATION

Obtunded, stupor, coma.

INTRODUCTION

Being hit by a car, human, large animal, falling tree or furniture, being shot at or in a dog fight are all reported causes of head trauma in small animals. The dog that knocks its head against a door frame and carries on walking will not have sustained clinically significant head trauma.

SIGNALMENT

A 6-year-old male crossbred dog.

CASE PRESENTING SIGNS

Presented in a mentally unresponsive state.

CASE HISTORY

A sudden onset of stupor or coma (Table 5.1) should not automatically be blamed on external trauma, unless, of course, the event is witnessed or has produced other physical evidence. The owner must always be questioned, however briefly, on the animal's recent health status.

In this case, the dog had been exercising off the lead and out of view. A train was heard to pass nearby. The dog was called but failed to return. The animal was found soon after, in lateral recumbancy in an unresponsive mental state. The owners presented the dog, saying it had been hit by a train.

CLINICAL EXAMINATION

Initial assessment of a traumatized animal is done quickly and is repeated at frequent intervals. Mental state, pupil size and pupil light reflex (PLR), eye movement (resting position, spontaneous movement, and vestibulo-ocular reflex, VOR) should be noted. Approximately 10% of traumatic brain-injured humans also have a cervical spine injury. Avoid rotating or hyperextending the neck.

The Glasgow Coma Scale was modified by Dr Andy Shores for use in small animals and this helps monitor progression of signs (Table 5.2). One study found that there was a 50% probability of survival during the first 48 hours after head trauma in dogs with a score of 8. Contrary to humans, the age of the animal did not predict survival. The study was not able to suggest a long-term prognosis based on either survival or functional capacity.

Examination in this case revealed:

- Coma
- Lateral recumbency
- Bilaterally symmetrical pinpoint pupils
- VOR was present
- Regular respiration with good thoracic excursions
- Absence of external signs of trauma
- Heart rate and pulse rate were 126 bpm
- Temperature was 37.7°C.

Neuroanatomic localization

The lesion was localized to the cerebrum or brainstem ARAS based on the coma.

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Differential diagnosis

It seemed highly unlikely that a dog could be hit by a train and not be visibly damaged by the experience. Head trauma from a blunt projectile may have occurred. Increased intracranial pressure from neoplasia, cerebral haemorrhage, or inflammation would be expected to have shown some earlier signs of mentation or behaviour change or have a less-acute onset but this is not always the case. Metabolic encephalopathy can alter mentation acutely and for extended periods of time (hours).

Table 5.1 Causes of stupor and coma

Brainstem	Bilateral cerebrum
Trauma	Metabolic encephalopathies
	Hypoglycaemia
	Hyperosmolar (DKA; hypernatraemia)
	Hypo-osmolar (water intoxication)
	Uraemia
	Acidosis
	Anoxia
	Portosystemic shunt
	Hyperkalaemia
	hypocalcaemia
Tumour	Toxicity
Inflammation	Post-ictal
Vascular	Hydrocephalus
Hydrocephalus	Herniation with secondary compression of brainstem

CASE WORK-UP

1. Assessing oxygenation
   - Pulmonary contusions are common post-trauma. Auscultation detects increased lung sounds distributed unevenly over the thorax, or decreased lung sounds with consolidation. There is no specific therapy. Treatment is supportive.
   - Neurogenic pulmonary oedema is rare but most often associated with severe head trauma. It progresses within hours or days to resolution or death. Improvement, if it is to occur, is usually seen within 48 hours. It results from a central sympathetic discharge from an increase in ICP. Treatment is aimed at reducing ICP, oxygenation, and diuretics.
   - Respiration patterns are often altered in coma. A lack of increased effort does not imply adequate oxygenation/ventilation.

Table 5.2 The modified Glasgow Coma Scale for small animals

Small animal coma scale	Score
Motor activity	
Normal gait, normal spinal reflexes	6
Hemiparesis, tetraparesis, or decerebrate activity	5
Recumbent, intermittent extensor rigidity	4
Recumbent, constant extensor rigidity	3
Recumbent, constant extensor rigidity with opisthotonus	2
Recumbent, hypotonia of muscles, depressed or absent spinal reflexes	1
Brainstem reflexes	
Normal pupillary light reflexes and oculocephalic reflexes	6
Slow pupillary light reflexes and normal to reduced oculocephalic reflexes	5
Bilateral unresponsive miosis with normal to reduced oculocephalic reflexes	4
Pinpoint pupils with reduced to absent oculocephalic reflexes	3
Unilateral, unresponsive mydriasis with reduced to absent oculocephalic reflexes	2
Bilateral unresponsive mydriasis with reduced to absent oculocephalic reflexes	1
Level of consciousness	
Occasional periods of alertness and responsive to environment	6
Depression or delirium, capable of responding but response may be inappropriate	5
Semicomatose, responsive to visual stimuli	4
Semicomatose, responsive to auditory stimuli	3
Semicomatose, responsive only to repeated noxious stimuli	2
Comatose, unresponsive to repeated noxious stimuli	1

Note: Asymmetrical abnormalities (e.g. pupil size) are assigned the lower score of the two possible. Score before medication and supportive care administered. Oculocephalic reflex, VOR; Semicomatose-stuporous.

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2. Assessing ventilation
   - Hypoventilation results from brain injury, thoracic trauma and pain, weakness secondary to spinal cord or brainstem injury. In addition, ventilation-perfusion mismatch can follow head trauma without evidence of pulmonary disease on the radiographs.
   - Short, shallow respiration with intermittent attempts at deep respiration accompanies pleural space disease (pleural effusions, pneumothorax).
   - Respiratory acidosis is a warning that respiratory failure will occur.
3. Assessing systemic perfusion
   - Hypoperfusion manifests as a faster heart rate (dog), weaker pulse, paler mucous membrane colour, slower capillary refill time, and lower rectal temperature. Hypovolaemic animals have a depressed mentation which improves with treatment. The mortality rate in humans with severe traumatic brain injury is doubled with a systolic blood pressure of <90 mmHg.

CLINICAL TIP

Systemic hypotension does not result from lesions rostral to the lower brainstem.

4. Assessing intracranial pressure (Table 5.3)
   - The closed skull gives the traumatized, swelling brain little room for accommodation. Increasing ICP forces blood and CSF out of the brain. As ICP equals or exceeds the systemic arterial pressure, the systemic blood pressure increases and a reflex bradycardia follows. These reflex changes are called the *Cushing's reflex* and are more common in end-stage disease.
   - Increases in ICP are monitored by neurological examination in veterinary patients. Papilloedema is rarely seen after acute head injury in humans.

Table 5.3 Assessing the coma patient

Function	Aim
Oxygenation	PaO_2 >90 mmHg (dog) PaO_2 >100 mmHg (cat) SaO_2 >95% PCV 25–30% Haemoglobin (Hb) 7–10 g/dl. (N = 13.8–21.4 g/dl)
Ventilation	10–20 breaths/min $PaCO_2$ of 25–35 mmHg
Perfusion	Systolic pressure >90 mmHg
Intracranial pressure	Improvement in neurological examination

CLINICAL TIP

Pulse oximeters overestimate oxygenation.
End-tidal CO_2 monitors underestimate $PaCO_2$.

Investigation of this case required:
- Sudden onset coma: blood and urine analysis to assess the metabolic state and clotting ability.
- Tetraplegia: radiographs of the axial skeleton to assess for fractures. The lack of voluntary movement, and response to pain made judgement of a subclinical spinal injury very difficult. Muscle tone was normal to slightly increased, which made a lower motor neuron lesion less likely.
- Possible external trauma: thoracic radiographs, abdominal radiographs, monitor PCV and urine production.
- Further history: the dog had been well prior to the coma.
- Imaging the brain: the sudden onset of coma without confirmed trauma or systemic disease.

DIAGNOSIS

Metabolic causes were ruled out by normal laboratory findings. The low normal body temperature was insufficient to produce such a profound depression of the mental state. Over the course of the following 12 hours, swelling of the right temporalis muscle developed. Blunt trauma, possibly from a stone, was suspected. Imaging of the brain was discussed but declined.

PROGNOSIS

The prognosis for a head trauma case is determined by the other injuries sustained and the degree of neurological deficit. The latter is judged by serial examination.

Dramatic improvements in brain function can be seen in the first week after treatment of acute head trauma and many animals return to being functional pets.

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Treatment and prognosis remain directed by examination findings. If the animal neurologically deteriorates despite medical treatment then imaging is indicated. CT classification of head trauma in humans has prognostic value but such studies do not exist in veterinary medicine.

Brain death is an indication for euthanasia.

Clinical criteria for brain death

1. Exclude: toxin/drug-induced, metabolic coma, hypothermia and hypovolaemia.
2. Examination:
   - Coma
   - No spontaneous respiration at PCO_2 >60 mmHg
   - Absent brainstem reflexes:
     - pupils dilated and unresponsive to light
     - absent vestibulo-ocular reflex
     - absent corneal reflex in both eyes
     - absent gag reflex.

The period of observation in humans is lengthy (6–72 hours). No time guidelines exist for animals.

Electroencephalogram (EEG) and brainstem auditory evoked response (BAER) have also been used in assessment of brain death. They are not necessary. They confirm the clinical assessment, nothing more.

Caloric testing is used in humans as another test of the vestibulo-ocular pathways through the brainstem. Irrigation of the ear canal with warm or cold water induces a density change in the endolymph of the inner ear. This creates a convection current within the semicircular canal that stimulates the hair cells. Direct thermal effects on the vestibular nerve may play a role. The response can be delayed up to 1 minute or more. Ice cold water in the external ear canal for 3–5 minutes induces fast phase nystagmus to the contralateral side but this may be absent in normal dogs. Testing with warm water (44°C) is apparently even less reliable.

The complications of head trauma include:

- DIC
- Gastrointestinal ulcers
- Aspiration pneumonia
- Sepsis
- Pulmonary oedema; neurogenic, cardiogenic
- Atelectasis
- Cardiac arrhythmias
- Hypertension (human) from excessive secretion of catecholamines begins within the first 10 days and may continue for 3 months, also a result of the Cushing's reflex from increased ICP
- Seizures may commence months to years later in an otherwise neurologically normal animal. Some 10% of humans with 'medium to severe' craniocerebral injury develop seizures, generally within 2 years after the trauma.

Syndrome of inappropriate antidiuretic hormone secretion (SIADH)

Excessive ADH secretion can result from various cerebral diseases. Water retention and continued urinary loss of sodium (20–30 mEq/l) results in a dilutional *hyponatraemia*. There is a decreased serum osmolality, increased urine osmolality, and decreased urine output. Treatment requires fluid restriction and diuresis.

Serum sodium should be increased no faster than 0.5–2 mEq/l/hr or 12 mEq/l/day in order to avoid osmotic demyelination (central pontine myelinolysis).

Cerebral salt wasting (CSW)

Caused by an increased secretion of atrial and brain natriuretic peptide which suppresses aldosterone synthesis. Increased renal excretion of sodium occurs (150–200 mEq/l), followed by water, resulting in *hyponatraemia* and hypovolaemia. Treatment is sodium replacement and fluid administration.

Central diabetes insipidus

Decreased release of ADH results in loss of large volumes of dilute urine, leading, if untreated, to dehydration, *hypernatraemia*, and increased serum osmolality.

In **this particular case** a reasonable chance of useful function returning was predicted, based on the functioning VOR and small symmetrical pupils.

TREATMENT

Treatment aims to limit damage from the four horsemen of the intracranial apocalypse: hypotension, hypoxaemia, hypercarbia and increased intracranial pressure (ICP).

1. Maintaining oxygenation
   - Supplemental oxygen should be provided to any critical trauma patient until proven unnecessary
   - Nasal O_2 catheter: 100 ml/kg/min provides 40% inspired O_2
   - Transtracheal O_2 catheter: 50 ml/kg/min provides 40% inspired O_2
   - Whole blood is used if the PCV <20% (Hb <8 g/dl) or is rapidly declining. Aim for a *PCV of 25–*

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30% and albumin >2.0 g/l. Rate of administration: 4–10 ml/kg/hr over 4–6 hours
- Haemoglobin-based oxygen carriers (oxyglobin) are administered when *haemoglobin (Hb)* is <7 g/dl. Aim for 7–10 g/dl (n = 13.8–21.4 g/dl). This is used in dogs at 5 ml/kg increments with a limit of 30 ml/kg/day. The half life is 30–40 hours. It improves oxygenation and has a vasopressor action.

2. Maintaining ventilation
   - Positive pressure ventilation is required if oxygenation or ventilation cannot be maintained or requires severe increases in respiratory effort
   - 10–20 breaths/min give the desired *$PaCO_2$ of 25–35 mmHg*.
3. Maintaining systemic perfusion
   - Give fluids based on cardiovascular need as judged by blood pressure, urine output and central venous pressure. Restore *systolic pressure to >90 mmHg*
   - *Small volume resuscitation* is recommended for hypotensive cats, and hypotensive dogs when the consequences of fluid overload are life-threatening, e.g. head trauma, pulmonary contusions, cardiogenic shock, oliguric renal failure, and hyperosmolar states (hypernatraemia, hyperglycaemia). It is also the recommended technique for cardiopulmonary resuscitation of animals that are euvolaemic before the arrest. Volume replacement and expansion should not cause cerebral oedema as long as volume overload and elevated right heart pressures are avoided and serum osmolality remains normal.

Methods

Dog
- Isotonic *crystalloids 10–20* ml/*kg* + *hetastarch 3–5* ml/*kg*. Given as an IV bolus
- Titrate additional crystalloid 10–20 ml/kg + 1–5 ml/kg colloid to raise blood pressure
- Stop when desired mean arterial pressure or maximal colloid dose is reached
- Consider vasopressors (phenylephrine or dopamine infusion) if the blood pressure and cardiac output cannot be restored.

Cat
- Isotonic *crystalloids 10–15* ml/*kg* + *hetastarch 2–3* ml/*kg*. Given as an IV bolus
- Titrate additional hetastarch 2–5 ml/kg to raise blood pressure to >40 mmHg
- Ensure body temperature is >36.5°C.

Larger volumes may be given to hypovolaemic animals during cardiopulmonary resuscitation if over-perfusion is not a major concern: *Lactated Ringers solution (LRS) or 0.9% NaCl* 90 ml/kg/hr (dog), 40–60 ml/kg/hr (cat) to effect. LRS is slightly hypotonic to plasma. If large volumes are required for treating hypovolaemia, a more isotonic solution or colloid should be used.
- One fourth of the bolus is provided within the first 5–15 minutes and the patient reassessed
- Improvement is usually seen by the time half the animal's vascular volume (dog: 45 ml/kg; cat: 20–30 ml/kg) has been given.

Most (70–80%) of the crystalloid solution moves to the ECF within an hour of IV administration in a normal animal.

Infusion rates can be reduced by 40–60% when colloids are used in addition.

Hetastarch and Dextran 70 have the same doses, dose limit, and half-life.
- 10–20 ml/kg IV to effect as a bolus in dogs
- 5 ml/kg IV increments over 5–10 minutes in cats
- Limits: Dog: 20–50 ml/kg/day. Cat: 5–20 ml/kg/day
- Half-life of 25 hours.

Hypertonic saline at 3% and 7% has been used in resuscitation at a dose of 4–6 ml/kg IV given slowly over 5 minutes. The duration of action is 15–60 minutes. Vagal-induced bradycardia and hypotension occur if it is given too rapidly.

Cardiac arrhythmias, often ventricular, commonly occur 12–36 hours after trauma secondary to direct injury, or shock/ischaemia. The pulse must be checked while the heart is auscultated. Poor tissue perfusion is an indication for treatment with lidocaine. Most cases resolve within 2–4 days. Severe hypothermia causes cardiac arrhythmias.

4. Control ICP

   Normal ICP for dog and cat: 5–12 mmHg. Treatments are given in an empirical manner as objective monitoring of ICP is not routinely performed. Fluid restriction is not used. Reduction of the colloid oncotic pressure alone does not increase oedema in the injured brain.
   - Mannitol:
     - 0.5–2.0 g/kg IV over 10–20 minutes
     - Limit: 3 boluses within a 24-hour period to avoid hyperosmolality
     - Effects within minutes and lasts 2–5 hours
     - Warm solution before use to dissolve crystals

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- Practical benefits outweigh theoretical concern about creating intracellular hyperosmolality or worsening intracranial haemorrhage.
- Frusemide
  - Doses and timing of administration vary from source to source
  - 0.75–1.0 mg/kg IV to reduce CSF production
  - 2–5 mg/kg IV before mannitol use for synergistic effect
  - 0.7 mg/kg IV or IM 15 minutes after mannitol administration
  - Some use it alone, without mannitol. There is a delayed onset of effect compared with mannitol.
- Barbiturate comas are used in humans to control unresponsive ICP. The dose is titrated based on EEG measurements and cardiovascular stability. This is not performed in animals as neither ICP nor EEG are monitored
- Hyperventilation to a $PaCO_2$ of 30 mmHg is the aim in human medicine. Vascular response to CO_2 levels may be absent or decreased in head trauma. Prolonged or excessive hyperventilation can lead to diffuse cerebral hypoxia
- Hypertonic saline can cause hypernatraemia.

5. Control pain
   - Opioids are used for pain control. There is little or no change to the ICP in most circumstances
   - Fentanyl: Dog: 0.002–0.005 mg/kg IV, IM, SQ q1–2 hr. Cat: 0.001–0.003 mg/kg IV IM SQ q2–4 hr
   - Fentanyl patch: 25 µg/hr: <5–10 kg; 50 µg/hr: 10–20 kg; 75 µg/hr: 20–30 kg; 100 µg/hr: >30 kg
   - Patches require 3–6 hours to produce maximum effect in cats, and 12–24 hours in dogs. Duration of action varies: 1–3 days. If the animal weighs <5 kg, keep half the patch covered
   - Morphine/methadone: dog/cat: 0.05–1.0 mg/kg IM, SQ, slow IV q4 hr.
6. Correct hypothermia
   - Hypothermia blunts the cardiac response to catecholamines
   - Mild hypothermia is used in humans to decrease the brain's metabolic demands and thus make it more capable of withstanding ischaemia. Tympanic membrane temperature is a more accurate reflection of brain temperature than oesophageal temperature.
7. Control seizures

   Emergency treatment:
   - Propofol: 1–2 mg/kg IV
     - Infusion: 0.1–0.6 mg/kg/min, to effect
     - Decreases ICP. Anaesthetic. High doses can drop blood pressure which can decrease cerebral perfusion. Avoid hypotension
   - Benzodiazepines: 0.5–1.0 mg/kg IV
     - Infusion: 0.5–2.0 mg/kg/hr in 0.9% saline
     - Little or no effect on ICP. Anticonvulsant
   - Phenobarbitone: 2–6 mg/kg IV
   - Anticonvulsant
   - Pentobarbitone: 2–15 mg/kg IV slowly, to effect
     - Difficult to obtain. Anaesthetic. Causes hypotension and hypothermia, neither being desirable. The dose required to decrease brain metabolism also causes hypotension. Excitement during recovery can resemble seizure activity resulting in unnecessary repeat doses.

   Anticonvulsant prophylaxis is not routinely commenced after head trauma. Head trauma sufficient to induce stupor or coma may lead to seizures later in life. Such animals may be otherwise neurologically normal. Anticonvulsants are only used if the seizure pattern warrants it.
8. Other factors
   - *Vomiting* occurs with increased ICP and vestibular derangement as both stimulate the vomiting centre. The vestibular system also projects to the chemoreceptor trigger zone (CTZ). Narcotics cause emesis. Delayed gastric emptying and/or gastric atony occur with head injury
   - *Serum glucose*. Hyperglycaemia aggravates ischaemia. Avoid hypoglycaemia. Glucose should only be used to treat hypoglycaemia
   - *Hypermetabolic state*. Severe traumatic brain injury has a 140% increase in resting metabolic expenditure. Place a feeding tube if the patient is unable to eat voluntarily for >2 days
   - *Nursing*: Elevate the head 10–30% above the heart, avoid compression of jugular veins, ensure the bladder empties, turn the patient frequently, monitor electrolytes and maintain normovolemia with isotonic crystalloid fluids. Lubricate the eyes frequently to prevent corneal ulcers
   - *Corticosteroids*: Brain oedema post-trauma is a mixture of cytotoxic (ischemia) and vasogenic (blood-brain barrier disruption) and yet use of corticosteroids in humans has been associated

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with a worse outcome. There is some debate as to the cerebro-protective benefit of methylprednisolone sodium succinate unrelated to its glucocorticoid activity. If coma has been caused by mass lesions (brain tumours, epidural hematomas) then corticosteroids are indicated.

BRAIN SURGERY

1. Penetrating bite wounds lead to abscesses. Debridement and broad spectrum antibiotics are indicated.
2. In the absence of progressive deterioration, removing haematomas may not significantly improve the case.
3. If the skull segment is depressed more than its own thickness and compounded, surgery should be considered.

Referral is recommended prior to any brain surgery, not after. Fatal brain injury occurs without skull fracture. Repairing a fracture is no guarantee of a successful outcome.

Treatment in this case consisted of pain relief and supportive care. The dog regained consciousness within 2 days and was able to walk with assistance within 2 weeks.

Follow-up

At 3 months after the coma had resolved, the dog had a generalized tonic-clonic seizure. The dog was normal on neurological examination. MRI of the brain was normal. CSF was also normal. Further seizures of a similar nature occurred infrequently (once or twice a year). No anticonvulsants were used.

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6 Metabolic encephalopathy: insulinoma

INITIAL PRESENTATION

Altered behaviour (agitation or lethargy), decreased mental alertness, seizures, exercise intolerance, weakness or collapse commonly occur. Stupor and coma may result.

INTRODUCTION

Cerebral dysfunction appears acutely, episodically, or as an insidiously worsening process depending on the underlying disease, its speed of onset and the duration and severity of the metabolic derangement. Signs may be triggered by additional stresses such as eating, exercise, excitement, drug therapy, surgery and concurrent disease.

Cases of metabolic coma typically have intact and symmetrical PLR and VOR reflexes. Respiration may have altered depth or rate but is regular, in comparison to structural diseases causing coma.

Distinct areas of the CNS may be preferentially susceptible, producing focal signs in a diffuse disease.

Mixed metabolic derangements occur, with no part seemingly severe enough to produce the clinical signs. Mild changes can have an additive effect on cerebral function. Each must be addressed.

Cardiac dysfunction secondary to severe electrolyte disturbances and acute metabolic acidosis may be rapidly lethal.

SIGNALMENT

A 9-year-old neutered female boxer dog.

CASE PRESENTING SIGNS

Episodic digging behaviour, twitching and vocalizing.

CASE HISTORY

A description of the animal's activity and responsiveness is vital. When and how often an abnormal event occurs and how long it lasts is useful for diagnosis and also for monitoring progression of disease and the success of future treatment.

The dog was presented for assessment of a 6-month history of episodic events which commenced with digging activity indoors and hiding in corners. Twitching of the face and body, vocalizing, defecating, and running about the house then followed. The dog was mentally unresponsive during the event and if restrained, its limbs were felt to flex and extend. The events lasted up to 2 hours and occurred at any time of day, with weeks or months between the events. The dog was described as being otherwise normal.

CLINICAL EXAMINATION

Neurological examination of this case was normal. No video of the event was available.

NEUROANATOMIC LOCALIZATION

The lesion was localized to the cerebrum due to the behaviour change. Episodic changes can occur with a persistent structural lesion of the brain or an intermittent metabolic defect. Episodic behaviour change also occurs with psychological conditions in small animals. The

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normal approach is to rule out organic CNS disease before diagnosing psychological causes.

In all three causes of episodic behaviour change, the animal may appear normal between the episodes. Structural or metabolic lesions can cause subtle abnormalities (e.g. change in house training, change in personality or sleep patterns) between the flamboyantly abnormal events, so it is vital to thoroughly quiz the owner.

An unresponsive mental state can also occur when an animal is agitated or distracted by severe pain, or an acute balance loss, or intense pruritus: the owner tries to distract the animal but the animal pays no attention to the owner. If physical examination of the animal fails to find any other abnormality, then the unresponsive mental state probably results from cerebral dysfunction of some kind.

Some aspects of this patient's episodes were suggestive of seizure activity; the localization would be the same.

DIFFERENTIAL DIAGNOSIS

Intracranial pathology or extracranial systemic disease was suspected in this case.

CASE WORK-UP

Blood glucose was measured on an in-house system and found to be low (2.3 mmol/l; range 2.0–5.5). It was rechecked with a commercial lab and found to be 1.9 mmol/l. Concomitant serum insulin level was high at 85 µIU/ml (5.0–20.0).

Causes of hypoglycaemia

- Excess insulin; endogenous, exogenous
- Liver insufficiency
- Glycogen storage diseases
- Neonates
- Hunting dogs, toy breeds
- Neoplasia
- Hypoadrenocorticism
- Sepsis.

Hypoglycaemia often presents with intermittent neurological deficits. Seizures are usually self-limiting as catecholamine secretion raises the blood glucose level. Behaviour change can go on for hours. Eating can both raise blood glucose levels and stimulate insulin secretion, either improving or triggering signs. Non-specific restlessness, anxiety, shaking, trembling, twitching and hunger are a result of excess sympathetic stimulation. Collapse, weakness, exercise intolerance, ataxia, an obtunded or stuporous state, and transient blindness are also reported. A peripheral neuropathy occurs secondary to insulin-secreting tumours in dogs.

Table 6.1 Blood glucose values

Normal	Abnormal	Clinical signs
Dog: 2.9–6.5 mmol/l Cat: 3.1–7.2 mmol/l	3.3–3.8 mmol/l	<2.5 mmol/l

DIAGNOSIS

Fasting hypoglycaemia or hypoglycaemia with neurological signs confirms the diagnosis. Repeated testing is required in some cases. Serum fructosamine levels assess glycaemic control during the previous 2–3 weeks. A below-normal level or one in the lower half of the normal range (e.g. <300 µmol/l) suggests periods of hypoglycaemia. Insulin levels should be measured when hypoglycaemia is present. Less than 20% of dogs with hypoadrenocorticism have hypoglycaemia (Table 6.1).

CLINICAL TIP

Hypoglycaemia found in a seizuring animal is the cause of the seizures until proven otherwise.

An insulinoma was diagnosed. Abdominal ultrasound did not reveal a pancreatic mass.

Exploratory laparotomy found small lesions within the liver and a solitary mass in the pancreas. Histopathology confirmed the diagnosis of an insulin-secreting beta cell tumour.

PROGNOSIS

Insulinomas commonly metastasize to the liver, mesentery, omentum and lymph nodes. Debulking the tumour improves the control of hypoglycaemia and survival time. Survival time postoperatively ranged from 0–5 years. Pancreatitis and diabetes mellitus are postoperative complications.

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TREATMENT

1. 0.5 g/kg dextrose IV = 1 ml/kg 50% dextrose. Dilute in saline 1:2 to avoid phlebitis. Give slowly. Improvement occurs within a few minutes.
2. Unresponsive neurological signs due to cytotoxic oedema and cell death: treat raised ICP (mannitol, furosemide), maintain normoglycaemia, control seizures. When hypoglycaemia is profound or prolonged, the signs may not reverse immediately after glucose injections despite normoglycaemia being reinstated.
3. Feed 3–6 times a day.
4. Prednisolone 0.25 mg/kg PO q12 hr, increase if required to control the clinical signs.
5. Diazoxide 5 mg/kg q12 hr. Dose adjusted to control clinical signs.
6. Glucagon 50 ng/kg IV cat/dog. CRI: 5–10 ng/kg/min IV, dose adjusted to maintain euglycaemia.
7. Octreotide 10–40 µg/dog SC q8–12 hr. Used with insulin-secreting tumours.

   The dog was treated with IV dextrose but remained hypoglycaemic. Prednisolone and divided feeds were commenced.

FOLLOW-UP

Clinical signs recommenced a few months post-surgery and resolved when the dose of prednisolone was increased. The animal was later euthanized at the owner's request following its first generalized seizure.

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7 Metabolic encephalopathy: hepatic encephalopathy

Ed Friend and Susan Fitzmaurice

INTRODUCTION

Severely reduced liver function is required to produce hepatic encephalopathy. This is most commonly due to vascular shunting. Low blood glucose levels may contribute to the cerebral malfunction caused by an array of substances which include ammonia.

Only two-thirds of canine congenital portosystemic shunts occur in small breeds such as Yorkshire terriers, Maltese terriers, poodles and miniature schnauzers. Domestic shorthair cats represent the majority of feline cases.

CASE HISTORY

Cerebral dysfunction occurs in approximately 95% of cases with signs generally commencing by 2 years of age. Intermittent episodes of abnormal behaviour (aimless wandering, head pressing, circling or a change in personality), ataxia, blindness and seizures may be triggered by gastrointestinal bleeding, infections, hypokalaemia, constipation and high protein diets. Improvement may be seen after antibiotic therapy. The animal may be slow to recover from a general anaesthetic. Seizures with a normal inter-ictal period may be the only abnormality.

CLINICAL EXAMINATION

Neurological deficits are usually symmetrical. Non-neurological signs include stunted growth, anorexia, depression, intermittent vomiting, diarrhoea and polydipsia. Inappropriately golden or copper-coloured irises have been noted in both the dog and the cat. Intermittent ptyalism occurs in 75% of affected cats, possibly related to either nausea or seizure activity.

CASE WORK-UP

Diagnostic efforts are aimed at demonstrating decreased liver function, and the presence of anomalous vascular communications. Urine analysis is commonly overlooked as a diagnostic tool but should be performed, as ammonium biurate crystals can be found in both species. The diagnostic yield from ultrasound is determined by the operator skill. Portography and transcolonic scintigraphy are also used. Microvascular dysplasia of the liver has a similar clinical presentation and requires liver biopsy for diagnosis. Inborn errors of metabolism cause hyperammonaemia without evidence of hepatic dysfunction.

DIFFERENTIAL DIAGNOSIS

- Congenital portosystemic shunt
- Severe primary hepatobiliary disease
- Congenital urea cycle deficiencies
- Organic acidaemias.

PROGNOSIS

Seizures may persist even after surgical ligation of the shunt.

TREATMENT

There is little correlation between blood ammonia levels and the clinical signs but treatment aims to reduce the formation and uptake of this metabolite and other encephalotoxins from the gut.

1. Antibiotics: neomycin, ampicillin, metronidazole.
2. Lactulose: Cat 0.5–5 ml PO q8–12 hr; Dog 5–25 ml PO q8–12 hr. Adjust the dose so as to produce soft stool while avoiding outright diarrhoea.
3. High-quality protein diet.
4. Treat/avoid GI ulceration, azotaemia, constipation, infection.
5. Seizures: propofol, diazepam (lower dose required), potassium bromide.

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8 Metabolic encephalopathy: osmolality

INITIAL PRESENTATION

Anorexia, lethargy, vomiting, ataxia, muscle weakness, tremors, altered mental state and behaviour, seizures and coma.

INTRODUCTION

Osmolality is the number of solute particles in a kilogram (kg) of solvent. Serum osmolality is chiefly determined by the sodium concentration as seen in the following equation:

$$\text{Serum Osm} = 2(\text{Na} + \text{K}) + \text{glucose}/18 + \text{BUN}/2.8$$

An estimate can be made by doubling the serum Na^+ and adding 10, if glucose and BUN are normal. Serum osmolality is calculated by the formula above or by freezing point depression. A difference between the two values indicates the presence of other osmotically active particles, e.g. ethylene glycol, mannitol. Decreasing the colloid oncotic pressure has minimal effect on brain water content in the normal brain.

Normal serum osmolality; cat/dog: 280–305 mOsm/kg.

In experimental animals, a reduction in plasma osmolality of as little as 5% under otherwise normal circumstances causes brain oedema and increases intracranial pressure (ICP).

Less than 260 or more than 330 mEq/l are likely to produce cerebral dysfunction but it is the speed of change in osmolality that is most likely to provoke neurological signs.

- Causes of hyper-osmolality: hypernatraemia, severe hyperglycaemia
- Causes of hypo-osmolality: hyponatraemia/water intoxication
- Clinical signs most commonly occur with rapid changes in osmolality.

Normal serum sodium: 140–150 mEq/l (dog), 150–160 mEq/l (cat). Lethargy commences with Na >170 mEq/l, or total osmolalities of ≥350 mOsm/kg. Signs of hyper-osmolality may be intermittent, worsening during periods of fluid loss from vomiting and diarrhoea. Osmotic dehydration and shrinkage of the brain may tear meningeal blood vessels.

Hypernatraemia is associated with:

Acute: Fluid loss via the GIT, burns, third space accumulation, renal failure or diuresis, impaired thirst or access to water with or without diabetes insipidus or salt poisoning (2–3 g/kg body weight).

Mild chronic: Chronic, untreated diabetes insipidus.

Severe chronic: Essential hypernatraemia caused by a diencephalic abnormality characterized by a lack of thirst and failure of ADH secretion in response to osmoreceptor stimulation.

Hyponatremia is caused by:

Acute: Sudden hypotonic water load, decreased renal capacity to excrete water, excessive/acute inappropriate ADH activity, psychogenic polydipsia or acute CNS disease (cerebral salt-wasting syndrome). It is uncommon in veterinary practice.

Chronic: Renal disease, decreased blood volume, enhanced ADH output (hypothalamic lesions, head trauma, meningitis, hypothyroidism or hypoadrenocorticism).

Signs occur with a sodium <120 mEq/l, and are highly likely to be present if sodium <115 mEq/l. Presentation

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is similar to hypernatraemia but also includes weight gain (fluid retention) and dyspnoea.

SIGNALMENT

A 10-month-old female Staffordshire bull terrier.

CASE PRESENTING SIGNS

Intermittent ataxia and collapse.

CASE HISTORY

This young dog had intermittent lethargy and persistent inappetance since she was acquired at 7 weeks of age. The thirst was highly variable, being alternately absent or excessive. A head tremor and collapsing on the forelimbs had first been seen 1 week prior to referral.

CLINICAL EXAMINATION

The dog was underweight with dry oral mucous membranes. She was quiet but mentally responsive. Neurological examination was normal.

NEUROANATOMICAL LOCALIZATION

A reasonable assumption in this case, based on the signs commencing and occurring simultaneously, is that the head tremor and forelimb collapse have the same cause. Neither sign is pathognomonic for a particular area of the nervous system, so a list of possible sites should be considered: weakness from LMN or UMN origin; cerebellar disease; cervical cord disease with neck spasm and UMN weakness worse in the forelimbs or multifocal lesions.

L-2-hydroxyglutaric aciduria (L-2-HGA), an inherited error of metabolism, has been reported in Staffordshire bull terriers aged between 6 months and 7 years. Seizures, ataxia, altered behaviour (dementia) and tremors occur.

Thirst is stimulated by an increased plasma osmolality acting on osmoreceptors found in the anterior hypothalamus. The same or different osmoreceptors in the anterior hypothalamus stimulate vasopressin (ADH, antidiuretic hormone) release from the posterior pituitary. Vasopressin increases permeability of the renal collecting ducts to water thus retaining water and decreasing plasma osmolality. Dehydrated animals stop drinking before the plasma hypertonicity is resolved, indicating some form of pharyngeal/gastrointestinal feedback system.

Hypovolaemia increases water intake by two methods unrelated to any change in plasma tonicity. Decreased ECF increases renin secretion and thus levels of circulating angiotensin II which act on brain receptors to stimulate thirst. Baroreceptors in the heart and blood vessels also play a role.

Lesions of the osmoreceptors in the hypothalamus stop the signal to drink, causing adipsia. Hypodipsic hypernatraemia has been described in cats and dogs. The animals are disinterested in drinking despite high plasma sodium levels.

Inappetance is a non-specific sign, meaning that it accompanies pathology of many different organ systems. It signals that the animal is not normal, but the history and examination should be used to find other signs which can narrow down the diagnostic field and enable the body systems to be listed in order of the most likely affected.

DIFFERENTIAL DIAGNOSIS

Causes of the adipsia, inappetance, lethargy, and episodic neurological signs of CNS or muscular origin:

- Hypothalamic lesion: hydrocephalus or other brain malformation, encephalitis with secondary hypernatraemia
- Hypothyroidism
- Hypoadrenocorticism
- Hepatic encephalopathy.

CASE WORK-UP

Hypernatraemia 198 mmol/l (reference range: 144–160), hyperchloraemia, azotaemia, urine specific gravity 1.048 were found.

Causes of hypernatraemia which were ruled out by the preliminary lab work and history include: gastrointestinal fluid loss, diabetes insipidus, renal failure and salt toxicity.

Sodium retention caused by excessive levels of aldosterone from an aldosterone-secreting adrenal tumour or bilateral adrenal hyperplasia was assessed by serum aldosterone concentration and abdominal ultrasound. Both were normal.

ADH serum levels were not assessed as the urine specific gravity was >1.030.

ACTH stimulation test was normal.

MRI of the brain found a congenital cerebral malformation of the corpus callosum and lateral ventricles.

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DIAGNOSIS

Essential hypernatraemia secondary to a brain deformity affecting the hypothalamic osmoreceptors.

PROGNOSIS

The prognosis depends on the ability to control the hyperosmolality. Adding water to the food intake has been successful.

TREATMENT OF HYPERNATRAEMIA

1. For hypernatraemia (Na^+ ≥170 mEqL) with normal perfusion and hydration, replace the solute free water using maintenance crystalloids (0.9% NaCl).
2. Free water deficit (L) = [(measured Na/desired level of Na) – 1] × body weight (kg) × 0.6.
3. Replace free water deficit over 24–48 hours. Lower 0.5–1 mOsm/kg/hr (or by 2 mEq sodium/l/hr in acute salt intoxication). The speed of correction should mirror the speed of development.
4. Add volume for metabolism and insensible losses: 2 ml/kg/hr. Dose increase by 1–2 ml/kg/hr if fever and hyperpnea are present. Rapid replacement can cause cerebral oedema.
5. Loop diuretics, e.g. furosemide, increase renal sodium excretion.
6. Watch for fluid overload and pulmonary oedema.
7. Add maintenance fluid ration to canned food.

This dog was treated by adding water to the daily diet and dividing feeding into four-times daily. The sodium level returned to normal.

FOLLOW-UP

Appetite and energy level increased. Neurological signs did not recur.

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9 Granulomatous meningoencephalitis

INITIAL PRESENTATION

Staring into space while lying on the sofa.

INTRODUCTION

Looking in one direction for an extended period of time is often a sign of cerebral disease. Staring at walls, into corners, or 'into space' are common presenting signs and distracting the animal may or may not be successful at stopping it. Neck pain limits head movement and general activity but such animals move their eyes towards stimuli and are mentally responsive. Recently-blind animals move slowly, bump into objects and do not respond to visual clues; clinical diagnosis is straightforward.

SIGNALMENT

An 11-year-old male labrador.

CASE PRESENTING SIGNS

Episodes of staring.

CASE HISTORY

Hour-long episodes of being mentally unresponsive while lying on the sofa and staring at the wall commenced 6 months prior to referral. The events occurred at least once a week at any time of day and only when indoors. The dog was normal between the events. No other abnormalities were reported.

CLINICAL EXAMINATION

The dog was neurologically normal.

NEUROANATOMICAL LOCALIZATION

Cerebrum, due to the behaviour change, which may have been partial seizure activity.

It may not have been neurological: the dog may have had undiagnosed arthropathy causing a reluctance to move but this was not evident in the history or physical examination. An age-related hearing loss may not have been noticed previously and have contributed to the dog's lack of response to being called. A trial of non-steroidal anti-inflammatory drugs (NSAIDs) had been used but this had had no effect on the frequency or duration of the events and had been discontinued.

DIFFERENTIAL DIAGNOSIS

Metabolic encephalopathy: intermittent loss of consciousness may be from hypoglycaemia which can be difficult to diagnose from one random blood sampling.

Liver failure, but there was no history or physical evidence of this and so was it discounted.

Seizure activity.

Increased intracranial pressure from neoplasia.

Cognitive dysfunction syndrome, a progressive, incurable loss of normal behaviour in the absence of any

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other known cause, was a consideration in this aged dog. Owners often describe their pet as 'senile'. Systemic or primary neurological diseases must be ruled out first before diagnosing this condition.

Presenting signs mimic those of cerebral disease or ageing change (deafness, reduced mobility):

- Inattentive, becoming lost in familiar environments, failure to recognize familiar people/animals
- Increased irritability/aggression/agitation/anxiety
- Inactive, aimless wandering, pacing at night, vocalizing at night
- Disturbance of sleep/wake cycle
- Loss of house-training.

CASE WORK-UP

Blood glucose and fructosamine levels were repeated and were normal.

MRI of the brain was normal.

CSF was abnormal with elevated WBC count and total protein: RBC = 1; WBC = 585; TP = 0.77 g/l. Cytology showed a high nucleated cell density comprising 93% neutrophils (mostly non-degenerate), 6% small lymphocytes; 1% macrophages/monocytes.

DIAGNOSIS

Neutrophilic pleocytosis accompanies tissue destruction within the CNS. It is not pathognomonic of infection. The long duration of signs with corresponding normal interictal period is highly unlikely to occur with bacterial CNS infection. MRI result ruled out neoplasia of the brain which, given the animal's age, was a consideration. Non-infectious inflammation or degeneration of the brain was considered. A late-onset storage disease was possible.

Normal MRI is possible with CNS inflammation. Granulomatous meningoencephalomyelitis (GME) has a highly variable appearance. An atypical form of GME was considered.

PROGNOSIS

Uncertain, however lack of progression of signs and normality between the events suggested it was good.

TREATMENT

Treatment options for non-infectious inflammatory CNS disease

Prednisolone 1–2 mg/kg PO q12 hr. Try to taper the dose after a period of normality or if iatrogenic hyperadrenocorticism develops.

Add-on therapy potentially allows for a reduction in dose or elimination of prednisolone. Myelosuppression by these drugs is a common side-effect:

1. Azathioprine
2. Radiation therapy
3. Procarbazine 25–50 mg/m^2 PO q24 hr
4. Cytosine arabinoside 50 mg/m^2 SQ q12 hr for 2 consecutive days. Repeat every 3 weeks
5. Cyclosporine:
   - 6 mg/kg PO q12 hr
   - 10 mg/kg PO SID q24 hr for 6 weeks then decrease to 5 mg/kg SID
6. Lomustine (CCNU)
   - 44–88 mg/m2 PO q6 weeks
   - 60 mg/m2 PO q6 weeks
7. Mycophenolate mofetil 20 mg/kg/day divided q8 hr or q12 hr.

In this case, prednisolone was commenced and no events were noted in the following 2 weeks.

Neurological examination remained normal. The dose of prednisolone was gradually reduced and stopped after 6 weeks of treatment.

Five months later, the dog became restless and vocalized at night when on the sofa. Selegiline and dietary antioxidants were used in case brain ageing change had produced a cognitive disorder. Treatment with selegiline improves cognitive function and slows progression of signs in dogs and cats with a clinical effect noted in most within one month of treatment. Food containing increased antioxidants and mitochondrial co-factors improves cognition in clinical studies.

FOLLOW-UP

The dog was euthanized as the owner could no longer cope with the disruption. Postmortem examination found granulomatous meningoencephalitis (GME).

Non-infectious inflammatory encephalitis

The vast majority of CNS inflammation is currently idiopathic, which pet owners can find extremely difficult to accept. Treatment is empirical and the prognosis is guarded but treatment should always be attempted. Not all dogs fall into the following categories. Resist the temptation to label everything GME (Table 9.1).

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Table 9.1 Differentiating non-infectious inflammatory meningoencephalitis

	NME	NE	GME
Age	6 months–7 years (juvenile or young)	1–10 years old	Any age. Often young–middle aged
Location	Cerebral hemispheres	Brainstem + cerebrum	Brain, spinal cord, optic nerve
Common signs	Cerebral: seizures etc., neck pain	Brainstem: central vestibular, seizures etc., neck pain	Caudal brainstem, cervical cord, (cerebrum); neck pain, cerebellovestibular (seizures)
Progression	Acute: <2 weeks Chronic: 4–6 months	Chronic; months	Multifocal: acute and rapid Focal: chronic and slow
CSF	Lymphocytic pleocytosis	Mononuclear pleocytosis (lymphocytes + monocytes)	Mononuclear pleocytosis (lymphocytes, plasma cells, fewer monocytes)
Pathology	Acute encephalitis or chronic cerebrocortical necrosis and inflammation. Grey and white Leptomeninges.	Necrosis of cerebral white matter Cortex and meninges are not involved	Angiocentric granulomatous inflammation of white matter Leptomeningeal involvement
Prognosis	Poor: <6 months Little response to prednisolone	Poor	Variable: poor to good

NME, necrotizing meningoencephalitis; NE, necrotizing encephalitis; GME, granulomatous meningoencephalitis.

NE: necrotizing encephalitis

- Yorkshire terrier, French bull dog, Alaskan huskies.

Subacute necrotizing encephalopathy in two young female Yorkshire terriers was recently found associated with a mitochondrial respiratory chain complex I and IV defect.

NME: necrotizing meningoencephalitis

- Pug, Maltese, chihuahua, Pekingese, Boston terrier, shih tzu, Coton du Tulear, papillon.

GME: granulomatous meningoencephalitis

- Any breed, often small, but not exclusively.

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SECTION 3

BEHAVIOUR CHANGE

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10 Behaviour change – an introduction

Consciousness = alertness + awareness.

- Alertness (the level of consciousness) is judged by reflex and behavioural responsiveness to stimuli and is governed by the ARAS and the cerebrum. Psychological change can alter responsiveness but does not produce states resembling stupor and coma.
- Awareness (the quality or 'content' of consciousness) is judged by behaviour. Behaviour is controlled by the cerebrum. Organic neurological disease and psychological factors can both cause a change in behaviour.

The goal when investigating a case of abnormal behaviour is to rule out organic neurological causes, e.g. metabolic encephalopathy, brain tumour, pain, before concluding that the behaviour change is psychological in nature.

In veterinary medicine, consciousness is generally regarded as a synonym for an alert mental state without taking into account what the animal may or may not be able to perceive, interpret or distinguish. If describing an altered consciousness (alertness or awareness), describe what is abnormal about the animal.

Normal behaviour is assessed against that occurring in others of the same species, breed and age group (Fig. 10.1). Consulting relevant textbooks is recommended.

Avoid anthropomorphism: withdrawing the upper lips horizontally to show canines and incisors has been given the description of *smiling or grinning*. It is done, often by Dalmatians, in response to a command or interaction with humans. It is described as a passive defensive behaviour or an anxiety-induced behaviour.

SLEEP BEHAVIOUR

Sleep consists of cycles of rapid eye movement (REM) and non-rapid eye movement (NREM) phases (Table 10.1).

Seizures can be triggered in slow wave sleep.

Parasomnias are defined as intrusions of abnormal movement or behaviour into the sleep pattern. Intermittent paddling of limbs and lifting the head, with or without urination can occur in REM (also known as REM sleep behaviour disorder) and can easily be stopped by waking the animal. It could be mistaken for seizure activity.

A sudden attack provoked by waking or disturbing the dog during sleep is thought to be a manifestation of territorial aggression or vulnerability and has been termed *sleep-associated aggression*.

Sudden shock-like myoclonic jerks of limbs or body when falling asleep are called *hypnic jerks* or *sleep starts*. It occurs to an exaggerated degree in miniature wire-haired dachshunds with Lafora's disease (familial myoclonic epilepsy).

Snoring is an indication to check for narrowed upper airways, e.g. polyps, long soft palate.

Compulsive repetitive yawning (chasm) as a sign of CNS disease, e.g. seizures, encephalitis, tumours of the fourth ventricle and frontal lobes, has not been reported in animals. There is reference to frequent yawning as a sign of otitis-media-interna but other deficits from pathology in this location are far more common. Perhaps head shaking and yawning in otitis media is a method for clearing a blocked eustachian tube.

Hypersomnia is the excessive but normal-appearing sleep at inappropriate moments, e.g. during meals, from which the patient awakens when stimulated.

Bulldogs with sleep apnoea exhibit hypersomnia but are presented for treatment of upper airway obstruction. Animals with narcolepsy generally also have cataplexy which causes the most obvious sign: episodic collapse.

Fever, systemic illness or cerebral disease can increase sleep duration and frequency. Pain, weakness and exercise intolerance cause inactivity.

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Lesions of the ascending reticular activating system and its projections to the thalamus can cause excessive daytime sleepiness.

Narcolepsy comprises hypersomnia and cataplexy, resulting in intermittent collapse and sleep.

It occurs sporadically in many dog breeds and is inherited in some: labradors, doberman pinschers, poodles and dachshunds. It is rarely reported in cats. Episodes are induced by exercise, eating and excitement and are reversed by stimulating the animal, e.g. patting or name-calling.

The cataplexy, the sudden atonia of limb muscles causing collapse, or a drop attack, lasts seconds or minutes and is the most obvious clinical sign of narcolepsy. Cataplexy alone does not affect consciousness.

Narcolepsy can accompany neurological disorders such as head trauma, tumours, and encephalopathies in humans and so affected animals should be appropriately investigated by a neurologist before assumptions of inheritance are made.

Insomnia is an inadequate amount of sleep or impaired quality of sleep. Transient causes in humans include a change in sleep environment, uncomfortable room temperature, stressful events, acute medical or surgical disease, stimulants or removal of CNS depressant medication. Elderly dogs pacing throughout the night may have cognitive dysfunction syndrome but other causes of restlessness should be considered. Hospitalized dogs can sleep an excessive amount when first discharged home.



Figure 10.1 Most domestic shorthair cats do not jump through hoops, lit or unlit.

CLINICAL TIP

Old age is not a disease.

INGESTION BEHAVIOUR

Pica is the ingestion, chewing or licking of non-food items such as metal, dirt, clay, sticks, rocks or fabric. The animal is intensely focused on and selective of the ingested item, which is described as a morbid craving in humans. It may be a form of obsessive-compulsive disorder (OCD). It is common in iron deficiency in humans. *Coprophagia*, the ingestion of faeces, is normal maternal behaviour. It is not known why the majority of dogs do this. *Eating grass* or vegetation is common in cats and dogs. Vomiting usually follows but subsequent appetite is rarely impaired. Animals are not known for their skills in botany and do not discriminate between toxic and non-toxic plants. Chewing houseplants, many of which

Table 10.1 Phases of sleep

Phase	Motor	Autonomic	Duration
NREM: Initial	Assumes rest position. Closes eyes. Sudden twitches, hypnic jerks	HR, RR, BP decrease Pupils constrict	A few minutes
NREM: Slow wave	Limb movement, parasomnias	HR, RR, BP decrease Pupils constrict	30 minutes
REM	Atonia. Twitching of face, paws, tail	Irregular respiration HR and BP increase with movement	10–15 minutes

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are toxic, may be attention-seeking behaviour. Dogs with portosystemic shunts may have a *dietary preference* for fruit and vegetables. *Fabric chewing/eating or wool sucking* cats are usually either Siamese or Burmese and often have been weaned early at 2–4 weeks. There may be fabric preferences which may include plastic. Most cats begin with wool sucking before progressing to other materials. This may be an OCD. *Blanket or flank sucking* occurs in doberman pinschers.

Polyphagia is the excessive ingestion of food compared to normal caloric requirements. An overly simplistic view of appetite control is that the feeding centre in the hypothalamus is constantly active unless switched off by the satiety centre. The feeding centre can be stimulated directly (primary) or more commonly, secondarily to a non-neurological cause.

Primary

- Psychogenic
- Labradors and Scottish terriers are over-represented in the over-consumers
- Reported in some cases of encephalitis/encephalopathy
- Destruction of the satiety centre in the ventromedial hypothalamus
- Stimulation of the feeding centre in the lateral hypothalamus.

Secondary

- Disease states: e.g. diabetes mellitus, hyperthyroidism, hyperadrenocorticism, exocrine pancreatic insufficiency, acromegaly, insulin-secreting tumour
- Drug-induced: corticosteroids, benzodiazepines, phenobarbitone, potassium bromide, megestrol acetate or cyproheptadine
- Inadequate caloric intake
- Increased metabolic rate: pregnancy, lactation or increased exercise.

CLINICAL TIP

Animals may drink or eat ravenously immediately after a seizure. This is not classed as polyphagia.

Animals with structural forebrain disease may *only eat from one half of the bowl*. This is a manifestation of *hemi-inattention or hemi-neglect* in which the animal ignores the side contralateral to the lesion. Most sensory information decussates to the contralateral cerebral hemisphere where it is interpreted. A lesion interrupting this system of perception results in neglect of the contralateral hemispace. Affected animals are usually slightly to moderately obtunded and have a tendency to turn in one direction rather than the other. Use of the expression *neglect* in this sense is reserved for a failure to orientate to a meaningful stimulus in the absence of any sensory or motor deficit (e.g. blindness, deafness or paralysis), which would prevent the animal responding.

Absence of appetite, *anorexia*, can be distinguished from reluctance to eat by history taking and observation. The former is usually a sign of systemic illness although some dogs will miss the occasional meal without any sign of illness. Patients hand-fed while hospitalized may reject normal rations when first discharged home.

Interest in food with a hesitation or *reluctance to eat* may be due to oral pain or pain on opening the mouth. Group dynamics between the household animals may induce fear or subordination and refusal to eat.

Prehension difficulties (oral pain, pain on opening the mouth, jaw weakness or tongue weakness) may *slow eating* and be erroneously interpreted by the owner as a reduced appetite. Prehension difficulties can also lead to messy eating, i.e. dropping food, scattering food over a greater area than normal, dropping water back into the bowl creating a frothy opaque food-flecked fluid.

Psychogenic polydipsia, excessive water consumption driven by psychological causes, is rare. It results in low–normal or decreased serum osmolality.

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11 Loss of house-training

INITIAL PRESENTATION

Behaviour change: loss of house-training.

INTRODUCTION

Occasional urinating or defecating in the house by the pet is often excused by the owner as 'having accidents'. If it breaks the life-long pattern of behaviour, it is abnormal. Loss of house-training is a common early sign of cerebral dysfunction. Medical causes such as reservoir (bladder or rectum) or sphincter incompetence and anxiety disorders should be ruled out.

SIGNALMENT

An 11-year-old male domestic shorthaired cat.

CASE PRESENTING SIGNS

Loss of house-training.

CASE HISTORY

The cat had been urinating and defecating in the house in different places for three months. Episodic periods of unresponsiveness, sitting quietly and staring into space, had occurred during the same period. No abnormalities were found on blood work. Thyroid function was normal. Blood pressure was normal.

CLINICAL EXAMINATION

The cat dashed out of the carrier and then sat, immobile, outwardly unresponsive to the environment. When coaxed, the cat walked normally but tended to circle to the right. Left-sided placing responses and hopping were slow. The menace response was absent in both eyes. PLR were normal. Pupil size was normal and so was fundic examination.

NEUROANATOMICAL DIAGNOSIS

Right cerebrum indicated by the behaviour change, obtunded state, circling to the right, left-sided postural deficits. The bilateral loss of the menace response could be ascribed to the dull mental state of the cat.

DIFFERENTIAL DIAGNOSIS

- Meningioma
- Other cerebral neoplasia.

The long time course and presentation as a behaviour change is quite typical of a slowly expanding intracranial lesion in a cat.

Urine found in house

Loss of consciousness with urination occurs more commonly with seizures than syncope. It also depends on bladder fullness at the time of the event. Finding puddles (or floods) of urine in the house caused by an otherwise house-trained dog may be the first sign of seizure activity noted by the owner. Loss of house-training with an apparently random pattern of urination is a common behaviour change induced by cerebral disease.

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Figure 11.1 Right-sided meningioma.

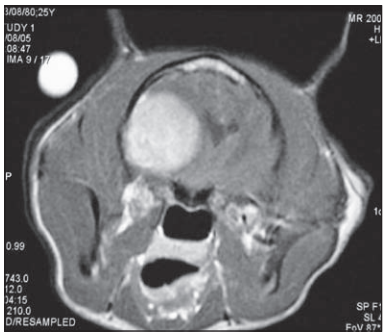


Figure 11.2 Note the shift of the falx cerebri to the left, and the compression of the lateral ventricles. The capsule on the right is used a marker.

CLINICAL TIP

Rather than ask if the pet is house-trained (the answer will be yes), ask if the pet has urinated in the house recently.

Weakness or pain may prevent the animal from going outside or to a litter tray to urinate. Cats may only be able to negotiate the cat flap in one direction.

Polydipsia/polyuria can result in the house-trained animal being unable to hold a large volume of urine for the usual length of time.

CASE WORK-UP

MRI: a large contrast-enhancing broad-based peripherally-attached mass on the right (Fig. 11.1).

DIAGNOSIS

Surgical removal of the mass confirmed the diagnosis of meningioma and resulted in a dramatic improvement in the cat's mental state which returned to normal. No seizures were noted postoperatively. The cat remains normal several years after the surgery (Fig. 11.2).

Brain tumours

What is wrong with my pet?

The presenting signs depend on the location of the lesion and the speed of onset. Gradually increasing intracranial pressure is accommodated by the collapse of the ventricles and compression of the blood vessels. This phase of expansion may be overlooked as signs can be vague: lethargy, depression. Owners ascribe them to the advancing age of their pet.

CLINICAL TIP

Old age is not a disease.

A sudden onset of signs is usually a result of secondary change such as oedema, haemorrhage, compression of the brainstem, or blockage of the ventricular system.

The presentation ranges from seizures to behaviour change to collapse, as most brain tumours occur in the cerebrum (telencephalon). Cats usually present with a behavioural change and altered alertness, whereas dogs most commonly present with seizures.

The inter-ictal phase may be normal. Questioning may reveal a loss of house-training or a subtle change in personality. Frontal lobe tumours often present as seizures with a normal inter-ictal phase: months can pass before an altered personality develops.

Intracranial neoplasia may present as neck pain. The associated lethargy/depression can be mistaken for that caused by severe pain.

The overall incidence of brain tumours in the canine population is quoted as 0.01% to 3%. To put that in context, the overall incidence of seizures, from any cause, in the dog population is about 3%. Every general practice sees a few new seizure cases a year, so it stands to reason each veterinary practice would also be seeing similar numbers of dogs with brain tumours. (The comparison of seizures to tumours is not to imply that

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the latter causes the former. It was made to give a sense of proportion to a clinical occurrence.) To re-use a bankrupt expression, brain tumours are 'not uncommon'.

... but he is so young!

Neoplasia is on the differential diagnosis list for any case of brain disease. The index of suspicion is increased in older dogs aged 9.4 years ±3.4 years and brachycephalic dogs over 2 years old. The young are not immune: primary brain tumours were reported in dogs as young as 3 months old. The age of onset was the same for primary and secondary canine brain tumours. Meningiomas are the most common brain tumour in cats, occurring in adults over 5 years old, sometimes at multiple intracranial sites.

... but he is so well!

Primary brain tumours are common. They rarely metastasize outside the CNS. Affected animals are generally well, although one study of primary canine brain tumours found 23% of affected dogs had evidence of unrelated malignant neoplasia elsewhere; 58% of dogs with a pituitary adenoma had extracranial neoplasia found on postmortem.

This means that:

1. Successful treatment of a brain tumour may be thwarted by disease elsewhere.
2. Tumours found at extracranial sites may not be related to the intracranial pathology.
3. Imaging of the thorax and abdomen should be performed prior to investing in specific treatment of a brain tumour. Unfortunately, thoracic radiographs underestimate the frequency of pulmonary metastases.

What could it be?

MRI had 100% success in detecting an intracranial mass in a study of canine brain tumours. CT was less sensitive, at 90%. Radiologists could predict the histological tumour type as their top differential on MRI images in only 71% of cases.

CSF may be normal (10%), show a mixed cell pleocytosis (58%) or albuminocytologic dissociation (30%) in canine primary brain tumours. Atypical cells in CSF have been found in CNS lymphoma cases. These figures indicate that:

1. The clinician thought the benefit of acquiring CSF was greater than the potential risk of herniation.
2. CSF results are rarely specific.
3. Neoplastic cells rarely appear in the CSF.

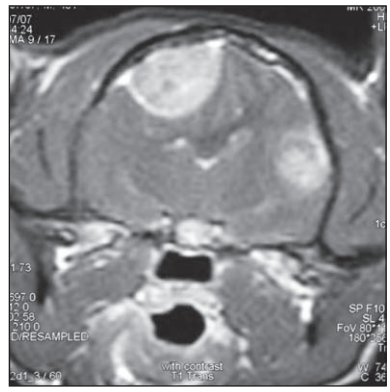


Figure 11.3 MRI of multiple meningiomas in an adult male DSH cat (T1WI + contrast).

Neoplasia may be focal or multifocal within the brain. Tumour type is suspected, based on species, breed, anatomic location, MRI appearance and (rarely) CSF (see Fig. 11.3 and Table 11.1). Histology is required for a definitive diagnosis.

How long has it been there?

Clinical signs may be present for 0–3 years prior to diagnosis. The median time was 26 days in dogs. The rate of onset was similar in primary and secondary brain tumours, although one source states that secondary tumours have a faster progression. The location of the tumour influences the rate of deterioration. Meningiomas grow slowly, which partially accounts for the long duration of clinical signs in affected cats prior to diagnosis (Fig. 11.4).

Is it malignant?

Malignant describes a worsening process that will lead to death. Brain tumours, regardless of their cytological characteristics, are malignant. Expansion of the tumour within the skull will ultimately cause fatal increases in ICP and death.

What are the treatment options?

Supportive

Tumours increase the extracellular fluid of the brain by several mechanisms.

1. Vasogenic. Structural injury and metabolic impairment of the capillary endothelium and neovascularization by vessels lacking tight junctions all increase blood–brain barrier permeability. The increased

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Table 11.1 Categorizing brain tumours

	Dog	Cat
Primary	Meningioma (45%) Astrocytoma (17%) Oligodendroglioma (14%) Choroid plexus tumours (7%) Primary CNS lymphoma (4%)	Meningioma: most common feline brain tumour Glioma: rare
Secondary: local extension	Tumours of the skull, extension from the nose, pituitary tumours, cranial nerve sheath tumour	Pituitary tumour, cranial nerve sheath tumour, tumours of surrounding tissue (sarcoma, carcinoma)
Secondary: distant spread	Haemangiosarcoma, lymphosarcoma, metastatic carcinoma (mammary gland, lung, anal sac, prostate, kidney, thyroid, bladder), malignant melanoma	Metastatic (mammary carcinoma, lymphosarcoma)



Figure 11.4 Slow growing tumours can reach a large size before producing clinical signs. Relative size of a feline meningioma to the skull.

interstitial fluid (oedema) formed spreads through the path of least resistance, the white matter. The formation and spread is directly influenced by the systemic arterial pressure.

Treatment: glucocorticoids, hyperosmolar fluids and surgical excision Avoid hypertension.

2. Compressive. Obstruction of the bulk flow of interstitial fluid into the ventricular system. This accounts for the effects of those tumours which do not alter the blood-brain barrier.

   Treatment: surgical excision.

3. Hydrocephalic. Obstruction of the ventricular system causes a retrograde increase in ventricular pressure. This removes the gradient down which the interstitial fluid flows and also produces periventricular oedema.

   Treatment: CSF shunting, decrease CSF production (prednisolone, hyperosmolar fluids, furosemide).

Anti-inflammatory doses of prednisolone 0.25–0.5 mg/kg PO BID are highly effective at reducing vasogenic oedema and hence ICP. Clinical improvement is noted within a few days.

It might be preferable to delay adding anticonvulsants until the mental state has improved after steroid treatment. The side-effects of sedation and ataxia may be mistaken for a worsening of the intracranial disease. If seizure frequency improves after steroid administration, it will be due to the reduction of oedema rather than a direct anticonvulsant action.

If the seizure frequency is such that anticonvulsant use is required, try starting the animal on a low phenobarbitone dose, increasing it over the following 2–3 weeks to the usual 2–3 mg/kg PO q12 hr.

Non-invasive supportive treatment of animals with brain tumours is limited to controlling oedema and seizures. There is little benefit to the animal in placing a ventricular shunt unless definitive treatment of the tumour is planned.

Life expectancy with supportive treatment alone is quoted as 1–4 months after diagnosis. Metastatic tumours and nasal tumours extending into the brain are only given supportive care.

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Table 11.2 Treatment of brain tumours

Tumour	Treatment	Longevity (months)
Meningioma: cat	Surgery	Months–years
Meningioma: dog	Surgery	Range: 0.5–22, Median: 7
	Radiation	Range: 5–12, Median: 5–9
	Surgery/radiation	Range: 3–58, Median: 16.5
Glioma: dog	Radiation	6
	Chemotherapy	7–11
Trigeminal neuroma: dog	Surgery (rarely reported)	Months
	Untreated	Range: 5–27
Pituitary tumour (canine)	Surgery (rarely attempted)	<40
	Radiation	Mean: 46
	Untreated	Mean: 18
Pituitary tumour (feline)	Radiation	Range: 8–20
Lymphoma	Chemotherapy	–
	Radiation	–
Skull multilobular osteochondrosarcoma	Surgery	Median: 26

Definitive

Surgical removal of feline meningiomas is possible and gives almost immediate clinical improvement, and an excellent postoperative quality of life. Longevity is measured in years.

Radiation therapy may induce late-onset brain necrosis, infarction, and leukoencephalopathy approximately 6 months after treatment. Dogs may be more likely to develop this than cats. See Table 11.2 for treatment of brain tumours.

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12 Defecation: faecal incontinence

INITIAL PRESENTATION

Appears aware but unable to control the urge; appears unaware and drops faeces when walking, lying down, in bed; has poor control of defecation when excited or during prolonged confinement.

CLINICAL TIP

Dogs with hindlimb weakness or lumbosacral joint (LSJ) pain may be unable to maintain the squatting position and often stand up and defecate while walking.

INTRODUCTION

Defecation reflex

- Filling of the rectum: the internal sphincter contracts pushing faeces back into the colon until the volume is sufficient. At this point, the internal anal sphincter (smooth muscle) relaxes and the external anal sphincter contracts. The external anal sphincter is composed of skeletal muscle innervated by the pudendal nerve formed from sacral segments and nerves S1–3.
- The stimulus for defecation is distention of the rectum.
- Awareness of rectal distension depends on the sacral afferents more than the cerebrum.
- Initiation of defecation by rectal distension activates parasympathetic efferents (pelvic nerve) that contract the colon and inhibit the external anal sphincter and pelvic muscles.
- Facilitation of defecation and expulsion is by the animal adopting the proper posture and the generation of increased abdominal pressure.

Prevention of defecation

1. Reflex by the internal anal sphincter contracts. This occurs, for example, in response to a sudden increase in abdominal pressure or increases in rectal filling.
2. Voluntary: mediated by higher centres (cerebrum).

Faecal incontinence

Faecal incontinence is the inability to control and retain faeces until voluntary conscious defecation is initiated.

SIGNALMENT

A 13-year-old male miniature wirehaired daschund.

CASE PRESENTING SIGNS

Faecal incontinence and urinating in spurts.

CASE HISTORY

A L1–2 IV disc extrusion had occurred 1 month earlier resulting in hindlimb paralysis, urine retention and lack of pain perception in both hindlimbs. Hemilaminectomy at the site decompressed the cord and voluntary movement of the tail had returned despite the paraplegia persisting. The owner reported that the dog urinated in spurts when taken outdoors and held in a standing position. On one occasion the dog pulled itself out of its bed to the backdoor where it had then urinated (which may or may not be a coincidence).

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CLINICAL EXAMINATION

Neurological exam found a quiet alert dog with voluntary tail movements (wagging) and paraplegia with a lack of pain perception in both hindlimbs. Muscle tone was present in both hindlimbs, as were the patellar reflexes and the withdrawal reflexes bilaterally. The cutaneous trunci reflex was normal bilaterally with no cut off. The bladder had some resistance to expression, which was easy to overcome. The bladder was as large as a ping-pong ball.

NEUROANATOMICAL LOCALIZATION

The lesion was localized to the T3–L3 cord.

The urinating in spurts and indoors may be due to:

1. Straining to urinate
   - Poor coordination between bladder contraction and sphincter relaxation creates a functional obstruction called reflex dyssynergia or detrusor-urethral dyssynergia. Urination has voluntary initiation but a stop–start spurting 'staccato' flow leaving a large residual volume which is difficult to manually express. Such poor coordination can result from a lesion anywhere rostral to the sacral cord. It is more common in large breed young adult male dogs.
   - Detrusor atony results from an overstretched bladder, sacral lesions or dysautonomia. Depending on the degree of bladder weakness, the animal may or may not be able to initiate urination. The urine stream produced can lack the normal force and speed of flow.
   - Increased sphincter tone or urethral spasm secondary to local inflammation, trauma, and pain.
2. Increased urgency to urinate
   - Detrusor hyperreflexia commonly occurs with inflammation of the bladder or urethra. The voiding reflex is triggered, despite the lack of bladder distension. This is sensed and responded to with urgency, hence the term 'urge incontinence'. The bladder is small or normal in size.

DIFFERENTIAL DIAGNOSIS

A combination of lower urinary tract infection secondary to urine retention, and reflex dyssynergia secondary to spinal cord injury from IVDD was suspected.

CASE WORK-UP

Urine culture and sensitivity from a cystocentesis sample was performed.

There was no indication to do an MRI of the surgical site at this stage. Recovery from paraplegia with lack of pain perception was always going to take weeks to months, if it occurred at all. Also, the dog had regained or seemed to have regained tail function and there was no spinal pain.

TREATMENT

Phenoxybenzamine and diazepam and frequent bladder expressions were recommended. Antibiotics were given to treat the urinary tract infection.

FOLLOW-UP

Four months later the dog re-presented for assessment of faecal incontinence. The dog was able to walk unaided and had voluntary control of tail movement and urination. Defecation occurred as the dog walked around. Urination control had returned to normal.

The neurological examination found a mentally alert ambulatory dog with hindlimb ataxia with intact spinal reflexes and intact hindlimb pain perception. Panniculus was also intact. Anal tone was present. The faecal matter looked normal.

Causes of faecal incontinence:

1. Reservoir failure
   - colorectal disease (inflammation, infection or neoplasia)
   - increased urge to defecate.
2. Sphincter failure
   - non-neurological: ano-rectal disease (trauma, neoplasia, fistula or surgery)
   - neurological
     - LMN: paresis or paralysis of the external anal sphincter. The external urethral sphincter is also innervated by the pudendal nerve so affected animals may also have urinary incontinence
     - Uncontrolled passive passing of faeces.
3. Failure of coordination (of filling reflexes)
   - seen in UMN spinal cord lesions (52% of dogs)
   - may be the presenting sign in animals with minimal neurological deficits (Grade 2) or as a sequela to severe (Grade 5) spinal cord injury

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- may wax and wane
- can defecate voluntarily when taken outside.

4. Loss of house-training: voluntary defecation in inappropriate places, e.g. behaviour change or cerebral disease.

In this case the faecal incontinence was thought to be a sequela to the previous spinal cord injury. Prognosis was guarded but the return of other neurological function gave hope that faecal control might return in time.

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13 Psychological

INTRODUCTION

High levels of activity are normal for certain breeds and ages of dog. It is often rewarded and reinforced inadvertently by owner attention. Hyperactivity is overdiagnosed in dogs. Most are overactive or under-exercised.

Anxiety can produce increased activity, pacing, vigilance, destructive behaviour, vocalization as well as panting, salivation, pupil dilation, drooling, shedding fur, urination and defecation. Signs of separation anxiety begin shortly after the owner's departure.

Low serum levels of phenobarbitone can cause hyperactivity. Hyperthyroid cats may appear more restless and aggressive.

Stereotypic, ritualistic, or obsessive-compulsive disorders (OCDs) are a select aspect of normal behaviour repetitively performed out of context and which replaces normal functioning during the episode. There may be a specific trigger initially but the condition progresses so that any state of stress, anxiety or high arousal will induce the stereotypic behaviour. The animal appears conscious but may not be responsive to the environment. The behaviour can be interrupted and no postictal phase of ataxia or depression follows.

Stereotypic movement is the most commonly reported category: tail-chasing, pacing, spinning/circling, fence-running, jumping in place, chasing light, freezing, staring and vocalizing and snapping at the air (fly catching/snapping; catching rain drops).

Over-grooming, limb or paw chewing, licking, flank-sucking and scratching may also be categorized as OCDs. Skin lesions and paresthesias should be ruled out.

Seizure activity is generally considered a prime differential diagnosis as the animal is often not responsive to the owner during the event. One dog was referred for poorly-controlled seizures which consisted of the dog sitting and spinning on its tail. This was neither a seizure nor an OCD: treatment of his dermatitis resolved the problem. Dogs with neck pain may episodically circle but should show signs of pain in the history or on examination, e.g. not shaking the head fully, root-signature lameness, etc. Nerve compression/inflammation/neoplasia can cause pain or a suspected paraesthesia giving episodic signs in a conscious animal.

SIGNALMENT

A 7-year-old neutered female Border collie.

CASE PRESENTING SIGNS

Pacing around the house, clawing at the carpet and staring, restless and circling.

CASE HISTORY

The dog began to pace soon after adoption from a rescue centre 1 year ago. She was aggressive if the owner tried to move her from the bed or furniture. She was taken to training classes, was provided with activity toys when left alone, and was given a lot of exercise on a lead. The aggression resolved and the pacing episodes lessened but continued and could last for hours. The pacing worsened in new environments. At 10 days prior to referral, the dog began circling, which was almost always to the right, and clawing at the carpets. This behaviour could last many hours during which nothing apparently could distract the dog. The dog was walked twice daily and was taken to training classes intermittently.

CLINICAL EXAMINATION

The dog endlessly paced around the perimeter of the examination room at a fast walking gait. She stopped, stared, and then resumed the pacing. She could be distracted from the behaviour. Neurological examination was otherwise normal.

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NEUROANATOMICAL LOCALIZATION

Behaviour is ultimately governed by the cerebrum. Lack of any definable organic cause along with the normal neurological examination and the history was suggestive of a psychological disturbance.

DIFFERENTIAL DIAGNOSIS

An anxiety response by the dog to a perceived stress.

CASE WORK-UP

Haematology and biochemistry were normal. MRI of the head was discussed but not performed. The pacing had started 1 year ago and had not changed, in essence, since it commenced. No other changes to the dog's ability had occurred. Behaviour change from organic intracranial disease worsens with time due to the increasing intracranial pressure or worsening parenchymal damage.

The animal was referred to a behaviourist for further assessment and treatment.

DIAGNOSIS

Anxiety disorder.

TREATMENT

The dog was rewarded for responding to commands, including recall so that she could be allowed off the lead. The owners were told to instruct the dog to settle if the pacing started. Clomipramine and a DAP (dog appeasing pheromones) collar were also used.

FOLLOW-UP

The dog did not improve.

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14 Intracranial arachnoid cyst

SIGNALMENT

A 6-month-old male Yorkshire terrier.

CASE PRESENTING SIGNS

Circling, lack of house-training.

CASE HISTORY

The pup had always circled a lot, especially when on the lead or when excited. He was not house-trained and bumped into furniture when running about the house but not when moving at a slower pace. The pup resented handling and would struggle to escape, and bite. Treatment with lactulose syrup and oral Synulox had not improved the clinical signs.

CLINICAL EXAMINATION

The pup was alert and appeared to be responsive to the environment and the owner. The dog circled to the left but did turn to the right on occasion. Slight hypermetria was seen in all limbs which was judged as normal for the breed. Hopping and placing were decreased on the right side. Cranial nerve examination found that the menace response was absent bilaterally and the symmetric pupils were small in ambient light but could dilate in darkness. PLR were normal. An open fontanelle could be felt.

NEUROANATOMICAL LOCALIZATION

The lesion was localized to the left cerebrum, due to the right postural deficits, the circling to the left, small pupils, lack of menace response, and lack of house-training.

DIFFERENTIAL DIAGNOSIS

- Cerebral malformation
- Encephalitis (distemper; necrotizing)
- Metabolic encephalopathy

CASE WORK-UP

Haematology and biochemistry were normal. Postprandial serum bile acid measurement is a sensitive detector of liver dysfunction and disruption of enterohepatic circulation from a portosystemic shunt. The bile acid stimulation test in this dog was normal. Lack of response to treatment for a suspected portosystemic shunt (PSS) helped to rule out this disease as a probable cause of the clinical signs. The neurological deficits on examination rule out a psychological cause for the circling. Cerebral malformation or inflammation is most likely at that age.

Encephalitis was a possibility. Canine distemper may cause neurological signs in the immunocompromised (e.g. young or unvaccinated) dog several weeks after infection. This acute CNS inflammation results from direct viral injury. Those able to contain the infection and avoid acute inflammation may go on to develop neurological signs later in life. A chronic encephalitis results and is caused by an inflammatory response and subsequent demyelination. Distemper may produce acute encephalitis 1–2 weeks after immunization, with a modified live virus (MLV) vaccine. Neurological signs are variable. Myoclonus is a common sequela.

Necrotizing encephalitis in the Yorkshire terrier typically appears between 1 and 10 years of age, resulting in cerebral and brainstem signs which worsen over several months. The signs in this case appeared to be static. The dog had been vaccinated and had not travelled outside the UK.

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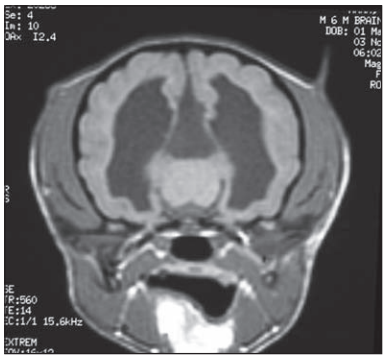


Figure 14.1 Transverse T1WI. CSF-filled triangular quadrigeminal cistern arachnoid cyst between the cerebral hemispheres.



Figure 14.2 Sagittal T2WI. Large CSF-filled structure dorsal to the cerebellum is the quadrigeminal cistern arachnoid cyst.

MRI of the head found a quadrigeminal cistern arachnoid cyst and enlargement of the lateral and third ventricles. The enlarged cistern may have compressed the midbrain aqueduct, causing secondary dilation of the third and lateral ventricles. CSF cell count, cytology and total protein level were normal.

DIAGNOSIS

Hydrocephalus. The quadrigeminal cistern arachnoid cyst could be an incidental finding.

Quadrigeminal cistern arachnoid cysts are rare but have been reported in small breed dogs presented for investigation of seizures or ataxia. A large infratentorial CSF-filled structure on the midline is visible on MRI caudal to the occipital lobes, dorsal to the midbrain and rostral to the cerebellum (Fig. 14.1). Intracystic haemorrhage may occur.

Compression of the occipital lobes by >14% or compression of both the occipital lobes and the cerebellum, as calculated from a mid-sagittal image, suggested that the cyst was more likely to be clinically relevant rather than an incidental finding (Fig. 14.2). CSF total protein levels were not increased in the cases of parenchymal compression by the cyst. Ventriculomegaly was an associated finding which may be due to a breed predisposition. Obstructive hydrocephalus is blamed for the development of clinical signs in humans with quadrigeminal cysts but this was not found to be the case in canine patients.

TREATMENT

The pup was commenced on 0.5 mg/kg prednisolone once daily for a fortnight but it made no difference to the dog's condition and the treatment was discontinued. The owner was happy with the dog's quality of life. Surgical drainage if behaviour or neurological deficits worsened was discussed, but was ruled out by the owner.

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15 Aggression

INTRODUCTION

Pain and psychological disturbances are more common causes of aggression than cerebral disease. Aggression from pain is either due to touching the painful area or the animal avoiding such contact. Treating the source of pain generally restores the animal's personality to normal.

Aggression from a behavioural, 'psychological' cause has many triggers, e.g. fear, anxiety, being challenged or threatened. Treatment is life-long.

There is no guarantee that treating a cerebral cause of aggression will reverse the clinical sign.

It is reasonably common for dogs to be presented for neurological assessment after biting all the family members, sometimes more than once, over a period of time. In many instances, the owner admits to wanting a guilt-free reason for euthanizing the pet and thinks that an MRI of the brain is the answer. Neurological and physical examinations and history typically fail to find any abnormality.

If the owner is willing to accept and manage the risks of aggression and will agree to treat either an underlying brain disease or a psychological disturbance then MRI would be of value to the dog as it would aid treatment choices.

SIGNALMENT

A 10-year-old neutered female labrador.

CASE PRESENTING SIGNS

Hindlimb ataxia. Behavioural change.

CASE HISTORY

There was a 2-month history of hindlimb ataxia. Once-yearly generalized seizures, which had first been noticed at 2 years of age. The last seizure occurred 2 months prior to referral at which time phenobarbitone was commenced. Since then, the dog had urinated in her bed, defecated indoors, not greeted visitors and had snapped at the owner twice.

CLINICAL EXAMINATION

The dog was alert and ambulatory with hindlimb ataxia. Hindlimb hopping and proprioception were reduced in both hindlimbs. Forelimb function was normal. Spinal reflexes were normal. Spinal pain could not be elicited by palpation.

NEUROANATOMICAL LOCALIZATION

The lesion was localized to the T3–L3 spinal cord segments owing to the hindlimb weakness as demonstrated by the decreased ability to hop, the reduced proprioceptive ability of the hindlimbs and the presence of intact spinal reflexes in both hindlimbs. Intact spinal reflexes signify that the lumbosacral intumescence is intact and so the cause of weakness must be further rostral. In an ideal world where diagnosis is easy, all animals with UMN paralysis or paraparesis would have 'textbook' hyperreflexia.

The seizures began years ago and the dog had not developed any behavioural abnormality until recently. Seizures may result from small frontal lobe lesions which with time will expand and cause inter-ictal neurological deficits. As a general rule of thumb, such deficits would have been expected to appear within 1 year. The age of onset of seizures and normal inter-ictal period is highly suggestive of idiopathic epilepsy. Post-ictal behaviour change and ataxia can occur but would not be expected to last 2 months. Low serum phenobarbitone levels can cause hyperactivity but not aggression. Polyuria and polydipsia are side-effects of phenobarbitone

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and this may result in urination in the house if the animal is not allowed outside to eliminate more frequently.

The recent behaviour change was thought to reflect the dog's response to being painful, even though a pain response such as vocalizing could not be elicited on examination.

DIFFERENTIAL DIAGNOSIS

- Intervertebral disc disease
- Spinal neoplasia
- Spinal cord neoplasia
- Discospondylitis
- Articular facet enlargement with cord compression.

CASE WORK-UP

MRI was chosen to image the spine and soft tissues (Fig. 15.1). It is important to include all the neuroanatomical area under consideration. In this case, the lesion was within the T2 vertebral body, compressing the adjacent spinal cord. If only the TL junction had been imaged to look for IVDD this lesion would have been missed.

DIAGNOSIS

Vertebral body neoplasia.

AETIOPATHOGENESIS OF EXTRADURAL VERTEBRAL BODY TUMOURS

Primary tumours arise from the surrounding tissue (bone, cartilage, fibrous, vasculature or lymphoid). Approximately 70–90% of cases occur in large breed dogs >20 kg with the mean age being 5–6 years. Most occur in the thoracic and lumbar spine. The most common primary tumour in cats and dogs is *osteosarcoma*. Surgical resection of canine vertebral osteosarcoma is reported, with postoperative survival being less than 3 days. If left untreated all dogs were dead or euthanized by 1 month after diagnosis as a result of problems related to the primary tumour.

Metastatic neoplasia is less common. Prostatic adenocarcinomas often produce a proliferative periosteal reaction along the ventral vertebral bodies of L5–L7, the sacrum, pelvis and femurs.

Lymphosarcoma of the spinal cord may be part of a multicentric distribution or occur as a primary tumour. In either situation, the lesion may be extradural or intramedullary. Some 95% of feline spinal lymphosarcomas were solitary thoracolumbar spinal lesions in young cats. Progression of signs is typically an acute deterioration after an insidious onset. Some 85% of affected cats also had extraneural lymphosarcoma. CSF is worth collecting as neoplastic cells may be seen on cytology: one of the rare occasions in which CSF analysis yields specific results. Sample CSF caudal to the lesion. The CSF may be normal if the tumour is extradural, i.e. does not involve the subarachnoid space. Surgical decompression of the spinal cord may be required to obtain a tissue diagnosis, however, such local treatment should not be viewed as curative. Chemotherapy with or without radiation produces rapid remission lasting up to 12 months. Untreated animals have an approximate survival time of 1–2 months.

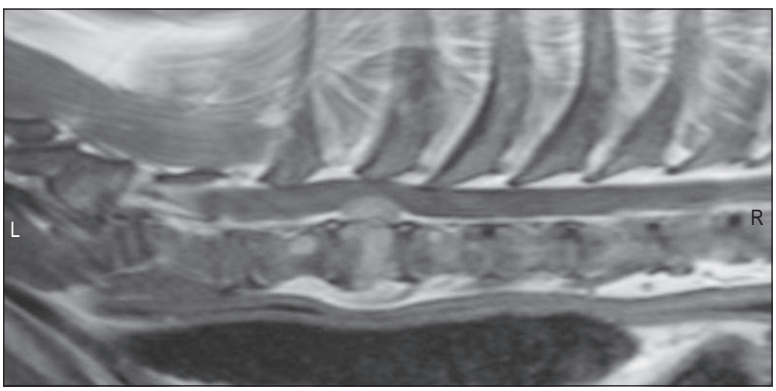


Figure 15.1 Sagittal MRI of spine. T1WI post-contrast. Tumour of the vertebral body compressing the spinal cord.

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Figure 15.2 The eye is drawn to the narrowed C6–7 IVD space. Note the fractured dorsal spines of C6 and C7 vertebrae.

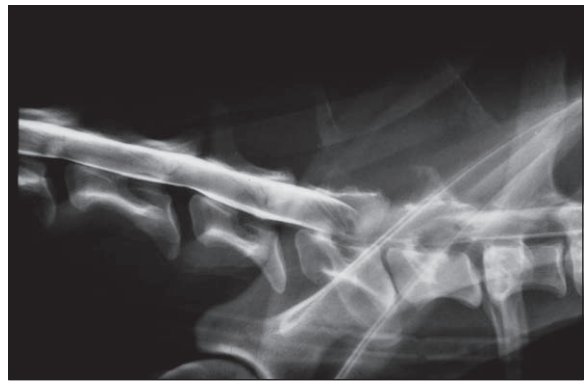


Figure 15.3 Dorsal extradural compression secondary to neoplasia.

Table 15.1 Differentiating tumour and infection

Radiographic sign	Neoplasia	Discospondylitis
Bony lysis and cortical break	*	
Endplate destruction	*	*
Collapse of adjacent disc space	*	*
Collapse/shortening of vertebral body	*	
Pathological fracture	*	
Bone sclerosis	*	*
Bone production	*	*
Cyst-like expansile lesion	*	
Paravertebral soft tissue mass	*	
Involvement of dorsal lamina, processes, articular facets	*	
Usually one vertebra	*	
Usually involvement of the disc space		*
Widening of canal or foramen	*	
Loss of bone density	*	
Extensive soft tissue involvement		*

Vertebral neoplasia usually does not cross the IVD space to involve the adjacent vertebra (Figs 15.2 and 15.3).

Table 15.1 shows the differentiating factors between tumour and infection.

Spinal pain, cord compression, and the possibility of fracture contribute to a very poor prognosis. *Multiple myeloma* is an exception; while chemotherapy is not curative, it does reduce the tumour burden, relieve bone pain and allow repair of osteolytic lesions thus prolonging length and quality of life; 25% of affected dogs have vertebral involvement detected by radiography. Multiple osteolytic lesions of the axial and appendicular skeleton have been reported in canine lymphoma and metastatic carcinoma.

Cartilaginous exostoses (osteochondroma/osteochondromatosis) are nodules composed of cartilage with or without bone which arise at the growth plates of vertebrae, ribs and long bones. Almost all arise from the dorsal lamina or spinous process. They may be solitary (rare) or multiple. Young dogs and adult cats are affected. Growth of the mass ceases at skeletal maturity in dogs. Resection relieves cord compression and allows histopathological diagnosis. Recurrence and malignant transformation are reported.

TREATMENT

The dog was euthanized. No postmortem was performed.

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SECTION 4

SEIZURES

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16 Seizures – an introduction

Seizures originate from the brain. They are the physical manifestation of abnormal synchronous electrical discharge in the brain. The brain can be stimulated to do so by intracranial or extracranial causes. The chief purpose of clinical examination and laboratory tests is to differentiate the two.

1. Extracranial causes; derangement elsewhere in the body, secondarily affecting brain metabolism:
   a. Metabolic: hypoglycaemia, portosystemic shunt, hyperosmolarity or hyperviscosity syndrome
   b. Toxic.
2. Intracranial causes:
   a. Structural: physical interruption of brain tissue; inflammation, malformation, degeneration, trauma, haemorrhage/ischemia or tumour
   b. Functional: derangement at a cellular level altering electrical impulses; idiopathic epilepsy.

DESCRIPTION OF THE SEIZURE

1. *Prodromal phase:* change in mood or behaviour lasting 1–24 hours before seizure activity. Not classified as seizure activity, e.g. clinging to/following the owner, appearing anxious or restless. Usually only noticed by those familiar with the animal's normal behaviour.
2. *Pre-ictal phase (aura):* commencement of seizure before impairment of consciousness occurs. Lasts seconds to minutes and is characterized in humans by altered sensory perception.
3. *Ictus/fit/seizure:* Commonly identified by a lack of consciousness/responsiveness and rhythmic involuntary motor activity lasting seconds to minutes. Variations exist. The point of describing or classifying a seizure is that different types may respond better to certain anticonvulsants and some seizures occur more often in specific diseases. A convulsion is a series of involuntary contractions of skeletal muscles. The term is commonly used as a synonym for seizure.
   a. *Generalized seizure*: lack of consciousness/responsiveness, involuntary motor activity of the whole body, autonomic signs (salivation, urination and defecation). Described as:
      - *Clonic:* rhythmic muscle contractions ('*paddling, running*')
      - *Tonic:* marked increase in muscle tone with varying intensity
      - *Tonic-clonic:* increase in muscle tone followed by/alternating with rhythmic muscle contractions
      - *Atonic:* loss of muscle tone. Rare
      - *Myoclonic:* shock-like muscle jerks, focal or generalized, occurring as single or multiple events.

   Generalized tonic-clonic seizures were regarded as the most common seizure type but a recent study found 65% of dogs had partial seizures. The difference in opinion is due to recognizing that the commencement of a generalized seizure may have a partial onset, and classifying it as such. Partial seizures may be missed in cats as the motor/behaviour signs can be subtle: sitting, staring, growling, salivating or facial twitching.

   A bystander can assess the animal's mental responsiveness to stimuli but not the level of awareness. This makes it difficult to classify some stereotypic activities as seizures or psychological disturbances. If an animal will not respond to being called or stroked because it is fearful, painful, or struggling to maintain balance, the event may be mistaken for seizure activity and treated inappropriately.

CLINICAL TIP

Myoclonus does not always indicate seizure activity.

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b. *Partial seizure*: focal involvement, with or without altered consciousness/responsiveness. It may secondarily generalize to involve the whole body. The sequential spread of motor activity to other body parts is called a *Jacksonian march* and results from spread of the electrical disturbance through the motor cortex. Generalization may happen so quickly that the partial/focal onset is overlooked.
   - *Simple partial seizure*: consciousness unimpaired, e.g. seeking attention from the owner, rhythmic contraction or flexion of a specific limb, turning the head in the same direction, tonic opening of the jaw, head tremor, anxiety, trembling, hypersalivation (ptyalism), vomiting, incoordination, sitting down/losing hindlimb tone, staring look, restlessness and dilation of pupils.
   - *Complex partial seizure*: impaired consciousness, e.g. disoriented, unable to recognize owners, unable to respond to commands, head pressing, apparent changes in perception, following imaginary objects, unprovoked aggression, unmotivated barking, repetitive stereotypic behaviour from which it cannot be distracted, confusion and abnormal behaviour, lip-smacking or chewing.

CLINICAL TIPS

- **Brief, recurring stereotyped changes in behaviour, motor signs, or autonomic signs may be partial seizures.**
- **Brief recurring stereotypic motor activity may be a 'movement disorder'.**

4. *Post-ictal phase*: Seen as a state of apparent confusion following a seizure in which the animal wanders, paces, or runs around, exhibiting restlessness, ataxia, bumping into things, not responding to the owner and may lose all house-training. Some animals appear mentally depressed for days or weeks and are lethargic and sleep a lot. The first evidence of seizure activity noticed may be the post-ictal behaviour change and finding urine or faeces in the house and saliva-soaked fur. One study found no correlation between the type, intensity, or duration of seizures and the duration of the post-ictal phase. It can be the most disruptive and worrying phase for the owner.

CLINICAL TIPS

- **Animals with a focal (partial) onset of seizures should be investigated for intracranial pathology.**
- **Intracranial vs. extracranial is the most important 'classification' of seizures.**

DIAGNOSIS

1. History is vital: description of event
   - Differentiate from: syncope, vestibular disease, weakness/collapse, behavioural disorders, rhythmic repetitive events in which the animal ignores the owner (pruritus; painful events), sleep disorders (hypnic jerks, narcolepsy/cataplexy).
   - The importance cannot be over-emphasized.
2. Neurological examination:
   - Abnormalities may be due to post-ictal change, commencement of anticonvulsants, the underlying pathology, or longstanding unrelated neurological deficits.
   - An abnormal inter-ictal examination in a seizuring dog was a strong predictor (90%) of an abnormal brain MRI. Normal inter-ictal examination however was not a failsafe guarantee of a normal brain: 22% of such dogs had an abnormal MRI.
   - Frontal lobe lesions can present initially as seizures with a normal inter-ictal examination.
3. Complete blood count, serum biochemistry profile, urine analysis
   - Aids differentiating extracranial causes from intracranial causes.
4. Imaging the brain
   - Differentiates intracranial causes
   - Some abnormalities may not be causal
   - MRI is preferred to CT in most cases for its high contrast resolution of soft tissue and lack of artefacts in the caudal fossa (area of the skull between the tentorium cerebelli and the occipital bone)
   - Age at onset of seizures in the dog does not predict the MRI outcome, thus dogs more than 6 years of age at the first seizure must not be condemned as having incurable brain disease and denied investigation/treatment. Equally, young dogs should not be assumed to have a good prognosis.

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5. CSF analysis
   - Requires GA so generally done at the time of MRI/CT
   - Pleocytosis may occur after repeated or prolonged seizures in humans. Electroconvulsive therapy may raise total protein levels. Post-ictal change to CSF in animals is therefore possible
   - Of seizuring dogs with abnormal CSF, only 74% had an abnormal MRI of the brain
   - Of seizuring dogs with normal CSF, 26% had an abnormal MRI of the brain.

THE OWNERS' PERSPECTIVE

A total of 60% of owners said that owning an epileptic dog had a negative influence on their daily life, causing stress and restricting their activity.

Chief concerns:

- Fear of dog having a seizure unattended
- Fear of finding the animal dead after a seizure
- Fear of the dog drowning during a seizure while swimming.

The emotional stress was reduced by:

- Regular contact and easy access to veterinarians
- Thorough information on seizures, the therapeutic regime, prognosis, and instructions for handling a seizuring animal.

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17 Skull tumour

INITIAL PRESENTATION

Seizures.

INTRODUCTION

Head trauma usually has to be severe to cause lasting signs.

SIGNALMENT

An 11-year-old female Jack Russell terrier.

CASE PRESENTING SIGNS

A recent onset of seizures

CASE HISTORY

The first seizure began 20 minutes after the dog's head was knocked by an opening door. A lump on the side of the dog's skull was noted by the owners at this time. For the following 5 months the seizures occurred spontaneously every 2 weeks and consisted of a 5 minutes generalized tonic-clonic seizure followed by a post-ictal period lasting several hours during which time the dog did not recognize or respond to the owner. On one occasion, the dog seizured a few hours after the veterinarian pressed the right-sided skull mass. The seizure pattern had changed recently with three seizures occurring during the week prior to referral.

Inter-ictally the dog remained house-trained but had been seen to bump into the furniture and miss its footing when ascending stairs. The claws were heard to drag on the linoleum floor. Episodes of twitching, jerking and clamping the jaws shut, and generally appearing quieter than normal were also observed. These inter-ictal changes had become more noticeable in the 2 weeks prior to referral. The dog had not been treated with any medication.

CLINICAL EXAMINATION

The dog was quiet but mentally responsive. The gait was normal. The menace response was absent on the left. The placing responses and hopping were all decreased on the left. A raised mass could be seen and palpated (gently) on the right parietal skull.

NEUROANATOMICAL DIAGNOSIS

The history of seizures, and left-sided deficits found on examination indicated a right-sided cerebral lesion.

DIFFERENTIAL DIAGNOSIS

The firm right-sided mass was thought to be a neoplasm of the skull. The knock on the head from a gently opening door would not be sufficient to induce seizure in a normal dog nor induce brain damage. The dog had lived in the household for 11 years and had skilfully avoided opening doors until recently, which suggests the dog was not as aware, as visual, or as balanced as normal. Pressing the mass appeared to transfer force to the underlying cerebrum.

CASE WORK-UP

Skull radiographs: an increased opacity was seen on the calvarium (Fig. 17.1).

MRI: a right-sided focal mass with a broad based peripheral attachment to the overlying skull with exten-

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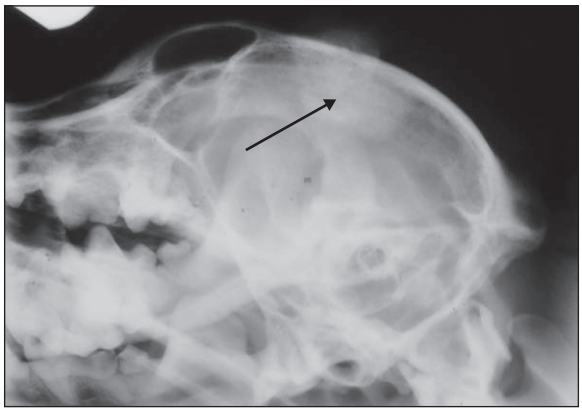


Figure 17.1 Skull radiograph (arrow to calcified mass).

sion into the overlying temporalis muscle was seen. A midline shift and compression of the right lateral ventricle was present. Minimal cerebral oedema was seen. Signal characteristics indicated that it was a calcified mass.

Thoracic radiographs were normal.

DIAGNOSIS AND TREATMENT

Multilobular osteochondroma was confirmed by surgical removal. The neurological deficits resolved.

Aetiopathogenesis of skull tumours

The presenting signs depend on the location of the mass and the degree of soft tissue displacement, e.g. seizures, behaviour change, trismus, exophthalmos or a firm palpable mass.

Osteosarcoma of the calvarium produces a visible external mass and extradural compression of the brain. Direct invasion of the CNS parenchyma is rare. Incomplete surgical resection is followed by tumour recurrence within 2–3 months. The most common cause of death is recurrence rather than metastasis.

Multilobular osteochondrosarcomas are slow growing, locally destructive tumours occurring in the calvarium, orbit, zygomatic arch, mandible, maxilla, and hard palate. Dogs and cats may be affected. CT is required to detect the large intracranial component. Surgical removal, even if incomplete, greatly improves survival times. Recurrence is slow to develop (median time: 14 months). Lung metastases also grow slowly (mean time of metastasis to euthanasia is 9 months).

An osteoma presents as a palpable non-painful lump, appearing radiographically as well-circumscribed dense bone.

Treatment of seizures

When to start anticonvulsant therapy:

- If status epilepticus has occurred
- If individual seizures occur at less than 2 week intervals
- If seizure frequency is increasing.

It is important to remember two facts:

1. The inter-ictal period varies animal to animal and within the same animal.
2. Anticonvulsants rarely ‘cure’ seizures.

It therefore makes sense to wait until the seizure pattern is known. Animals experiencing short, single seizures with a minimal post-ictal phase once every few months may not benefit in a measurable way from anticonvulsants. The owner may tolerate the pet’s intermittent seizure more than the drug side-effects.

In this case, the seizures every 2 weeks were frequent enough for most owners and vets to consider medication.

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18 Meningioma

INITIAL PRESENTATION

Seizures.

SIGNALMENT

A 15-year-old male standard wirehaired dachshund.

CASE PRESENTING SIGNS

Seizures.

CASE HISTORY

Two seizures had occurred, 2 months apart. The dog was normal inter-ictally. Laboratory work was normal.

CLINICAL EXAMINATION

Neurological exam was normal.

NEUROANATOMICAL LOCALIZATION

Seizures indicate cerebral disease. Extracranial causes of cerebral disease had been eliminated.

DIFFERENTIAL DIAGNOSIS

Frontal lobe meningioma was suspected given the age.

CASE WORK-UP

On MRI a small contrast-enhancing mass was found on the surface of the left frontal lobe at the caudal limit of the frontal sinuses (Fig. 18.1).

CSF WBC = 2; TP = 0.32 g/l cytology was normal.

Thoracic radiographs were normal.

DIAGNOSIS

The characteristics were highly suggestive of a meningioma. Increased total protein is common with intracranial neoplasia.

TREATMENT

Surgical removal of the mass was suggested but declined.

FOLLOW-UP

Seizures occurred once every 6–7 weeks until the pattern changed, 5 months after the diagnosis The inter-ictal period shortened to 1–2 weeks. The post-ictal period lengthened and consisted of 2–3 hours of pacing and vocalizing. Phenobarbitone was commenced and serum levels were in the low therapeutic range. The seizure pattern did not improve, so the dose and subsequent serum level was increased. Seizure frequency increased again, with single events occurring on successive days. The dog appeared lethargic but was otherwise neurologically normal.

The dog was euthanized 9 months after the first seizure was seen.



Figure 18.1 MRI of the frontal lobes. T1WI + contrast. Suspected meningioma on the left.

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19 Cerebral haemorrhage

INITIAL PRESENTATION

Dull, seizures, circling.

INTRODUCTION

Haemorrhage into the nervous system may present as meningeal irritation causing neck pain or diffuse spinal pain. Lesions of the CNS may be multifocal or focal.

SIGNALMENT

A 1-year-old female Staffordshire bull terrier.

CASE PRESENTING SIGNS

Seizures.

CASE HISTORY

One month prior to referral the hindlimbs gave way during exercise. Ataxia, circling and falling backwards were noted the following day. The dog recovered and had its first seizure the day before referral.

CLINICAL EXAMINATION

The dog was obtunded and ambulatory, circling to the left and had a left head turn (Fig. 19.1).

No ataxia was noted. The menace response was present bilaterally. Both pupils were miotic but a PLR could be detected bilaterally. The fundus looked normal.

Hopping, proprioception, and spinal reflexes were all normal. No spinal pain was found. The left upper lip was swollen and bruised (Fig. 19.2).

No petechiae, joint swelling, or other bruising was found.

NEUROANATOMICAL DIAGNOSIS

Left cerebrum.

The history of falling over backwards was suggestive of cerebellar disease but no sign of this was present on examination.

DIFFERENTIAL DIAGNOSIS

Vascular/haemorrhagic or inflammatory disease were considered most likely given the age. Neoplasia or decompensated hydrocephalus were also possible.

CASE WORK-UP

The bleeding time was prolonged at 7 minute (N <4 minute).

Clotting times were at the high end of normal.

Haematology: normal platelet count. Bruising developed at the venipuncture site.

Baerman examination of faeces – larvae seen were identified as *Angiostrongylus* sp.

MRI of the brain showed two suspected haemorrhages, one in the dorsal cerebellum and one in the left cerebrum stretching from the olfactory bulb caudally creating a mass effect and some secondary oedema (Figs 19.3 and 19.4).

CSF was not taken because it would not have added anything to the diagnosis and there was an unnecessary

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Figure 19.1 Left head turn.



Figure 19.2 Bruised lips.

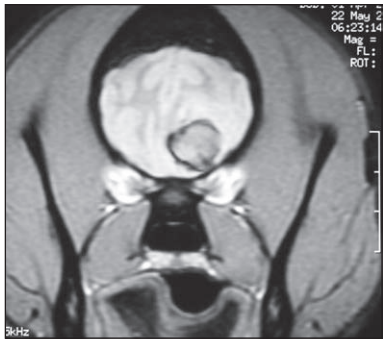


Figure 19.3 MRI. GRE left cerebral haemorrhage.

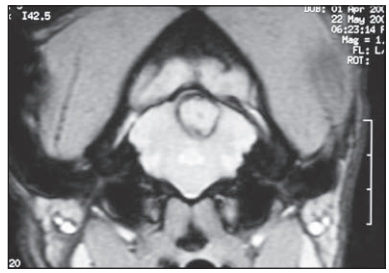


Figure 19.4 MRI. GRE cerebellar haemorrhage.

risk of haemorrhage if meningeal vessels were damaged during sampling.

DIAGNOSIS

The diagnosis was an acquired clotting defect giving rise to intracranial haemorrhage.

TREATMENT

- Treatment for *Angiostrongylus vasorum* (lungworm)
- Vitamin K antagonism (rodenticide)
- Prednisolone, for intracranial oedema.

The dog had not received aspirin, which interferes with platelet function.

Angiostrongylus vasorum (French heartworm)

Despite the name, this parasite (*metastrongylid nematode*) has a worldwide distribution.

The definitive hosts ingest the infective L3 larvae by eating the intermediate host (snails and frogs). The L3 larvae travel through the portal circulation to the liver then right cardiac ventricle and pulmonary arteries where they mature. Eggs are shed and travel to the pulmonary capillaries, hatch into L1 larvae which penetrate the airways, are coughed up, swallowed, and excreted in the faeces to be ingested by the intermediate host.

L1 larvae may migrate to aberrant sites via the circulation but clinical signs are mostly:

1. Respiratory system: cough, dyspnoea
2. Coagulopathy: haemorrhages in sclera, conjunctiva, gingiva, brain, subcutaneous tissue and body cavities. There may be bruising and haematomas may form at venipuncture and surgery sites
3. Collapse.

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Figure 19.5 Scleral haemorrhage in another case of *Angiostrongylus vasorum* infection presenting with signs of neck pain.

Neck /back pain is reasonably common so these animals can present as though they have meningitis. CSF analysis may demonstrate erythrophagia but is unnecessary for the diagnosis as evidence of haemorrhage is usually present on physical/retinal examination or can be found on the coagulation profile, bleeding time measurement or haematology (Fig. 19.5).

The dog is only infected by ingesting stage 3 larvae within the intermediate host.

The dog was neurologically stable, with normal heart rhythm, respiration and lung sounds for 24 hours. Bilaterally dilated pupils suddenly developed and respiratory arrest occurred half an hour later. Pulmonary haemorrhage was evident on intubation.

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20 Hydrocephalus

INITIAL PRESENTATION

Seizures.

INTRODUCTION

What appears strikingly abnormal to a vet does not always ring warning bells for the general public. In fact, one owner presented his 'rottweiler puppy' for first vaccination only to be told it was a guinea pig. Always comment on the abnormal, even if it is not the reason for the animal's presentation.

SIGNALMENT

A 3-month-old female golden retriever.

CASE PRESENTING SIGNS

Seizures.

CASE HISTORY

Frequent generalized seizures commenced 4 days prior to referral. Since then the pup had not been able to find treats thrown to her, she had lost interest in her toys and had begun urinating and defecating in the house. The pup had previously been house-trained successfully. She had always bumped into things while walking and this was thought to be normal behaviour for a puppy. The owners had noticed the pup's domed head (Fig. 20.1) at 8 weeks of age when they had bought her from the breeder.

CLINICAL EXAMINATION

The alert ambulatory puppy walked normally but occasionally bumped into table legs. The menace response was absent bilaterally. Both pupils were small but possessed normal pupillary light reflexes. Proprioception and hopping responses were normal in all limbs. The head was enlarged. No open fontanelles were palpated. Slight bilateral ventrolateral strabismus was present but eye movement was normal.

NEUROANATOMICAL LOCALIZATION

Cerebrum. The menace response is present from 10–12 weeks of age in puppies. The absent menace response may reflect the age of the dog or bilateral cerebral involvement. Seizures, loss of learnt behaviour and miotic pupils are all signs of cerebral disease. Bumping into objects suggests altered vision in this puppy with an otherwise normal gait. Ventrolateral strabismus with normal eye movements is a characteristic finding of congenital hydrocephalus.

DIFFERENTIAL DIAGNOSIS

Congenital hydrocephalus, possibly decompensated by intraventricular haemorrhage or a recently acquired encephalitis.

CASE WORK-UP

Haematology, biochemistry, urine analysis, and bile acid stimulation performed by the referring vet were all normal.

Enormously enlarged lateral ventricles, an enlarged third ventricle and a narrowed aqueduct through the midbrain were found on MRI of the head (Fig. 20.2). The 4th ventricle appeared to be a normal size.

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Figure 20.1 Note the steep rise between the nasal and frontal bones.

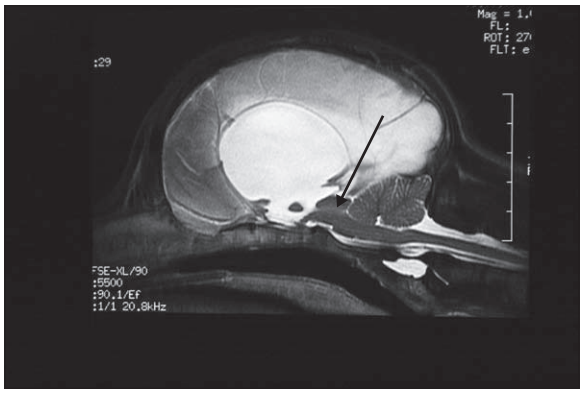


Figure 20.2 MRI sagittal brain T2WI. Narrowed mesencephalic aqueduct (arrow).

The ventricular fluid had the MRI characteristics of CSF, and the ventricles were symmetrical. There was no contrast enhancement.

CSF analysis: elevated total protein level of 0.41 g/l but the cell count and cytology were normal.

CLINICAL TIP

Open fontanelles are not diagnostically significant *per se* but may occur with hydrocephalus.

DIAGNOSIS

Congenital hydrocephalus

Ventriculomegaly is the enlargement of the ventricular system. Hydrocephalus is the combination of ventriculomegaly with cerebral dysfunction.

Aetiopathogenesis of hydrocephalus

Congenital and acquired forms of hydrocephalus result from either blockage of CSF flow through the ventricular system or poor absorption of CSF at the arachnoid villi. Excess secretion of CSF by a choroid plexus papilloma is a rare cause of acquired hydrocephalus. Impaired CSF circulation has been proposed as the cause of hydrocephalus in dogs with the genetic disorder primary ciliary dyskinesis.

A congenital malformation might be expected to show signs from birth or ambulation but this is not always the case. Hydrocephalus is typically associated with dome-headed toy breed puppies showing cerebral signs but it can become clinically apparent in the young adult dog of an atypical breed. The thin rim of remaining cerebral tissue is susceptible to injury which could account for a sudden onset of signs (Fig. 20.3).

As with any neurological disease, the signs follow anatomical derangement. The instigating lesion of acquired hydrocephalus may be sited at any point along the ventricular system and show initial signs related to that structure, e.g. cerebellopontine, midbrain, meningeal, with secondary cerebral involvement occurring 'upstream'.

Congenital hydrocephalus generally presents with cerebral signs: altered mentation and behaviour, pacing, circling and seizures. The rate of progression of signs is highly variable from a gradual worsening to acute decompensation. Enlargement of the head with a dome-shape is a visually striking clue. Open fontanelles are not pathognomonic for hydrocephalus.

Bilateral ventrolateral strabismus ('setting sun' sign) is common and may result from skull deformity causing shallow orbits (Fig. 20.4). Ocular and pupil movement are normal which rules against midbrain or CN III dysfunction as being the cause.

Ultrasound imaging through fontanelles may detect dilated ventricles. A radiographic ground-glass uniform opacity of the enlarged calvarium results from flattening of the internal ridges and depressions (Fig. 20.5). The gyri and sulci of the brain are still present. CT and MRI easily detect enlarged ventricles, trans-ependymal oedema, sites of obstruction, and causative lesions such

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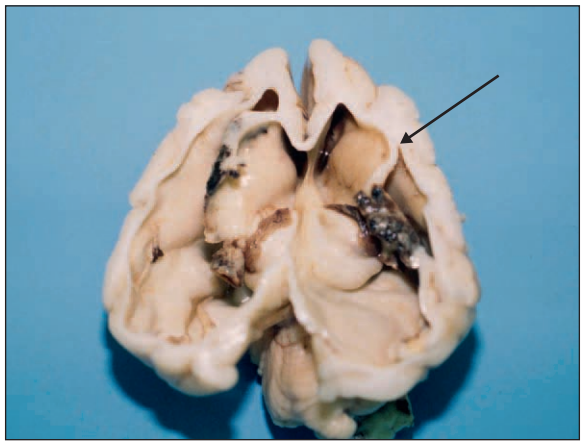


Figure 20.3 Hydrocephalic brain: note the thin rim of cerebral cortex (arrows).



Figure 20.4 Hydrocephalic 3-month-old Jack Russell terrier puppy. Ventrolateral strabismus.

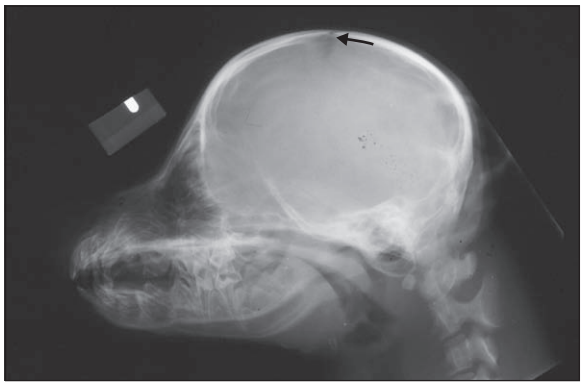


Figure 20.5 Skull radiograph of another hydrocephalic puppy. Note the 'ground glass' smooth appearance of the calvarium (arrow, open fontanelle).

as inflammation, intraventricular haemorrhage or tumour.

Although congenital hydrocephalus is thought to be due to narrowing of the mesencephalic aqueduct during fetal development, the cause of this is not evident at the time of diagnosis. Failure of lateral aperture formation was recorded in a 3-month-old great Pyrenees puppy.

PROGNOSIS

There is no correlation between clinical signs and the degree of ventricular dilation. As with most neurological diseases, the prognosis cannot be determined by imaging and so the clinician must rely on the examination findings, progression of signs, and knowledge of any underlying disease process.

TREATMENT

Surgical drainage of hydrocephalus in acquired cases can relieve signs and prevent worsening while the primary lesion undergoes treatment. In a recent retrospective study of nine dogs with acquired hydrocephalus treated by ventriculoperitoneal (VP) shunting, seven were euthanized due to a lack of improvement or worsening of signs 2 days to 19 months (mean 8.6 months) after the operation.

Placing a shunt into the ventricle of a congenital hydrocephalic is difficult to recommend based on the uncertain prognosis and the need for shunt readjustments due to blockage, under-drainage, migration, infection and growth of the animal. If no benefit from medical management is seen within 2 weeks or the patient deteriorates, then surgery should be considered. Reported success rates range from 50% to 90%. In the retrospective study mentioned, five dogs with congenital hydrocephalus were treated with VP shunting, with three of these dogs euthanized due to severe seizures 2–9 months postoperatively.

Conservative treatment has not been successful long-term. Temporary relief may be provided by:

1. Corticosteroids
   - Oral prednisolone 0.25–0.5 mg/kg BID decreases CSF production, reducing to 0.1 mg/kg every other day which is given for 1 month, then stopped if possible.
   - IV dexamethasone reduced CSF production immediately after 0.15 mg/kg and attained maximal reduction of 50% in 50 minutes.

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- If no benefit from corticosteroid use is observed within 2 weeks, then other therapy should be considered, e.g. shunts.

2. Osmotic diuretics
   - Mannitol. 1–2 mg/kg IV over 15–20 minutes. This may be repeated 2–4 times over 48 hr. Monitor hydration.
3. Carbonic anhydrase inhibitor
   - Acetazolamide 10 mg/kg PO q6–8 hr reduces CSF pressure by reducing CSF production. Short-term use.
4. Proton pump inhibitor
   - Omeprazole.

Treatment in this case was limited to phenobarbitone and corticosteroids. The pup became more playful. Surgical placement of a ventricular shunt was offered but declined. The pup seizured again and was euthanized.

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21 Idiopathic epilepsy

INITIAL PRESENTATION

Seizures.

INTRODUCTION

The definition of idiopathic epilepsy is the ocurrence of seizures for which an underlying cause has not been found. It is a diagnosis made by exclusion of other aetiologies.

A presumptive diagnosis is made after:

- at least two seizures have been observed
- the physical and neurological examinations are normal during the inter-ictal period
- no behavioural abnormalities are observed while the animal is at home
- normal lab tests (haematology, biochemistry, urine) and diagnostic imaging (CT, MRI) .

SIGNALMENT

A 4-year, 10-month-old male golden retriever.

CASE HISTORY

A generalized tonic-clonic seizure occurred while the dog was walking on the beach. This was followed by a normal inter-ictal period and then another seizure, similar in nature, 2 weeks later. Phenobarbitone had been commenced. No exercise intolerance was reported. The dog was described as being otherwise normal.

CLINICAL EXAMINATION

The dog was alert ambulatory, with a normal gait, postural reactions and proprioception. The right menace response was absent but facial muscle function was normal.

The right pupil was slightly larger than the left. When light was shone into the right eye, neither pupil constricted. When light was shone in the left eye the right and left pupils constricted. The right optic disc was darker than the left suggesting a lack of myelin. The other CNN were normal as were the spinal reflexes.

NEUROANATOMICAL LOCALIZATION

The absent menace response and PLR in the right eye indicated a CN II lesion. Seizures are a sign of cerebral disease. The dark optic disc suggesting demyelination of the optic disc suggested a long-term lesion, and so the two lesions may have been unrelated.

DIFFERENTIAL DIAGNOSIS

- Optic nerve atrophy
- Idiopathic epilepsy
- Lesion of the optic chiasm compressing the thalamus would be expected to alter mentation and vision.

CASE WORK-UP

- MRI of the head was normal
- CSF was normal
- Ophthalmology consultation diagnosed right optic nerve atrophy.

DIAGNOSIS

Two separate lesions. The cause of the seizures was not found. Idiopathic epilepsy.

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Seizures occur in 1–3% of dogs, with idiopathic epilepsy accounting for about 20–80% of those seizures, depending on the reference. It has a proven inheritance in a number of canine breeds but can also affect crossbreds. Do not breed from affected animals. Idiopathic epilepsy also occurs in cats. The incidence of idiopathic epilepsy in cats varies greatly. One source states that 60% of seizuring cats had idiopathic epilepsy.

There is no established range for the age of onset of idiopathic epilepsy in cats; it may be from 3 months to 16 years. In a study of 12 cats, the age range at onset of seizures was 0.25–12 years with a median age of 2 years. The age of onset in dogs is between 1 and 5 years of age.

PROGNOSIS

A total of 15% of affected dogs show a spontaneous remission (freedom from seizures for more than 3 years without anticonvulsants) or remission with treatment (seizure free for more than 3 years while on anticonvulsants).

Approximately 25% of idiopathic epileptic dogs are refractory to treatment. Examine all aspects of the case for errors (incorrect diagnosis, or inadequate use of drugs); 20–40% are refractory to initial monotherapy with anticonvulsants.

Idiopathic epilepsy is not thought to shorten the lifespan of affected dogs; however another source states that 19% of dogs die of causes directly related to their seizure disorder.

Seizure pattern

- The type, number and frequency of occurrence varies from animal to animal regardless of the aetiology.
- The physical characteristics of seizures are relatively constant over time in any particular animal. The frequency of seizures may change over time. If seizure appearance or frequency changes, the animal should be reassessed for underlying disease that may alter drug metabolism or intracranial function.
- Seizures usually occur at rest or when sleeping.
- Association with time of day or feeding may help diagnose hypoglycaemia or hepatic encephalopathy.
- Triggers are rarely found. Seizures are usually spontaneous.
- Brain tumours may initially present as seizures in dogs but more commonly present as a behaviour change in cats.
- Seizures from brain tumours may be infrequent.
- Idiopathic epilepsy is typically generalized tonic-clonic which may commence as a partial seizure.
- Partial seizures follow an identical pattern characteristic to the individual dog.
- Partial seizures have been linked to focal, structural brain lesions.

Seizure threshold

- There is no clinical evidence that the use of acetyl promazine is associated with increased seizure activity in dogs with seizure disorders.
- Housing, feeding, habits, time of day/year play a minor, if any, role.
- Emotional stress (unexpected noise or unusual event) might precipitate a seizure in a predisposed animal.
- In humans the seizure frequency can increase during the menstrual cycle. This is activated by oestrogens and suppressed by progesterone. This is called *catamenial epilepsy*. It has been proposed, but not proven, that oestrus in the dog may have the same effect.

TREATMENT

How to start anticonvulsant therapy

1. Warn the owners about side-effects and review their expectations.
2. The initial oral dose is based on body weight and is usually at the lower end of the dose range.
3. Loading doses for rapid rise in serum level should only be given while the animal is hospitalized, as the sedation and ataxia produced is profound.
4. Adjustments of dose are made in the light of the serum level at steady state and its effect on the seizure pattern. It is impossible to judge the effect of an anticonvulsant without taking a history. Owners must keep seizure diaries.
5. Sedation and ataxia generally last 7–14 days in the naïve patient. Gradually introducing the recommended dose may limit this.
6. Polyphagia can lead to animals raiding bins, opening refrigerators or snatching food from ovens. Increase the volume of the diet without increasing calorie intake. Divide meals.
7. Bromide must be given with food, the salt content of which should be kept constant. Phenobarbitone absorption is 10% less when given with food, although therapeutic levels can still be obtained.

Table 21.1 gives an example of a seizure diary.

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Table 21.1 Example of a seizure diary

Date	Time (am/pm)	Activity at onset	Duration	Recovery period	Notes
28/2	am	Sleep	1 minute, every hour, ×3	Dazed in between, OK next day	Clusters; rectal diazepam
31/3	pm	Resting	1–2 minutes, single	Ataxic 30 minutes then slept	Urination
2/4	–	–	–	Current dose: 60 mg BID	Phenobarbitone level: 28 mg/dl

CLINICAL TIPS

- Managing a seizure patient requires correlation of the seizure pattern, serum levels and the presence of side-effects.
- Advise owners to give a missed dose as soon as possible and then continue with the schedule.

Effects of anticonvulsants

Desirable:

- Reduce the frequency, number, duration and motor activity of a seizure.
- Shorten the post-ictal period.

Defining 'control':

'Good' or 'well-controlled' is defined as:

- ≥50% reduction in number of seizure compared with the untreated state.
- A decrease in the frequency of seizures to <1 per month for at least 1 year.
- These two factors are maintained for at least two-thirds of the time since diagnosis.

Owners generally tolerate monthly seizures, and for a period, weekly seizures. They are less likely to accept cluster seizures, status epilepticus and the adverse effects of anticonvulsants (Table 21.2).

CLINICAL TIP

Owners and vets vary in their definition of seizure control. Clarify expectations to improve compliance.

FOLLOW-UP

When to monitor anticonvulsant levels

- The aim is to have the drug within the therapeutic range. After initiating therapy or changing dose; serum levels can vary between animals on the same dose rate.
- At a steady state (after 5 half-lives have passed)
- If seizure control is poor or worsens
- When toxic signs develop
- When there is alteration in liver or kidney function
- If non-compliance is suspected, either by the animal (spitting it out, avoiding it in food, vomiting/regurgitation) or the owner
- Legal requirements for supplying the drug to an animal 'under your care'.

Sampling:

- Peak (4 hr) and trough (12 hr) do not vary significantly in 91% of dogs (phenobarbitone) at steady state
- After 5 half-lives (i.e. steady state)
- Serum separation tubes falsely decrease serum phenobarbitone/benzodiazepine levels
- Clorazepate can increase the phenobarbitone serum level and hence side-effects
- No change in ALT and ALP in cats after 25 days of phenobarbitone by mouth
- Check ALT and AST prior to and 5 days after starting diazepam in cats (hepatic necrosis).

When to add another anticonvulsant

- Monotherapy is preferred
- Do not give up on a drug too soon: assess its effect through several seizure cycles (e.g. at least through three typical inter-ictal periods). Explain to the owner that 'to wait and see' is an unavoidable part of seizure management
- After increasing the first drug to the limit of tolerance

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Table 21.2 Treatment of seizures: prophylaxic use of anticonvulsants

Drug	Phenobarb	Diazepam	Bromide	Levetiraceram	Clorazepate	Felbamate	Gabapentin	Zonisamide	Pregabalin
Dose (mg/kg)									
Dog	2–5 BID	–	K^+ 20–35 Na^+ 17–30 mg/kg/day	10–20 TID	0.5–1 TID	15 BID/TID	10 TID	10 BID	2–4 mg/kg q8 hr
Cat	2.5 BID	0.2–2 BID/TID	30 mg/kg/day	20 TID	–	–	–	–	–
Metabolism	Hepatic	Hepatic	–	–	Hepatic	Hepatic	Hepatic	Hepatic	
Excretion			Renal	Renal		Renal			
Half-life (hours)									
Dog	40–90	2–4	24 day	4		5–6	3–4	15	
Cat	34–43	15–20	10 day	–		–	–		
Therapeutic range (µg/ml, mg/l)									
Dog	15–35	0.5–0.7	700–2300	?		?		10–40	2.8–8.2
Cat	10–30	0.5	1–1.6 mg/ml	–		–		–	–
% Control									
Dog	50–70	–	72 (BR); 80–90 (PB+BR)	64 (L+PB +BR) transient		?	55%	58 (Z + others)	23–79% (mean: 50%)
Cat		80	35	70 (L+PB)		–	(G+PB+BR)	–	(PG+PB+KBR)

L, Levetiracetam; PB, Phenobarbitone; BR, Bromide; PG, Pregabalin; G, Gabapentin.

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- Top recommended serum level is reached
- The additional drug is generally added on to the current therapy. Depending on the reason (ineffective first drug; unacceptable or adverse effects) the dose of the initial drug may be slowly decreased.

When to wean off anticonvulsants

- If the animal was mistakenly diagnosed as seizuring and medication is not correcting the problem
- Remission rate: 15% (dogs) spontaneous remission and remission with treatment (seizure free for >3 years while on anticonvulsants) of idiopathic epileptic dogs
- Spontaneous remission: freedom from seizures for >3 years without anticonvulsants
- If the animal has not seizured for 6–12 months
- How: slowly (over months). Decrease one drug at a time.

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22 Post-ictal behaviour change

INITIAL PRESENTATION

Multiple seizures, behaviour change.

INTRODUCTION

Status epilepticus is defined as repeated seizure activity without an intermission or seizure activity lasting more than 5 minutes, or two or more seizures with incomplete recovery between seizures. It is generally not known why it happens to some and not other animals. A low serum drug level was responsible in only 5.7% of dogs in one study. Side-effects include neuronal injury, hyperthermia, hypoxia, acidosis, hypotension, renal failure, disseminated intravascular coagulation and cardiopulmonary failure. Animals may have a personality change lasting approximately 1–2 weeks post-ictally.

SIGNALMENT

A 3-year-old neutered male Jack Russell terrier.

CASE PRESENTING SIGNS

Behaviour change following multiple seizures.

CASE HISTORY

A single seizure had been noted 1 month prior to referral. The dog had been resting when it stiffened in its basket, and began to paddle the limbs and salivate and urinate while remaining unresponsive to voice or touch. The recovery period lasted half an hour, during which time the dog appeared agitated and paced around the house. This passed and the dog returned to normal. Multiple seizures occurred during the 4 days prior to referral. Since then the dog had been urinating in doors, pacing, twitching, was disinterested in the owners' presence and did not appear frightened of loud noises as he normally was.

CLINICAL EXAMINATION

The dog paid attention to the environment but was less responsive to the owners than usual. His gait was normal as he continuously walked about the room. The menace response was intermittently present. The rest of the examination was normal.

NEUROANATOMICAL LOCALIZATION

The lesion was localized to the cerebrum because of the seizures and menace deficit. The latter may be absent if the animal is not normally responsive or is mentally depressed as the menace response is not a reflex. The gait is generally normal in cases of cerebral disease. The change in behaviour is also suggestive of cerebral derangement.

DIFFERENTIAL DIAGNOSIS

The age of onset suggests:

- Idiopathic epilepsy
- Portosystemic shunt
- Encephalitis
- Hydrocephalus.

Lead toxicity is more common in young dogs. Cerebral haemorrhage secondary to ingesting *Angiostrongylus vasorum* in snails was possible.

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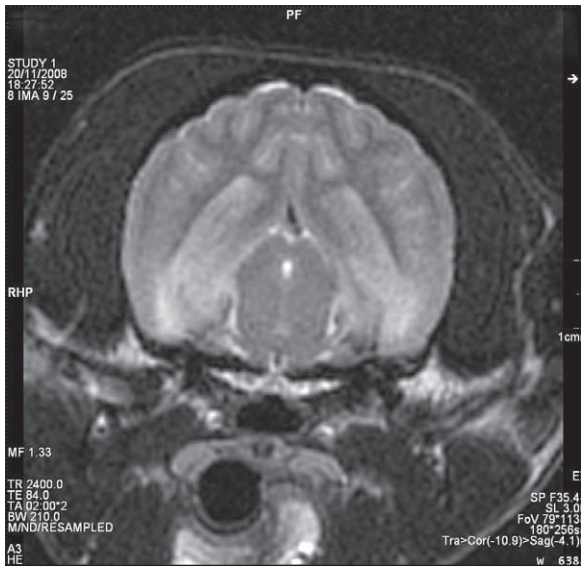


Figure 22.1 Transverse T2WI. Bilateral oedema in the temporal lobes.

CASE WORK-UP

Haematology, biochemistry, urine analysis, clotting and bleeding times and bile acid stimulation test were normal. CSF sampled at the time of MRI was normal.

MRI of the brain found increased signal intensity on T2WI in the temporal lobes with little mass effect (Fig. 22.1).

DIAGNOSIS

Prolonged post-ictal behaviour change following suspected idiopathic epilepsy.

Transitory T2WI hyperintensity of the cortical grey matter, subcortical white matter or hippocampus has been found in humans after generalized tonic-clonic seizures or status epilepticus and represent vasogenic or cytotoxic oedema. This has also been reported in the canine piriform and temporal lobes and improvement in the MRI lesion occurred completely or partially in 10–16 weeks time.

Hyperintense lesions on T2WI in the cerebrum, cerebellum or brainstem has been histopathologically correlated with demyelination in the acute stage of canine distemper in clinical cases. T2WI hyperintensity of the temporal lobe was also seen in acute canine distemper without demyelination being present but these dogs in the study had seizures in the preceding 4 days and thus the MRI charge could be expected to be a post-ictal change.

PROGNOSIS

Good. The dog's behaviour was expected to improve.

TREATMENT

In this case the dog had received several IV doses of diazepam 0.5 mg/kg in the hours prior to referral. The dog did not have any generalized seizures once referred but remained agitated and had transitory facial twitching which was thought to be partial seizure activity.

Phenobarbitone was commenced at 3 mg/kg BID. Some sedation and ataxia was noted for 1 week but this lessened. One week after the last seizure had occurred, the dog responded to the owner with normal affection.

Treatment for status epilepticus

- *Lorazepam* is a long-acting benzodiazepine with high affinity for the GABA receptor complex. Lorazepam (0.2 mg/kg IV) and diazepam (0.5 mg/kg IV) – give the same seizure-free interval after use.
- *Diazepam*: onset of antiepileptic activity within 2–5 minutes after IV administration in the dog. Diazepam constant rate infusion of 2–5 mg/hr in 5% dextrose solution or 0.5 mg/kg/hr in 0.9% saline IV. Adsorption to the plastic infusion set did not lower efficiency at this rate
- *Midazolam*: 0.35 μg/kg/min IV (dog)
- *Phenobarbitone* IV anticonvulsant action after 15–20 minutes. Dose of 2–4 mg/kg IV can be repeated at 30 minutes intervals up to a total cumulative dose of 20 mg/kg
- *Pentobarbitone* anaesthesia 3–15 mg/kg given to effect. Excitement during recovery may mimic seizure activity.
- *Levetiracetam:* Dog toxicity trial; single IV dose of 210 mg/kg gave no adverse effects. Dog single 60 mg/kg IV bolus over 2 minutes (N=6) no side-effects. IV use with diazepam IV potentiates the anticonvulsant effects of both
- *Rectal diazepam:* dose 0.5 mg/kg used when the initial seizure occurs. Up to three times
- *Propofol:* 1–8 mg/kg IV increments to effect and 0.1–0.6 mg/kg/min continuous rate infusion.

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Side-effects of anticonvulsants (D, dog; C, cat)

1. Transient sedation and ataxia for 7–14 days: bromide (D, C), phenobarbitone (D, C), diazepam (C)
2. Hyperactivity: phenobarbitone-low serum levels (D); bromide (D), felbamate (D) – high serum levels
3. Pelvic limb stiffness and ataxia: bromide (D)
4. Polyphagia: phenobarbitone (D), diazepam (C), bromide (D)
5. Polyuria/polydipsia: phenobarbitone (D, C), bromide (D)
6. Vomiting: bromide (give with food) (D, C), zonisamide (D)
7. Anorexia: bromide (D)
8. Diarrhoea: bromide (D)
9. Induction of liver enzymes (ALT, ALP): phenobarbitone (D). ALKP more than ALT. Normalizes 5 weeks after discontinuation of treatment
10. Decreases thyroxine (T4) and free thyroxine (fT4) and increases TSH without signs of hypothyroidism: phenobarbitone (D)
11. Hepatotoxicity: primidone (D), diazepam (C)
12. Pancreatitis?: bromide with or without phenobarbitone (D)
13. Dermatitis: bromide
14. Coughing: bromide (C)
15. Anaemia
16. Blood dyscrasia: phenobarbitone (D, C)
17. Coagulopathy (C)
18. Refractory/tolerance to the drug: benzodiazepine (D), phenobarbitone (D), levetiracetam (D).

FOLLOW-UP

At 2 weeks after the last seizure, the dog was responsive to the owners and appeared to be normal. Neurological examination was normal. A lack of house-training persisted. The owner confessed that she had never been strict about house-training the dog. Serum phenobarbitone level was in the low–normal range (20 mg/dl. Normal 15–35). The dose would be increased if clusters of seizures occur at the next seizure occurrence.

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SECTION 5

CRANIAL NERVE FUNCTION

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23 Blindness

INITIAL PRESENTATION

Bumps into things, reluctant to move, hesitant to use stairs, stumbles on uneven ground, sleeps all day, unable to locate moving or stationary objects, cannot catch objects, always sniffing the ground, low head carriage, develops aggressive behaviour.

INTRODUCTION

Blindness: the lack of vision, is a result of pathology in the afferent pathway of the visual system (Fig. 23.1).

It may appear worse at night (*nyctalopia*) or during the day (*hemeralopia*: blindness with good night vision). Animals adapt remarkably well to a loss of vision as long as the environment remains constant. A lack of adaption in which the animal still behaves in a quiet tentative manner, bumping into objects, may indicate a cerebral lesion. Long-term blindness may appear to be sudden in onset following a change in living arrangements, e.g. staying in kennels or visiting new areas. Physical obstruction of the eyes by blepharospasm or bilaterally protruded third eyelids secondary to enophthalmos is capable of mechanically rendering the animal sightless. Localizing the anatomic site for blindness requires examination of the fundus, PLR and vision.

PLR can persist in an eye blinded by retinal detachment, retinal degeneration or optic neuritis. The pupil in such cases is more dilated than normal.

Enlargement of the optic disc occurs in papilloedema and, more commonly, optic neuritis. *Papilloedema* describes an enlarged pale pink oedematous optic nerve head (disc) with indistinct margins over which retinal blood vessels 'kink'. It has been reported as a sign of increased intracranial pressure. Papilloedema does not cause visual deficits.

Optic neuritis results in acute bilateral blindness and may be retrobulbar and hence invisible on fundic exam, or may involve the optic disc.

Atrophy of the optic nerve is reflected in a shrunken pale grey optic disc.

SIGNALMENT 1

A 6-year-old neutered female lurcher.

CASE PRESENTING SIGNS

Inappetance and episodes of staring into space.

CASE HISTORY

The dog had been inappetant for 2 weeks when it started to have periods of standing under the dining room table and staring into space. Ptosis of the right eye occurred 5 days later. No oral lesions were found. Haematology, biochemistry, urine analysis and abdominal radiographs had failed to find the cause of the reduced appetite.

CLINICAL EXAMINATION

The dog was mentally depressed with a menace response that was absent in the right eye and decreased in the left. PLRs were absent bilaterally with a mydriatic left pupil. There was decreased movement of the right eye in all directions, right enophthalmos and right ptosis. Sensation to the face was normal. Retractor bulbi reflex was present bilaterally.

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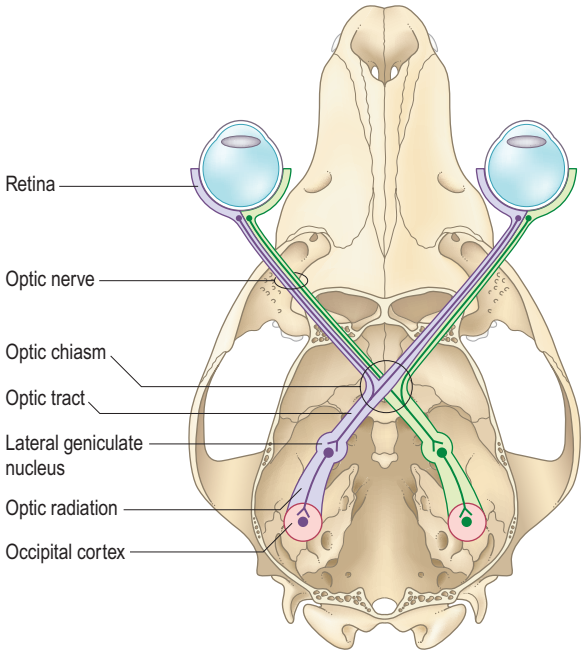


Figure 23.1 Pathway for visual perception.

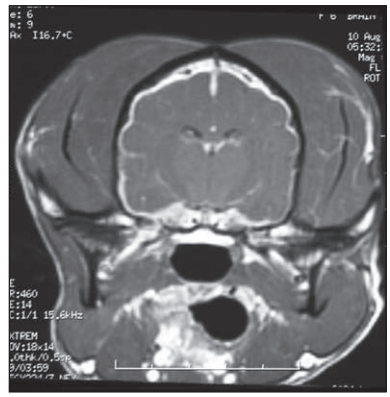


Figure 23.2 Transverse T1WI+C: thickened dura mater, interfering with cranial nerve function.

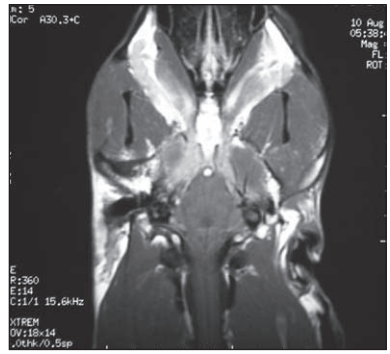


Figure 23.3 Dorsal T1WI+C: as in Figure 23.2.

Gait, posture, hopping, proprioception and spinal reflexes were normal. No spinal pain was detected.

NEUROANATOMICAL DIAGNOSIS

The lesion was localized to the right CNN II, III, IV and VI, and the sympathetic nerve supply to the right eye (Horner's syndrome) and to the left CNN II and III. The lesion was thought to lie in or near the cavernous sinus, or optic and orbital canals (see page 42).

DIFFERENTIAL DIAGNOSIS

- Meningioma on the floor of the skull
- Pituitary tumour
- Lymphoma
- Neosporosis was possible.

CASE WORK-UP

MRI of the head showed thickened contrast-enhanced dura mater (pachymeninges) around the brain and across the floor of the skull (Figs 23.2 and 23.3).

Contrast-enhancement of the meninges on MRI has been associated with inflammation (infectious and idiopathic) and neoplasia in small animals. Pial (pia mater) involvement is seen as enhancement following the gyri into the cortical sulci. This is more common in inflammatory/infectious meningitis in humans but a small study of affected animals failed to prove this relationship. Dural enhancement is that which does not enter the sulci. Magnet strength, pulse sequence and dose of contrast agent influence the intensity of meningeal enhancement on MRI. Absence of enhancement does not rule out inflammation. Postoperative meningeal enhancement occurs in humans both at the surgical site and at a distance from it. This could potentially be a diagnostic problem postoperatively in a deteriorating patient.

CSF collected at the cisterna magna from this dog had a normal cell count and cytology but the total protein was elevated at 0.78 g/l.

Thoracic radiographs, to check for visible metastases, were normal. Repeat haematology and biochemistry

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only revealed a lower urea (2.6 mmol/l; N 3.5–7.0) due to inappetance.

DIAGNOSIS

A definitive diagnosis could not be made as biopsy was declined and the dog euthanized.

Pachymeningitis of the spinal and brainstem dura mater is a rare condition reported in pointer dogs presenting with neck pain and cranial nerve deficits (CNN V, VII and VIII) as well as Horner's syndrome and seizures. Neutrophilic CSF with a raised total protein level was found.

Ossification of the dura mater forming radio-opaque lines is an incidental finding in older dogs.

Thickened cranial meninges occur in humans causing entrapment of cranial nerves, and blockage of veins, arteries and CSF flow resulting in cranial nerve deficits, focal neurological deficits and raised ICP. Infections, neoplasia and autoimmune disorders are implicated. A group of patients remains undiagnosed despite meningeal biopsy.

SIGNALMENT 2

A 10-year-old neutered female domestic shorthaired cat.

CASE PRESENTING SIGNS

Blindness, pacing and mental dullness.

CASE HISTORY

There was a 3-week duration of pacing and a 1-week duration of hindlimb ataxia and mental depression. The cat walked through its food bowl and into objects, pacing around the house for hours and sat facing corners. The cat's eyes had a 'glazed' appearance and yet the cat responded to its name and seemed to know when feeding time was due. The cat had been sneezing and a left ocular discharge was present.

CLINICAL EXAMINATION

Initial examination found an obtunded cat which paced around the examination room and stood in corners for prolonged periods. Posture and gait were remarkably normal. Left-sided placing was slightly reduced. Menace response was absent bilaterally. Pupil size was medium to large with the left slightly larger than the right pupil. Light shone in the left eye failed to constrict either pupil. Light shone in the right eye induced both pupils to constrict to a small degree. Fundic exam was normal. Spontaneous horizontal nystagmus fast phase to the right was present at rest. Vertical nystagmus was seen when the neck was dorsiflexed.

NEUROANATOMICAL DIAGNOSIS

The lesion was localized to the left optic nerve, the right optic nerve and/or left and right oculomotor nerves. The central vestibular apparatus was also affected, probably on the left given the lack of paradoxical vestibular signs. The obtunded mental state indicated either brainstem or widespread cerebral involvement. The pacing and pausing without apparent reason indicated cerebral disease.

The lesion could have been multifocal, or there may have been an expanding focus near the optic chiasm affecting vision and pupil control, causing raised ICP and compression of the brainstem.

CASE WORK-UP

MRI found thickened meninges on the rostral two-thirds of the brain, also involving the optic chiasm. The frontal sinus mucosa was also thickened but no communication between the brain and sinus was seen (Fig. 23.4). CSF was normal. Toxoplasma titres were negative. FIP titre was negative.



Figure 23.4 Transverse T1WI+C: thickened dura mater.

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Table 23.1 Systemic fungal disease with CNS involvement

Organism	Neurological signs	Other signs	Diagnosis
Blastomycosis (*B. dermatitidis*)	Rare in dogs Cats: diffuse or multifocal CNS	Lung, lymph node, skin, eye, bone	Cytology Antibodies CSF/serum
Histoplasmosis (*H. capsulatum*)	Uncommon Paresis, ataxia, CNN	Lung, lymph node, GIT, liver, spleen, eye, bone marrow, (skin)	Cytology
Coccidioides spp (*C. immitis*, *C. posadasii*)	Uncommon CNS, meninges	Lung, bone, skin, abdominal viscera, eye, heart, prostate	Antibodies CSF/serum (serious biohazard)

DIFFERENTIAL DIAGNOSIS

Meningoencephalitis, possibly bacterial or fungal spread from the sinus. Meningioma.

Systemic fungal disease

Fungal infections which gain access via inhalation or ingestion and then disseminate throughout multiple organs are known as systemic mycoses. Weight loss, lymphadenopathy, fever, and respiratory signs are common. A geographical distribution of the pathogens exist. Travel history is important. CSF protein is raised, and a mixed pleocytosis (± high percentage of eosinophils) is common. Cryptococcal organisms are commonly seen in the CSF of affected patients (Table 23.1).

Cryptococcosis

- Agent: *Cryptococcus neoformans*
- Exposure: inhalation. Bird droppings and eucalyptus bark and leaves
- Species: common in cats. Dogs. Immunosuppression not required.
- Organs: respiratory (nasal, nasopharynx, pulmonary), cutaneous (nodules, ulcerative, facial deformity), ocular (uveitis, optic neuritis) neurological (diffuse or focal meningoencephalitis)
- Neurological:
  - Access: Extension from the nose is common. Haematogenous to skin, eyes and CNS.
  - Presentation: In 20% of affected cats and 50–80% of affected dogs neurological signs may be the first sign of disease and result from involvement of the brain, cord, meninges or a combination.
- Diagnosis: 60–90% have organisms in the CSF. Fungal culture. Latex agglutination detecting capsular antigen in serum, CSF. Antibodies are found in healthy and diseased animals. Cytology on CSF, exudates, aspirates, bronchoalveolar lavage (BAL).
- Treatment: prolonged (months); fluconazole, itraconazole, 5-flucytosine, ketoconazole, amphotericin B
- Prognosis: guarded for resolution of CNS infection.

Nasal mycosis

Aspergillosis

- Agent: *Aspergillosis fumigatus*
- Exposure: ubiquitous. Not typically infectious. Normal nasal inhabitant
- Species: dogs (opportunistic pathogen). Rarely cats
- Organs: respiratory (chronic nasal disease, sinus, facial pain)
- Neurological access:
  - Extension through cribriform plate
  - Presentation: meningoencephalitis. Seizures. (Systemic *aspergillosis* is rare, fatal and affects multiple organs. There is no history of nasal involvement. A different organism is involved.)
- Diagnosis: difficult. Biopsy. Serology unreliable
- Treatment: topical clotrimazole
- Prognosis: poor/grave for CNS involvement.

DIAGNOSIS

Meningoencephalitis, meningioma.

TREATMENT

The cat's condition worsened and the animal was euthanized.

Histopathology of the brain found a highly undifferentiated tumour of the meninges extending into the neuropil. A poorly differentiated meningioma was the final diagnosis.

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24 The control of pupil diameter

Abnormal pupil function in the pet can lead the owners to report large 'black' eyes, a staring expression, or sunken eyes which have 'disappeared' behind the prolapsed third eyelids and ptosis of Horner's syndrome. The face may be described as having an 'odd expression'. Dilation of conjunctival vessels from sympathetic paralysis is reported as a red eye. Anisocoria may be noticed.

Pupils are normally symmetrically sized and shaped. Calm animals in an examination room have a pupil diameter of approximately 5 mm. Sympathetic drive actively dilates the pupils. Constriction is mediated by the parasympathetic branch of CN III via the ciliary nerves (Fig. 24.1). Structural change to the iris occurs with increasing age, and ocular pathology such as uveitis, glaucoma, lens luxation and synechia. *Dyscoria* describes a misshapen pupil. The feline iris is supplied by lateral and medial short ciliary nerves. If the lateral short ciliary nerve is affected, the pupil cannot constrict laterally and this gives a *reverse-D-shaped* pupil in the right eye and a *D-shaped* pupil in the left eye.

Unequal pupil size is called *anisocoria*. The difficulty is deciding which side is abnormal. Blue irises have a larger pupil size than brown, which can give rise to anisocoria in heterochromia iridis. The animal should be examined in ambient light, darkness and with the strong light source of an ophthalmoscope.

Ambient light

- Afferent lesions (CN II) create small subtle (2–3 mm) differences between the pupils.
- Efferent lesions (CN III) give rise to a large (7–8 mm) difference between the pupils.

Darkness

- Afferent lesions: both pupils dilate fully due to a lack of light stimulation and the presence of normal sympathetic tone.
- Efferent lesions:
  - Parasympathetic (CN III): equal and maximally dilated pupils
  - Sympathetic: the affected pupil is smaller as it fails to dilate fully.

Pupillary light reflex

- The *indirect pupillary light reflex* is produced by shining light into the contralateral eye. The indirect PLR is slower than the *direct PLR* due to the majority of afferent fibres decussating (twice) to supply the directly stimulated eye.
- The swinging flashlight test detects afferent lesions.

Hippus is a rhythmic, repetitive constriction and dilation of both pupils occurring independently of illumination, convergence or psychic stimuli. Its presence does not necessarily signal disease and does not have a localizing or prognostic value to date. Hippus has been reported in levothyroxine overdose and mucopolysaccharidosis Type II in the dog.

CLINICAL TIP

Assessing the PLR in conjunction with the menace response will localize the lesion to the shared afferent pathways of vision and pupil control, or to those anatomic areas distinct to each function.

MIOSIS

Miosis is an abnormal reduction in pupil size.

Unilateral miosis must be differentiated from contralateral mydriasis. It may result from Horner's syndrome, or unilateral ocular disorders causing pain, e.g. keratitis.

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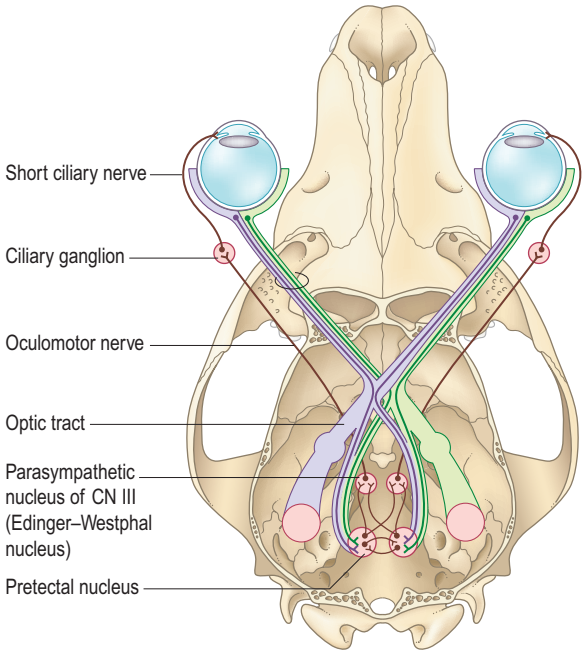


Figure 24.1 Pathway for pupil constriction.

Bilateral miosis occurs in dogs secondary to opiate use. Anterior uveitis, metabolic encephalopathies and structural brain lesions also cause bilateral miosis and would be accompanied by other diagnostic signs.

MYDRIASIS

A widely dilated pupil is described as *mydriatic*. *Mydriasis* may be unilateral or bilateral (Fig. 24.2).

It typically results from oculoparasympathetic paralysis either pharmacological (atropine) or structural in nature. The parasympathetic (ps) nucleus of CN III lies close to the CN III nucleus innervating the extraocular muscles. Midbrain lesions would therefore produce *internal ophthalmoplegia* (impairment of iris sphincter muscle) as well as *external ophthalmoplegia* (impairment of the extraocular muscles) in addition to deficits of gait and mental alertness. The preganglionic ps fibres have a superficial arrangement in CN III and are vulnerable to compression as CN III passes through the orbital fissure. Unilateral mydriasis is one of the first signs of imminent brain herniation due to compression of these peripherally located parasympathetic fibres.



Figure 24.2 Bilateral mydriasis.

Pharmacological paralysis may be bilateral or unilateral, depending on the method of drug administration, while CN III lesions are usually unilateral. Sympathomimetic drugs such as adrenaline produce mydriasis, as do opioids in cats (*cf.* miosis in dogs).

Pharmacological testing of parasympathetic lesions

Both eyes are tested with equal amounts of eye drops. The normal eye is used as the control.

Pilocarpine HCl 2% does not have a localizing value but indicates that the lesion is neurological. If the mydriatic pupil does not constrict but the control eye does, iridial disease or a pharmacological block (atropine) is responsible for the mydriasis (see Table 24.1).

Table 24.1 Localizing the origin of mydriasis

Drug	Pre-ganglionic lesion	Post-ganglionic lesion	Normal pupil (control)
Pilocarpine HCl 0.1% (direct-acting)	No constriction	Constriction	No constriction
Pilocarpine HCl 2% (direct-acting)	Constriction (>20 minutes)	Prompt constriction (sooner, greater, longer than control)	Constriction (within 20 minutes)
Physostigmine 0.5% (indirect-acting)	Constriction (40–60 minutes before the normal eye)	No constriction	Constriction

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25 Alteration in pupil function

INTRODUCTION

These two cases illustrate altered pupil function localized to different regions of the nervous system.

SIGNALMENT 1

An 8.5-year-old male Staffordshire bull terrier.

CASE PRESENTING SIGNS

Bumping into things.

CASE HISTORY

The dog suddenly became mentally dull and ataxic 5 days prior to referral. The dog bumped into objects, growled at the owner, slept a lot more and urinated indoors. Appetite and thirst had decreased. The dog fell to the left when getting off the sofa.

CLINICAL EXAMINATION

The dog was obtunded with a lowered head posture. The left menace response was absent and the pupils were bilaterally mydriatic and lacked a PLR. Fundic examination was normal. The dog walked slowly and on one occasion was seen to lean to the left. Hopping was slightly slower in all limbs. Proprioception was normal.

NEUROANATOMICAL LOCALIZATION

The lesion was localized to the midbrain to explain the dilated pupils lacking a PLR. An absent menace can be due to a depressed mental state, however the change should be bilateral. A left cerebellar lesion could explain the left menace deficit and falling to the left. A right cerebral lesion or left optic nerve lesion could also account for the menace deficit in this dog in which case the lesion may be in the region of the optic chiasm and compressing the thalamus.

DIFFERENTIAL DIAGNOSIS

- A focal lesion of the brainstem with secondary effects on the cerebellum or cerebrum
- Neoplasia
- Haemorrhage
- Inflammation.

CASE WORK-UP

MRI found a contrast-enhancing lesion which was isointense on T1 and of variable intensity on T2WI. There was secondary dilation of the third and lateral ventricles (Fig. 25.1). Within the third ventricle choroid plexus papilloma (CPP) and ependymoma are intraventricular in location and enhance uniformly. Meningiomas can be intraaxial.

DIAGNOSIS

Midbrain compression causing mydriasis.

SIGNALMENT 2

A 3.5-year-old neutered male collie cross dog.

CASE PRESENTING SIGNS

Lethargy, vomiting and protrusion of the third eyelids.

CASE HISTORY

The dog had been lethargic for 1 week, lagging behind on exercise and lacking interest on a walk. He had vomited twice. Two days prior to referral, bilateral protrusion of the third eyelids occurred, followed the next day by squinting in the light and bilateral dry nasal

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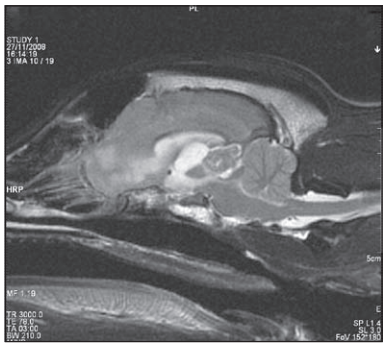


Figure 25.1 Sagittal T2WI showing lesion compressing midbrain and aqueduct causing dilation of the third ventricle.

discharge. The dog appeared to be urinating more often but was not polydipsic.

CLINICAL EXAMINATION

The dog had a depressed mental state, dry oral mucous membranes and bilateral dry nasal discharge. There were normal menace responses and absent PLRs bilaterally. The pupils were midsized with the right slightly smaller than the left. The third eyelids were prolapsed. Right enophthalmos and right ptosis were present.

Gait, posture, proprioception, hopping and spinal reflexes were all normal.

The Schirmer Tear Test (STT) was 0 mm bilaterally.

A 10% phenylephrine testing of the right eye produced a normal palpebral fissure size and third eyelid appearance in 5 minutes.

NEUROANATOMICAL LOCALIZATION

Dry mouth (xerostomia)

Lack of saliva impedes the formation of a food bolus but xerostomia is not a reported cause of dysphagia in small animals. A unilateral facial nerve lesion commonly results in dry eye but it would be impossible to clinically recognize decreased saliva production from the same lesion.

Cause

1. Panting
2. Dehydration
3. Decreased salivary gland secretion
   - Parasympathetic failure: drugs, dysautonomia, CNN VII and IX lesions
4. Uraemia.

Dry nose (xeromycteria)

Dry nose presents as a dry nasal plane (unilateral or bilateral), epithelial crusts and a slight mucopurulent discharge.

Serous glands of the nasal plane have a parasympathetic innervation by the facial nerve. The afferent stimulus for nasal secretion is transmitted by the trigeminal nerve. Pathological dryness of the nose, xeromycteria, results from:

1. Failure of parasympathetic innervation
   - Parasympatholytic drugs such as atropine
   - Dysautonomia
2. Facial nerve paralysis
3. Epithelial damage, e.g. Canine distemper virus can cause hyperkeratosis of the nasal plane and digital pads
4. Trigeminal nerve lesion.

Reduced tear production: dry eye

The precorneal tear film's three components are: (1) a superficial lipid layer which prevents evaporation of (2) the middle aqueous layer that is bound to the cornea by (3) the inner mucoid layer.

The aqueous layer is predominantly composed of water secreted chiefly by the lacrimal gland. Parasympathetic branches of CN VII innervate the lacrimal gland. Two-thirds of aqueous tear production is in response to sensory stimuli (reflex secretion) and the sensory arc is carried by the ophthalmic branch of CN V. Basal tear production is governed by CN VII.

Dry eye, xerophthalmia, results from impaired secretion of the lacrimal glands and presents with a dull appearance to the cornea, dry, sticky mucopurulent discharge adherent to the cornea, corneal ulcers, pigmentation and neovascularization – 'red eye' (conjunctivitis).

Causes

1. Failure of secretion by the lacrimal gland:
   - Lack of afferent innervation: trigeminal nerve (neurotropic keratitis)
   - Lack of efferent innervation: facial nerve
   - Destruction of the gland: drugs, autoimmune or idiopathic.
2. Increased evaporation by failure of eyelids to close (CN VII or generalized muscle weakness).
3. Non-neurological: inflammation: idiopathic or immune-mediated; drugs or general anaesthesia.

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Diagnosis

The STT measures the aqueous tear film.

- Dry eye + dry mouth (xerostomia): immune-mediated destruction of both glands is rare. It may present as dysphagia.
- Dry eye + dry nose (xeromycteria): could be neurogenic (CNN V or VII). Consider dysautonomia.
- Dry eye + dry nose + dry mouth: consider dysautonomia.

The lesion in this dog was localized to the autonomic nervous system.

DIFFERENTIAL DIAGNOSIS

Canine dysautonomia

Widespread dysfunction of the sympathetic and parasympathetic nervous systems occurs sporadically in cats and dogs and is called *dysautonomia* (Table 25.1). Feline dysautonomia used to be known as Key–Gaskell syndrome. Animals less than 3 years of age of either sex are most frequently affected with signs developing acutely within 2 days to 2 weeks. Parasympathetic signs predominate. Mydriatic pupils unresponsive to light are a consistent finding. Degenerative change also occurs in cranial nerve nuclei, dorsal root ganglia and ventral horn cells accounting for the involvement of skeletal muscle.

Diagnosis is based on clinical signs. Pharmacological testing of the pupil may help.

Treatment is supportive. Prognosis is guarded but not uniformly hopeless.

Table 25.1 Clinical signs of dysautonomia

Organ	Clinical sign
Eye	Dilated unresponsive pupils
	Photophobia
	Blepharospasm
	Protrusion of the third eyelid
Glands	Dry mouth, nose, eye
GIT	Slow gastric emptying
	Generalized ileus
	Constipation (cat)
	Diarrhoea (dog)
	Megaoesophagus
	Vomiting, retching, regurgitation, dysphagia
	Anorexia, weight loss
Cardiac	Bradycardia unresponsive to exercise or stress
Urinary	Urinary and faecal incontinence
	Distended bladder
	Dysuria
Other	Depression, lethargy
	Dilated anal sphincter
	Hind limb (HL) proprioceptive deficits

AUTONOMIC FUNCTION TESTING

Pupillary reaction

Pilocarpine 0.1% test

1. 1% pilocarpine diluted 1:10 with irrigating solution
2. Apply 1 drop to one eye
3. Monitor every 15 minutes for development of miosis
4. Result:
   - No change after 60 minutes: normal
   - Miosis after 60 minutes: parasympathetic denervation; 87% of affected dogs responded in one study
5. If no response after 90 minutes, apply 1–2% pilocarpine. If the iris cholinergic receptors are blocked by atropine there will no response.

Another method uses 1–2 drops of 0.05% pilocarpine solution placed in one eye. Dysautonomic dogs are said to show rapid constriction with some normal animals responding within 45–60 minutes.

Tear production

Schirmer Tear Test I measures tear production. Dry eye <5 mm/60 seconds. Normal: 15–20 mm/60 seconds (see page 30).

Heart rate variability

Atropine response test

1. Measure heart rate
2. 0.02 mg/kg atropine IV
3. Heart rate measured every 5–10 minutes for up to 60 minutes
4. The maximum change in the heart rate is measured. Healthy dogs are expected to have an increase in heart rate of more than 40 bpm or an end-test heart rate of more than 140 bpm.

In dysautonomia, no significant change in heart rate occurs during a 60 minutes period.

Blood pressure variability

Orthostatic hypotension test

1. Place the dog in lateral recumbency on a firm but moveable surface.

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2. Measure the indirect blood pressure in the fore- and hindlimb simultaneously.
3. Elevate the dog by 30°, with the forelimbs higher than the hind.
4. Results may be inconclusive. Ideally, the normal animal will show little change in blood pressure when tilted and the dysautonomic dog will demonstrate a drop in blood pressure in the forelimbs and an elevated pressure in the hind.

Sympathetic skin response

Intradermal histamine test

1. Clip an area of skin on the lateral thorax.
2. Inject 0.05 ml of histamine (1 : 10 000) intradermally next to an equal quantity of 0.9% saline solution as a control.
3. Monitor for 30 minutes for development of a:
   - Wheal: local oedema from an increased capillary permeability caused by the direction actions of histamine on the blood vessels.
   - Flare: redness extending from the site of injury due to arteriolar dilation caused by a sympathetic neuron reflex.
4. Normal dog: visible wheal and flare.

Dysautonomia: absence of a flare response in 97% of dogs. Wheal developed in 50% of dogs.

Bladder function

Bethanechol challenge

1. 0.0375 mg/kg bethanechol chloride injected SC.
2. Subjective evaluation of dog's ability to evacuate bladder 30 minutes after the injection.
3. Detrusor atony secondary to prolonged distension may hamper bladder contraction.

DIAGNOSIS

Canine dysautonomia was diagnosed based on the lack of pupil constriction, reduced oral, nasal and lacrimal secretions and vomiting. No megaoesophagus was found on thoracic radiographs.

FOLLOW-UP

The dog improved over 2 months with supportive care. The right third eyelid occasionally protruded. STT remained abnormal on the right (6 mm) but had improved on the left (16 mm).

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26 Secretions

INCREASED TEAR PRODUCTION: EPIPHORA

Presenting signs

Overflowing tears, tear staining of the medial canthus.

Cause

Reflex lacrimation occurs in response to eye pain which should be evident on examination. Owners sometimes report epiphora occurring a few days before ipsilateral facial paralysis develops. Parasympathetic stimulation from organophosphate toxicity results in SLUD (salivation, lacrimation, urination, defecation).

Obstruction of the tear duct is a common cause of tear staining.

Paradoxical ('crocodile') tears occur in the human during and immediately after eating as a rare phenomenon secondary to aberrant reinnervation of CN VII when the fibres innervating the salivary glands are redirected to the lacrimal gland. This may be tested with 1% tropicamide placed on the tongue as a sialogogue. Excess tearing is seen in the ipsilateral eye after approximately 1 minute.

WET NOSE

Presenting signs

Clear watery (serous) nasal discharge from one or both nostrils may be normal. It is an unreliable indicator of general wellbeing hence the thermometer's supremacy. Systemic causes are usually bilateral.

Causes

- Early sign of nasal irritation by fungi, parasites, foreign bodies or neoplasia
- Viral or allergic
- Rhinorrhoea: CSF leaking from the nose caused by Congenital fistula or head trauma
- Over-hydration.

SALIVATION

The parotid, mandibular, sublingual and zygomatic glands secrete saliva into the oral cavity under the influence of the parasympathetic nervous system. The parotid and zygomatic glands are innervated by CN IX. The facial nerve (CN VII) innervates the mandibular and sublingual glands. In both cases, the post-ganglionic fibres travel in association with the mandibular branch of the trigeminal nerve (CN V).

Salivation is a reflex which is stimulated by taste (CNN VII, IX) and tactile sensation in the mouth (CN V) and gastric end of the oesophagus (CN X).

Higher centres initiate salivation, as demonstrated by anticipation of a food reward, or as part of an anxiety response.

EXCESSIVE SALIVATION

Presenting sign

Drooling, dribbling, strings of saliva hanging from mouth, frequent swallowing.

Excessive salivation is more commonly due to an inability to swallow (pseudoptyalism) than an excessive production (ptyalism, hypersalivation, sialism, sialismus, sialorrhoea, sialosis). Drooling is one of the first signs of facial paralysis noticed by the owner. Finding their pet with wet fur under the chin is often the first evidence of seizure activity.

Conditions causing oropharyngeal irritation or pain are far more common than neurological lesions as the cause of excessive salivation.

Excessive production

- Nausea
- Medication: stimulated by taste; opioids (dog); organophosphates
- Hepatic encephalopathy (cats)

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- Purring
- Hyperthermia
- Seizures
- Anxiety
- Anticipation of food.

Overflow of saliva

- Inability to close jaws (CN V) or move lips (CN VII)
- Cannot or will not swallow (dysphagia)
- Seizures.

DRY MOUTH (XEROSTOMIA)

This has not been reported as a primary problem. Lack of saliva impedes formation of a food bolus but xerostomia has not been a reported cause of dysphagia in small animals. A unilateral facial nerve lesion commonly results in dry eye but it would be impossible to clinically recognize decreased saliva production from the same lesion.

Cause

1. Panting
2. Dehydration
3. Decreased salivary gland secretion
   - Parasympathetic failure: drugs, dysautonomia, CNN VII and IX lesions
4. Uraemia.

DRY NOSE (XEROMYCTERIA)

Dry nose presents as a dry nasal plane (unilateral or bilateral), epithelial crusts and a slight mucopurulent discharge.

Serous glands of the nasal plane have a parasympathetic innervation by the facial nerve. The afferent stimulus for nasal secretion is transmitted by the trigeminal nerve. Pathological dryness of the nose, xeromycteria, results from:

1. Failure of parasympathetic innervation
   - Parasympatholytic drugs such as atropine
   - Dysautonomia
2. Facial nerve paralysis
3. Epithelial damage, e.g. canine distemper virus can cause hyperkeratosis of the nasal plane and digital pads
4. Trigeminal nerve lesion.

REDUCED TEAR PRODUCTION: DRY EYE

The precorneal tear film's three components are: (1) a superficial lipid layer which prevents evaporation of (2) the middle aqueous layer that is bound to the cornea by (3) the inner mucoid layer.

The aqueous layer is predominantly composed of water secreted chiefly by the lacrimal gland. Parasympathetic branches of CN VII innervate the lacrimal gland. Two-thirds of aqueous tear production is in response to sensory stimuli (reflex secretion) and the sensory arc is carried by the ophthalmic branch of CN V. Basal tear production is governed by CN VII.

Dry eye, xerophthalmia, results from impaired secretion of the lacrimal glands and presents with a dull appearance to the cornea, dry, sticky mucopurulent discharge adherent to the cornea, corneal ulcers, pigmentation and neovascularization – 'red eye' (conjunctivitis),

Causes

1. Failure of secretion by the lacrimal gland:
   - Lack of afferent innervation: trigeminal nerve (neurotropic keratitis)
   - Lack of efferent innervation: facial nerve
   - Destruction of the gland: drugs, autoimmune or idiopathic.
2. Increased evaporation by failure of eyelids to close (CN VII or generalized muscle weakness).
3. Non-neurological: inflammation: idiopathic or immune-mediated; drugs or general anaesthesia.

Diagnosis

The Schirmer Tear Test (STT) measures the aqueous tear film.

- Dry eye + dry mouth (xerostomia): immune-mediated destruction of both glands is rare.
- Dry eye + dry nose (xeromycteria): could be neurogenic (CNN V or VII). Consider dysautonomia.
- Dry eye + dry nose + dry mouth: consider dysautonomia.

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27 Alteration of eyelid position and movement

INTRODUCTION

The palpebral fissure is the opening between the eyelids. It forms at 7–10 days in the newborn cat and dog. Opening and closing the palpebral fissure is called blinking. Blepharospasm is a sustained or intermittent, involuntary forced closure of the palpebral fissure. A 'squint', in medical parlance, describes strabismus, not the palpebral fissure narrowing of blepharospasm.

PRESENTING SIGNS

- Ptosis: half-closed eyes, droopy eyelid(s). The contralateral eye may appear exophthalmic by comparison.
- Absent blink: staring appearance, sleeps with the eyes open, secondary corneal pathology
- Blepharospasm: Holds eye(s) shut, 'avoids the light', photophobia, excessive blinking
- Protrusion of the third eyelid: the eye 'disappears'
- Narrower or wider palpebral fissure: the eye appears smaller, recessed, sunken, or bigger, bulging or enlarged.

Innervation of the palpebral fissure

The size of the palpebral fissure is chiefly controlled by CN III and sympathetic neurons which elevate the upper eyelid (Fig. 27.1). Failure of one or both causes a drooping of the upper lid called ptosis (Table 27.1).

Eye closure is achieved by contraction of the orbicularis oculi muscle, innervated by the facial nerve (CN VII) (Table 27.2).

CN VII paralysis prevents blinking. The lack of orbicularis oculi muscle tone may cause the lower lid to droop, thus slightly widening the palpebral fissure in facial paralysis.

Stimuli for blinking

Reflex blinking occurs in response to:

- Visual stimuli (CN II), e.g. dazzle reflex, menace response
- Touch or pain (CN V), e.g. blink reflex, ocular irritation
- Sound (CN VIII), e.g. sudden loud startling noise.

Reflex eyelid contraction can occur in synchrony with the rate of spontaneous nystagmus of vestibular disease. Both features appear, and resolve, in concert with each other.

Excessive blinking or involuntary eye closure (blepharospasm) is most commonly caused by ocular pain. Additional clues such as epiphora and conjunctival hyperemia should be present. Photophobia, the dislike and avoidance of light, is due to ocular pain in the presence of bright light. It is a sign of meningeal irritation.

Dysfunction of the central controls of voluntary blinking may give rise to an increased blink rate, eyelid spasms or eyelid tic. This is known as 'essential' blepharospasm and is a form of dystonia reported in humans. Sometimes other facial muscles are involved and hemifacial spasm occurs.

Causes of a narrow palpebral fissure

- Enophthalmos
- Oculomotor nerve paralysis
- Horner's syndrome
- Blepharospasm
- Hemifacial spasm
- Tetanus.

Causes of a wider palpebral fissure

- Exophthalmos
- CN VII paralysis
- Cerebellar nuclei lesion.

Third eyelid protrusion

The third eyelid is kept in position by the forward position of the globe. Only a small portion is normally visible ventromedially in the palpebral fissure. Smooth muscle from the periorbita attaches to the base of the third eyelid and aids its retraction. Movement of the

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Table 27.1 Opening the palpebral fissure

	Muscle	Innervation	Action
Upper eyelid: open	Levator palpebrae superioris	CN III	Elevation and retraction of upper eyelid; (chief elevator of eyelid)
	Levator anguli oculi medialis	Palpebral branch of CN VII	Lifts nasal portion of eyelid and erect hairs of eyebrow
	Müller's smooth muscle: medial component	Postganglionic sympathetic nerves (infratrochlear division of nasociliary nerve)	Elevates upper eyelid, and retracts dorsal portion of third eyelid
Lower eyelid: open	Müller's smooth muscle; inferior component	Sympathetic fibres travelling in Maxillary division of CN V	Depresses lower eyelid and retracts lower half of the third eyelid
	Pars palpebralis of the sphincter colli profundus	Buccal branches of CN VII	Depressor of the lower eyelid during opening

Table 27.2 Closing the palpebral fissure

Muscle	Innervation	Action
Orbicularis oculi	Branch of CN VII	Sphincter-like closure of palpebral fissure. Depresses the eyebrow
Retractor anguli oculi lateralis	Branch of CN VII	Draws lateral palpebral angle caudally

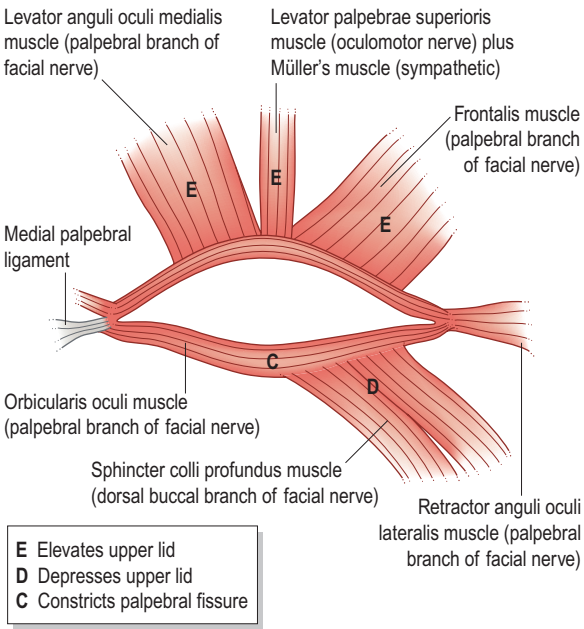


Figure 27.1 Musculature of the eyelids.

Table 27.3 Third eyelid protrusion

Causes of third eyelid protrusion	Examples
Enophthalmos	Corneal or conjunctival irritation Atrophy of masticatory muscles Atrophy of extraocular muscles Loss of orbital fat Dehydration Denervation of orbital smooth muscles (Horner's syndrome) Tethering of the globe by retrobulbar neoplasia Congenital microphthalmia, phthisis bulbi
Sympathetic paralysis	Feline dysautonomia (accompanied by mydriasis) Horner's syndrome (accompanied by miosis)
Retraction of the globe by the retractor bulbi muscle.	Tetanus Facial paralysis (absent blink reflex)
Systemic illness	Cats with systemic illness (Haws syndrome)
Chemical sedation	

third eyelid is passive in the dog. The cat has smooth muscle, which draws the third eyelid across the globe (Table 27.3).

SIGNALMENT

A 1-year-old neutered male Burmese cat.

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CASE PRESENTING SIGNS

Abnormal gait.

CASE HISTORY

Hindlimb ataxia commenced suddenly 3 weeks prior to referral. The cat became progressively more lethargic, developed dilated pupils and prolapsed third eyelids.

CLINICAL EXAMINATION

The cat was mentally dull and walked around the room, circling to the right with a high-stepping forelimb gait. There was a slight unsteadiness when walking, which occasionally appeared worse in the hindlimb. The cat lent slightly to the right and fell to the right when first removed from the cat carrier. Placing was slightly slower in the right fore- and hindlimbs but this was inconsistent. The eyes were partially open when walking but held closed at rest.

When the lids were parted, the pupils looked symmetric, large and lacked pupillary light reflexes bilaterally. The dazzle reflex was present and the fundus looked normal bilaterally. No abnormal nystagmus or strabismus was seen. Facial movement and sensation and the gag reflex were all normal, as were spinal reflexes.

NEUROANATOMICAL LOCALIZATION

On the basis of the dullness, blepharospasm, mydriasis, lack of PLR, circling to the right, leaning and falling to the right, the neuroanatomic site was thought to be multifocal with midbrain, cerebellar, and possibly cerebral involvement (Fig. 27.2).

DIFFERENTIAL DIAGNOSIS

- Inflammation
- Malformation.

CASE WORK-UP

The feline infectious peritonitis (FIP) titre was high at 3100 (N <10).

On MRI there was:

- A contrast-enhancing focal midbrain lesion with some enhancement of the third ventricle
- Secondary hydrocephalus of the lateral and third ventricles
- Meningeal and ependymal contrast-enhancement.

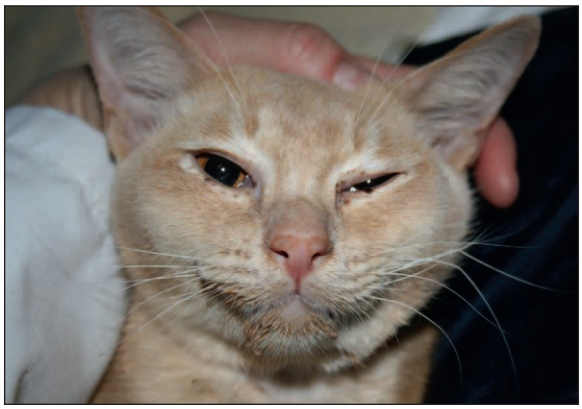


Figure 27.2 Blepharospasm, mydriasis, obtunded.

DIAGNOSIS

The contrast-enhancement of the meningeal and ependymal surfaces was suggestive of FIP.

AETIOPATHOGENESIS OF FIP

Feline infectious peritonitis affects the CNS and eye with minimal effusions into body cavities. The systemic infection causes anorexia, weight loss, fever and depression. Neurological deficits are commonly cerebellovestibular in nature but may present as a focal spinal cord lesion giving rise to paresis and ataxia. Diagnosis is made on histopathology but an index of suspicion is created from the clinical presentation, imaging and lab work.

MRI may find contrast-enhancement of the meninges or the ependymal lining of the ventricles. Inflammation can block the ventricular outflow giving rise to hydrocephalus. Proteinaceous CSF has an altered MRI intensity, improving its visibility on FLAIR.

CSF test results are variable. They may demonstrate a high protein level and pleocytosis (mononuclear or mixed), or be normal.

Serology titres do not correlate with the severity of the disease and can fluctuate. Persistently high (≥640) feline coronavirus (FCoV) antibody titre indicates an increased risk of persistent FCoV infection. Titres ≥16 000 are suggestive of FIP.

A serum albumin : globulin ratio <0.5 increases the index of suspicion for FIP. While an increase in the acute phase protein alpha1-AGP ≥1500 μg/ml is not specific for FIP, a level <500 μg/ml is usually found in healthy cats. A non-regenerative anaemia, lymphopaenia and

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Table 27.4 Viral infection of the feline CNS

Organism	Neurological signs	Other signs	Diagnosis	Treatment
FIV	Uncommon. CNS/PNS Behaviour change, seizures, dementia, ataxia	Stomatitis, neoplasia, anaemia, uveitis	Antibodies	
FIP	Common. CNS	Vasculitis, kidney, liver, spleen, body cavities, eye	Histopathology Antibody in CSF	Prednisolone 4 mg/kg PO SID
Feline panleukopaenia/ parvovirus	Kitten. Cerebellar hypoplasia, seizures, behaviour change	Panleukopaenia enteritis		GIT: supportive. CNS: none
Feline Borna virus	Rare. Hindlimb ataxia, paresis, hyperaesthesia, seizures, behaviour change	Anorexia, fever, increased salivation		Fatal within 6 months: Supportive

mild neutrophilia with a left shift are often found on haematology.

FIP is most common in cats 6 months to 3 years of age originating from a multi-cat household. All neurologically affected cats die. Immunosuppressive doses of prednisolone (4 mg/kg/day PO) may slow progression.

Feline immunodeficiency virus encephalomyelitis is more frequently noted on histopathology than as a clinical manifestation. It is estimated that 5–30% of clinically affected FIV-positive cats develop encephalopathy. Behavioural change, twitching of the face and tongue, seizures, nystagmus, ataxia, and intention tremor have been reported. There is minimal if any progression of signs.

Feline leukaemia virus predisposes the cat to developing lymphoma. Multiple skeletal lesions of osteochondromatosis occur in young infected cats. Olfactory neuroblastoma is a rare FeLV-associated neoplasm of the nasal cavity which invades through the cribriform plate and may cause neurological signs (Table 27.4).

PROGNOSIS

Poor.

TREATMENT

Prednisolone was commenced to treat the suspected vasculitis associated with FIP.

OUTCOME

The cat became recumbent, stuporous and was euthanized.

Postmortem examination revealed a severe pyogranulomatous meningoencephalitis resulting in a narrowed mesencephalic aqueduct and interventricular foramina. Transtentorial herniation of the occipital lobes displaced the cerebellum to herniate via the foramen magnum. Inflammation was angiocentric. The diagnosis was FIP.

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28 Horner's syndrome

INITIAL PRESENTATION

Eye appears sunken or 'disappears' from view. Odd appearance to the animal's face.

INTRODUCTION

Horner's syndrome describes the constellation of signs resulting from paralysis of the oculosympathetic pathway anywhere along its considerable length. It is a common cause of anisocoria in small animals.

Signs

Miosis: Anisocoria is most obvious in darkness or during excitement or fear when the failure to dilate fully is noted. It is less obvious when the animal is fatigued or drowsy as pupils naturally constrict as sleep approaches.

Ptosis: Denervation of the smooth muscle of the upper eyelid creates ptosis. The palpebral fissure may be narrowed further by denervation of the smooth muscle of the lower lid which causes it to be raised.

Enophthalmos: Loss of sympathetic tone to orbital smooth muscle and the resulting unopposed action of the retractor bulbi muscle causes the eye to sink further into the orbit.

Protrusion of third eyelid: Denervation of smooth muscle in the eyelid along with an enophthalmic position results in a prominent third eyelid.

Vasodilation of facial, aural, or conjunctival blood vessels may be noted. Ipsilateral coat colour change has been reported in Siamese cats secondary to the local temperature increase.

Figure 28.1 shows a left Horner's syndrome.

Pathway

The sympathetic innervation of the eye originates in the hypothalamus, descends through the brainstem and spinal cord to synapse within thoracic spinal cord segments T1–T3. This section of the pathway is referred to as the *First Order*. Axons exit via T1–3 nerve roots and pass through the thorax and several ganglia and travel rostrally up the neck in the vagosympathetic trunk to synapse in the cranial cervical ganglion. This anatomical division is referred to as the *Second Order*. Finally, the axons travel through the middle ear, along the floor of the skull and exit via the orbital fissure to innervate the smooth muscle of the eye, including the pupil dilators. The postganglionic pathway from the cranial cervical ganglion to the pupil dilators is referred to as the *Third Order*.

Lesions of the oculosympathetic pathway (Fig. 28.2) can be categorized as first, second, or third order according to their anatomical site which can be most reliably discovered by finding concomitant neurological deficits. Pharmacological testing may help localize the lesion. It makes use of the phenomenon of denervation hypersensitivity which develops 8–10 days after a postganglionic lesion develops. A denervated receptor responds to a dilute concentration of neurotransmitter whereas a normal receptor would not. This may be helpful in differentiating second from third order lesions. thoracic radiography is used to investigate second order lesions.

SIGNALMENT 1

A 10-year-old male Labrador retriever.

CASE HISTORY

This dog had a 7-day history of left third eyelid protrusion, miosis and ptosis with reddened conjunctiva. The eye was fluorescein negative. The eye looked most abnormal when the dog was resting.

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Figure 28.1 Left Horner's syndrome.

CLINICAL EXAMINATION

The dog showed left ptosis, enophthalmos, and miosis in light and darkness. The menace response and PLR were normal bilaterally.

NEUROANATOMICAL LOCALIZATION

The lesion was localized to the sympathetic nerve supply to the left eye and 10% phenylephrine drops reversed the signs of Horner's syndrome within 20 minutes, which suggested denervation hypersensitivity was present and hence a third order Horner's syndrome.

DIFFERENTIAL DIAGNOSIS

Lack of other neurological deficits indicated that idiopathic Horner's syndrome was the most likely cause. There had been no history of otitis externa, balance loss or facial paralysis in this dog which made middle ear disease less likely.

CASE WORK-UP

Thoracic radiographs were taken to further investigate the pathway of the sympathetic nerve supply to the eye. This would be classed as a second order lesion, and theoretically should have been ruled out by the pharmacological testing. Radiographs were normal.

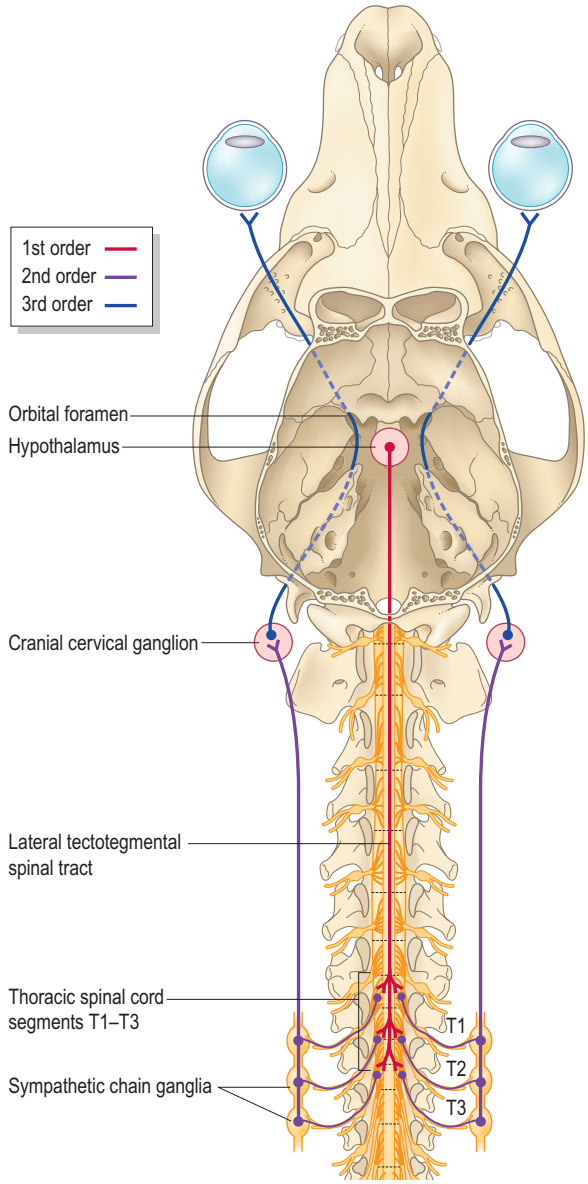


Figure 28.2 Oculosympathetic pathway.

Pharmacological testing of Horner's syndrome

Place 1–2 drops of 10% phenylephrine in both eyes. The normal eye acts as a control. Note the time the drops were applied. Examine the pupil size in a darkened room.

- Third order lesion: pupil dilation ≤20 minutes
- Second order lesion: pupil dilation ≈45 minutes
- First order lesion: pupil dilation ≈60–90 minutes

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This is not 100% reliable for localizing lesions; 2.5% dilutions and 0.1% phenylephrine (stock 10% solution diluted 1 : 100 with saline solution) have also been used in the past.

DIAGNOSIS

Idiopathic Horner's syndrome

Approximately 45% of canine and 25% of feline Horner's syndrome cases are idiopathic. Idiopathic Horner's syndrome commonly occurs in golden retrievers, and the lesion is thought to be postganglionic. Signs may resolve spontaneously within 1–8 months.

The prognosis for Horner's syndrome is determined by the site and the cause of the lesion. Horner's syndrome occurring with cervical spinal cord disease signifies damage to the lateral tectotegmentospinal tract and has not been used as a prognostic sign for recovery.

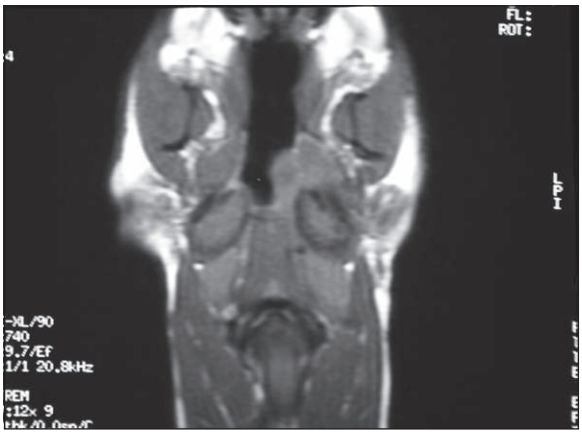


Figure 28.3 Dorsal T1WI. Hyperintense material rostral and caudal to the left tympanic bulla, and caudal to the right bulla.

FOLLOW-UP

No further clinical signs developed in the dog.

SIGNALMENT 2

A 12-year-old neutered female Siamese cat.

PRESENTING SIGNS

Nystagmus and left Horner's syndrome.

CASE HISTORY

This cat had had otitis externa for 12 months and left Horner's syndrome for 4 months prior to referral. The cat had been mentally depressed and inappetant for the last 3–4 days.

CLINICAL EXAMINATION

Clinical examination revealed a mentally depressed cat with left enophthalmos, left ptosis but no anisocoria in either light or darkness. The ears had a foul smell and were painful when palpated. An intermittent pendular nystagmus was present and thought normal for the breed. Pus was aspirated from the right external ear canal.

NEUROANATOMICAL LOCALIZATION

The neurological deficit was localized to the sympathetic nerve supply to the left eye. No evidence of vestibular disease was present. Pain probably accounted for the depression. Extension of bacterial otitis media to the cranial vault could also cause mental depression.

DIFFERENTIAL DIAGNOSIS

- Otitis media-externa
- Polyp
- Osteomyelitis of the tympanic bullae
- Extension of infection into the cranial vault.

CASE WORK-UP

MRI: material in right and left ear canals and bullae (Figs 28.3 and 28.4). Swelling/inflammation around the left pterygoid bone. Contrast-enhancement of thickened dura on the left cerebrum.

CSF: normal. (WBC 0, RBC 0; Total protein 0.19 g/l with cytology of scattered lymphocytes and monocytes).

TREATMENT

The cat became a lot brighter on IV fluids and buprenorphine pain relief. She ate well.

Surgery: combined ventral bulla osteotomy and total ear canal/lateral bulla osteotomy of the left and right ears. There was an eroded bulla wall ventrally. A polyp was found in the dorsolateral compartment of the bulla. Pus was found in the middle ears.

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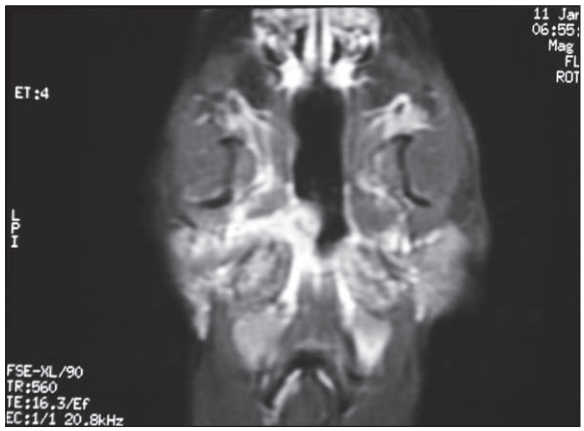


Figure 28.4 Dorsal T1WI+C. Contrast-enhancement of soft tissue around the left bulla and right bulla.

The cat developed facial paralysis bilaterally after the surgery.

An interesting phenomenon has been noted in which hyperactive function of the postganglionic sympathetic nerve to the eye was suspected in cats after their ears had been flushed

Iatrogenic pupillary dilation (Pourfour du Petit syndrome)

Unilateral mydriasis and exophthalmos was reported in cats within 2 hours of endoscopically examining the middle ear and flushing the tympanic cavity using a Spruell needle. The PLR was brisk but incomplete on the affected side. The ipsilateral palpebral fissure was wider and eyelid closure was normal. The signs were thought to be due to a mild stimulation of the postganglionic sympathetic nerves as they passed through the tympanic cavity resulting in a hyperactive function rather than paralysis. Signs resolved within 1–7 days. Ipsilateral Third Order Horner's syndrome developed in one cat 4 days after the initial signs. This condition is rare (i.e. three cats over a 2-year period from one author performing a common procedure).

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29 Idiopathic facial paralysis

INITIAL PRESENTATION

Acute

Dropped or drooping flaccid upper and lower lips, dropped ear, lopsided face, odd look to face, food collecting in the mouth between the flaccid lip and the teeth, dropping food, drooling saliva (and secondary head shaking), inability to blink, sleeping with eyes open, widened palpebral fissure, dry eye, corneal ulcers, dry nose and deviation of the nose can all occur (Fig. 29.1).

Chronic

Elevation of the lip and ear, widening of the palpebral fissure and contracture of the facial muscles can occur.

INTRODUCTION

The facial nerve innervates the:

- Muscles of facial expression: ears, eyelids, lips, cheeks and nose
- Sublingual and mandibular salivary glands
- Nasal and lacrimal glands.

It also transmits sensory fibres from the taste buds of the rostral tongue but dysfunction is not noticed.

Facial nerve lesions typically cause paralysis of the muscles of facial expression. In time, the flaccid muscles atrophy, undergo *fibrosis and contracture* and thus assume a more normal position. Voluntary and reflex movement of the contracted muscles is still absent. The contracture does not relax under GA.

LMN paresis or paralysis is the most common sign of dysfunction, however 'positive' signs of nerve irritation have been reported:

Hemifacial spasm is a unilateral regular tonic-clonic contraction of one or more muscles of facial expression. It results from an irritation of the facial nerve but has been reported in dogs in concert with intracranial masses physically distinct from the facial nerve. The spasm relaxes under general anaesthesia which helps distinguish it from the fibrosis and contracture secondary to chronic facial paralysis.

Animals present with facial asymmetry, elevation and wrinkling of the upper lip, caudal displacement of the labial commissure, blepharospasm, elevation of the ear, deviation of the nose to the ipsilateral side and hyperactive palpebral (blink) reflex.

Myotonia causes a delayed relaxation of muscle following stimulation. This could give the appearance of a hemifacial spasm. Focal tetanus is limited to the limbs. The face is affected bilaterally in generalized tetanus. Other differentials of hemifacial spasm include: myokymia, blepharospasm, facial tic and partial seizure.

Epiphora is an increased tear production and appears as overflowing tears or tear staining of the medial canthus.

Reflex lacrimation occurs in response to eye pain which should be evident on examination. Owners sometimes report epiphora occurring a few days before ipsilateral facial paralysis develops. Parasympathetic stimulation from organophosphate toxicity results in SLUD (salivation, lacrimation, urination, defecation).

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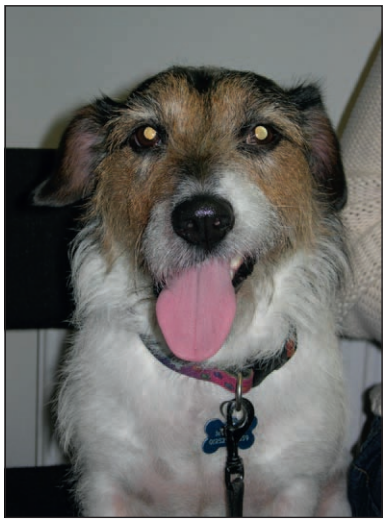


Figure 29.1 Acute paralysis of R VII. The right ear is slightly more ventral. The right upper lip is noticeably lower.

Obstruction of the tear duct is a common cause of tear staining.

Paradoxical ('crocodile') tears occur in the human during and immediately after eating as a rare phenomenon secondary to aberrant reinnervation of CN VII when the fibres innervating the salivary glands are redirected to the lacrimal gland. This may be tested with 1% tropicamide placed on tongue as a sialogogue. Excess tearing is seen in the ipsilateral eye after approximately 1 minute.

The serous glands of the nasal plane are innervated by the facial nerve. A wet nose with clear watery (serous) nasal discharge from one or both nostrils may be normal and has not been recognized as an early sign of impending facial nerve paralysis. It is an unreliable indicator of general wellbeing – hence the thermometer's supremacy. Systemic causes are usually bilateral and include: over-hydration, early sign of nasal irritation by fungi, parasites, foreign bodies, neoplasia, viruses, or allergies. *Rhinorrhoea* is the leakage of CSF by the nose and is reported following head trauma.

SIGNALMENT

An 8-year-old neutered female cross-breed dog.

CASE PRESENTING SIGNS

Right-sided facial paralysis.

CASE HISTORY

The dog suddenly developed a lop-sided face, drooling from the right side of the mouth and increased tear production from the right eye, 5 days prior to referral. The dog had become less active. There was no history of ear disease. Treatment with corticosteroids and amoxicillin had not improved the condition.

CLINICAL EXAMINATION

The quiet but alert dog with a normal gait, postural reactions and proprioception was examined. The dog was visual but lacked a right menace due to the lack of a right blink. The left facial muscles were normal. Facial sensation was normal bilaterally. Eye movement was normal. STT was 20 mm bilaterally. No epiphora was seen.

NEUROANATOMICAL LOCALIZATION

The lesion was localized to the right facial nerve, peripheral to the brainstem.

DIFFERENTIAL DIAGNOSIS

Causes of facial nerve paralysis:

- Idiopathic: cat and dog
- Surgery: to the external ear canal or bulla osteotomy
- Otitis media: inflammatory polyps(cats, dogs), tumour (extension; SCC, benign adenoma, fibroma or cholesteatoma); bacterial infection (*Staphylococcus, Pseudomonas, Proteus* and *Streptococcus* spp)
- Neuromuscular diseases: polyradiculoneuritis, myasthenia gravis, botulism and hypothyroidism
- Idiosyncratic reaction to sulphonamides (dog)
- Petrosal bone fracture.

In this case, otitis media, neoplasia of the bulla, meningioma of the brainstem, or idiopathic facial paralysis were considered most likely. In most cases of otitis media there has been some history of otitis externa, head shaking or scratching. Osteomyelitis or neoplasia of the bulla can result in the animal being unwilling to open its mouth or chew food, which presents as inappetance, not chewing hard food and not fetching items. The peripheral location of meningioma means that they can present with peripheral CNN involvement, before ataxia and paresis develop.

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Figure 29.2 Note the 'bulging' flaccid lips on the left, particularly the lower, and the deviation of the nose to the right.

CASE WORK-UP

CSF was normal as was an MRI of the head.

Toxoplasma and Neospora serology were negative. The dog was euthyroid.

DIAGNOSIS

Suspected idiopathic facial paralysis.

FOLLOW-UP

Five months later left facial paralysis occurred suddenly and the nose deviated to the right. No other deficits were found on this examination, so the lesion was thought to be idiopathic left facial nerve paralysis (Fig. 29.2).

AETIOPATHOGENESIS

Idiopathic facial paralysis

Unilateral facial paralysis is a common problem. The contralateral side may become paralysed within 1–2 months. The onset is sudden, and animals are usually presented the same day with a history of having had 'a stroke'. Cortical lesions may produce subtle facial weakness but LMN lesions produce the common causes of facial weakness in small animals. It is very common in spaniels, and is unrelated to the presence of syringomyelia. The diagnosis is made by excluding known causes. Some 75% of canine and 25% of feline cases of facial paralysis are idiopathic. The lesion lies within the facial nerve peripheral to the brainstem and consists of axon and myelin loss without inflammation.

Spontaneous recovery may occur in 4–8 weeks but paralysis may be permanent. Paralysed nasal muscles give rise to uneven tension between the two sides and the nose is pulled toward the unaffected side. If the paralysed flaccid muscles later contract the nose straightens.

Antiviral therapy and corticosteroids are not used to treat this condition in small animals.

Prehension can be impeded by the flaccid lip becoming trapped between the teeth causing pain. Always check the upper lip for wounds. Dry eye is a possible sequela from paralysis of the parasympathetic supply to the lacrimal glands. The Schirmer tear test should be rechecked.

Corneal protection is maintained by lacrimation in response to pain (CN V), retraction of the globe (CN VI) and movement of the third eyelid. However, lagophthalmos, the inability to close the eyelid(s) leaving a partial opening, does increase the risk of corneal abrasion and fluorescein staining should be carried out routinely.

Enhancement of the facial nerve has been seen on MRI but the prognostic value of this is currently unproven.

Fluid accumulation within the tympanic bullae is noted as an incidental finding in various dog breeds, generally the brachycephalics such as the CKCS, cocker spaniels, bulldogs and boxers. A diagnostic dilemma arises when these dogs are being assessed for facial paralysis, Horner's syndrome, or peripheral vestibular disease. When is the fluid within the tympanic bulla clinically significant? When should a bulla osteotomy be performed? Should the material be removed by myringotomy? Contrast-enhancement of the bulla's mucosal lining is not necessarily clinically significant. Bulla osteotomy is not 100% benign and risks causing deafness, facial paralysis and Horner's syndrome. Decision-making is currently based on clinical judgement of the signalment, history, presence or absence of otitis externa and the progression of neurological deficits. The collection of fluid within the tympanic bulla has been termed primary secretory otitis media without proof that the viscous mucus present is from a secretory malfunction.

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30 Dropped jaw

INITIAL PRESENTATION

Inability to prehend food or toys, slow, messy eating: drops food and dribbles water around the bowl, drooling, salivating (pseudoptyalism), jaw hanging open, unable to close mouth, tilting or throwing the head back to eat.

INTRODUCTION

Prehension is the grasping of food or objects with the mouth. The inability to prehend food has been called oral dysphagia.

The most obvious deficit occurs with bilateral trigeminal nerve paralysis. The mandibular branch of the trigeminal nerve (CN V) innervates the muscles of mastication. Unilateral paralysis does not interfere with feeding. Bilateral paralysis causes the jaws to hang apart at rest. The animal attempts to eat but food falls from the mouth. Food placed at the back of the mouth can be swallowed.

Tongue paralysis as a sole deficit is rare. Weak tongue retraction has been noted as an examination finding, not a presenting complaint, in myasthenia gravis. A pit bull pup with episodic paralysis (from suspected hyperkalaemic periodic paralysis) precipitated by exercise had flaccid neck, limb and tongue muscles. A fractured hyoid bone, strenuous tongue manipulation in small dogs and brainstem tumours are also reported causes. The rare conditions of muscular dystrophy, congenital myotonia, and acromegaly cause hypertrophy of the tongue and its protrusion from the mouth.

The lips play a part in prehension although not as prominently as in primates or ungulates. Nevertheless, always consider facial paralysis as a cause of dropping food. The flaccid paralysis of upper lips leads to them being caught between teeth during prehension. This is painful, and the mouth is opened again. Food is also trapped between gums and lips despite a functional tongue and jaw.

Oral or pharyngeal pain or obstruction are common causes of a failure to prehend food. Teeth chattering has been reported in cats with dental disease.

SIGNALMENT

A 5-year-old female Hungarian vizsla.

CASE PRESENTING SIGNS

Inability to close the mouth.

CASE HISTORY

The dog suddenly developed a dropped jaw 1 week prior to referral.

The owners had noticed the water in the drinking bowl being frothy the day before the jaw was visibly hanging open. The next day the right third eyelid became prominent. Muscle loss on the head was noted bilaterally. The dog tried to pick things up but could not do so. No regurgitation, choking or vomiting were reported.

CLINICAL EXAMINATION

The dog was alert and ambulatory with normal proprioception and hopping. Right Horner's syndrome and a dropped jaw with absent jaw tone were found (Figs 30.1 and 30.2). Sensation was present in all areas of the face. STT was normal. Facial muscle movement appeared to be normal. Atrophy of the masseter and temporalis muscles was present bilaterally.

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Figure 30.1 The lower jaw hangs open.



Figure 30.2 Right Horner's syndrome.

NEUROANATOMICAL DIAGNOSIS

The mandibular branch of the trigeminal nerve and the right sympathetic nerve supply to the eye.

The trigeminal nerve (CN V), as the name indicates, has three branches:

1. Ophthalmic branch
   - Sensory to the eyeball, eyelids, nasal mucosa and skin
   - Exits orbital foramen of skull
   - Gives rise to the lacrimal nerve which innervates the lacrimal gland: postganglionic parasympathetic fibres from CN VII travel with this nerve to the lacrimal gland
   - Carries postganglionic sympathetic fibres to the pupil.
2. Maxillary branch
   - Sensory to the eyelids, nasal mucosa, nose, upper teeth and upper lip
   - Exits skull via the round foramen, alar canal and rostral alar foramen
   - Also transmits postganglionic parasympathetic fibres to the lacrimal, palatine, and nasal glands.
3. Mandibular branch
   - Sensory to the buccal cavity, tongue, teeth of lower jaw, lower lip and some skin of the head
   - Motor fibres to the muscles of mastication: masseter, temporalis, medial and lateral pterygoids, rostral digastricus and mylohyoideus
   - Passes through the oval foramen.

DIFFERENTIAL DIAGNOSIS

A mandibular neuritis/neuropathy was suspected in this dog.

Mandibular neuropathy is a common cause of an acute inability to close the mouth. Also known as trigeminal neuritis, it is a spontaneous, non-painful, idiopathic inflammation of the trigeminal nerve motor branch bilaterally resulting in jaw paralysis. Sensation to the face is intact in most animals. Horner's syndrome may be associated with mandibular neuropathy but the cause is not clear. The condition spontaneously resolves in 2–3 weeks. Corticosteroids are not required. Golden retrievers were over-represented in one study of this condition.

The jaw may be fixed in an open position by temporomandibular luxation. Lockjaw refers to the trismus of tetanus rather than jaws in a fixed open position. A tonic opening of the jaws is common during seizure activity.

CLINICAL TIP

Note any gagging, inability to swallow, and regurgitation as these indicate other cranial nerve involvement or muscle weakness.

CASE WORK-UP

- Muscle enzymes were normal
- Haematology was normal
- MRI of the head found bilaterally symmetric trigeminal nerves without enlargement of either oval foramen
- Tympanic bullae were normal
- CSF was normal.

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DIAGNOSIS

The diagnosis by exclusion is a mandibular neuropathy. Horner's syndrome can occur when the trigeminal nerve is damaged.

TREATMENT/PROGNOSIS

The dog was hand-fed canned food made into balls and placed at the back of the throat. The jaws were partially tied together with a fabric bow and the water bowl was elevated, to aid the lapping of water. No medication was given.

The dog improved spontaneously, returning to normal within 10 days (see also Chapter 36, p 158).

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31 Middle ear disease

INITIAL PRESENTATION

Initial presentation may include: facial paralysis, Horner's syndrome, pain on opening the mouth.

INTRODUCTION

The middle ear lies beyond the tympanic membrane (ear drum), and consists of the mucosa-lined tympanic bulla, a bony 'balloon' lying on the ventrolateral surface of the temporal bone. It contains the three auditory ossicles which transmit sound waves from the external ear to the internal ear. The tiny muscles attached to either end of the ossicle chain contract in response to loud noise, limiting the transmission of sound to the inner ear as a protective mechanism. The auditory (eustachian) tube connects the middle ear to the nasopharynx. In cats, the large ventromedial and small dorsolateral compartments are separated by a very thin bony lamella. The facial nerve and sympathetic nerve supply to the eye are closely associated with the cavity of the middle ear.

The initial presentation may include:

- Facial paralysis, Horner's syndrome or pain on opening the mouth
- Signs of associated chronic otitis externa (ear pain with or without head tilt, shaking or scratching the head, smelly ears)
- Signs of associated otitis interna (peripheral vestibular disease).

SIGNALMENT

A 2-year-old neutered male British blue cat.

CASE PRESENTING SIGNS

Head tilt and nystagmus.

CASE HISTORY

2–3 months of left otitis externa. A week of depression and 2 days of a left head tilt, nystagmus, ataxia, being unable to jump and stumbling to the left.

CLINICAL EXAMINATION

The cat was alert, ambulatory, ataxic and had a left head tilt. When walking, the cat staggered or fell to the left. Hopping and proprioception were normal in all limbs. Spontaneous horizontal nystagmus with a fast phase to the right was seen.

NEUROANATOMICAL LOCALIZATION

Left peripheral vestibular apparatus.

DIFFERENTIAL DIAGNOSIS

- A middle ear polyp
- Bacterial otitis with secondary irritation of the inner ear.

Secondary meningitis can occur by extension which may have caused irritation of the peripheral portion of the CN VIII.

Inflammation of the middle ear most commonly results from extension of bacterial or yeast infections from the external ear canal. The middle ear acts as a reservoir for reinfection of the external ear canal.

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An intact ear drum may be present with either otitis media or otitis externa. Bacteria may ascend via the auditory tube from the nasopharynx or gain access haematogenously.

Nasopharyngeal polyps are found in the tympanic bulla and may extend into the external ear canal or into the nasopharynx. The index of suspicion should be raised when presented with a cat affected by vestibular disease, chronic otitis externa, snoring, stertor, facial paralysis or Horner's syndrome. Surgical removal of the polyp is required for successful treatment. Chronic otitis media can lead to extension of infection into the cranial vault.

Cholesteatoma develop within a chronically inflamed middle ear and require surgical removal.

CASE WORK-UP

Lateral oblique, open-mouth, DV or VD views of the skull allow visualization of the tympanic bullae. Increased opacity within the lumen, and bony proliferation or thickening of the tympanic bulla are evidence of middle ear involvement but these changes may not be apparent for several weeks. Some 25% of otitis media cases will be missed by radiographic assessment only. Lysis of the bulla is suggestive of neoplasia rather than osteomyelitis. When the otitis is mild, radiographs and CT have the same diagnostic yield. Fewer cases are missed on CT than radiography when the bulla changes increase in severity.

MRI gives fabulous soft tissue detail which is important for assessing anatomy associated with the neurological deficits caused by otitis media-interna. It can detect extension of infection into the calvarium. Paraaural abscesses, possibly associated with past unsuccessful ear surgery, are noted. The extent of tumours of the soft tissue and skull can be assessed.

In this case, the cat was admitted to hospital to prevent injury from falling. MRI of the head was performed (Fig. 31.1).

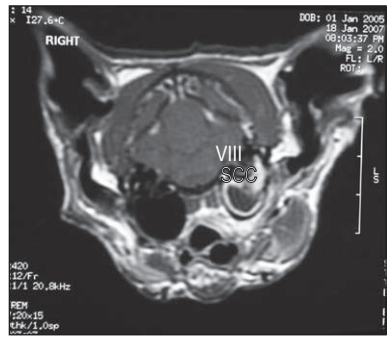


Figure 31.1 MRI T1WI + contrast. Enhancement of soft tissue in and around the left tympanic bulla. VIII marks the vestibulocochlear nerve. SCC points to the semicircular canal.

DIAGNOSIS

Otitis media, osteomyelitis with polyp.

TREATMENT

Ventral bulla osteotomy removed pus and polyp material from the middle ear.

Causes of otitis externa should be addressed. Cultures of the external ear canal often differ from those of the middle ear. Myringotomy under general anaesthesia with aspiration of material for culture and sensitivity has been recommended. Empirical treatment with cephalosporins, amoxicillin, and clavulanic acid for two weeks is an option, followed by further investigation if no improvement is seen. Any soft tissue masses within the bulla should be surgically removed and sent for histopathology.

FOLLOW-UP

There was resolution of the vestibular signs.

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32 Otogenic intracranial infection

INITIAL PRESENTATION

The initial presentation may include vestibular disease, facial paralysis or Horner's syndrome.

INTRODUCTION

Chronic ear infection can result in pyogranulomatous otitis media-interna with osteomyelitis of the tympanic bulla. Extension into the cranial vault is detected on MRI as a globoid or plaque-like mass extending from the bulla into the cerebello-medullary angle. This compresses the brainstem, and causes meningoencephalitis. Abscess formation may be seen as a thickened rim around the mass. Material is seen within the bulla.

Somewhat surprisingly, there may be no clinical indication of intracranial extension. Head or neck pain, depression, lethargy, seizures, and CN V deficits are not routinely found. A mild to moderate increase in rectal temperature (39–40°C) may be present. Haematology is normal or shows a mild neutrophilia. It should be suspected in any age or breed of cat or dog with chronic, recurrent, or existing otitis media-interna that develops vestibular signs (peripheral or central) over a period of hours to days. Most cases have been reported in the cat.

SIGNALMENT 1

An 8-year-old neutered female domestic shorthaired cat.

CASE PRESENTING SIGNS

Loss of balance.

CASE HISTORY

There had been a few days of lethargy before the spontaneous nystagmus, right head tilt and ataxia occurred. Treatment with dexamethasone and amoxicillin/clavulanic acid for 5 days appeared to lessen the ataxia but the cat became inappetant and would not move from its bed. No history of ear disease was reported but the cat was seen to scratch at the right ear before the balance loss occurred.

CLINICAL EXAMINATION

Examination found an alert ambulatory ataxic cat which lost balance when looking up, lurching to the right but not falling. A right head tilt was obvious. Spontaneous nystagmus was not present at rest or when the neck was dorsiflexed. The vestibulo-ocular reflex was slow to the right. Right facial paralysis was present. STT was normal bilaterally. No anisocoria was present. The placing response was slow in the right forelimb. Hopping was slightly reduced in the right fore- and right hindlimbs. Spinal reflexes were normal. No spinal pain was found.

NEUROANATOMICAL LOCALIZATION

The lesion was localized to the right facial nerve and the right vestibular apparatus. The right-sided postural deficits indicated right brainstem involvement. The CNN VII and VIII could be involved together by middle and inner ear lesions respectively, or by a right brainstem lesion picking off the nerves. The lack of spontaneous nystagmus in an ataxic cat with a head tilt is unusual and may indicate cerebellar involvement as the cause of the vestibular signs.

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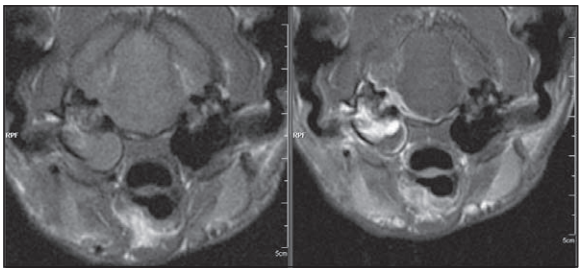


Figure 32.1 Transverse T1WI; pre-contrast (left) and post-contrast (right). Extension of infection from ear to meninges.

DIFFERENTIAL DIAGNOSIS

- A carcinoma of the right tympanic bulla extending to the inner ear and brainstem
- Otitis media-interna with extension to the brainstem
- Meningioma of the right brainstem
- Nerve root tumours of CN VIII are rare.

CASE WORK-UP

MRI found that both compartments of the right middle ear were filled with material hypointense on T1WI and with areas of hyperintensity on T2WI. There was contrast-enhancement of the bulla, the muscle external to the bulla, and meninges of the brainstem and cerebellum (Fig. 32.1).

CSF was abnormal, with an elevated total protein of 0.60 g/l (normal range: 0.0–0.25) and increased WBC of 13/μl (normal <5). Cytological examination showed 81% neutrophils, 15% monocytes and small/medium lymphocytes. No bacteria were seen. Culture of the CSF was not performed. These results indicate tissue destruction, in this case probably from inflammation, most likely bacterial.

In one study of otogenic abscesses, CSF was consistently abnormal in acute presentations, revealing a neutrophilic pleocytosis and elevated protein levels. CSF may be normal in chronic cases.

DIAGNOSIS

Otitis media-interna with meningitis.

TREATMENT

Treatment of otogenic abscesses consists of removal of necrotic tissue by ventral bulla osteotomy. Antibiotics are continued empirically for 1 month if a negative culture of bulla contents, CSF or both is obtained or for 3 months if a positive culture occurs. Multiple organisms are frequently isolated in acute and chronic infections: *Staphylococcus* spp., *E coli*, *Pseudomonas aeruginosa*, *Proteus mirabilis*, *Enterococcus* spp. and *Streptococcus* spp. Anaerobic organisms, and *Pasteurella multocida* are additionally found in cats.

This cat had a right ventral bulla osteotomy which allowed removal of a polyp from both compartments of the middle ear. Horner's syndrome developed post-operatively. The cat was given cephalexin for 4 weeks and prednisolone for 4 weeks on a decreasing dose.

PROGNOSIS

The prognosis was good to excellent with surgical treatment and antibiotics.

FOLLOW-UP

The balance had improved to normal but Horner's syndrome was still present 1 month postoperatively.

SIGNALMENT 2

A 12-year-old neutered female Siamese cat.

CASE PRESENTING SIGNS

Nystagmus.

CASE HISTORY

Otitis externa had commenced in the right, then the left ear 6 months ago. Horner's syndrome on the left appeared at the same time. The cat had been anorexic and mentally depressed for 3 days prior to referral. The cat had been referred for investigation of nystagmus.

CLINICAL EXAMINATION

A mentally depressed febrile cat with foul-smelling, painful external ear canals was found to have intermittent pendular nystagmus, left enophthalmos, a decreased left palpebral fissure and anisocoria with a miotic left pupil in ambient light and in a darkened room. The blink reflex was weak on the left despite apparently normal facial sensation. Normal vestibulo-ocular reflexes were present.

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NEUROANATOMICAL LOCALIZATION

The left facial nerve and the sympathetic innervation of the left eye. Osteomyelitis of the tympanic bulla on one or both sides could account for the pain and fever.

The nystagmus was pendular, intermittent, and considered normal for the breed. No signs of vestibular disease were present.

DIFFERENTIAL DIAGNOSIS

- Extension of bacterial otitis externa to the middle ear(s)
- Polyp
- Carcinoma.

CASE WORK-UP

IV fluids and pain relief improved the cat's mental state and appetite.

Haematology and serology results were normal.

MRI: fluid-like material was found in both tympanic bullae and ear canals. There was extensive inflammation of bone and soft tissue of both bullae and the left pterygoid bone, with extension into the cranial vault. Contrast-enhancement of thickened dura mater on the left cerebrum was noted (Fig. 32.2).

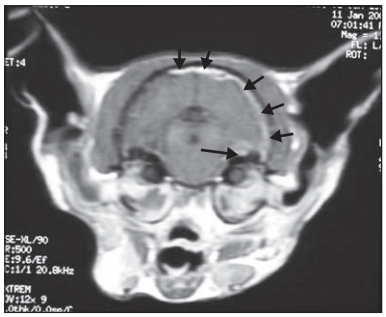


Figure 32.2 T1WI + contrast. Meningeal thickening (arrows).

CSF was normal. Aerobic and anaerobic culture were negative.

Opaque white liquid in the right ear canal was aspirated under the general anaesthetic and appeared to be viscous pus.

TREATMENT

Surgery to remove purulent material and polyps from both tympanic bullae. The cat improved postoperatively.

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33 Deafness

INTRODUCTION

Air conducts sound waves to the tympanic membrane. This energy is transmitted via the malleus, incus and stapes ossicle chain of the middle ear to the fluid of the cochlear duct and thus its hair cells. Failure of the transmission of sound by the external or middle ear is called conductive deafness.

The cochlear duct is found in an osseus spiral-shaped passageway within the petrous-temporal bone known as the cochlea. The bony cochlea along with the vestibule and semicircular canals containing the vestibular apparatus comprise the inner ear. Movement of the hair cells creates action potentials which travel via the cochlear portion of CN VIII into the medulla oblongata. The auditory pathway decussates multiple times as it ascends to the auditory cortex. Failure of the generation of action potentials or failure of their transmission to the auditory cortex is called sensorineural deafness.

Deafness in an otherwise normal animal is a result of a peripheral lesion (external, middle or inner ear, or cochlear nerve peripheral to the brainstem) rather than a central (brainstem pathways or higher) one. To create complete bilateral deafness from a brainstem lesion would be incompatible with life (Fig. 33.1).

SIGNALMENT

A 2-year, 11-month-old female English springer spaniel.

CASE PRESENTING SIGNS

Not responding to commands.

CASE HISTORY

For the last month the dog had been running in the wrong direction when called and was thought to be deaf. When she shook, she fell down.

CLINICAL EXAMINATION

On the first examination, the dog had a normal gait and posture with normal postural reactions (hopping and proprioception) and cranial nerve examination. Wax obscured the left ear drum.

NEUROANATOMICAL DIAGNOSIS

The dog was suspected of being unilaterally deaf, given the poor ability to localize the direction of sound. Falling when shaking suggested a loss of balance and so vestibular involvement was suspected despite the lack of a head tilt or abnormal nystagmus.

DIFFERENTIAL DIAGNOSIS

Cause of deafness

Inherited sensorineural deafness occurs in cats and dogs, and is linked to genes controlling coat colour. Merle or dapple coats have dark, irregular blotches on a white background, e.g. dalmatians, and such breeds have a high incidence of deafness. White-coated animals with or without blue irises have a high risk of deafness.

Agenesis or degeneration of the hearing receptor is responsible and is present from birth or soon after in the majority of breeds. BAER testing is generally performed at 5–8 weeks of age before puppies from at-risk breeds are sold and re-homed. There is no treatment for inherited deafness. Hearing aids magnify sound and are of no benefit as the hearing receptor is destroyed.

Acquired sensorineural deafness occurs as an ageing change. Ototoxicity from aminoglycosides, diuretics, antiseptics and chemotherapy has been reported in animals.

Conditions which block the transmission of sound through the external or middle ear cause hearing loss of varying severity. Deafness is a complication of tympanic bulla surgery.

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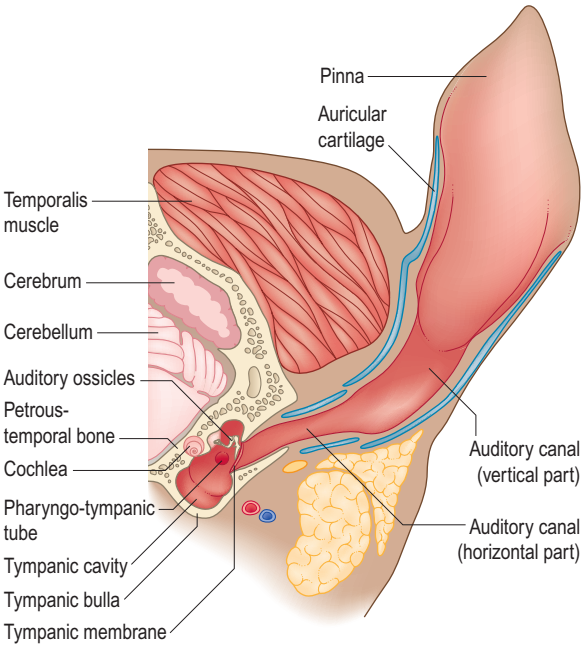


Figure 33.1 Diagram of the ear.

In this case: Otitis interna was considered most likely in a dog of this age.

CASE WORK-UP

Hearing testing

Behavioural assessment

The behavioural response to sound is not diagnostically accurate but is the ultimate purpose of hearing in a pet–owner relationship. Even normal-hearing animals ignore test sounds after the second or third occurrence. A startle response to noise can be stimulated by vibration, e.g. a slamming door or a running engine, as well as sound. Movement of the pinna in response to sound is the minimum expected response from a hearing animal.

Electrical assessment

A series of clicks is transmitted to one ear at a time by way of ear phones. The clicks evoke an action potential in the cochlear duct which is then transmitted through the brainstem to the auditory cortex. The first 10 seconds of this transmission records the passage through the brainstem. For this reason the test is known as the brainstem audio-evoked response (BAER) test.

The stimulus artefact, a deflection at the start of a BAER trace, indicates that the stimulus is delivered by the headphones and picked up by the recording electrodes under the skin of the head. If the sound successfully enters the brainstem, a series of 5–7 waveforms is recorded.

Congenital deafness destroys the hearing receptor in one or both ears. This prevents sound entering the brainstem. In this situation the trace shows a stimulus artefact followed by a flat line.

Movement of the head moves the recording electrodes which create artefactual waves on the trace. The BAER test counteracts this by averaging all the responses to the clicks, so that over time, the random artefacts are averaged out, leaving the true result. This is called signal averaging and is used in electrodiagnostics to filter out random waveforms which would obliterate the low voltage evoked recordings. Repeated movement continually creates artefacts which means the test must be restarted, or the machine must be given time to average-out the spurious waveforms. The animal, generally a puppy, must remain still. Various techniques are used but some pups require sedation in order to achieve a meaningful trace (Fig. 33.2). A congenitally deaf animal lacks waveforms, so it is vital that the trace convincingly reflects this. Sedation does not interfere with the transmission of sound through the ear and brainstem.

An animal is described as either 'bilateral hearing present', 'unilaterally deaf in the left or right ear' or 'bilaterally deaf'. The term '50% deaf' has no clear meaning and must not be used (Fig. 33.3).

BAER tests initial anatomical pathways of hearing but is no guarantee that the bilaterally hearing animal will pay attention to the owner.

In this case: The BAER detected traces bilaterally, indicating that the cochlea and CN VIII (cochlear nerve) were functional.

DIAGNOSIS

The dog was seen to lurch to the left when shaking its head. Otitis interna was suspected. Enrofloxacin was commenced and an MRI of the head was scheduled.

PROGNOSIS

Dogs with unilaterally absent BAER are behaviourally indistinguishable in most cases, from dogs with normal bilateral hearing and so make excellent pets. The inheritance of deafness is not straightforward. The general recommendation is to breed only from bilaterally hearing

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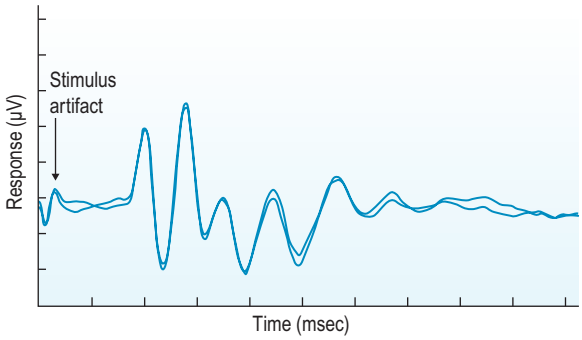


Figure 33.2 Normal BAER trace.



Figure 33.4 Transverse T1WI. Increased hyperintensity of the white matter.

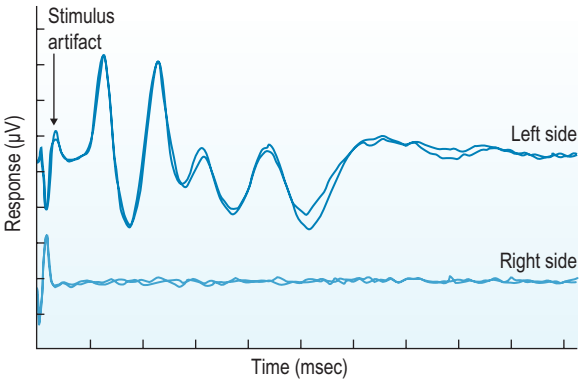


Figure 33.3 Normal trace in the left ear. Absent trace in the right ear. Unilaterally deaf in the right ear.

animals but this pairing will still produce unilaterally or bilaterally deaf offspring on occasions.

The percentage of bilaterally deaf Dalmatians is low (7%). Unilateral deafness in Dalmatians is more prevalent at 21%. Surprisingly, not all owners are aware of the problem. Bilaterally deaf adult Dalmatians may be presented for being disobedient ('hyperactive', 'wilful', untrainable). Such dogs may be re-homed numerous times. Dalmatian breeders in the UK often choose to have bilaterally deaf Dalmatians euthanized at diagnosis rather than risk the animal spending its life in animal shelters. Bilaterally deaf dogs can be trained with hand signals by a dedicated owner.

FOLLOW-UP

Repeat examination 3 weeks later found that the dog had positional vertical nystagmus but had otherwise remained unchanged.

Vertical nystagmus indicates a central vestibular disease with a lesion within the cerebellum or vestibular nuclei of the brainstem. Not responding normally to sound may mean that the dog had a problem centrally processing the information (cerebral disease).

The slow progression was suggestive of a degenerative disease such as fucosidosis, an inherited disorder in English springer spaniels. Neosporosis and GME can give rise to progressive multifocal disease but would be expected to worsen over a shorter period of time. Hydrocephalus secondary to a caudal fossa mass could give rise to both cerebral and cerebellar signs.

MRI recorded an unusual grey-white matter contrast with the white matter appearing more hyperintense relative to the grey matter (Fig. 33.4). No contrast enhancement was seen. This is suggestive of oedema, or abnormal myelination.

The dog was commenced on supplements used in mitochondrial disease but this did not halt progression of signs.

One month later, the dog was still able to hear sound but could not locate the origin of it. She did not appear to recognize the owner; 2 months after the MRI the dog was ataxic.

A DNA test for fucosidosis found the dog was homozygous for the mutant gene and therefore affected.

AETIOPATHOGENESIS OF FUCOSIDOSIS

Fucosidosis is a lysosomal storage disease of autosomal recessive inheritance in English springer spaniels of either sex. A deficiency of the enzyme alpha-fucosidase results in accumulation of incompletely metabolized substrates

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in visceral and nervous tissue. Intracytoplasmic vacuoles may be seen in leukocytes from peripheral blood, bone marrow and CSF.

Signs reflect neurological involvement and are first noted between 4 months and 2 years of age. The main problems are ataxia, personality change and impairment of hearing and vision. The initial presenting complaint may be a resistance to handling and being over-anxious. This does not improve with time.

Affected animals die or are euthanized by 4 years of age because of worsening signs. There is no cure.

DNA testing is available.

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34 Sneezing

INITIAL PRESENTATION

Persistent episodic sneezing. *Ptarmus* (spasmodic sneezing).

INTRODUCTION

Sneezing is a protective reflex mediated by the medulla oblongata. Sensory input from pain fibres (trigeminal nerve) in the nasal mucosa initiate the reflex. A rapid inspiration followed by the explosive passage of air through the nose and mouth expels irritants. Any nasal disease may present as an acute onset of persistent sneezing. Nasal discharge typically develops.

Sneezing with its subsequent loss has been reported in humans secondary to infarction of the medulla.

Reverse sneezing is an outburst of noisy forceful inspiration triggered by nasopharyngeal irritation which typically occurs in small breed dogs of any age. Episodes last seconds and can be associated with excitement or drinking. The problem is self-limiting and does not result in anoxia or collapse. Faulty interplay of the epiglottis and soft palate is thought to be the cause. It is not a sign of neurological disease.

SIGNALMENT

A 7-year-old neutered female Shetland sheepdog.

CASE PRESENTING SIGNS

Chronic sneezing and an asymmetrical head.

CASE HISTORY

Conjunctivitis of the right eye, sneezing, and rubbing the right face were first noted 7 months prior to referral. The rubbing had since stopped however the dog had trains of about 7 sneezes once a day. Nasal disease had been thoroughly investigated and no pathology had been found. Atrophy of the right temporal muscles had begun in the last month.

CLINICAL EXAMINATION

The dog was alert and ambulatory with normal postural reactions and proprioception in all limbs. Pupil size was symmetric and the PLRs were normal. Right enophthalmos was present which made observing eye movement difficult but right eye movement was present. Atrophy of the right masticatory muscles with the sensation absent in the right side of the face in all three branches of the trigeminal nerve and a right dry eye were found. The right ear and nostrils both moved. The nasal planum was wet and symmetrical. The lips were symmetrical.

NEUROANATOMICAL LOCALIZATION

Right trigeminal nerve (atrophy, sensation). The enophthalmos can occur with loss of masticatory muscles or Horner's syndrome, both of which occur with trigeminal nerve lesions. The former was considered most likely as miosis was not present. Face rubbing, sneezing and decreased tear production are also signs of trigeminal sensory dysfunction.

DIFFERENTIAL DIAGNOSIS

- Trigeminal neuroma was suspected given the long time course and slow progression

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- Meningioma
- Skull tumour in the region of the oval foramen.

CASE WORK-UP

MRI showed a large uniformly contrast enhancing mass in the area of the right trigeminal nerve root which was compressing the adjacent brainstem. Minimal oedema of the surrounding parenchyma was noted.

CSF was abnormal with a large increase in total protein of 0.70 g/l (N <0.25). WBC count and cytology was normal.

DIAGNOSIS

Trigeminal nerve root tumour.

Sneezing is an unusual presentation of a trigeminal nerve lesion.

Excessive salivation, *coughing* and dysphagia have been reported in a *sensory trigeminal neuropathy* in a 2-year-old rough collie dog in which bilateral sensory loss over the trigeminal distribution was present.

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35 Coughing

INTRODUCTION

Coughing is a voluntary or reflex action in which deep inspiration is followed by forced expiration against a closed glottis. This requires strong contraction of the respiratory muscles and the larynx. The sudden opening of the glottis releases air in an explosive manner. Its passage through the vocal folds creates the characteristic sound. It may be followed by a terminal retch.

It is stimulated by irritation of the pharynx, larynx, trachea, extrapulmonary bronchi and rarely by other intrathoracic structures. The reflex is mediated by the medulla oblongata. Afferent input is supplied by the glossopharyngeal nerve (CN IX) innervating the pharynx and the vagus (CN X) which contains sensory input from the pharynx, larynx and thoracic structures. The motor arc is governed by the intercostal muscles (T1–12), the diaphragm (phrenic nerve, C5–7) and the larynx (CN X).

A weak cough, termed 'bovine cough' in human neurology, results from a failure to build up sufficient intrathoracic pressure due to a vagal lesion (recurrent laryngeal nerve) or generalized weakness.

A goose-honking sound is associated with tracheal collapse.

The Valsalva manoeuvre is forceful exhalation against the resistance of a closed mouth and nose, as occurs during vomiting, straining to defecate, and coughing with the subsequent release of resistance and exhalation. This gives rise to autonomically mediated cardiovascular reflexes with changes in heart rate and blood pressure and is the basis for 'cough syncope'. Cough syncope may result from the increased intrathoracic pressure blocking venous return to the heart or strong vagal stimulation inducing vasodilation and bradycardia (vasovagal syncope).

LARYNGEAL PARALYSIS

By Ed Friend

Laryngeal paralysis is failure of abduction of the arytenoid cartilages by the dorsal cricoarytenoid muscles during the respiratory cycle. This causes a reduction in the diameter of the rima glottidis during the inspiratory phase, and clinical signs are most prominently exercise intolerance due to a reduced capacity to ventilate. Most dogs are classified as 'idiopathic' laryngeal paralysis and no underlying cause can be identified, but disease occurs as a result of loss of innervation of the dorsal cricoarytenoid muscle by the recurrent laryngeal nerves. The traditional view is that nerve dysfunction occurs as a result of the relative extreme length of these nerves. It has been more recently observed, however, that idiopathic laryngeal paralysis is part of a generalized neuropathy. Many other areas of the animal may therefore be affected, although the most prominent clinical sign is usually the laryngeal dysfunction. The underlying neuropathy in most cases is a disease that progresses over a period of weeks to years.

SIGNALMENT

Idiopathic laryngeal paralysis typically occurs in large and giant breed dogs over the age of 9 to 10 years. It is commonly seen in Labrador retrievers, golden retrievers, collies and English and Irish setters. A congenital form of the disease has been described in the Bouvier des Flandres, Dalmatian, husky and bull terrier, with animals showing clinical signs at less than 1 year of age.

All animals presenting with laryngeal paralysis should be investigated for the presence of an underlying cause (see later in this chapter), and this should be especially thorough if the signalment is not typical. Other causes

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that should be ruled out are traumatic or surgical injury (usually to the neck), masses that interfere with the pathway of the recurrent laryngeal nerves (e.g. mediastinal or neck mass), endocrinopathies and infectious diseases. The prognosis for dogs following surgery that do not have 'idiopathic' laryngeal paralysis is likely to be worse and so it is imperative that the underlying cause is found out before surgery is embarked upon in these cases.

HISTORY

Dogs presenting with laryngeal paralysis usually have a gradual onset of respiratory stridor, cyanosis and decreasing exercise tolerance; there may be worsening of signs in hot weather as the dog will be attempting to shift more air through the larynx to thermoregulate. Syncope is sometimes seen with light as well as vigorous exercise and animals may present in severe respiratory distress. It is only with careful questioning that a more chronic history becomes apparent. A change in bark will have been apparent in some cases.

There may also be a history of coughing, sometimes with lethargy and pyrexia. There may be a low level of irritation due to small amounts of saliva and food entering the trachea, or if more severe aspiration occurs, aspiration pneumonia may result. This has previously been thought to occur because the vocal folds are not guarding the rima glottidis as effectively as normal; however it may also be due to reduced function of pharyngeal nerves and muscles caused by the generalized neuropathy. Owners may notice this as increased coughing during eating or drinking, or if the animal struggles or is slower to eat.

Megaoesophagus is sometimes reported with this condition, and so a history of regurgitation should be ruled out. If this is diagnosed, concurrent aspiration pneumonia should always be considered and the prognosis is more guarded.

The generalized neuropathy may also lead to pelvic limb weakness. This is usually mild and may be misdiagnosed as osteoarthritis as the animals are large breed and quite old. Owners sometimes report scuffing of the nails as the dog walks on hard surfaces, or stumbling on tight turns. Weakness on rising is very common. More severely affected dogs may be unable to rise and progression of this aspect of the disease is sometimes a cause for the animal to be euthanized.

CLINICAL EXAMINATION

General examination is usually unremarkable. The most prominent finding is noise on laryngeal auscultation, predominantly (but not usually exclusively) on the inspiratory part of the cycle. This will usually be significant enough to be detected on thoracic auscultation.

Heart sounds and other cardiac parameters such as pulse strength and quality and heart rate should all be normal; it is imperative that cardiac disease is eliminated as a cause for the clinical signs. Mucous membranes are often cyanotic because of respiratory compromise. Lung sounds may be harsher in the presence of aspiration pneumonia and referred laryngeal noise is common.

Muscle atrophy, especially of the pelvic limbs, is very common. A basic neurological examination will reveal pelvic limb weakness in most cases, along with conscious proprioceptive deficits. Pharyngeal sensitivity is reduced in some dogs, which will have a very poor gag reflex.

MANAGEMENT OF A LARYNGEAL PARALYSIS DOG PRESENTING WITH SEVERE RESPIRATORY COMPROMISE

- Supplement with oxygen in a way that does not further stress the animal, e.g. oxygen cage.
- Calm the animal; lightly sedate to reduce anxiety if appropriate (anxiety increases the rate and depth of respiration, often worsening the ability to ventilate in laryngeal paralysis cases).
- Place an intravenous line. Maintenance intravenous fluids may later be required as the animal may not wish to eat or drink, but this is also useful to administer medications or anaesthetics.
- Check rectal temperature and cool if raised (cold water towels with or without cool intravenous fluids) – hot weather may often precipitate an episode of respiratory distress in an animal that has previously been coping with laryngeal paralysis.
- Physical examination and further diagnostic tests to rule out concurrent diseases that may worsen the respiratory distress, e.g. concurrent cardiac disease, pleural effusion, etc.
- Induce anaesthesia and intubate if very severe. This should be avoided if possible as the animal will be unlikely to make a good recovery without surgery to help the laryngeal paralysis (see later), i.e. an experienced surgeon has to be present who can perform the surgery. Tracheostomy tubes can be used as a last resort to help recover the animal if surgery cannot be

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performed, but there is a significant morbidity and mortality associated with their use. They also complicate the definitive surgery when this is eventually performed, as the animal may be more likely to cough postoperatively, which may disrupt the laryngeal sutures.

DIAGNOSTIC TESTS

Routine haematology and biochemistry are performed, to look for concurrent disease in these older patients, in particular concurrent endocrinopathies. It was once thought that hypothyroidism may be a direct cause of laryngeal paralysis, but it is now thought these two conditions may occur independently in the same individuals. If suspected, a T4/TSH assay may be useful.

Conscious orthogonal view thoracic radiographs should be obtained. Conscious radiographs are initially taken to attempt to rule out megaoesophagus, as the presence of air in the oesophagus of an anaesthetized animal is a normal finding. A contrast oesophagram is indicated if there is a strong suspicion of oesophageal dysfunction from the patient's history. These films should be studied carefully to ensure that there are no masses interfering with the path of the recurrent laryngeal nerve, and that there is no concurrent aspiration pneumonia.

Final diagnosis is by laryngoscopy under a light plane of anaesthesia; propofol is commonly used to achieve this after light pre-medication. This final step of diagnosis is best performed immediately before surgical treatment, to avoid additional anaesthetic risk to the patient. The vocal folds in the normal animal should be vigorously abducted by the movement of the arytenoid cartilages on every inspiration, and should return to the resting state on every expiration. In dogs with laryngeal paralysis, they will usually hang in the resting state without moving, although they may move 'paradoxically' by being drawn inwards (towards the midline) by the negative pressure of inspiration. The vocal folds are often inflamed and slightly swollen in cases of laryngeal paralysis.

The disease is usually bilateral by the time of diagnosis, but unilateral disease may be detected in early cases when slightly increased laryngeal noise without exercise intolerance may be present. Surgery is not indicated at this stage, as exercise intolerance is the principle clinical sign we wish to improve with surgery (see below).

False positives on laryngoscopy are common, especially if the plane of anaesthesia is too deep (the animal must be under light anaesthesia, and so a gag to hold the mouth open is often useful), or if the larynx is being examined after intubation (intubation will usually lead to temporary cessation of arytenoid movement).

Examination under anaesthesia is usually used to confirm the clinical diagnosis of laryngeal paralysis, although it must be remembered that there are other differentials for upper respiratory tract obstruction. Some are not likely with the signalment of a typical case of idiopathic laryngeal paralysis, such as laryngeal collapse or overlong soft palate. Others are more likely in the age and breeds that are affected, but are very rare: laryngeal tumours and pharyngeal wall salivary mucoceles, for example.

Other diagnostic tests are not commonly performed, but may be useful in some individuals. For example, a broncho-alveolar lavage for cytology and culture and sensitivity may be useful if there is an aspiration pneumonia; further imaging such as ultrasound or CT scan may be required if neoplasia is suspected (along with biopsy where appropriate); electromyography and muscle biopsies for the thorough evaluation of an underlying neuropathy.

TREATMENT

Specific treatment for the underlying neuropathy in idiopathic laryngeal paralysis does not exist, but examination by a neurologist should be encouraged. If there are neurological deficits in the limbs, non-specific adjunctive treatments can be recommended. These are often aimed at increasing the remaining muscle bulk as much as possible to increase strength, such as swimming or regular moderate walks. Gentle physiotherapy (such as moving joints through their range of motion and muscle massage to increase blood flow) may be helpful. Therapy that may help concurrent orthopaedic disease is useful, such as weight loss, analgesics and joint remedies.

Pharyngeal and/or oesophageal dysfunction does carry a guarded prognosis, as aspiration pneumonia can result. Affected dogs seem to cope better with tinned-consistency food, rather than dry or liquid food. Dogs with oesophageal disease may benefit from being fed from a height and being held upright after feeding.

Aspiration pneumonia should be treated with a prolonged (>4 week) course of antibiotics, that are ideally chosen on the basis of culture and sensitivity results from a broncho-alveolar lavage. The most severely affected dogs require hospitalization for supportive care, intravenous fluid therapy and intravenous antibiotics. Response

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to treatment is based on resolution of clinical signs and improvement of radiographic changes. Many dogs, however, continue with a low level of disease that can never be completely eliminated (presumably due to ongoing aspiration) which is why the prognosis is guarded.

The treatment of choice for exercise intolerance, severe cyanosis and syncope caused by laryngeal paralysis is currently a left arytenoid lateralization ('laryngeal tie-back'). This permanently abducts the arytenoid cartilage and therefore the vocal fold, and thereby increases the size of the rima glottidis. This is a technically difficult procedure that has a high complication rate and so referral to a surgeon experienced in this procedure is recommended.

After laryngoscopy to confirm the diagnosis and any additional tests such as chest radiography, the animal is positioned in right lateral recumbency. The left lateral neck and laryngeal region are clipped and prepared for aseptic surgery. A sandbag placed under the neck can help elevate the larynx and make the dissection easier. The skin is incised just ventral to the jugular vein; dissection through the soft tissues reveals the thyropharyngeus muscle that is then split. The muscular process of the arytenoid is identified, and is mobilized. The degree of mobilization is critical to the success of the surgery and requires considerable experience. The arytenoid is then sutured with non-absorbable monofilament suture material to either the thyroid or the cricoid, depending on surgeon preference (Fig. 35.1).

The muscular process is rotated externally and caudally as the suture is tightened, thereby increasing the rima glottidis diameter. Closure is routine, but particular care must be taken to accurately close the thyropharyngeus muscle, as this is an important pharyngeal constrictor and is vital for deglutition.

The aim of surgery is to improve ventilation, and so exercise intolerance and syncope will be greatly improved in most animals after surgery. Surgery will not however help many of the other clinical signs that may be associated with this disease, such as coughing, increased respiratory noise, dysphagia, pelvic limb weakness and so on. In fact, surgery may temporarily worsen the ability to swallow, as incision through the thyropharyngeus muscle is required. Healing of this muscle should occur over a period of several weeks. Thyropharyngeus disruption is one of the main reasons the author does not recommend bilateral arytenoid lateralization, or if surgery on the first side fails, surgery on the right side should be delayed if possible until the left thyropharyngeus muscle has healed. Limb function can occasionally be worsened by surgery, presumably as a result of prolonged recumbency exacerbating concurrent joint disease. These dogs should be encouraged to ambulate as soon as possible after surgery to avoid this complication.

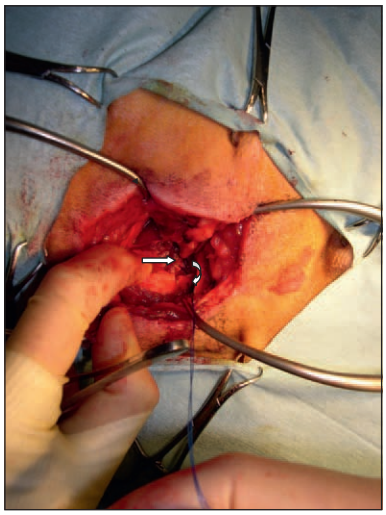


Figure 35.1 A mattress suture of polypropylene has been passed through the muscular process of the arytenoid cartilage (straight arrow) and then through the caudal part of the thyroid cartilage (curved arrow).

It is imperative that dogs are thoroughly screened before surgery, to ensure that only patients that will benefit from surgery are selected. For example, if the predominant clinical sign is coughing and difficulty rising, it is possible that surgery will not benefit the animal. This may change, however, as the disease progresses and so the animal should be re-examined regularly to assess its ability to move air through the larynx. If there is considerable cyanosis and exercise intolerance, along with coughing and pelvic limb weakness (for example), surgery is still indicated but a more guarded prognosis should be given.

POSTOPERATIVE CARE

1. Monitor postoperatively to ensure the animal is able to breathe normally. This aspect is usually improved compared with before surgery however, if the surgeon is experienced.
2. Postoperative antibiotics are required if the pharyngeal mucosa is inadvertently perforated during surgery, or if there is concurrent aspiration pneumonia present (see earlier).

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3. Analgesia – opioids for 24–48 hr are mandatory, along with non-steroidal anti-inflammatory drugs (if not contraindicated).
4. Barking should be discouraged, and so some dogs should be discharged home sooner than is ideal if they cannot be kept quiet.
5. Tinned consistency food should be fed, when the animal has fully recovered from the surgery.
6. Exercise should be limited for 4 weeks after surgery.

CASE EXAMPLE

A 12-year-old male golden retriever.

CLINICAL HISTORY

This dog was presented with an acute onset cough and lethargy. Appetite had been reduced for the previous 24 hr and the exercise ability had reduced over the previous 6 months.

Although the dog had slowed down a bit over the previous few months, he was still very lively and could easily manage walks of over half an hour. No cyanosis or episodes of syncope had occurred. The cough had started 3 days earlier, and was increasing in frequency. The owner mentioned that for several months the dog had coughed a little while eating and drinking and was a little slow on rising. There was no history of regurgitation.

CLINICAL EXAMINATION

On examination, mild pyrexia (39.4°C) with increased respiratory stridor (predominantly inspiratory) centred over the larynx was found. The dog was panting in the consulting room, but he was able to ventilate reasonably well. There was mild cyanosis. He had marked temporal muscle atrophy and mild pelvic limb weakness and muscle atrophy which might have been related to a generalized neuropathy (Fig. 35.2).

CASE WORK-UP

Haematology and biochemistry were unremarkable. Chest radiographs were taken under general anaesthesia, and showed a marked alveolar pattern in the cranioventral lung fields These changes were consistent with an aspiration pneumonia. There was a small amount of air in the oesophagus, but consistent with a normal anaesthetized animal. A broncho-alveolar lavage was performed, which was negative on bacterial culture, but showed increased numbers of neutrophils (Fig. 35.3).



Figure 35.2 Appearance at the time of presentation. Note the temporal and pelvic limb muscle atrophy and mild cyanosis.

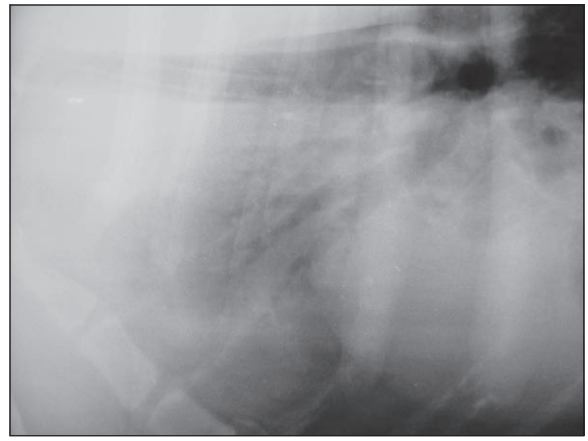


Figure 35.3 Alveolar pattern in the cranial lung lobe consistent with the presence of aspiration pneumonia.

DIAGNOSIS

A diagnosis of laryngeal paralysis was made and so he was referred for surgery.

TREATMENT

In this case, surgery was inappropriate at the time of presentation, as the acute worsening of clinical signs was related to the presence of an aspiration pneumonia.

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Treatment was initiated with intravenous fluid therapy and intravenous antibiotics (amoxicillin-clavulanate 20 mg/kg TID and metronidazole 10 mg/kg BID). He made a rapid improvement: his cyanosis improved and his appetite returned to normal. He was discharged with a 4-week course of oral antibiotics (amoxicillin-clavulanate 21.4 mg/kg BID and metronidazole 11.4 mg/kg BID). Follow-up radiographs to monitor progression were taken every 4 weeks initially and then every 2–3 months at the referring vet.

The amount of respiratory compromise shown by the dog meant that laryngeal tie-back surgery was not indicated at this stage. Follow-up appointments were made at the referral centre, as progression of the laryngeal signs is likely. In these cases, tie-back surgery should only be performed when absolutely necessary, as surgery may increase the likelihood of aspiration.

PROGNOSIS

A guarded prognosis was given for the complete resolution of the aspiration pneumonia, but if laryngeal function deteriorates and signs of cyanosis develop, laryngeal tie-back surgery is still indicated.

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36 Change in voice and swallowing

INTRODUCTION

Dysphagia

A difficulty or inability to swallow is called *dysphagia* and it presents as: gagging, retching, choking, extending or flexing the neck during swallowing, repeated attempts to swallow, coughing when eating or drinking, nasal discharge, and pseudo-ptyalism.

Swallowing (*deglutition*) is initiated by sensory information from the oral cavity (CN V), pharynx (CN IX) and epiglottis (CN X).

Food, voluntarily pushed to the pharynx by the masticatory muscles (CN V), the palatine muscles (CN V), and caudal tongue movement (CN XII), stimulates the reflex which is coordinated by the swallowing centre within the medulla oblongata.

Cessation of respiration and closure of the glottis (CN X) is synchronized with contraction of the pharyngeal muscles (CNN IX, X) to avoid aspiration.

Folding of the epiglottis over the glottis is achieved by contraction of the geniohyoideus (CN XII) and mylohyoideus (CN V) muscles, pulling the larynx forward.

An inability to swallow saliva causes drooling (*pseudoptyalism*). *Oral dysphagia* is the inability to prehend food and form a bolus.

Voice production

Laryngeal paralysis can present with the following signs: an altered volume, pitch or character of voice (dysphonia), lack of voice (aphonia), inspiratory dyspnoea, sonorous respiration, high-pitched wheezing sound (stridor), gagging or coughing when eating or exercise intolerance with or without collapse.

Voice is generated by air passing through the larynx. Alteration of this process changes voice quality. An inability to create and expel a thoracic reservoir of air results in *hypophonia* (quiet voice) or *aphonia*. Weakness or paralysis of the respiratory muscles from UMN lesions (e.g. C1–5 spinal cord disease or tetanus), or LMN origin (e.g. myasthenia gravis or polyradiculoneuritis) is generally accompanied by non-ambulatory tetraparesis or tetraplegia.

Airflow through the larynx is impeded by paralysis of the dorsal cricoarytenoid muscle. This muscle, innervated by a branch of the vagus nerve (CN X), functions as chief abductor of the paired vocal folds and arytenoid cartilages which together create the glottis, the cranial opening of the larynx. Narrowing of the airflow creates stridor, dysphonia, and most importantly, inspiratory dyspnoea. Obstruction of the upper airway by nasopharyngeal polyps or a long soft palate creates a turbulent airflow and loud, stertorous respiration (e.g. snoring or snorting) but rarely results in dyspnoea.

Presentation is prompted by dyspnoea, whereas specific questioning of the owner is usually required to discover changes in the pet's vocalization.

Inability to close the glottis risks aspiration of pharyngeal contents (saliva, food and water). The chief laryngeal adductor is the thyroarytenoid muscle, also innervated by the vagus. Coughing and gagging while eating and drinking is a reflex protective of the airways. Sensation to laryngeal mucosa is supplied by the internal branch of the cranial laryngeal nerve, a division of CN X.

Unilateral laryngeal paralysis would be noticed in athletic, working animals during exercise, but as most pets are relatively sedentary it takes bilateral paralysis to produce clinical signs.

Concomitant signs of aspiration pneumonia may be present: fever, tachypnoea, cough or anorexia. Severely dyspnoeic animals choose to breathe rather than eat as to do both simultaneously would be impossible.

Increased muscle tone in myotonia congenita produces stridorous breathing and a high-pitched bark along with the generalized appendicular muscle stiffness. Dyspnoea and cyanosis have been reported during the increased muscular effort of whelping, or a sudden change of posture. Laryngospasm is a complication of tetanus.

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SIGNALMENT

A 7-year-old male miniature schnauzer.

CASE PRESENTING SIGNS

A hoarse bark, difficulty eating, drooling, retching, gagging.

CASE HISTORY

Signs progressively appeared over 3 weeks, beginning with drooling from the right side of the mouth, retching and gagging and spending a long time drinking. A high-pitched hoarse bark occurred 1 week later. Right Horner's syndrome, atrophy of right masticatory muscles, right forelimb lameness and intermittent neck pain were reported the following week.

CLINICAL EXAMINATION

Clinical examination revealed an alert ambulatory dog with right fore lameness. Hopping was normal in all limbs. The right forelimb placing response was reduced. The right supraspinatus and infraspinatus muscles were atrophied. Muscle tone was reduced in the right forelimb. Ptosis, miosis and enophthalmos of the right eye with protrusion of the third eyelid were present. Sensation to the right nostril and right upper lip was reduced. Atrophy of the right temporalis and masseter muscles was present. Jaw tone was reduced. The gag reflex was decreased. There was slight atrophy of the right side of the tongue. Saliva drooled from the right side of the mouth.

NEUROANATOMICAL LOCALIZATION

Right CN V (M&S), CNN IX, X, and XII. LMN signs to right fore (brachial plexus or nerves). History of neck pain (meninges; cord; vertebrae or discs). Lame right forelimb (nerve compression or inflammation or neoplasia). A multifocal disease affecting peripheral and cranial nerves was suspected. LMN changes to the right fore and the right Horner's syndrome can be caused by a lesion within the ipsilateral brachial plexus or nerve roots.

DIFFERENTIAL DIAGNOSIS

- Ganglioradiculoneuritis
- Neoplastic infiltration of nerves.

CASE WORK-UP

Serology was negative for *Toxoplasma*, *Neosporum caninum* and *Borreliosis*.

Thyroid function was normal: Total T4 12 nmol/l (13–52), TSH 0.13 ng/ml (<0.41).

Muscle enzymes were elevated at: AST 306, CK 1082, ALT 224.

EMG; spontaneous potentials in the right trapezius, supraspinatus, infraspinatus, carpal flexors, masseter and temporalis muscles.

CSF: (25/5/03); total protein 0.57 g/l, WBC = 1, RBC = 0. Cytology: a few reactive lymphocytes.

CSF: (4/6/03): total protein 1.88 g/l; WBC = 60; RBC = 1. Cytology: 85% large atypical cells consistent with neoplasia. These resembled lymphocytes or histiocytes.

MRI showed severe muscle atrophy of the right temporalis, digastricus, masseter, pterygoid muscles which were hyperintense due to fat infiltration. It also showed a small discrete enhancing nodule in the approximate region of the trigeminal nerve where it emerges from the trigeminal canal (Fig. 36.1). There was a similar structure on left but it was much smaller. The right tympanic bulla was half-full of fluid.

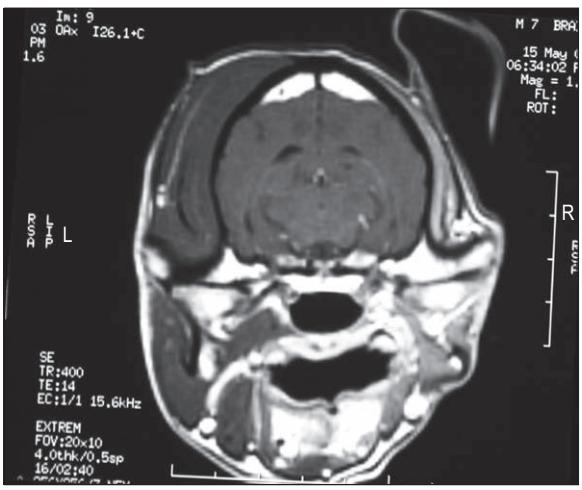


Figure 36.1 Transverse T1WI+C: atrophy of right masticatory muscles and enlargement of the right trigeminal nerve.

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DIAGNOSIS

Focal enlargement of the right trigeminal nerve with multifocal nerve involvement, possibly with lymphoma.

TREATMENT

The vet discussed chemotherapy for suspected lymphoma but the owner declined and the dog was euthanized.

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37 Gait – an introduction

Weakness (paresis) or paralysis, ataxia, and lameness are the most commonly observed alterations in gait.

PARALYSIS AND PARESIS

Paralysis is the absence of voluntary movement.

Paresis is reduced voluntary movement.

Paresis may manifest as:

- Difficulty rising from sitting
- Not moving
- Collapse, falling
- Unable or unwilling to exercise.
- Unable to jump into the car, go up stairs or climb onto furniture
- Unable to support self on one hind limb while urinating
- Unable to maintain a squatting position to urinate/defecate
- Slow moving or shuffling gait
- Dragging/scuffing/scraping/abrading the dorsum of the paw on the ground
- Dragging self using forelimbs alone
- Decreased limb extension: plantigrade stance, crouching gait
- Increased limb extension (stiff or stilted) with short steps followed by collapse
- Synchronous use of both hindlimbs (bunny hopping).

Some of these descriptions also apply to animals with orthopaedic disease causing lameness.

The affected limbs are discovered by observation and by provoking movement with postural reaction testing.

DEFINITIONS

The animal is described as:

- Tetraplegic or quadriplegic: paralysed in all four limbs
- Non-ambulatory tetraparetic: weak in all four limbs. Able to move all limbs but unable to stand and walk unaided
- Ambulatory tetraparetic: weak in all four limbs but able to walk unaided
- Hemiplegic: paralysed in the forelimb and hindlimb of one side
- Hemiparetic: weak in the forelimb and hindlimb of one side
- Paraplegic: hindlimb paralysis
- Paraparetic: hindlimb weakness
- Monoplegic: weakness of one limb. Usually described by stating which limb is affected and how badly, e.g. paralysis of the right forelimb or weakness of the left hindlimb.

CLINICAL TIP

Do not forget that weakness or paralysis may also occur in other parts of the body causing dysphagia, regurgitation, dysphonia, absent blink, inspiratory dyspnoea, inability to prehend or altered neck posture. Always check the history and the cranial nerve function.

LOCALIZATION

A chain of synapsing neurons is called a *pathway*. A pathway in the CNS may be known as a *tract, fasciculus, peduncle, or lemniscus*. Small bundles of neurons in the PNS are called *fascicles* which combine to create *nerves*.

Descending tracts from the brain transmit the impulses which make the animal move. The impulses gain access to the muscles via nerves.

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- Damage to the descending pathways causes paresis/paralysis
- Damage to the nerves causes paresis/paralysis
- Damage to the connection between nerves and muscles, the neuromuscular junction, causes paresis/paralysis
- Damage to muscle tends to cause paresis rather than paralysis.

CLINICAL TIP

Most damage to the cerebrum does not cause obvious paresis or paralysis. Such animals walk with a normal gait. Postural reactions can detect a subtle paresis.

Differentiating the cause of paralysis requires examination of the limb's nerve function: muscle tone and reflexes. If the tone and the reflexes are intact, present, normal (2+) or increased (3+, 4+) on examination it means that those nerves, and the spinal cord segments of their origin, are functioning and are not responsible for the weakness or paralysis.

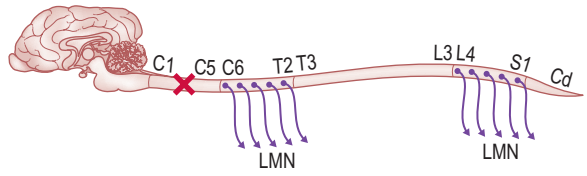
Fantastic! A functioning reflex arc means a whole chunk of the nervous system works! The cause of the weakness therefore lies in the descending tracts cranial to the weak or paralysed limb.

But…if the tone and reflexes are reduced (1+) or absent (0) on examination it means that those reflex arcs do not work and somewhere in that reflex pathway is to be found the lesion causing weak or paralysed limbs (Fig. 37.1) (see p 20, Table 2.3).

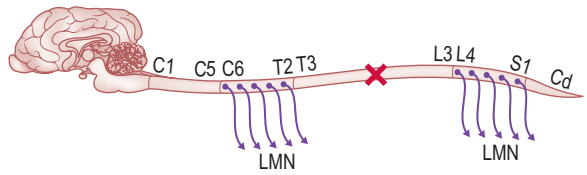
CLINICAL TIPS

- Muscle atrophy occurs with denervation and with disuse.
- Disuse atrophy is generalized and takes weeks to appear.
- Denervation atrophy is limited to the muscles supplied and is usually severe, occurring within days.

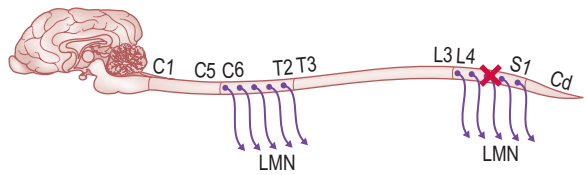
Imagine the motor control of the animal as a car, with the descending tracts being the engine. The nerves, neuromuscular junction (NMJ) and muscle are the wheels. If the wheels are removed, the engine can rev as long as it wants but the car will not move.



(A) Tetraparesis, ataxia of all four limbs, increased spinal reflexes and muscle tone



(B) Paraparesis with increased spinal reflexes and muscle tone in the hindlimbs (normal forelimb function)



(C) Paraparesis with decreased spinal reflexes and muscle tone in the hindlimbs (normal forelimb function)

Figure 37.1 Motor deficits in spinal cord lesions.

In anatomical terms, the engine is known as the upper motor neuron (UMN) and the wheels as the lower motor neuron (LMN).

UMN and LMN are used as adjectives when describing paresis and paralysis.

CLINICAL TIP

Review the technique on examination of reflexes. Practise at every opportunity to gain insight into normal variation.

Monitoring progression of spinal cord disease requires assessment of voluntary movement AND the spinal reflexes.

Imagine the common scenario of an acute severe thoracolumbar spinal cord lesion from trauma or intervertebral disc disease (IVDD). The animal initially presents with hindlimb paralysis and increased hindlimb reflexes. Several days later the hindlimbs become flaccid and areflexic. What presented as a UMN lesion now appears to be a LMN lesion! Why would this be? Has the animal suddenly got tick paralysis? Have the peripheral nerves been damaged by avulsion of the original trauma?

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No. Always consider that there may be one cause for all the signs. What has happened is that the original insult to the T3–L3 spinal cord has induced myelomalacia. This destructive process has extended caudally along the spinal cord, destroying the cord segments containing hindlimb reflex arcs. The animal still has the original lesion to the descending tracts, but once the reflex arcs are destroyed the limb becomes flaccid and areflexic. Remember the car: if the wheels are removed, the car will have LMN paralysis. The LMN paralysis will be the overwhelming clinical presentation.

CLINICAL TIP

The secret of localization is to view the process as a jigsaw. View all pieces in relation to each other to get the whole picture (of health or dysfunction).

IPSILATERAL OR CONTRALATERAL TO THE LESION?

The descending motor tracts originate in various parts of the brain. If the pathways are damaged ABOVE the red nucleus in the midbrain the weakness/paralysis will be CONTRALATERAL to the lesion.

If the lesion is BELOW the red nucleus, the weakness/paralysis will be IPSILATERAL to the lesion.

VARIATIONS

- Giant and large breed dogs with C1–5 spinal cord segment lesions can present with UMN hindlimb paresis and ataxia.
- Large breed dogs with caudal cervical cord C6–T2 lesions may not show obvious LMN signs to the forelimbs.

'Spinal walking'

Stretch reflexes become hyperactive after spinal cord transection. This gives rise to a positive supporting reaction induced by pressing on the pads of such an animal. The limb extends, following the point of pressure as it is withdrawn. This explains why paralysed animals can stand.

Pattern generators exist in the spinal cord and are the basis for 'spinal walking'. Automatic, hindlimb reflex walking can commence weeks to months after a severe T3–L3 spinal cord lesion (Grade 5). The cord transection appears functionally complete but tract remnants in these animals have survived and transmit impulses from the brainstem locomotor region. As few as 5–10% of the descending axons are sufficient to drive the local circuits producing basic locomotion in cats and rats. The gait is rhythmic but not coordinated with the forelimbs.

ASSESSING THE SEVERITY OF PARALYSIS

Spinal cord lesions are the most common cause of paralysis in small animals.

Cerebral lesions generally cause only subtle gait change and are accompanied by other signs such as behaviour change, alteration of mental state or seizures.

Brainstem lesions can result in significant gait change but are generally also accompanied by cranial nerve dysfunction and altered mental alertness.

The spinal cord contains ascending and descending tracts which show clinical deficits in the following order:

1. Ascending tracts: proprioception: most susceptible: ataxia proprioceptive deficits are commonly the first abnormality.
2. Descending tracts: motor: paresis/paralysis
3. Ascending tracts: pain perception: least susceptible: analgesia.

In a lesion which slowly develops, the most susceptible tracts will be affected first so the signs will be ataxia ± paresis. In a rapidly developing lesion the signs will progress through ataxia to paresis then paralysis in a short period of time. In many cases the rapid onset lesion will quickly produce paralysis with no progression of dysfunction being noted by the owner.

Spinal cord lesions are graded based on the level of dysfunction exhibited (Table 37.1). Grade 4 can be further defined with statements about urinary continence or incontinence. Incontinence in pets affects the quality of life of the owner and hence the animal's longevity.

Another grading system, the modified Frankel score, uses the opposite ranking with paralysis (0) and normal strength and coordination (5).

Table 37.1 Grading morbidity of spinal cord lesions

Grade	Dysfunction
1	Neurologically normal
2	Ataxia, ambulatory paresis
3	Non-ambulatory paresis
4	Paralysis, pain perception present
5	Paralysis, no pain perception

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CLINICAL TIP

In cases of acute spinal cord disease, if the animal can walk, it can feel pain in those limbs.

It is always worth repeating the maxim that flexion of the limb in response to pinching is an involuntary REFLEX action. It does NOT measure an animal's ability to perceive pain.

PROGNOSIS FOR RECOVERY

Prognosis for a patient to return to normal ambulation is determined by a combination of:

1. Grade of dysfunction
2. Duration of clinical signs.

Generally speaking, the more severe the deficit (grade 5 vs. grade 1), the more guarded the prognosis. 'Guarded' means 'a cautious appraisal' of the animal's chances of achieving a defined goal. It is the scientific equivalent of 'sitting on the fence'. The term 'grave prognosis' indicates that one should 'hope for the best but prepare for the worst'.

When giving an opinion of prognosis make it clear to which goals you are referring. Owners usually want to know a time frame in which this improvement will occur, e.g. 'there is a good prognosis for return of urinary continence and this is expected to happen within 2–3 weeks with the current treatment', e.g. 'there is an excellent prognosis for return of ambulation within 2 weeks but normal strength and coordination will take longer, perhaps up to several months'.

What is a 'good' prognosis?

Owners commonly ask for percentage success rates or survival times for different treatment methods. Journals quote these numbers for a group of afflicted animals, e.g. the most common length of time for recovery in animals with disease X is … days/months. Look at the details and note that for some, the time is shorter and for other animals, the time is longer. Individual variation exists. The owner can only be offered an estimate of success or longevity.

Also be aware that the published case studies represent treatment by clinicians who may have more expertise or better nursing facilities so that the published success rates may be higher than those attainable in other practices. Equally, the articles may represent a biased case selection, with a larger number of seriously ill animals being included in a study than would normally appear in a general practice population.

A pragmatic assessment would invoke the famous physics thought experiment known as Schroedinger's Cat. All outcomes are possible until the result is known.

The term 'good prognosis' is as subjective as the term 'seizure control'. Scientific papers define these terms but interpretation varies between papers. Pet owners do not read veterinary journals and have their own priorities and goals which the clinician must discover.

A grading system similar to the Karnofsky scale (Table 37.2) for disability may be useful in quantifying disability (and quality of life) in animals.

Table 37.2 Karnofsky scale for disability grading (modified)

(%)	Human	Cat/dog
100	Normal	Normal
90	Minor signs, normal activities	Minor signs. Remains playful, active
80	Normal life possible with effort	Reduced activity
70	Cares for self	Ambulatory paresis/ ataxia, eats, drinks, normal urination and defecation. Grooms
60	Cares for self with occasional assistance	Uses ramps or is carried up steps. Cannot climb. Harness or sling walks to support body weight. Falls
50	Requires frequent care and considerable assistance	Cannot walk unaided. Cannot urinate unaided. May use a K9 cart
40	Disabled, requires special care at home	Paralysed. Incontinent. Needs hand-feeding. Frequent turning and bathing
30	Severely disabled, hospitalized	No independent activity. Can ventilate
20	Very sick, hospitalized	No independent activity. Side-effects of disability, e.g. aspiration pneumonia, hypoventilation
10	Moribund	Moribund

Note: this scale represents a suggested method of quantifying ability in small animals but its utility remains unproven. After Karnosfsky et al 1984 Cancer 1:634.

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38 UMN: spinal empyema

INITIAL PRESENTATION

Inflammation of the spinal cord and its meningeal covering presents as ataxia, paresis and spinal pain along with less specific signs of lethargy, fever (37–39.5°C) and anorexia.

INTRODUCTION

Focal spinal cord disease is an uncommon presentation of inflammatory CNS disease. Any segment is at risk but inflammation of the cervical cord is most often reported. Dogs less than 3 years old and hound and toy breeds are most at risk of developing meningomyelitis. The onset of signs is highly variable, lasting days to over 1 week. Improvement without treatment is unusual.

The diagnosis of meningomyelitis is made by:

- CSF sample: increase the diagnostic yield by sampling both cranial and caudal to the localized lesion. Pleocytosis is found in the majority of cases. Tissue inflammation occurs to some degree in other conditions such as compression, infarction, and neoplasia. The general trend is that inflammatory CNS disease has greater pleocytosis than other conditions. Variation prevents this being a cast iron rule.
- MRI: may be normal. Contrast enhancement of parenchyma and/or the meninges is highly suggestive of the condition. The cord may be swollen.
- Myelogram: thought to be contraindicated in inflammatory disease because of the risk of contrast agent worsening the inflammation. Contraindicated in known inflammatory CNS disease as it adds nothing to the diagnosis or the treatment. It is used in an emergency only if the chief differential is a suspected extradural lesion requiring surgery. May be normal or show an intramedullary pattern.
- Serology: positive titres indicate exposure to an organism but do not necessarily indicate the presence or absence of clinical disease caused by the organism.
- PCR on CSF.

SIGNALMENT

A 1-year-old male English bulldog.

CASE PRESENTING SIGNS

Unwilling or unable to walk. In pain.

CASE HISTORY

The dog had been painful for 2 days, pacing, vocalizing and crying when moved or when the neck was touched, before becoming recumbent and depressed. Temperature was 39.5°C (normal: 37.5–39°C). Two weeks prior to becoming unwell, the dog had been bitten on the left ear. The wound had healed with time, cleaning and antibiotics.

CLINICAL EXAMINATION

The dog was quiet and lay in sternal recumbency. When held up, the dog neither cried nor attempted to move. Hopping and proprioceptive testing were negative in all limbs. When placed in lateral recumbency the dog could right itself slowly. Spinal reflexes were normal in all limbs. CNN were normal. The neck muscles were tense and resisted flexion.

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CLINICAL TIP

Inflammatory disease usually causes multifocal neurological signs, so recheck often for the development of other neurological deficits.

NEUROANATOMICAL LOCALIZATION

The lesion was localized to the cervical spinal cord segments C1–5.

DIFFERENTIAL DIAGNOSIS

- Discospondylitis
- Haemorrhage or inflammatory disease were considered most likely in a dog of this age with pain and fever
- Vertebral malformation with or without a cord malformation was a possibility.

CASE WORK-UP

Haematology showed a monocytosis of 2.09 (range 0.2–0.72 × 10^9/l). There was a mild elevation of muscle enzymes: AST 150(N = 0–45 IU/l), ALT 403(0–40 IU/l), CK 455(0–400 IU/l).

The buccal mucosal bleeding time was 2 minutes and 30 seconds (normal <4 minutes). The PT was 15 seconds (12–17) and APPT 94 seconds (71–102).

Toxoplasma and Neospora titres were negative.

MRI of the cervical spine found contrast-enhancement of the epaxial muscles at the level of the C1 and C2 vertebrae, extending between C1–2 and compressing the spinal cord dorsally (Fig. 38.1). Contrast-enhancement of the meninges was present. Discospondylitis of C1 was suspected.

CSF was not collected from the cisterna magna as it would have meant pushing the needle through the area of pathology. CSF could have been collected from the lumbar site but was not.

A dorsal surgical approach was made and soft beige coloured material was found dorsal to the arch of C1. This was debulked. Histopathology found myositis with oedema and fibrosis. The inflammatory cells were a mixed population. No infective organisms or foreign bodies were found. A very heavy growth of beta haemolytic *Streptococcus* sp was cultured from the tissue biopsies and was sensitive to amoxicillin/clavulanic acid.

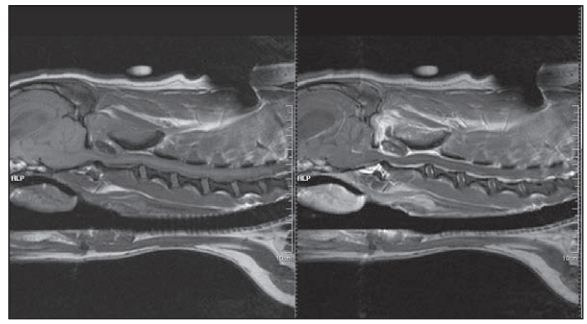


Figure 38.1 Sagittal T1WI and T1WI+C. Dorsal compression of the cord at the level of the atlas.

DIAGNOSIS

Acute cellulitis, secondary to a foreign body or an infected dermoid cyst. Haematogenous spread was possible given the previous bite wound.

Spinal epidural empyema, the collection of pus in the extradural space, is rare in small animals. The most common presenting signs are spinal pain, fever and rapidly worsening weakness. Leukocytosis and neutrophilia are common. Raised total protein and nucleated cell count in the CSF are also reported. Extradural compression of the cord may be focal or multifocal, with T3–L3 segments being the most commonly affected. The highest yield of positive cultures is obtained from the surgical site or blood culture. Any breed or age of dog may be affected. MRI characteristics of the epidural empyema are those of inflammation and oedema: hyperintensity or mixed signal intensity on T2WI, hypointensity on T1WI. A small area of signal void may represent haemorrhage, which can be visualized as hypointensity on GRE sequences, or a foreign body.

A dermoid sinus is created from an incomplete separation of the neural tube and skin on the dorsal midline, forming a tube-like tract lined with hair follicles and glands. The sinus opening is small and marked by a tuft of fur. When infected the sinus is painful and may cause neurological deficits appropriate to the location(s) in the cervical, cranial thoracic or sacrococcygeal regions. They may be located at either end of the dorsal ridge in Rhodesian ridgebacks, and have been reported in small breed and other large breed dogs. Treatment is surgical excision.

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TREATMENT

Decompression of the spinal cord was not performed at the same time as surgical biopsy due to the extensive material around the cord, and the potential to worsen the neurological condition due to cord trauma or spinal destabilization. The dog rapidly improved on IV antibiotics postoperatively and became ambulatory within days. Pain relief was supplied by methadone IM every 4 hours and could be discontinued soon after antibiotic therapy commenced and neurological improvement was seen.

FOLLOW-UP

Hindlimb ataxia and forelimb proprioceptive deficits were present a week after the operation. These resolved within another month while the dog was still receiving antibiotics. Another MRI was planned for 8 weeks after the operation to monitor resolution of the lesions. Had improvement not been seen, surgical decompression of the cord would have been performed.

BACTERIAL INFECTION OF THE CNS

Bacterial meningitis is rare in small animals. They are not affected by specific bacteria with a predilection for the nervous system as occurs in humans.

Extension from otitis media-interna is reasonably common and is visible on MRI as contrast enhancement extending around the brainstem. Affected animals do not consistently show signs of meningitis. A typical history would be chronic otitis externa with a sudden onset of neurological signs (peripheral vestibular disease with or without facial paralysis and Horner's syndrome) localized to the middle and inner ear. Ipsilateral paresis and proprioceptive deficits are uncommon. Bulla osteotomy and systemic antibiotics resolve the extension to the brain in most cases.

Bite wounds penetrating the skull or tail wounds causing an epidural infection are reported. Surgical drainage of an abscessed site followed by 6–8 weeks of broad-spectrum or culture-specific antibiotics contribute to an excellent prognosis.

Fever is inconsistently present. Approximately half the cases have a peripheral leukocytosis or leukopenia or thrombocytopenia. CSF is abnormal in 90% of cases, with degenerate and toxic neutrophils and a raised total protein. Unfortunately, CSF cultures are generally negative even in proven bacterial infection. There are no specific recommendations on how to improve this. Blood and urine cultures are also diagnostically disappointing. All are submitted, but treatment is commenced on an empirical basis. Gram-negative infections are most common. Single or multiple organisms may be present (Table 38.1). Intravenous antibiotics are used until improvement is seen, usually taking 3–5 days, and are followed by oral antibiotics until resolution occurs in approximately 2 weeks.

Table 38.1 Systemic bacterial infection with CNS involvement

Organism	Neurological signs	Other signs	Diagnosis	Treatment
Leptospirosis (*Leptospira interrogans*)	Benign meningitis. Rare	Kidney, liver, lung, eye, coagulopathy	IgG/IgM Fresh urine-dark field, culture PCR	Penicillin, doxycycline (potential zoonosis)
Borreliosis (*Borrelia burgdorferi*)	Not well documented	Joint, kidney	Difficult. Antibodies common, disease uncommon. PCR on CSF	Doxycycline Amoxicillin 3rd generation cephalosporins
Mycobacteriosis (classic tuberculosis)	CNS, occasionally	Skin, lung, GIT, bone, eye	Acid-fast organisms in tissue or exudates. Culture	Euthanasia (potential zoonosis)
Listeriosis (*L. monocytogenes*)	Rare Meningoencephalitis	–	–	
Brucellosis (*B. canis*)	Rare Uveitis, meningitis, discospondylitis	Minimal in neutered animals despite chronic bacteremia	Antibody Blood culture	Difficult. Gentamicin and minocycline (zoonosis)

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Treatment

1. Antibiotics
   - *Empirical*: third generation cephalosporin (Gram-negative) with metronidazole (anaerobic)
     - Cefotaxime 25–50 mg/kg IV q8 hr
     - Metronidazole 10 mg/kg slow IV q8 hr
   - *Empirical*: high dose IV aminopenicillin (Gram-positive aerobes and anaerobes, some Gram-negative)
     - Ampicillin 22 mg/kg IV q6 hr.
2. Corticosteroids

Anti-inflammatory dose of prednisolone PO or dexamethasone IV. Short term.

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39 UMN: spinal cord infarction

INITIAL PRESENTATION

Sudden onset of asymmetrical weakness and proprioceptive deficits.

INTRODUCTION

Spinal cord disease may present with either upper motor neuron (UMN) or lower motor neuron (LMN) signs, or a combination of the two, depending on the location of the lesion.

SIGNALMENT 1

A 2-year, 6-month-old neutered female miniature schnauzer dog.

CASE PRESENTING SIGNS

Sudden onset of asymmetrical weakness.

CASE HISTORY

This dog began dragging the dorsum of the right hind paw. Later the same day the right forelimb started to collapse when weight-bearing. The dog seemed a little depressed but brightened up after pain relief was given by the local vet. There had been no prior illness or injury reported. The signs had not apparently improved or worsened during the 3 days prior to referral.

CLINICAL EXAMINATION

The dog was alert and non-ambulatory. When held up, voluntary movement was seen in all limbs. Hopping was normal in the left forelimb, slow in the left hind, and absent in the right fore and hind limbs. Spinal reflexes were normal to increased in all limbs. Muscle tone was increased in all limbs. No spinal pain was elicited.

NEUROANATOMICAL LOCALIZATION

The lesion was localized to the cervical spinal cord segments C1–C5, on the right hand side.

DIFFERENTIAL DIAGNOSIS

The sudden onset and strikingly lateralized nature of the signs of this spinal cord disease are very suggestive of spinal cord infarction caused by fibrocartilagenous thromboembolism. Compressive cord disease such as intervertebral disc disease (IVDD) and neoplasia is less focal as a general rule and tends to progress. Sudden onset IVDD is usually painful.

CASE WORK-UP

MRI of the cervical cord 4 days after the onset of signs found an area of hyperintensity on T2WI within the right side of the spinal cord at the level of the C4–5 vertebrae. CSF was normal, but this may have reflected sampling rostral to the lesion (Fig. 39.1).

Haematology and biochemistry were normal. Clinical signs had improved overnight and the animal was ambulatory with proprioceptive deficits of the right fore and hind paws at each step.

DIAGNOSIS

The diagnosis is based on a combination of factors as no one factor is diagnostic in itself. The sudden onset, progression, lateralization and improvement, coupled with signal changes on MRI suggestive of intramedullary oedema or tissue necrosis supported the clinical diagnosis of a fibrocartilagenous thromboembolism.

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Figure 39.1 Transverse T2WI. Focal hyperintensity within the right side of the cervical spinal cord.

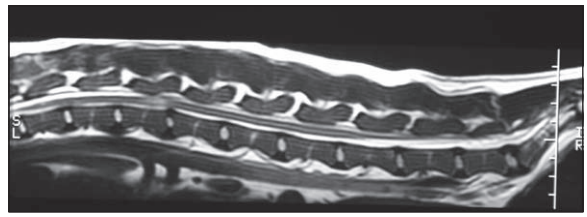


Figure 39.2 L1–2 IVD protrusion with syrinx present cranially in a dog suspected of having a lesion within the L7–S1 cord segments.

SIGNALMENT 2

A 4-year-old neutered female golden retriever.

CASE PRESENTING SIGNS

Sudden onset of asymmetrical weakness with right hemiparesis.

CASE HISTORY

The golden retriever was let out into the garden in the morning and did not return. The owner found the dog sitting down, unable to rise but trying to pull itself along with the forelimbs. The dog had been out of sight for less than 5 minutes.

CLINICAL EXAMINATION

Abnormal findings consisted of a paralysed left hindlimb and a non-ambulatory paretic right hindlimb. The patellar reflex was present in the left hindlimb and reduced in the right hindlimb. The withdrawal reflex was absent in the left hind- and slightly reduced in the right hindlimb, as compared with the forelimb withdrawal reflexes. Pain perception was present in both hindlimbs. No spinal pain was found.

NEUROANATOMICAL LOCALIZATION

The lesion was localized to the lumbosacral intumescence based on reduced hindlimb reflexes.

DIFFERENTIAL DIAGNOSIS

- Infarction
- Haemorrhage
- IVD disease.

CASE WORK-UP

MRI found a very small syrinx at the level of the L1 vertebra which is not anatomically correct for the clinical signs. No abnormalities were found on blood work or lumbar CSF (Fig. 39.2).

Hindlimb movement returned and the dog was ambulatory on three limbs within 3 weeks with nursing and hydrotherapy. The left hind improved to an ambulatory state but had residual proprioceptive deficits. The patellar and withdrawal reflexes had all improved.

DIAGNOSIS

A spinal cord infarction was suspected based on the history and the speed of improvement.

AETIOPATHOGENESIS

Fibrocartilaginous thromboembolism (FCE) is very common in medium to large breed dogs aged 1–7 years and presents as a sudden onset paralysis. Signs may worsen from paresis to paralysis within 12–24 hours but not beyond. It commonly commences at exercise such as jumping for a ball, getting into the car, or just after being let out into the garden. A sudden cry is often heard but no further evidence of pain is shown. Many dogs suddenly rendered paraplegic struggle to sit, and frantically push away with their fully functional forelimbs. When examined, the animals generally do not show spinal pain. However one study found that 21% of dogs did show spinal pain on examination.

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Smaller breed dogs and cats are less commonly affected but the presentation is the same as for larger dogs.

Examination often reveals a striking asymmetry of deficits between the affected limbs. Lesions can occur anywhere along the spinal cord length. Reflexes and muscle tone reflect the lesion location except for those occurring in the lumbosacral intumescence which can produce an interesting mixture of deficits: the clinical picture is one of flaccid paraplegia and yet some reflexes are intact. It could be that the non-focal nature of the thromboembolism is more noticeable in an intumescence. Others have ascribed this melange of reflex changes to spinal shock.

Characteristics of an FCE

- Sudden onset
- Lack of progression of signs
- Asymmetry of signs
- Non-painful.

The cause of fibrocartilaginous material embolizing spinal vasculature is not known. The affected animals are generally healthy. Investigation is aimed at ruling out other causes of acute spinal cord disease:

- Haemorrhage
- Thromboembolic disease
- Intervertebral disc disease
- Neoplasia
- Inflammatory disease.

MRI, if available, is useful as it examines the intramedullary region. Images may be normal on standard MRI sequences during the first 48 hours after the onset of signs. Myelography is technically difficult if cord swelling is present. CSF changes, when they occur, are not specific.

Treatment consists of nursing the recumbent patient. Corticosteroids are not required and do not alter the prognosis. The prognosis is generally excellent with many dogs showing improvement within 2 weeks. Most return to a functional 'good quality' life. Some 88% of non-ambulatory patients regain the ability to walk within 1 month.

FCEs affecting the lumbosacral intumescence are said to have a poorer prognosis but improvement is still possible. Owners must be prepared for a recovery phase lasting months, during which time hydrotherapy, physiotherapy, bladder and bowel care are required.

CLINICAL TIP

Recurrence is rare. It is not possible to give advice on prevention as the cause of FCE is not known.

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40 UMN: vertebral malformation

INITIAL PRESENTATION

An acute onset of signs with chronic underlying pathology.

INTRODUCTION

Congenital vertebral malformations are commonly found in neurologically normal dogs. Do not presume a causal relationship between neurological signs and a vertebral malformation on the basis of non-contrast radiographs. Malformation of the vertebral body rarely causes a problem.

A hemivertebra has a wedge-shaped vertebral body and most commonly occurs in the thoracic or coccygeal vertebrae of screw-tailed breeds such as pugs and bulldogs. Adjacent vertebrae alter their shape to conform, resulting in scoliosis or kyphosis. Thoracic hemivertebrae may lack a rib.

Butterfly vertebrae have a cleft in the vertebral body with or without lateral spread of the two halves, and funnel-shaped endplates. They are not clinically significant and are most common in the screw-tailed breeds.

Spina bifida is a developmental anomaly with a midline cleft in the dorsal vertebral arch through which spinal cord and meninges may penetrate.

Block vertebrae are composed of two or more vertebrae fused into one. Bone density is normal. The overall size may be shorter.

A transitional vertebra is one occurring at the junction of two regions that takes on the characteristics of the arch and articular facets of the adjacent division.

SIGNALMENT 1

A 2-year, 6-month-old neutered female English mastiff.

CASE PRESENTING SIGNS

Acute hindlimb paresis.

CASE HISTORY

The dog had always been clumsy since she was acquired as an 8-week old puppy. She has struggled to rise from sitting and has abducted all limbs when walking on tiled flooring all her life. No hip disease had been found to account for this. Three days prior to presentation the dog had slipped on a wet tiled floor and fallen, collapsing to the ground. She was unable to rise, but could crawl. Two days later she was able to stand and walk unaided but had hindlimb ataxia. The dog did not appear to be in pain.

CLINICAL EXAMINATION

The dog was slow to rise from sitting but could do so unaided. She showed hindlimb ataxia with the right hindlimb circumducting at each step. The forelimbs had a shortened stride length. Hopping was normal in the forelimbs but slow in the hind, especially the right hindlimb. Proprioception was normal in the forelimbs but slow in the hindlimbs. When standing, the dog shifted most of its weight to the forelimbs. Muscle tone was increased in both hindlimbs. Spinal reflexes were increased in the hind and normal in the forelimbs.

NEUROANATOMICAL LOCALIZATION

The lesion was localized to the T3–L3 spinal cord or the cervical spinal cord. Large and giant breed dogs with cervical cord lesions can present with hindlimb UMN signs.

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DIFFERENTIAL DIAGNOSIS

- Spinal cord compression by vertebral or meningeal malformation was considered the most likely given the chronically abnormal gait
- Malformation of the articular facets.

1. 'Wobbler syndrome': cervical spondylomyelopathy or cervical vertebral malformation/malarticulation

Compression of the cervical spinal cord in large and giant breed dogs commonly presents as hindlimb ataxia with or without paresis. The term *Wobbler* vividly describes the ataxia and 'syndrome' indicates that there are many potential causes for the lesion. Forelimb gait may appear normal, hypermetric, or have a shortened, 'choppy' stride. As expected from compressive lesions, manifestations of pain such as a low head carriage, resistance to neck movement, and a painful reaction to neck palpation can occur. Tetraplegia is unusual.

Onset of signs is insidious over weeks to months. Acute signs follow IV disc extrusion or a further insult superimposed onto a chronic lesion. Age of onset varies from breed to breed. Approximately 80% of cases are either young Great Danes (<2 years old) or middle-aged Doberman pinschers aged 6–8 years with males more commonly affected than females. Female Borzois have been reported with Wobbler syndrome. Compression most commonly occurs at vertebral level C5–6 and C6–7 in the Doberman.

Anterior stenosis of the vertebra canal at C2–3 and C3–4 is reported in young Bassett hounds under 8 months of age.

Diagnosis requires imaging. Abnormal angulation 'slipping', 'tipping' or 'tilting' of the vertebrae on non-contrast radiographs can be seen in dogs without cervical spinal cord disease and are not considered pathognomonic of Wobbler syndrome. Stenosis cannot be diagnosed without myelographic proof.

Myelography allows depiction of static and dynamic compression of the spinal cord in various positions. It is not an entirely benign procedure so should be reserved for those cases which have progressively worsening neurological function or neck pain that does not improve with cage rest. In one study, 20% of dogs experienced transient neurologically deterioration for 2–3 days after a myelogram, regardless of the site of injection or the degree of neck manipulation.

The most severe lesion is decided by the greatest degree of compression seen on the myelogram but 53% have a poor correlation between the myelographic compression and the severity of the clinical signs.

A recent study found that restricted activity and use of a thoracic harness rather than a neck collar, with or without the use of corticosteroids gave rise to similar levels of clinical improvement (70–75%) over the same time period as surgically treated dogs. Survival times were also similar between the conservative and surgical groups.

Surgery has been the mainstay of treatment but serious thought should be given to using a conservative alternative.

2. Spinal subarachnoid cysts (SAC)

- Scalpel blade-shaped dilations of the subarachnoid space in cats and dogs are known as subarachnoid cysts despite the lack of an epithelial lining. Paresis and ataxia progressively worsen over weeks to years prior to presentation. Most are located in the dorsal cervical subarachnoid space between C1–3 vertebrae of Rottweilers less than 1 year old but older dogs, and smaller breeds are also affected. Neck pain is not typically present. Thoracic SACs have the same clinical behaviour, tend to occur between T11–13 vertebrae, and have been reported in the cat. SACs may be multiple.
- Intradural extramedullary compression of the cord is found on myelography in all cases. Filling of the defect may be more obvious when in lateral recumbancy during a myelogram, than in dorsal recumbency for MRI.
- Surgical marsupialization is the treatment of choice and generally has a good outcome in young adults with a duration of signs of less than 4 months.

3. Extradural synovial cysts

- Dorsolateral extradural compression of the cervical spinal cord in young adult male large breed dogs occurs secondary to compression from cystic synovium of the adjacent intervertebral articular facet. Signs are progressive (<6 months) and may be relieved by surgical removal of the cystic tissue.

4. Infarction

5. Spinal calcinosis circumscripta (cervical tumoral calcinosis)

- Large focal partially mineralized masses between C1 and C2 or the cranial thoracic spine are occasionally

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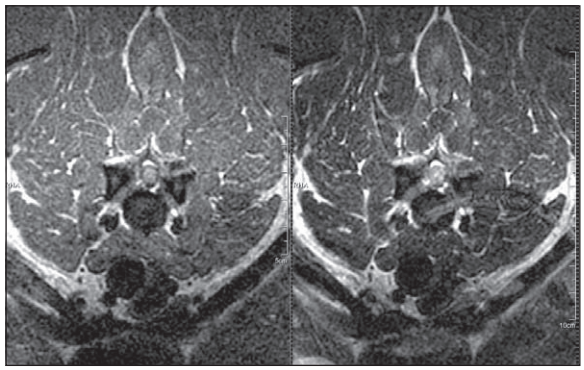


Figure 40.1 Transverse T1WI (left) and T2WI (right). Lateral compression of the spinal cord.

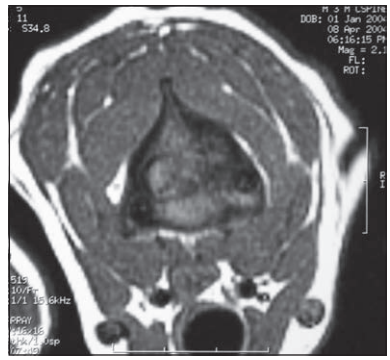


Figure 40.2 Transverse T1WI through axis. Compression of spinal cord.

found in young large breed dogs. Progressive expansion of the mass causes epidural spinal cord compression with or without spinal pain.

6. Atlantoaxial instability, subluxation, luxation

- C1–2 malformation and subsequent subluxation following trauma is more common in, but not limited to, toy breed dogs. Congenital agenesis of the dens has been reported in the rottweiler.

CASE WORK-UP

MRI of the neck was performed first and lateral compression of the spinal cord by the articular facets was seen at the level of C7–T1. This was worse on the right side (Fig. 40.1).

MRI of the T3–L3 spinal cord did not detect any lesions.

DIAGNOSIS

- Vertebral malformation with secondary cord compression
- Acute minor trauma on a chronically compromised spinal cord.

PROGNOSIS

The dog was improving quickly following the fall indicating a good prognosis for resolution of the acute signs. No medication had been used. There was a poor prognosis for reversal or improvement of the chronic ataxia and weakness.

FOLLOW-UP

The dog had improved back to its original state of disability by 13 days.

SIGNALMENT 2

A 13-week-old male Airedale puppy.

CASE PRESENTING SIGNS

This puppy had suddenly become non-ambulatory 2 days after purchase. The pup did not seem in pain.

Neurological examination found an alert non-ambulatory tetraparetic dog with absent proprioception in all limbs, and increased spinal reflexes. The lesion was localized to the cervical spinal cord segments C1–5. Malformation of the axis was found (Fig. 40.2). Voluntary motor control returned within 2 weeks. Treatment was 0.25 mg/kg prednisolone BID but the animal may have improved simply from the passing of time.

The dog remains ambulatory 3 years after first examination.

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41 UMN: intervertebral disc disease

INITIAL PRESENTATION

- Pain
- Proprioception deficits
- Paresis/paralysis.

INTRODUCTION

The anulus fibrosis of the intervertebral disc may rupture or bulge, and compress the adjacent spinal cord, nerve root, or both. Normal activity is sufficient to precipitate IVDD in a degenerate disc (Figs 41.1 and 41.2 and see Table 41.1).

Signs may occur suddenly or gradually. They can resolve with time and rest, or progressively worsen. Pain is a common sign, occurring in 60–70% of affected dogs (Table 41.2).

Cats are also affected by spontaneously occurring Type I disc disease. Type II is found as an incidental finding at post-mortem. The range of clinical presentations is similar to the dog. Pain may or may not accompany feline IVDD. The age range for feline IVDD is wide: 4–17 years, with most affected before 8 years of age.

The following cases of two dogs with hindlimb ataxia illustrate the differences in localizing lesions between large and small breed dogs.

SIGNALMENT 1

A 6-year-old neutered female rottweiler.

CASE PRESENTING SIGNS

Hindlimb ataxia.

CASE HISTORY

The dog suddenly became depressed, lethargic and developed hindlimb ataxia.

CLINICAL EXAMINATION

Clinical examination showed a quiet but mentally responsive ambulatory dog with an arched back, lowered head posture, hindlimb ataxia and a shortened stride in both forelimbs. While sitting, the dog was seen to knuckle the right fore and not correct this abnormal posture. Hindlimb hopping was decreased. Spinal reflexes were increased in the hindlimbs and normal in the forelimbs. No thoracic, lumbar, or lumbosacral pain could be elicited. The abdomen was relaxed. The neck was tense but the dog was muzzled and this may have been a pain response or a behavioural response to being handled.

NEUROANATOMICAL LOCALIZATION

The lesion was localized to the cervical spinal cord.

DIFFERENTIAL DIAGNOSIS

- Intervertebral disc disease (IVDD)
- Meningioma
- Vertebral tumour

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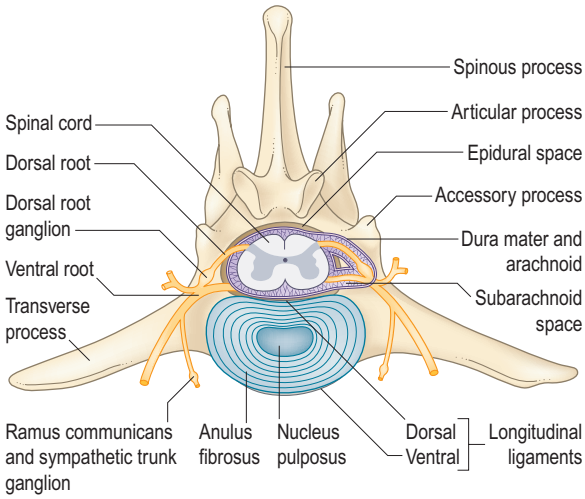


Figure 41.1 Cross-section through disc space.

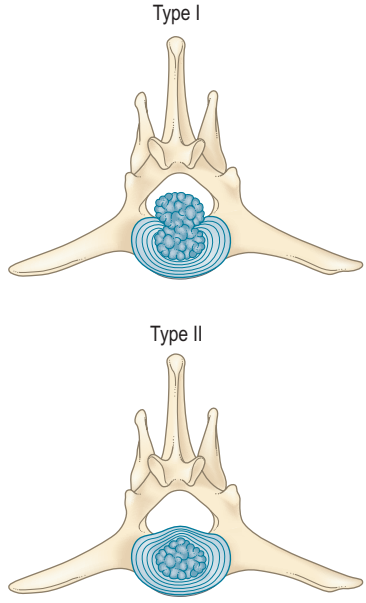


Figure 41.2 Type I and type II disc disease.

- Chronic lesion with an acute worsening e.g.: compression from articular facets or subarachnoid cysts.

Disc disease is the most common cause of spinal cord disease in dogs and was considered the most likely diagnosis in this case.

CASE WORK-UP

Non-contrast radiography

Approximately 50–80% of IVDD cases can be accurately diagnosed on non-contrast spinal radiographs, provided the exposures and positioning are optimum. Even radiologists have trouble detecting a second site of involvement. This level of accuracy is insufficient for surgical treatment. Spinal surgery is not like an exploratory laparotomy.

Spinal radiographs are performed when spinal pain is present and infection, neoplasia or trauma is thought to be the underlying aetiology. General anaesthesia is required for optimum positioning. If the animal has neurological deficits sufficient to warrant surgery, then contrast radiography (myelography), MRI, or CT of the relevant anatomical area is required. It is desirable to refer the patient to a clinician capable of localizing the lesion, imaging the spinal cord, and performing any surgery required. It is a waste of time and money to perform non-contrast spinal radiography on a paraplegic dachshund with back pain only to wake the animal up immediately afterwards and refer it on.

Radiographic signs of IVDD

- Narrowed or wedge-shaped IV space
- Narrowed articular process joint space
- Decreased size of the IV foramen
- Calcified disc material within the IV foramen
- Calcified disc material in the spinal canal
- Gas in the IV space (vacuum phenomenon)
- Displacement of calcified dura towards the cord (automyelogram).

Table 41.1 The nature of the anulus pathology has been categorized as type I or type II

	Type I	Type II
Pathology	Tear, rupture	Bulge, prolapse
Breed	Chondrodystrophoid (2–7 year olds) Non-chondrodystrophoid (8–10 year olds) Cat	Non-chondrodystrophoid Cat

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Table 41.2 Signs of disc disease

	Cervical	Thoracolumbar
Location	All, except C1–2	Caudal to T9–10
Common presentation	Neck pain Forelimb lameness	Back pain Non-ambulatory paraparesis Paraplegia
Pain perception	Intact	May be absent
Respiration	± Hypoventilation	Unaffected

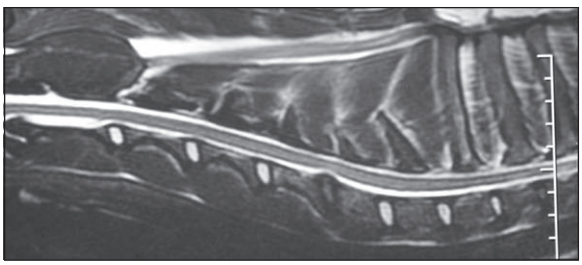


Figure 41.3 Sagittal T2WI. Extradural compression of the cord at the C5–6 IVD space.

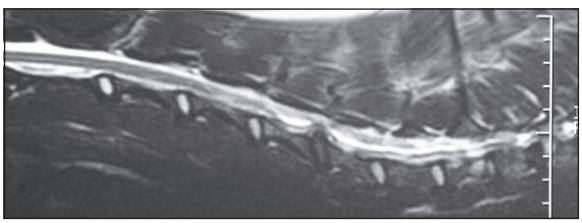


Figure 41.4 Sagittal T2WI. Extradural compression noted in a more lateralized 'slice'.

Myelography

The intervertebral site of thoracolumbar disc disease is accurately located by myelography in 80–100% of cases. Disc disease often lateralizes, i.e. the IVD material will be greater on one side of the cord than the other. Myelography was successful in locating the side of the lesion in 30–100% of cases. In 20% of myelograms, the site of the TL IVDD could not be found at all. Disappointingly, lateralization of clinical signs does not always correspond with the lateralization of the disc material.

For all the limitations of myelography, it remains a useful technique for the emergency spinal case that is a potential candidate for surgical decompression (Figs 41.3–41.5).

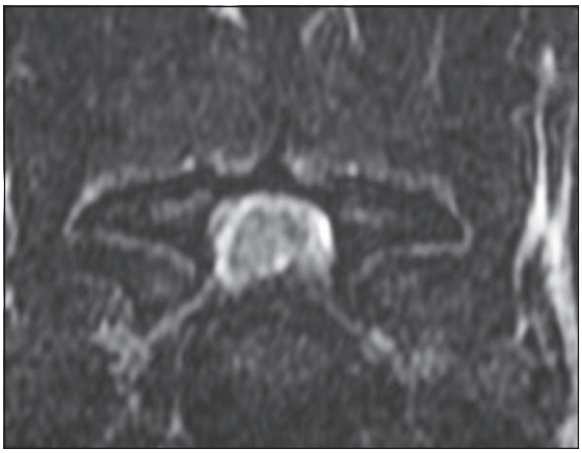


Figure 41.5 Transverse T2WI through the C5–6 IVD space with ventral compression of the cord by hypointense material.

This case had narrowed C5–6 IVD space on non-contrast radiographs.

MRI illustrated a C5–6 IVD extrusion.

CSF was normal.

DIAGNOSIS/TREATMENT

C5–6 ventral slot enabled removal of extruded disc material from the spinal canal.

PROGNOSIS

An approximate rule of thumb is that paraplegic dogs that perceive pain have more than an 80% chance of walking again if they are treated surgically. Those without pain perception and treated surgically have a 50% chance of walking again within 3 months and have a high incidence (30–40%) of intermittent faecal and urinary incontinence. A voluntary tail wag may be present in a paraplegic dog.

The prognosis for cats with IVDD appears to be the same as for dogs. Consult surgical texts for further information.

Ascending myelomalacia is the autolysis of the spinal cord following acute, severe spinal cord injury, whether from IVDD or a fracture. Approximately 10% of dogs with paraplegia and lack of pain perception will develop this irreversible condition within 5–7 days of the onset of paralysis whether the spinal cord is decompressed or not. Destruction of the cord produces LMN signs

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in the hindlimbs, a progressive loss of the cutaneous trunci reflex, and eventually tetraparesis. Treatment is euthanasia.

Depending on the study, recurrence rates for subsequent Type I thoracolumbar IVDD in dogs range from 2.6% to 26.5%. Prognosis for recovery is as above.

SIGNALMENT 2

A 4.5-year-old neutered female miniature shorthaired dachshund.

CASE PRESENTING SIGNS

Hindlimb paralysis.

CASE HISTORY

The dog had become depressed, and exhibited a lower head posture and arched back 4 days prior to referral. She had been allowed to jump and run about and developed hindlimb paralysis 24 hours prior to referral.

CLINICAL EXAMINATION

Clinical examination showed a quiet, depressed dog with hindlimb paralysis and absent hindlimb pain perception. Forelimb function was normal. Patellar and withdrawal reflexes were normal in the hindlimbs. CNN were normal.

NEUROANATOMICAL LOCALIZATION

The lesion was localized to the T3–L3 spinal cord.

DIFFERENTIAL DIAGNOSIS

The signalment and history were highly suggestive of IVD disease. Spinal or spinal cord tumours are rare. Haemorrhage into the cord and subarachnoid space can cause pain and progressively worsening signs. Infarction is not generally painful and does not progress to paralysis over 4 days (see Table 41.3).

CASE WORK-UP

Myelography found extradural compression of the cord at T13–L1 on the right side, extending rostrally and caudally (Fig. 41.6).

Table 41.3 Comparison of clinical signs in acute pancreatitis and thoracolumbar IVDD

Acute pancreatitis	Acute IVDD
Tense abdomen	Tense abdomen
Depression	Depression
Anorexia	± Inappetance
Vomiting	± Proprioceptive deficits
Diarrhoea	± Paraparesis or paraplegia



Figure 41.6 VD myelogram with right contrast column deviating medially and narrowed T13–L1 IVD space.

DIAGNOSIS/TREATMENT

A right hemilaminectomy at T13–L1 allowed removal of extruded disc material.

The dog had not regained voluntary motor control in either hindlimb and remained urinary incontinent 4 years after initial presentation.

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42 UMN: spinal fracture

INITIAL PRESENTATION

Spinal pain, paralysis/paresis, proprioceptive deficits, urinary incontinence.

INTRODUCTION

Most fractures are due to road traffic accidents (RTA) and most occur in the thoracolumbar spine. Associated injuries are significant: 34% of dogs with lumbar fractures have cardiopulmonary injuries such as pneumothorax and pulmonary contusions; 48% have other fractures, of which pelvic fractures were most common and approximately 23% have abdominal injuries.

Neurological deficits may not correspond to the site of the fracture due to the associated cord oedema, haemorrhage or myelomalacia. Repeat examinations at least twice daily, paying particular attention to the quality of spinal reflexes and muscle tone in the limbs, any 'cut-off' in the cutaneous trunci reflex, and movement of the thorax. An UMN paraplegia changing into LMN paraplegia indicates that the lesion is extending into the lumbosacral intumescence. Cranial extension of the lesion within the thoracic spinal cord will not change the hindlimb reflexes or muscle tone but the cutaneous trunci reflex will be abnormal and thoracic movement will weaken. Ascending myelomalacia is usually recognized before the animal dies of respiratory failure. Schiff–Sherrington syndrome occurs with sudden onset T3–L3 spinal cord lesions and may give the impression of a UMN cervical lesion.

Assessing the degree of voluntary motor function is difficult when the animal is kept immobile to stabilize the fracture site. Calling the animal or offering food may induce voluntary movement in limbs. Pelvic fractures add to the animal's immobility.

Palpating the spine may detect a depression over the site of dorsal spinous process fracture or malalignment of vertebrae. Luxation of vertebrae without a fracture is uncommon.

SIGNALMENT

A 6-month-old male collie dog.

CASE PRESENTING SIGNS

Hindlimb paralysis.

CASE HISTORY

The dog had been hit by a car the previous day.

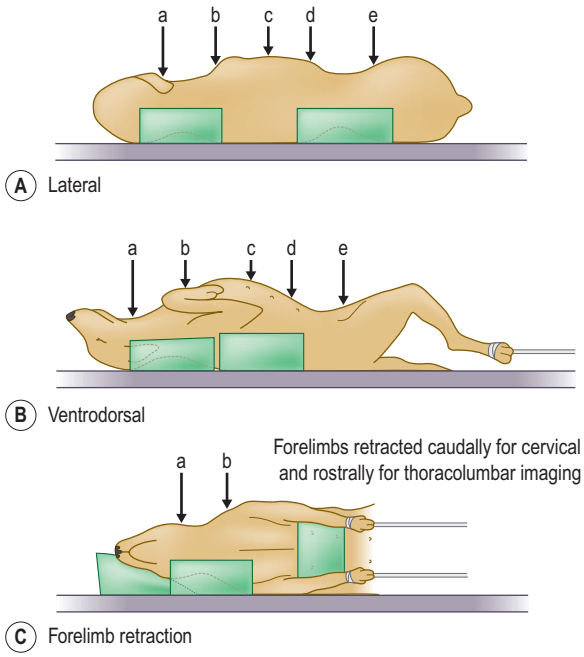
CLINICAL EXAMINATION

The dog was mentally alert with hindlimb paralysis, normal to slightly increased hindlimb reflexes, no pain perception in either hindlimb, absent cutaneous trunci reflex caudal to the scapulae and absent pain perception caudal to the scapulae. The bladder was large and difficult to express. Respiratory movements were chiefly abdominal.

NEUROANATOMICAL LOCALIZATION

The lesion was localized to the T3–L3 cord. Lack of pain perception localized the lesion further to the cranial thoracic cord.

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Area	Primary beam
a Cranial cervical	C2 – C3
b Caudal cervical	C5 – C6
c Thoracic	T8
d Thoracolumbar	T13 – L1
e Lumbar	L4 – L5

Figure 42.1 Positioning spinal radiographs.

DIFFERENTIAL DIAGNOSIS

Trauma.

CASE WORK-UP

Radiography

Positioning must reflect the neuroanatomical diagnosis (Fig. 42.1). Trauma, infection and neoplasia may produce multifocal lesions which require the whole spine to be radiographed. Assess changes in vertebral shape, density, alignment and surrounding soft tissues. Always check the dorsal articular facets as these will remain fractured even if vertebral bodies realign after the trauma (Fig. 42.2 and see Table 42.1).

Lateral and VD views are required to assess alignment of the spine. Support the spine when turning the animal under general anaesthesia or sedation as the relaxation of spinal muscles removes a natural splint. Spontaneous reductions of fractures and luxations can occur immediately after the trauma (Fig. 42.3). This creates a disparity between the severity of neurological signs and the changes seen on radiographs. Always check the articular, transverse and dorsal spinous processes and ribs for fractures. The L7 vertebral body may be displaced by more than 50% and show minimal signs due to the mobility of the cauda equina found at that vertebral level.

Damage to the vertebral arch (dorsal lamina and pedicles), articular facets and associated ligaments with or without a vertebral body fracture is regarded as an unstable configuration.

Wedge compression fractures or oblique fractures of the vertebral body are usually stable but bone fragments may enter the spinal canal.

MRI, CT, or myelography is required to assess the compression of the spinal cord. MRI has the additional advantage of imaging the intramedullary region (Fig. 42.4).

DIAGNOSIS

Trauma.

PROGNOSIS

Approximately 50% of thoracolumbar spinal fracture patients are euthanized due to severe neurological deficits.

The chance of ambulation returning in a paraplegic without pain perception starts at 50% and drops to 0% if the spine is displaced by 100% or greater, or if pain perception has been absent for more than 2 days.

Presence of pain perception does not ensure complete neurological recovery.

TREATMENT

There is no pharmacological cure for spinal cord injury. Do not rely on corticosteroids.

Treatment

1. Identify the cause, e.g. external trauma, pathological fracture, AA subluxation, IVDD and FCE.
2. Stabilize spinal fractures.
3. Administer pain relief.
4. Nurse the recumbent patient
5. Monitor the progression of neurological deficits.

Applying an external splint to limit ventral and lateral flexion can produce the same degree of improvement as

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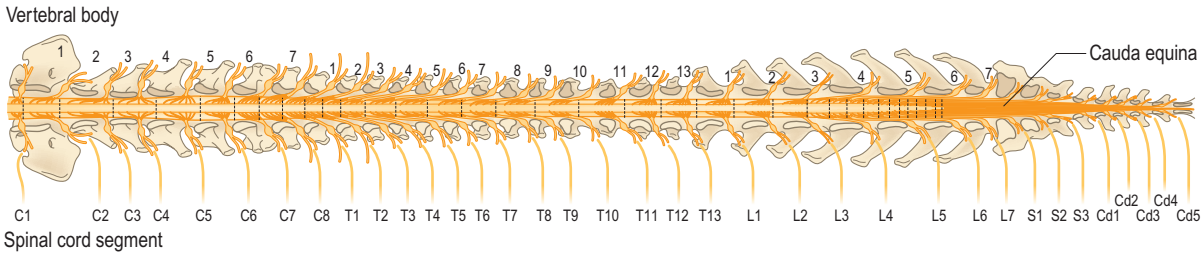


Figure 42.2 Alignment of the spinal cord and vertebral bodies. Note that there are eight cervical spinal cord segments and only seven cervical vertebral bodies.

Table 42.1 Termination of the spinal cord within the spine

	Position of S1–3 segments	Level of cord termination
Dog	L5 vertebra	Cranial half of L7 vertebra
Smaller dog		Caudal half of L7 vertebra
Cat	L6 vertebra	Within the body of L7

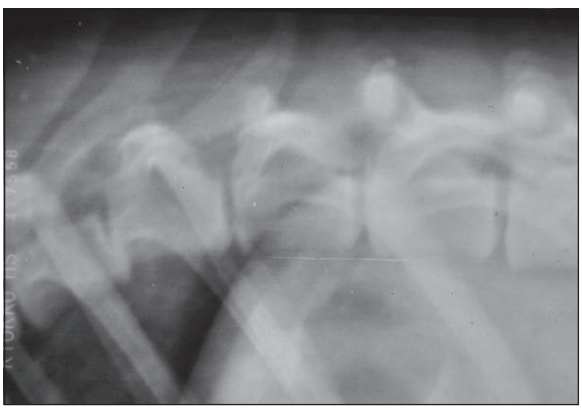


Figure 42.3 Note the luxation of the thoracic vertebrae.

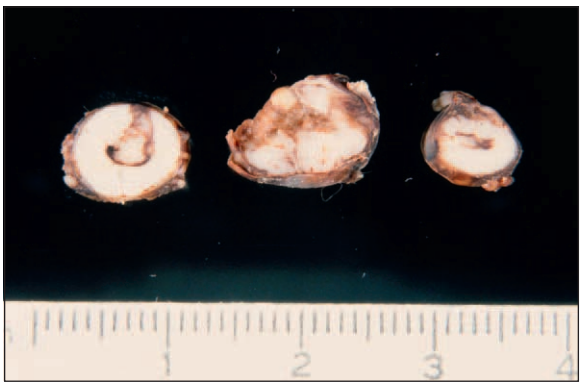


Figure 42.4 Destruction of thoracic spinal cord.

internal surgical fixation of thoracolumbar spinal fractures in dogs. Medical treatment takes approximately twice as long to reach the same neurological grade as that achieved by surgery but is cheaper and requires a shorter hospital stay.

Medical treatment is not an easy option: the owner must be vigilant in checking for pressure sores, bandage soiling and urine scalding. It may not be tolerated by cats, and may not withstand the pressures generated by large-breed hyperactive dogs.

Surgery is recommended if the spinal cord requires decompression, if the spine requires alignment and stabilization or if the animal deteriorates with medical care.

Radiograph at 6 weeks to check healing.

Methylprednisolone sodium succinate (MPSS) came into favour in the early 1990s because of a study performed in paralysed human patients. It is of no measurable benefit to small animals in clinical practice, and causes more side-effects than improvement. It is a normal reaction of people to remember events which confirm a prejudice, so before recounting all the cases that appeared to improve with use of MPSS the clinician should consider how many of those patients would have benefited from time, cage rest and general support alone. MPSS is no longer recommended as a treatment for spinal cord injury in the clinical setting.

In this case: The dog was euthanized because of the poor prognosis for return of voluntary function in the hindlimbs.

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43 LMN paresis and paralysis – an introduction

The motor functions of the CNS are expressed through the lower motor neuron (LMN) (Fig. 43.1), which consists of:

- Cell bodies in the ventral horn of the spinal cord
- Ventral nerve root
- Spinal nerve
- Neuromuscular junction
- Motor endplate
- Skeletal muscle.

Lower motor neuron cell bodies are also present within certain cranial nerve nuclei which supply skeletal muscles of the head region: muscles of facial expression, masticatory muscles, extraocular muscles, tongue, pharynx, larynx, and oesophagus. The visceral efferent (autonomic) supply to the salivary and lacrimal glands is also classed as a lower motor neuron as it connects the CNS to an effector organ (Fig. 43.2).

Malfunction of the LMN components produce diseases with similar characteristics:

- Paresis, paralysis
- Reduced or absent muscle tone
- Reduced or absent reflexes
- Muscle atrophy.

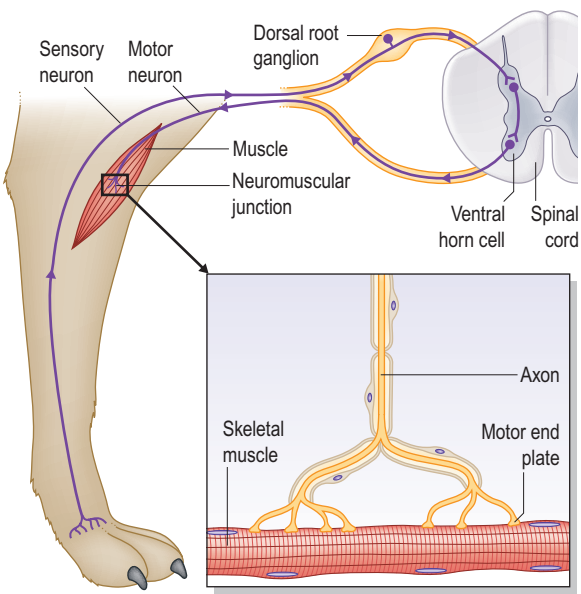


Figure 43.1 Components of the LMN.

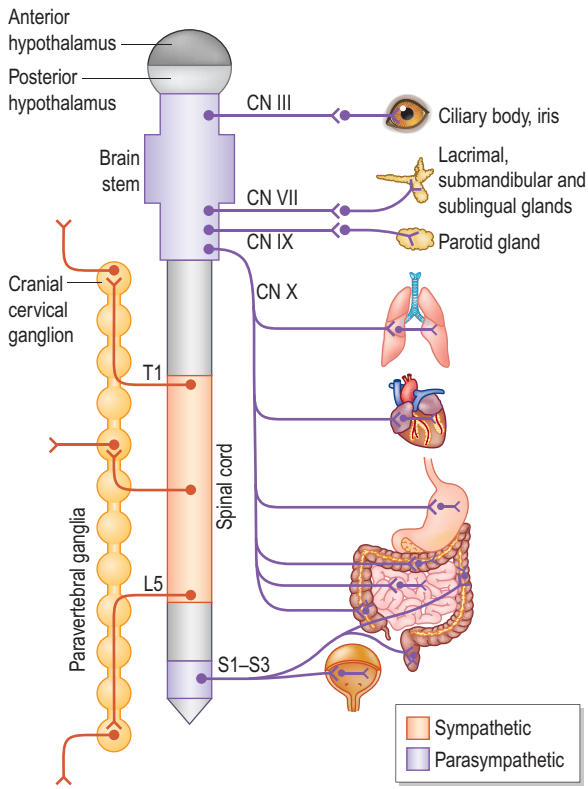


Figure 43.2 The autonomic LMN.

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CLINICAL TIP

Myopathies tend not to cause paralysis.

The presenting signs of a LMN disease depict weakness of the innervated muscle:

- Gait: exercise intolerance, stiff, stilted, shortened stride, paresis or paralysis
- Posture: neck ventroflexion, muscle tremors, flaccid tail, crouched, arched back and plantigrade stance
- Palpation: severe muscle atrophy
- Cranial nerve: inability to prehend or swallow food, regurgitation, facial weakness and a change in the voice.

CLINICAL TIP

Hypertrophy, increased tone and muscle pain may accompany certain myopathies.

Ataxia and proprioceptive deficits do not occur in neuromuscular disease that spares sensory nerves. An animal may be too weak to correct a knuckled-over limb unless the body weight is supported.

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44 LMN paresis and paralysis: acquired myasthenia gravis

INITIAL PRESENTATION

Dysphagia, regurgitation, retching, dysphonia, ptyalism, inability to blink, cervical ventroflexion (cat), tremors, stiffness progressing to generalized weakness with exercise, hindlimb weakness, recumbency and eventually respiratory failure.

INTRODUCTION

In the normal animal, the arrival of an action potential in a nerve terminus causes the voltage-gated calcium channels to open. The calcium influx induces a coordinated release of acetylcholine (Ach) into the synaptic cleft. The Ach binds to the Ach receptor on the skeletal muscle endplate, opening the membrane to sodium ions which depolarizes the post-synaptic membrane. Muscle contraction follows. The enzyme acetylcholinesterase rapidly removes the Ach from the synaptic cleft, allowing depolarization to recur (Fig. 44.1).

Acquired myasthenia gravis is an immune-mediated destruction of the acetylcholine receptors of the skeletal muscle endplate that interferes with neuromuscular transmission causing weakness.

SIGNALMENT

A 1-year, 2-month-old female bull mastiff cross.

CASE PRESENTING SIGNS

Sudden collapse.

CASE HISTORY

A husky, harsh-sounding bark and drooling were noted 5 days prior to referral. The dog began to retch and brought up a clear sticky phlegm the following day. Exercise intolerance quickly developed, limiting the dog to walking only a few yards before going stiff, trembling and sitting down. The dog sat rapidly while attempting to squat when urinating or defecating.

The next day the dog collapsed and was presented for examination.

CLINICAL EXAMINATION

The dog was alert and could stand without assistance (Fig. 44.2). She quickly developed an arched back, all her limbs extended, and she quickly sat and then lay down. Proprioception was normal. Hopping was normal in those brief periods of standing. Spinal reflexes were normal to increased. Muscle tone was normal. No atrophy or muscle pain was noted. Pain perception appeared normal in all limbs. The blink reflex was weak bilaterally with the lids only partially closing. Facial sensation was normal.

NEUROANATOMICAL LOCALIZATION

Generalized LMN disease.

DIFFERENTIAL DIAGNOSIS OF ACUTE LMN SIGNS

Immune-mediated myasthenia gravis

Signs reflect skeletal muscle weakness and may be focal (facial, pharyngeal, laryngeal, oesophageal) or generalized (focal signs + appendicular/axial weakness). The most common initial signs in dogs are generalized weakness with megaoesophagus or megaoesophagus alone.

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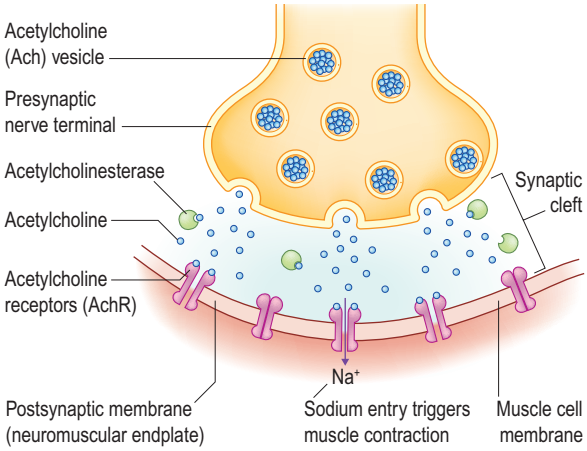


Figure 44.1 The neuromuscular junction.



Figure 44.2 Unable to stand for long, the dog would collapse to the ground.

In one study, 13% of seropositive dogs had generalized weakness without oesophageal or pharyngeal weakness. Cats chiefly present with generalized weakness and a decreased palpebral reflex. Extraocular muscle weakness is not reported in the small animal. Acquired myasthenia gravis is more common in the dog than the cat.

There is a higher prevalence in certain dog and cat breeds. A sudden onset, rapidly worsening generalized form, described as acute and fulminating, occurs in approximately 15% of cats and dogs with myasthenia gravis.

Signs may worsen with cold weather, oestrus cycles, pregnancy, concurrent infection and vaccination.

The age of onset ranges from 7 weeks to 15 years of age. Two peaks occur: at approximately 2–3 years and 9–10 years.

Tick paralysis

Envenomation by the *Ixodes* (Australia) or *Dermacentor* (USA) ticks causes a sudden onset, rapidly ascending flaccid paralysis by preventing release of Ach from the nerve terminus. Cats appear resistant to the salivary neurotoxin of *Dermacentor* spp. Clinical cases of tick paralysis in Australia coincide with the larger tick population in the spring and summer. Dogs and cats are affected most commonly in late winter and early spring prior to developing humoral immunity.

Ascending flaccid paralysis occurs 5–9 days after the attachment of the *Dermacentor* tick, progressing to tetraplegia within 3 days. Spinal reflexes are absent (patellar reflex lost before withdrawal reflex). A bark reduced in volume and changed in character, facial weakness and jaw weakness are found.

Signs commence 5–7 days after the attachment of the *Ixodes* tick, depending on the rate of feeding rather than the absolute number of ticks. High ambient temperatures shorten the course of the disease while in cold weather, signs may not commence until 2 weeks after tick attachment. Ascending flaccid paralysis progresses from hind to forelimbs within a few hours, leaving the animal in lateral recumbency. Spinal stretch reflexes such as the patellar reflex are quickly lost, followed by the withdrawal reflex which weakens as the disease progresses. The gag reflex is absent leading to pseudoptyalism. Voice change and regurgitation are common and early findings. Hypothermia develops. Centrally mediated sympathetic stimulation produces peripheral vasoconstriction and systemic hypertension with secondary pulmonary oedema. Tachycardia or reflex bradycardia and pupil dilation also occur. The pupillary light reflex eventually fails. Respiratory rate falls but tidal volume remains unchanged. Respiration is laboured, with a grunt on expiration from closure of the vocal folds. Cats have a similar presentation with initial signs of agitation, coughing, retching, a changed voice and prominent mydriasis.

Some dogs affected by *Ixodes* ticks have focal weakness of laryngeal, pharyngeal and oesophageal muscles without appendicular weakness. Megaoesophagus with or without dysphagia may be the sole presenting sign.

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Table 44.1 Signs in LMN disease

Acute LMN weakness	Spinal reflexes	Facial	Jaw	Pharynx	Larynx	Oesophagus	Autonomic
MG	Normal	+	–	+	+	+	–
Botulism	Absent	+	+	+	–	+	Pupil, tear production, GIT, urine retention
Ticks (USA)	Absent	+	+	+	+		–
Ticks (Aus)	Absent	+	+	+	+	+	BP, HR, pupils
PRN*	Absent	+	–	–	+	–	–
Rabies	±	+	+	+	+	±	–

*PRN: acute idiopathic polyradiculoneuritis.

Diagnosis is aided by clinical improvement seen after tick removal. Engorged ticks may have fallen off by the time the animal is presented.

Envenomation

Snakes, spiders, lizards, hornets, and worms found in Greenland shark meat also interfere with neuromuscular transmission. Familiarity with the local toxic fauna is recommended.

Rabies

Rabies is transmitted by bites from an infected animal. The time between the bite and demonstration of neurological signs can vary from 1 week to 6 months. Behaviour change (pica, nervousness and fearfulness), ataxia and paresis (limbs, jaw and tongue) are reported, with death occurring within 7–10 days. The chief point to remember about rabies is that it has a highly variable clinical presentation.

Botulism

Ingestion of the preformed type *C. botulinum* toxin in carrion and rotten food produces a sudden onset of ascending flaccid paralysis, the severity of which is linked to the amount of toxin ingested. The toxin prevents release of Ach from the nerve terminal. The onset of signs occurs within 0.5–6 days after ingestion. Hindlimb followed by forelimb paralysis occurs. Spinal reflexes are reduced or absent. Facial weakness, decreased jaw tone, reduced gag reflex and megaoesophagus are prominent. Autonomic manifestations may occur: mydriasis decreased PLR, decreased lacrimation, urine retention and constipation.

Diagnosis is made from the clinical signs and a history of eating carrion or rotten meat such as found in parks or fed in hunt kennels. It is an uncommon cause of paralysis. Recovery occurs within 1–3 weeks. Death from respiratory paralysis or aspiration pneumonia is possible.

Acute idiopathic polyradiculoneuritis

Inflammation of the ventral nerve roots occurs in dogs, with or without a raccoon bite or scratch. Ascending flaccid paralysis develops spontaneously or 7–12 days after raccoon exposure. Vaccination has been associated with acute polyradiculoneuritis in a young dog. Progression within 3–10 days to non-ambulatory tetraparesis or tetraplegia is accompanied by loss of voice and facial weakness. Affected dogs are able to eat if given access to food. Respiratory paralysis is more likely with rapidly worsening tetraparesis. The perineal reflex is present while other spinal reflexes are absent. Neurogenic atrophy is a feature, dysphagia is not. Hyperesthesia to innocuous touch has been reported.

Diagnosis is aided by finding an elevated level of total protein in the lumbar spinal fluid, reflecting the position of the nerve roots within the subarachnoid space (Table 44.1). Widespread denervation potentials are recorded on electromyography (EMG).

Polymyositis

Myopathies are characterized by exercise intolerance, a stiff, stilted gait, lameness alternating between limbs, lameness worsening with exercise (*cf.* orthopaedic disease), muscle swelling or atrophy and pain. Weakness may occur but proprioception is normal and spinal reflexes are generally intact. Dysphagia, regurgitation, masticatory muscle atrophy and a weak bark also occur. AST, ALT and CK are not uniformly elevated in myopathies. If raised, their decline may be used to monitor any response to treatment.

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Autoimmune polymyositis is an autoimmune disease of unknown aetiology mostly affecting adult large breed dogs of either sex. Infectious myopathies caused by protozoa or tick-borne pathogens are rare but efforts must be made to rule them out with serology. Equally, drug-induced myopathies should be considered when taking a history. Paraneoplastic myopathies are rare. A generalized immune mediated disease may present as polymyositis.

In this case preservation of spinal reflexes, muscle tone and proprioception was suggestive of myasthenia gravis. Generalized weakness from cardiac insufficiency or hypoadrenocorticism could produce exercise intolerance and collapse.

CASE WORK-UP

Diagnostics investigate the cause and the sequelae of the weakness.

Tensilon test

Edrophonium chloride (Tensilon) is a rapid-onset, short-acting anticholinesterase. It prolongs the presence of Ach in the synaptic cleft, allowing further stimulation of the remaining post-synaptic Ach receptors thus improving neuromuscular transmission and muscular strength.

1. Place IV catheter
2. Assemble ventilatory support: ET tube, Ambu-bag or anaesthetic circuit
3. Have ready: atropine 0.02–0.04 mg/kg IV, to treat side-effects of salivation, diarrhoea, retching, vomiting, bronchial secretions and bradycardia. Pre-treatment of cats has been recommended
4. Draw up the dose of Tensilon:
   - Dog 0.1–0.2 mg/kg
   - Cat 0.25–0.5 mg total dose
5. Give 1/5th dose IV. If tolerated, inject the remaining drug after 30 seconds
6. The effect begins 20–30 seconds after injection. Improvement of function lasts up to 10 minutes.

CLINICAL TIP

Improvement in muscle strength can occur in myasthenics after rest. Tensilon-induced improvement lasts longer.

A positive test is not specific for myasthenia gravis. Improvement may occur in some myopathies and neuropathies.

A negative test does not rule out myasthenia gravis. Half the dogs with acute fulminating MG do not respond to Tensilon.

Acetylcholine receptor (AChR) antibody titre

The titre is measured in serum before the use of corticosteroids. The serum concentration of AChR antibodies does not predict the severity of the disease.

Positive result:

- Dog >0.6 nmol/l
- Cat >0.3 nmol/l.

About 2% of dogs with generalized myasthenia gravis are seronegative. Seroconversion may occur. Using an ELISA test may give a high percentage of false negatives.

Radiography

Some 90% of affected dogs have megaoesophagus: 15–25.7% of affected cats had a cranial mediastinal mass; 3% of affected dogs have thymomas.

Aspiration pneumonia is a major cause of death or euthanasia and thus acts as a prognostic sign.

Electrodiagnostics

Repetitive nerve stimulation may elicit a decremental response. Electromyography and evoked potentials are useful for investigating the small percentage of seronegative animals. Referral is required for this investigation.

Third degree heart block has been reported in some dogs with MG, polymyositis and thymoma.

In this case:

1. Muscle enzymes were normal. Toxoplasma and Neospora serology were negative. ACTH stimulation was normal
2. Tensilon test was positive
3. Thoracic radiographs showed megaoesophagus but no pneumonia was noted
4. Serum to measure AchR antibody level was taken, and later found to be positive at 1.8 nmol/l (N < 0.6), confirming the diagnosis of immune-mediated myasthenia gravis.

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DIAGNOSIS

Immune-mediated myasthenia gravis.

PROGNOSIS

- Some 48% of focal/generalized MG dogs are euthanized or died shortly after admission due to aspiration pneumonia.
- Spontaneous clinical and immunological remission occurs within an average of 6.4 months (range: 1–18 months) in 88.7% of dogs
- The antibody titre does not correlate with the severity of the disease
- Recurrence may happen months or years later
- There is an excellent correlation between the resolution of the clinical signs including megaoesophagus, and the fall of the antibody titre to below 0.6
- Prognosis is improved with: speedy recognition of regurgitation (*cf*. vomiting), accurate diagnosis, correct treatment, dedicated owners and good clinical judgement.

TREATMENT

No controlled studies have been done to determine the optimum treatment for acquired MG. The best outcome requires 'good clinical judgement' and dedicated owners.

Search for concurrent disease

- ACTH stimulation, thyroid status (endogenous canine TSH, and serum free T4 levels by dialysis), muscle enzymes (CK, AST, ALT), electrolytes, serum biochemistry, urine analysis and haematology
- Thoracic radiographs
- Tracheal wash for culture
- Remove thymoma.

Anticholinesterases

- Pyridostigmine bromide (Mestinon): 1–3 mg/kg PO, q8–12 hr
- Neostigmine bromide (Prostigmin): 2 mg/kg/day PO in divided doses or 0.01–0.04 mg/kg IM q6–8 hr. The syrup form should be diluted 50 : 50 in water to avoid gastric irritation
- Side-effects: SLUD (salivation, lacrimation, urination, diarrhoea). Treat with low doses of atropine.

Nutrition/hydration

- Elevate food and water during feeding. Elevate the forequarters of the animal for 10–15 minutes afterwards. Feed at the time of peak effect of the anticholinesterase drug.
- Install a gastrostomy feeding tube to guarantee food and drug intake.
- Consider IV fluids to maintain hydration.
- Reducing the acidity of stomach contents may reduce lung inflammation from aspiration.

Corticosteroids

- Prednisolone 0.5 mg/kg q48 hr. This can be gradually increased over 2 weeks to immunosuppressive doses: 2–4 mg/kg/day
- Weakness may worsen when corticosteroids are commenced (Table 44.2).

Immunosuppression

- Azathioprine, mycophenolate mofetil.

Monitoring

1. Serum antibody level decreases after 7–10 days of immunosuppressive doses of corticosteroids.
2. Treatment continues while the antibody titre is positive. Monitor every 4–8 weeks.
3. If clinical signs have resolved while the animal is on corticosteroids and anticholinesterase, gradually withdraw the corticosteroids first.
4. Weakness may indicate under-dose or over-dose with anticholinesterase. If SLUD side-effects are present, the animal may be over-dosed. Improvement with the Tensilon test indicates under-dosing. If no improvement is seen after Tensilon injection, and an increase in anticholinesterase medication has not made a definite improvement, the animal is probably over-dosed and a decrease in medication is advised.
5. Pupil constriction and bradycardia are seen in humans during a cholinergic crisis. A medium-to-dilated pupil with tachycardia is present during a myasthenic crisis. Respiratory failure is present in both crisis states.

Treatment of this case

1. Pyridostigmine q8 hr which was increased to every 6 hr.
2. Immune suppression with prednisolone BID.
3. Elevated feeding. The water bowl was raised onto a platform.
4. The dog improved on this regime and was playful, and managed short lead walks. If allowed off lead

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Table 44.2 Drugs to avoid in myasthenia gravis

Antibiotics
Aminoglycosides
Polymyxin
Tetracyclines
Lincosamides
High-dose penicillin
Sulphonamides

Anaesthetics
Methoxyflurane

Anticonvulsants
Phenytoin
Barbiturates
Benzodiazepines

Psychotropic
Neuroleptics
  Phenothiazines
  Butyrophenones
Amitriptyline

Endocrine
ACTH
Corticosteroids
Oxytocin
Thyroid hormones
Methimazole

Cardiovascular
Anti-arrhythmics
  Quinidine
  Lidocaine
  Procainamide
Beta-blockers
Calcium channel blockers

Analgesics, anti-inflammatory agents
Morphine derivatives
D-penicillamine
Antimalarials

Neuromuscular blocking agents
Succinylcholine
D-tubocurarine

Other
Magnesium salts
  Laxatives
  Antacids
Iodinated contrast agents
Organophosphates
Carbamates

the dog weakened after a few minutes jogging and had to be rested during the lead-walk back home. No coughing and no regurgitation of food or phlegm occurred. Throat-clearing 'gurgling' noises were absent. The blink reflex was normal.

5. Mycophenolic acid was commenced, with the aim of reducing the dose of prednisolone to prevent muscle atrophy. Vomiting and melaena quickly developed and the drug was withdrawn.
6. Prednisolone was withdrawn gradually after 3 months of treatment. There had been generalized muscle loss and a pot-belly had developed. Pyridostigmine was then withdrawn and the dog remained clinically normal. A repeat AchR antibody titre was negative. Muscle bulk returned.

REGURGITATION VS. VOMITING

Regurgitation

Regurgitation is the passive expulsion of material from the mouth, pharynx, or oesophagus. Abdominal contractions are absent. The animal extends the neck and retrograde passage of white liquid or froth (swallowed saliva) or undigested food takes place seconds to hours after feeding. Ptyalism due to nausea is absent. However, pseudoptyalism due to dysphagia may be present.

Oesophageal obstruction or weakness of the pharyngeal or oesophageal muscles present as regurgitation. Weight loss, increased appetite, coughing, respiratory distress and aspiration pneumonia may also be present with or without signs of generalized muscular weakness. Pharyngeal weakness has the additional sign of dysphagia.

The oesophagus is composed of skeletal muscle innervated by the vagus nerve which supplies both the sensory and motor fibres. The distal third of the feline oesophagus is composed of smooth muscle, innervated by parasympathetic branches of the vagus nerve (CN X).

Weakness of oesophageal skeletal muscle causes flaccidity and dilation of the oesophagus known as megaoesophagus. Congenital and acquired megaoesophagus are far more prevalent in the dog than the cat.

Causes of decreased oesophageal peristalsis

1. Focal or generalized myasthenia gravis, botulism, tick paralysis (Australia)
2. Hypoadrenocorticism

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3. Dysautonomia
4. Idiopathic
5. Polymyositis
6. Peripheral neuropathy, e.g. lead toxicity (cat and dog)
7. Brainstem lesion affecting the vagal nerves or nuclei bilaterally
8. Increased muscle tone from myotonia or tetanus
9. Sensory lesion from ganglioradiculoneuritis.

Hypothyroidism was not found to be a risk factor for acquired megaoesophagus.

Diagnosis of regurgitation

Description

- White froth
- Undigested food
- Absence of bile.

Chronic vomiting can result in oesophagitis and thus regurgitation.

Examination

Gurgling of liquid retained within the dilated oesophagus may be heard. Closing the mouth, and squeezing the abdomen, or the cranial thorax, increases intrathoracic pressure which transfers to the thoracic oesophagus. Air within the thoracic oesophagus is forced into the cervical oesophagus and produces a visible enlargement on the left side of the neck.

Imaging

Non-contrast radiographs of the neck and the thorax are required. A dilated oesophagus filled with air, liquid or ingesta may be seen. The cat with neuromuscular weakness demonstrates a dilated proximal oesophagus only due to the distribution of smooth muscle in this species. Aspiration pneumonia may be present. Barium contrast studies, liquid or meal, are used for the diagnosis of intra- or extraluminal obstruction but pose a hazard to the dysphagic animal which may inhale them. Endoscopy is useful for diagnosis and to investigate partial strictures and oesophagitis.

Lab work

Investigation of neuromuscular weakness, e.g. AChR antibody titre, ACTH stimulation, T4 and TSH levels, muscle enzymes, electrolytes, haematology, biochemistry, urine analysis, electromyography and muscle biopsy.

Management of regurgitation

Elevated feeding. Divide the calorie intake into smaller portions given frequently. The forequarters of the animal are raised vertically while it is eating, if possible, by placing the food and the animal's forelimbs on a chair or bench. If this is not tolerated raise the forequarters after eating by lifting the forelimbs off the ground. In the absence of peristalsis the aim is for gravity to drive the food into the stomach. Elevate the forequarters after eating for as long as the animal/owner will tolerate it – if possible for 10–15 minutes. The ability to swallow liquid or solid food varies from dog to dog and is assessed by trial and error.

Prokinetic drugs, cisapride and metoclopramide improve oesophageal motility in the cat but not the dog. Anticholinesterase inhibitors are indicated in myasthenia gravis. Treating the underlying disease gives the best hope for resolution. A gastrostomy feeding tube may be required if weight loss cannot be reversed by oral feeding or if aspiration pneumonia and anorexia occur.

Vomiting

Presenting signs

Licking lips, salivation, depression, yawning, followed by forceful, non-productive, vigorous abdominal contractions (retching) which end in expulsion of stomach and upper GI contents via the mouth. Undigested food: gastric (or oesophageal) contents. Digested food: intestinal contents.

Vomiting (emesis) is a complex reflex coordinated by the medulla oblongata. The vomiting centre in the medulla consists of scattered groups of neurons which integrate the somatic, visceral, gastrointestinal and respiratory components of vomiting. It receives stimuli from:

1. Neural input: viscera (CN X, sympathetic nerves), pharynx (CN IX), vestibular apparatus (CN VIII), cerebral (fear/stress/pain)
2. Direct stimulation: meningoencephalitis, increased ICP
3. Humoral input: drugs, toxins or metabolites by stimulating the chemoreceptor trigger zone (CTZ) which projects to the vomiting centre.

Note: the vestibular apparatus also projects to the vomiting centre via the CTZ in the dog but not in the cat.

Cyclic vomiting responsive to phenobarbitone treatment is thought to be a form of seizure activity.

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Vomiting is a very common presenting sign for acute vestibular disease, whether peripheral or central in nature. Vomiting occurs a few times, the first day of vestibular dysfunction, and then generally stops. Persistent vomiting with persistent vestibular signs (spontaneous nystagmus, ataxia, falling, leaning or rolling) indicates continuing vestibular irritation and warrants investigation of the balance disorder. A head tilt may continue despite resolution of ataxia and nystagmus so cannot be used as a measure of clinical improvement.

Vomiting may be confused with expectoration (forceful coughing) and regurgitation.

Treatment

The cause should be diagnosed. Antiemetics are used to prevent morbidity from dehydration and electrolyte loss. The CTZ and vomiting centre have a number of receptors and by manipulating them, vomiting may be suppressed with medication.

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45 LMN paresis and paralysis: brachial plexus avulsion

INTRODUCTION

Failure of nerve transmission due to a functional disturbance from compression, ischaemia, or blunt trauma is called *neurapraxia*. The larger motor and proprioceptive axons are affected preferentially with preservation of pain perception. Structural integrity of the nerve's Schwann cells and endoneurial connective tissue persist, giving a good prognosis for recovery. *Neurotmesis* is the complete or partial transection of the nerve's axon, myelin sheath and endoneurial connective tissue. All axon types are affected and pain perception is lost. The destruction of the connective tissue and Schwann cell scaffolding prevents effective nerve regeneration. Electrodiagnostics and repeat clinical examination monitor clinical improvement.

SIGNALMENT

A 2-year, 9-month-old female Staffordshire bull terrier.

CASE PRESENTING SIGNS

Left fore weakness.

CASE HISTORY

Non-weight-bearing left-fore paresis began suddenly after being hit by a car 8 months prior to referral. The dog occasionally licked the left forepaw. No improvement had been noticed.

CLINICAL EXAMINATION

The dog was alert and ambulatory with a non-weight-bearing left forelimb. Severe generalized atrophy of the left fore was present (Fig. 45.1). The left elbow was held flexed. Muscle contracture prevented extension of the right carpus, elbow and shoulder. The left cutaneous trunci reflex was absent when either side was stimulated. The left pupil failed to dilate fully in darkness. Pain perception was absent in the radial and ulnar nerve distributions but was present in the distribution of the musculocutaneous nerve.

NEUROANATOMICAL LOCALIZATION

Left brachial plexus, including the ventral nerve roots supplying the lateral thoracic nerve (C8–T1) to account for the cutaneous trunci deficit, and those supplying oculosympathetic innervation to the eye (T1–3) to explain the left miosis in darkness.

DIFFERENTIAL DIAGNOSIS

Brachial plexus avulsion was diagnosed based on the history and clinical findings.

A rarely-occurring acute-onset flaccid paralysis of both forelimbs from axon and myelin loss in the brachial plexus has been reported in dogs and a cat. The cause is unknown but may have an immune-mediated basis. Acute, progressively worsening bilateral forelimb paresis is more commonly caused by a cervical spinal cord lesion. Neither was realistically suspected in this case but they are worth mentioning as other causes of forelimb paresis.

Nerve sheath tumours most commonly occur in the brachial plexus causing chronic lameness prior to the onset of flaccid paralysis of the affected limb. Occurrence in the cervical cord segments C1–C5 or the thoracolumbar cord segments T3–L3 results in cord compression which may be acute in onset.

CASE WORK-UP

Electromyography (EMG) is the recording of abnormal spontaneous activity in muscle cells. It evaluates the electrical stability of the muscle membrane which is

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Figure 45.1 Severe atrophy of the left forelimb.

altered by denervation and by myopathies. EMG is an extension of the physical examination and is useful for demonstrating abnormal neuromuscular function when the history and examination findings are equivocal. Areas of abnormality can be delineated prior to muscle biopsy in an effort to obtain a representative diagnostic sample.

EMG is performed under general anaesthesia to obtain patient compliance. Insertional potentials are generated by the movement of the recording needle through muscle membranes. It ceases once the needle is stationary. Normal muscle is electrically silent under GA. Spontaneous potentials are categorized based on amplitude and frequency. Fibrillation and positive sharp waves occur in both nerve and muscle diseases. Myotonic potentials wax and wane, giving the auditory impression of a revving motor, or the decrescendo of a dive bomber plane. EMG is assessed by the sound generated and the pattern of the abnormal spontaneous waveforms. 'Frying eggs' or 'rain on roof' are evocative descriptions of fibrillation. The greater the amount of abnormal potentials detected, the greater the pathological involvement of the muscle. The degree of EMG change, while indicating the anatomic distribution of the disease process, does not correlate with the severity of the clinical deficit. It is particularly useful when aiming to distinguish between disuse atrophy and neurogenic atrophy of muscle. Denervation potentials, a general term for spontaneous activity recorded following denervation of muscle, can be detected 7–10 days after denervation has occurred.

Electrodiagnostic equipment is expensive and does not represent a good investment for the general practitioner.

In this case, the diagnosis could be made without the need for EMG. The lack of functional improvement since the injury indicates a grave prognosis for recovery of function. The musculocutaneous nerve function kept the limb flexed and prevented abrasion of the paw. Self-mutilation had not occurred.

No treatment was given.

Brachial plexus avulsion is caused by major trauma, such as a RTA. Nerve roots lack a supportive perineurium so are the point of weakness when force is applied to the forelimb. Ventral roots are more susceptible to trauma than dorsal roots, which explains the persistence of pain perception in a flaccid paralysed limb. The flaccidity and non-weight-bearing state may not be recognized as neurological damage if a fracture is present. Humeral fractures rarely result in radial paralysis. Always check for ipsilateral Horner's syndrome which follows avulsion of the T1–3 nerve roots. Avulsion of C8, T1 nerve roots removes the ipsilateral motor arc of the cutaneous trunci reflex. These two deficits will aid diagnosis.

Absence of pain perception occurs if the dorsal roots are avulsed. Neurogenic atrophy, from ventral root trauma, occurs within 7 days. This is profound and far exceeds that expected from disuse of a fractured limb. Denervation can be detected by EMG approximately 1 week after avulsion although EMG is generally not required to make a diagnosis. Imaging of the plexus has little value. Contrast-enhancement of contused nerve roots may be seen. Prognosis is determined by the neurological deficits present. Recovery after 6 weeks was reported in a dog with brachial plexus contusion.

In the absence of fractures, the non-weight-bearing lameness is not painful. Self-mutilation of the denervated limb is rare.

Avulsion carries a grave prognosis for return of function. Stretching or bruising of the plexus carries hope of recovery. Surgical reattachment of nerves is not performed. Treatment is aimed at preventing limb contraction during a hoped-for recovery phase.

Amputation of the limb is rarely required. Partial avulsion may allow tendon transposition. Seek specialist advice from surgeons familiar with the technique.

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46 LMN paresis and paralysis: flaccid tail

INITIAL PRESENTATION

Flaccid tail, low tail carriage, tail paralysis.

INTRODUCTION

Sacrococcygeal muscles dorsally and ventrally, and the lateral intertransversarius coccygeus muscles extend and flex the tail. Innervation is supplied by sacral and caudal nerves.

Tail paralysis is common in cats secondary to stretching or avulsion of the nerves during trauma.

A focal tail myopathy occurs in working dogs and is known as limber tail, limp tail, frozen tail or cold tail, with the latter sobriquets probably owed to its increased occurrence in cold weather. The acute flaccid paralysis of the tail from its base or a few inches distal to the base occurs post-exercise in English pointers, Labradors, beagles, setters, foxhounds and other hunting breeds. It may or may not be painful. Recovery occurs within days to weeks without specific treatment. It may recur. The tail may hang to one side during recovery. Biopsies are not performed because of the potential to damage nerves while harvesting enough muscle to be diagnostically useful, besides which, the condition is quite distinctive and self-limiting.

Interestingly, a dog with LMN or UMN tetraplegia may still wag its tail voluntarily. This is generally taken as a good prognostic sign for recovery of ambulation but has not been subject to rigorous study.

SIGNALMENT

A 12-year-old neutered male domestic shorthaired cat.

CASE PRESENTING SIGNS

Flaccid tail.

CASE HISTORY

The cat suddenly appeared to be in pain and resented handling by the owner. The cat spent unsupervised periods outdoors, but there had been no evidence of external trauma to the cat. Two days later the tail was noticed to hang vertically and lose all movement. The hindlimb posture appeared crouched. The cat was able to voluntarily urinate and defecate. A week of meloxicam, buprenorphine, cage rest and time did not restore the cat to normal.

There had been two similar episodes of pain and a 'floppy' tail 1 year and 2 years prior to this recurrence, from which the cat had spontaneously recovered. The cat was described as being otherwise well.

CLINICAL EXAMINATION

The cat was alert, ambulatory with a normal gait and normal hindlimb posture. The tail hung vertically. Hopping and placing were normal in all limbs. Muscle tone and spinal reflexes were normal in all limbs. Anal tone was present. Gently touching the caudal lumbar spine made the cat cry out in a pain response (Fig. 46.1).

NEUROANATOMICAL LOCALIZATION

Cauda equina.

DIFFERENTIAL DIAGNOSIS

1. External trauma: tail-pull injury, sacral fracture
   Sacral fractures are uncommon in the dog and cat, usually result from a RTA and are generally accom-

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Figure 46.1 Flaccid tail. Note the normal extension of the tarsi. The thorax was shaved for placed of a Fentanyl patch for pain control.

Table 46.1 Clinical signs and prognosis for feline tail-pull injury

Tail	Anal tone	Bladder	Prognosis
Painful tail base	Normal	Normal	Good, without surgery
Paralysed, anaesthetic	Normal	Normal	Good, without surgery
Paralysed	Normal	Urine retention	Fair. Most expected to recover
Paralysed	Decreased	Difficult to express	75% recovery rate
Paralysed	Absent	Easy to express	50% recovery rate

panied by pelvic injury such as a fractured ilium (dog) and sacroiliac subluxation (cat).

Neurological signs are most common secondary to fractures which traverse the spinal canal. Sciatic nerve deficits, tail paralysis, urine retention or dyssynergia may result. Sciatic deficits may not be noticed immediately due to the animal's immobility from orthopaedic injury. Always check the degree of tarsal flexion when testing the withdrawal reflex as this is a measure of sciatic function.

Sacrocaudal fractures in cats result from RTAs and bite injuries. The common sites are S3–Cd1, and S2–3 but neurological signs may not correlate with the site of injury. This discrepancy is thought to be due to traction of the nerves, hence the name – tail-pull injury (Table 46.1).

Most cats recover hindlimb function, urination and tail function within 1 month. A recent study found that cats with intact tailbase sensation had control of urination within 3 days. About half the cats without tail base sensation post-trauma did recover control of urination by day 30. Full tail function can take months to return, if at all. It is not clear if surgical treatment of the fracture will hasten recovery. Medical treatment includes pain relief, and treating constipation that can result from pelvic nerve injury. Bladder care is vital. If the tail is continuously soiled, then amputation may need to be considered.

2. IVD disease
3. Neoplasia: vertebral or in the cauda equina
4. Infection: bite wound
5. Feline hyperaesthesia syndrome
   This constellation of signs occurs in any cat of any age with an onset usually between 5 and 8 years of age. Intermittent signs include violent licking and biting at the hindquarters and tail to the extent of drawing blood. Other signs include rippling skin dorsally, muscle spasms or twitching in thoracolumbar spinal musculature, agitation, restlessness, running, excessive vocalization, papillary dilation and attacking objects or people. Palpation of the thoracolumbar epaxial muscles may cause pain. Causes of pain or skin irritation should be ruled out before diagnosing this condition.

CASE WORK-UP

A narrowed L7–S1 disc space was seen on non-contrast radiographs.

MRI: Ventral compression of the cauda equina at the level of the L7–S1 IV disc space with what appeared to be calcified material (see Fig. 46.2).

DIAGNOSIS

Intervertebral disc extrusion.

TREATMENT

L7–S1 dorsal laminectomy enabled the removal of friable IV disc material ventral to the cauda equina. Pain relief was achieved with methadone.

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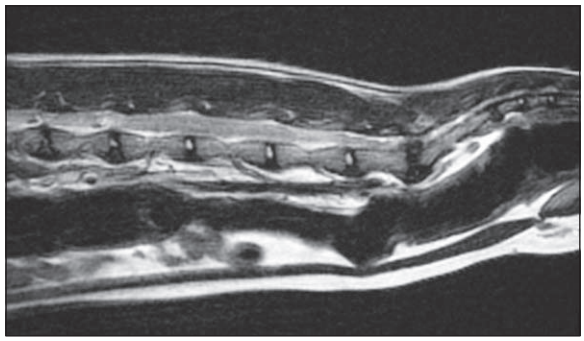


Figure 46.2 Sagittal spine T1WI. Ventral compression of the cauda equina at the L7–S1 IVD space.

FOLLOW-UP

One week after the surgery the cat was no longer in pain. The tail was still quite flaccid but could be held up in a normal position briefly by the cat.

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47 Ataxia: spinal ataxia

INITIAL PRESENTATION

In spinal cord disease, the ascending proprioceptive tracts may be affected before the descending motor tracts resulting in ataxia as the presenting sign. Damage to the ascending spinocerebellar tracts in spinal cord lesions results in cerebellar-like dysmetria. Spinal cord lesions will cause ataxia of the limbs caudal to the lesion.

INTRODUCTION

Coordination relies on balance, position sense and the ordering of received sensory information into smooth movement. Failure of coordination, from any of the components, results in *ataxia* a term used to describe irregular, unpredictable voluntary movement.

Causes of ataxia

Position sense is referred to as *proprioception* and is achieved through muscle, tendon and joint receptors, associated sensory nerves, and ascending tracts within the spinal cord (Fig. 47.1).

These tracts project to the ipsilateral cerebellum (unconscious proprioception) and to the contralateral cerebral cortex (conscious proprioception). This constitutes 'general proprioception'.

When this system fails, the gait becomes ataxic and limb position alters. This is most noticeable with spinal cord and brainstem lesions with deficits ipsilateral to the lesion. Cerebral lesions are subtle and tend not to affect the gait but can reduce the contralateral placing response. Sensory neuropathies are rare. Ataxia and anaesthesia of the limbs with self-mutilation are the two chief clinical presentations of sensory deficits. Lesions of muscle, tendons, and joints present as lameness not ataxia.

Presenting signs of abnormal proprioception

- Supporting weight on the dorsum of the paw ('knuckling over', 'knuckling')
- Scraping, scuffing, catching the dorsal surface of the digits/claws on the ground
- Excessive adduction of a limb, causing the limbs to cross each other
- Excessive abduction of a limb, or both limbs causing a wide-based stance, or more extreme, a splaying of the limbs ('Bambi on ice')
- Circumduction (swinging out to the side; abduction in an arc-shaped trajectory) of the limbs, particularly when turning
- Hypermetria of forelimbs ('goose-stepping', 'saluting gait')
- Stumbles, tripping over steps, own adducted limb, on uneven ground or surfaces with little traction, e.g. wood flooring
- Delayed initiation of a step (delayed protraction) resulting in a longer stride than normal. This gives the hindquarters a crouched appearance. This may result in collapse when turning.

Paresis may be responsible for the inability to support the weight and walk normally, resulting in stumbling, tripping or collapse. Supporting the animal's body weight while testing placing or the knuckling

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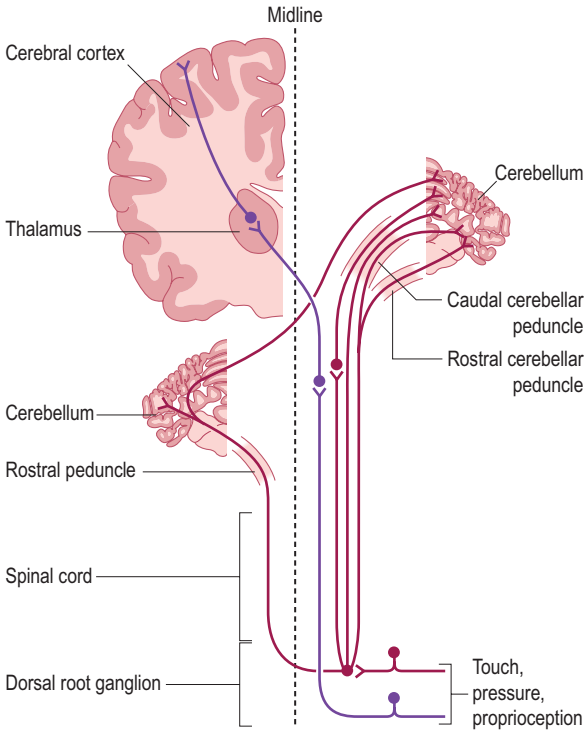


Figure 47.1 Pathways of conscious and unconscious proprioception.

responses should distinguish a proprioceptive deficit from paresis.

Owners commonly describe their pet as 'drunk, wobbly, clumsy or unsteady'.

CLINICAL TIP

Ataxia may be overlooked and go unmentioned by owners who ascribe it to their new pet's young age.

SIGNALMENT 1

A 3-year-old male springer spaniel.

CASE HISTORY

The dog suddenly started yelping when touched and would not jump into the car 3 weeks prior to referral. He was normally an active dog and spent the working day around horse stables. His condition did not improve with self-imposed rest and he remained lethargic and inappetant. When walking, the dog kept his head lowered and his back arched.

Hindlimb ataxia began 6 days before presentation and slowly worsened. The dog was unable to walk more than a few steps before the hindlimbs crossed and tripped each other (Fig. 47.2).



Figure 47.2 The head is lowered to shift weight forward. Hindlimb extension when sitting is common in UMN paraparesis or paraplegia.

CLINICAL EXAMINATION

The dog was quiet but mentally responsive. He was unable to stand without assistance. The back was arched, and pain was elicited by palpation of the lumbar spine. Hindlimb paresis and ataxia were present when the body weight was supported with a sling. The right hind knuckled over more than the left. Spinal reflexes were intact. The forelimbs were normal. No neck pain was found.

NEUROANATOMICAL LOCALIZATION

T3–L3 cord, slightly worse on the right.

DIFFERENTIAL DIAGNOSIS

IVDD, external trauma, meningitis or discospondylitis could all give rise to a sudden onset of pain. A migrating grass seed within spinal muscles with cellulitis would cause back pain but extension to the spinal canal would be unusual. No vertebral fractures could be seen on the radiographs taken by the local veterinarian.

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CASE WORK-UP

MRI showed ventral extradural compression of the spinal cord at L3–4. A small syrinx was present extending rostrally (Fig. 47.3). This was thought to be secondary to the trauma of cord compression.

DIAGNOSIS

L3–4 disc extrusion.

TREATMENT

A right-sided L3–4 hemilaminectomy to remove extruded disc material was carried out. The strength and proprioception improved on a daily basis. The dog was walking unaided without ataxia 6 days postoperatively.

SIGNALMENT 2

A 5.5-month-old female German shepherd dog.

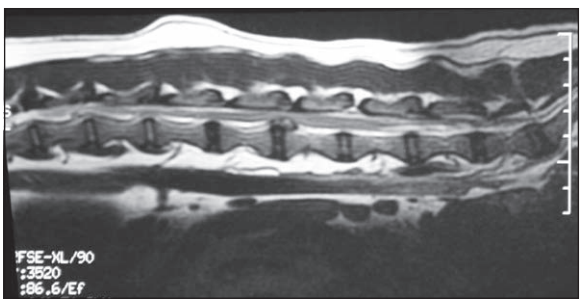


Figure 47.3 Sagittal spine MRI T2WI. Extradural compression of the cord at L3–4 IVD space. The syrinx is not obvious in this image.

CASE PRESENTING SIGNS

An abnormal gait of unknown duration.

CASE HISTORY

The dog was acquired from a 'lost dogs home' 1 week prior to referral and no past history was available.

The dog liked to exercise but tripped and fell on uneven ground and when turning. She was unable to jump into the car and was slow to descend stairs. The dorsal surface of the fore- and hindlimb paws were scraped against the ground, wearing down the claws.

CLINICAL EXAMINATION

An alert, fearful and aggressive dog with a normal posture, hindlimb ataxia and slight forelimb hypermetria was examined.

Proprioception was slow, and hopping was slightly reduced in all limbs. The patellar reflexes were 3+ bilaterally. Withdrawal reflex was present in all limbs. Panniculus reflex was normal. The dog moved its neck spontaneously in all directions and did not seem to be painful. The mental state and cranial nerves were normal. No neck pain could be detected.

NEUROANATOMICAL LOCALIZATION

Cervical spinal cord segments C1–5. Cerebellar disease produces ataxia and hypermetria but does not result in tetraparesis (Table 47.1). Conscious proprioceptive deficits cause the paws to be dragged dorsally, wearing down the dorsal surface of the claws.

Table 47.1 Differentiating forelimb hypermetria

Lesion	Cervical cord disease	Cerebellar disease
Pathogenesis	Spasticity Altered proprioception	Fine motor coordination Altered proprioception
Distinguishing feature	Extended forelimbs	Excessive flexion of forelimb joints
Description	Hypermetria, 'Over-reaching', 'floating'	Hypermetria, 'goose-stepping', 'prancing', 'high-stepping'
Strength	Weakness	No weakness
Other characteristics		Other cerebellar signs: menace deficit, truncal ataxia, intention tremor, exaggerated limb movement on hopping (± vestibular signs)

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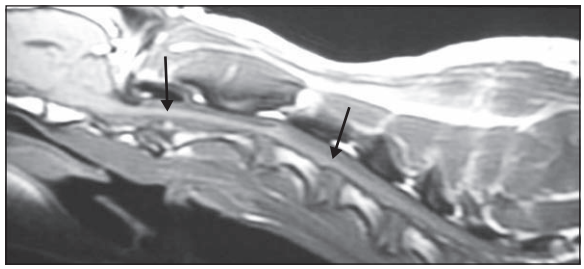


Figure 47.4 Sagittal T1WI. Widening of the dorsal subarachnoid space. Possibly a subarachnoid cyst. (Arrow points to the spinal cord).

DIFFERENTIAL DIAGNOSIS

- Malformation (vertebrae; cord; meninges)
- Inflammation
- Degeneration
- Tumour
- Vascular problem

The time course of the clinical signs was not known.

Diseases chiefly characterized by increased muscle tone and spasticity present with a stiff, stilted gait composed of short strides and limited flexion of the limb.

'Dancing' Dobermann dogs have alternate flexing and extending of the hindlimbs not the forelimbs and this is not referred to as hypermetria.

CASE WORK-UP

A treatment trial of prednisolone for 1 week made no difference to the dog's gait. Clinical signs remained unchanged.

MRI showed the cord to be narrow from the level of C2 vertebra to C4 vertebra. Syrinx formation caudal to C4 vertebra (not visible on these images). There was no contrast enhancement (Figs 47.4 and 47.5).

DIAGNOSIS

Malformation of the spinal cord.

TREATMENT

None. The dog's condition remained stable.

SIGNALMENT 3

A 7-year-old male German shepherd dog.

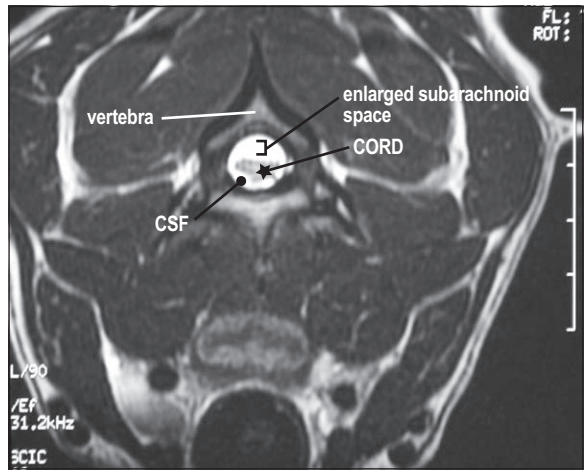


Figure 47.5 Transverse T2WI. Dorsoventrally flattened spinal cord (star), surrounded by CSF (dot). Wide subarachnoid space dorsally (bracket).

CASE PRESENTING SIGNS

Hindlimb ataxia and paresis.

CASE HISTORY

Hindlimb ataxia and paresis commenced suddenly 8 days prior to referral and worsened over the following 2 days. The gait improved slightly but not to normal with the use of prednisolone. The dog seemed to be painful.

CLINICAL EXAMINATION

Hindlimb ataxia and paresis were present. The lumbar spine was arched. The patellar reflexes were increased bilaterally and the withdrawal reflexes were normal in all limbs. The abdomen was tense when palpated and the dog yelped when pressure was applied over the thoracolumbar junction.

NEUROANATOMICAL LOCALIZATION

The lesion was localized to the T3–L3 spinal cord segments.

DIFFERENTIAL DIAGNOSIS

Spinal cord compression by IVDD, neoplasia, or enlargement of the articular facets was considered most likely given the age of the dog and sudden onset of signs.

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A common cause of hindlimb ataxia in the GSD is degenerative myelopathy but this has a gradual onset and does not cause the dog pain.

Degenerative myelopathy (DM)

Degeneration of all tracts of the spinal cord throughout its length commonly occurs in German shepherd dogs of both sexes over 5 years of age. The thoracic cord segments are most severely affected and the presenting signs of ataxia and UMN paresis reflect this. In accordance with the UMN lesion, the patellar reflexes are usually normal to increased. About 15% of cases will have decreased or absent patellar reflexes. Forelimb function is normal. Signs begin with hindlimb ataxia and progress over 6–12 months during which time the ataxia and paresis may relentlessly worsen or decline gradually interspersed with periods of relative constancy of signs. One hindlimb may be slightly worse than the other. Spinal pain is not a feature. Urinary and faecal continence is maintained.

The chief differential diagnosis is Type II intervertebral disc disease of the thoracolumbar spine which may not cause spinal pain nor respond to a treatment trial of prednisolone.

The diagnosis of DM is currently one made by excluding other lesions. Given the age of the animal under investigation Type II IVDD and DM could co-exist. The owner must be made aware of this if surgery on Type II IVDD is considered.

Myelography has the potential to worsen the paresis and ataxia so MRI is preferred. Lumbar CSF samples may have a raised protein level but are not diagnostically useful as the sole method of investigation. A DNA mutation found to be a major risk factor in developing DM has been discovered. Further study will clarify the percentage of 'gene positive' dogs that develop DM within their lifetime.

The cause remains enigmatic.

Specific treatment does not exist. Physiotherapy improves longevity by limiting disuse atrophy. Most animals are euthanized when nonambulatory paraparesis occurs. If the animal is kept alive long enough, forelimb paresis and incontinence eventually develop.

This has also been termed degenerative radiculomyelopathy due to the pathology being described in the lumbar dorsal nerve roots.

Degenerative myelopathy has also been reported in other middle-aged to older, large breed dogs such as the Siberian husky, Bernese mountain dog, Rhodesian ridgeback, boxer and Chesapeake Bay retriever. Pembroke

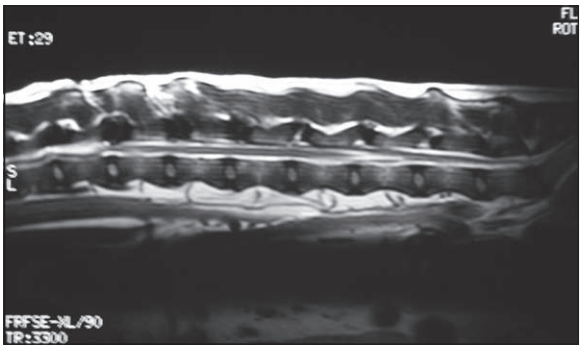


Figure 47.6 Sagittal T2WI. Dorsal compression of the cord at T13–L1.

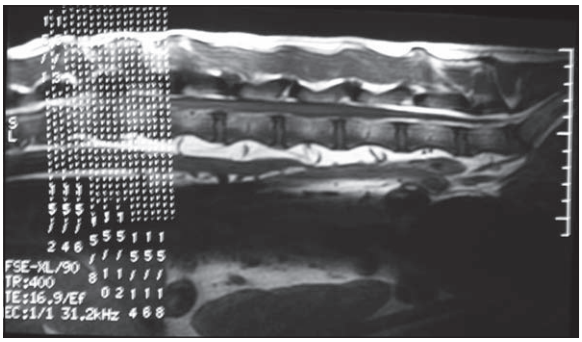


Figure 47.7 Sagittal T2WI. Scout image of transverse slices taken through an area of interest.



Figure 47.8 Transverse T2WI. Syrinx within the cord rostral to the compressive lesion. No specific treatment is given for the syrinx itself.

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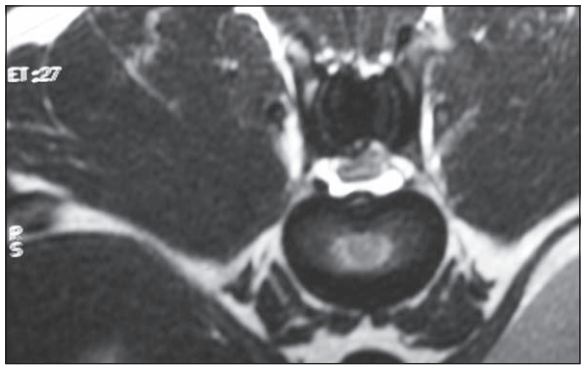


Figure 47.9 Transverse T2WI. Compression of the spinal cord.

Welsh corgis have a familial degenerative myelopathy with progression of signs occurring in animals over 1.5 years old. Euthanasia was performed at the mean age of 13 years by which time 70% had forelimb paresis. It has been reported in the cat but all other possible diseases must be ruled out first before presuming this to be the diagnosis.

CASE WORK-UP

MRI showed dorsal compression of the cord by the T13–L1 articular facets (see Figs 47.6–47.9).

DIAGNOSIS

Osteoarthrosis of the articular facets with secondary cord compression.

TREATMENT

A dorsal laminectomy removed compression from the spinal cord. Six weeks later, the dog was neurologically normal.

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48 Ataxia: cerebellar ataxia

INITIAL PRESENTATION

When the coordination fails, smoothness of movement is lost and is replaced by:

- Dysmetria, an exaggerated movement of limbs which appears as a jerking staggering gait, e.g. hypermetria of the forelimbs. Ipsilateral to the lesion
- Delayed onset of voluntary movement with normal strength
- Oscillation of the trunk at rest: truncal ataxia
- Intention tremor of the head
- Falling forward, backward or to either side
- Spasticity.

Strength is preserved. General proprioception is preserved ('knuckling response', placing). There may be an ipsilateral menace deficit. Vestibular signs may occur with some cerebellar lesions.

INTRODUCTION

The cerebellum's main function is to coordinate muscular activity by controlling the rate, range, force and direction of movement of the head, trunk and limbs. Smooth controlled movement results. The cerebellum receives proprioceptive information from muscle, tendon and joints, balance information from the vestibular apparatus, visual information and input from the generators of voluntary motor activity. It regulates motion and muscle tone by influencing the descending motor tracts of the spinal cord.

SIGNALMENT

A 3-month-old female domestic shorthaired kitten.

CASE PRESENTING SIGNS

Hindlimb ataxia and a tremor.

CASE HISTORY

The kitten was normal when acquired at 8 weeks of age. A male littermate was also bought and this animal had remained normal. Hindlimb ataxia was noted at first vaccination 3 weeks prior to referral and had worsened in the intervening period. The kitten remained ambulatory and could feed.

CLINICAL EXAMINATION

The kitten was mentally alert and ambulatory with hindlimb ataxia, 'knuckling-over' of the right fore, and falling to either side. The head posture was normal except for the fine tremor. Weakness and absent proprioception were found in the hindlimbs. Spinal reflexes were normal.

NEUROANATOMICAL LOCALIZATION

The lesion was localized to the cerebellum and brainstem or cervical spinal cord. A purely cerebellar lesion could not account for the hindlimb weakness detected on hopping reactions. The proprioceptive deficits of the right forelimb and both hindlimbs could be due to a cervical cord or brainstem lesion. A spinal cord lesion was thought most likely as the mental alertness appeared to be normal. The fine tremor of the head at rest was

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thought to be due to a cerebellar lesion, although the classic overshooting of an intention tremor was lacking. Myelination defects and CNS inflammation may also cause a tremor.

DIFFERENTIAL DIAGNOSIS

Abiotrophy

Postnatal degeneration of the cerebellar cortex and other components occurs chiefly in the dog. The age of onset and rate of progression are breed-related. Most cases are noticed once ambulation commences (3–12 weeks) but later onset at 6–12 months or adult onset (7–13 years) are also reported. MRI measurements detected cerebellar atrophy in affected American Staffordshire terriers. Definitive diagnosis is made by postmortem. Investigative efforts aim to rule out other potential causes of cerebellar dysfunction (Table 48.1).

Neuroaxonal dystrophy (NAD)

NAD is a degenerative disease of the CNS which presents as cerebellar dysfunction in young cats or dogs under 7 months old. Late onset disease occurs in the rottweiler (1–2 years old) and German shepherd dog (15 months old). A menace deficit, nystagmus and conscious proprioception deficits are reported in the affected rottweilers. Most cases slowly worsen. There is no treatment.

Lysosomal storage disease

Cerebellar ataxia and tremors are common presenting signs in the affected young animal.

Feline panleukopenia virus

In-utero or perinatal infection results in cerebellar hypoplasia which manifests as dysmetria, truncal ataxia, and a fine head tremor as the kitten begins to ambulate. Clinical signs do not progressively worsen but become more obvious as the kitten grows active. Quality of life is deemed satisfactory by pet owners.

Infarction

There is sudden onset of non-progressive cerebellar signs, possibly unilateral. MRI findings are highly suggestive of the condition. Diagnostic efforts focus on eliminating causes of thromboembolic disease. Prognosis is good. Half the affected dogs were spaniels or spaniel crosses in one study.

Neosporosis

Neospora caninum is a protozoal pathogen of dogs which has been reported as causing rapidly progressive fatal neurological disease. As expected with an infec-

Table 48.1 Onset of signs in cerebellar abiotrophy

Onset (1st signs)	Slow	Progressive	Stabilize or unknown
By first vaccination (≤16 weeks of age)	Samoyed	Azores cattle dog Australian kelpie Beagle Bernese mountain dog Border collie Bull mastiff Coton de Tulear Irish setter Jack Russell terrier Kerry blue terrier Labrador Rhodesian Ridgeback Mixed breed cat	Coton de Tulear Miniature poodle Rough coated collie
By neutering (≤6 months old)	Old English sheepdog Gordon setter	Airedale Finnish harrier	
≥1 year of age	Brittany spaniel American Staffordshire terrier Epagneul Breton dog Siamese cat Domestic shorthair cat		

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tious disease, lesions are multifocal but the predominant sign in young dogs is progressive paraparesis from myelitis. Progressively worsening ataxia of 2 months to 2 years duration has been reported in adult dogs aged 3–10 years. Clinical signs indicated a cerebellar lesion. MRI found a smaller cerebellum and contrast-enhancing thickened meninges overlying the cerebellum. Serum antibody titre (IFAT) for *Neospora* was >1:800 in 4/4 dogs. CSF analysis showed a mononuclear pleocytosis. PCR testing of CSF for *Neospora* was positive in 3/4 tested. Clindamycin 10–20 mg/kg and trimethoprim-sulfadiazine 15 mg/kg q12 hr improved 4/4 dogs, however one subsequently worsened. Histopathology confirmed the diagnosis.

Dysmyelination or hypomyelination of the CNS

There are rare, congenital disorders in which puppies are affected between 2 and 8 weeks of age and show a generalized (whole body) tremor present at rest and with movement. Signs decrease or disappear with sleep, and worsen with exercise, excitement and goal-directed movement. The severity is breed-dependent. Some are rendered recumbent while in others the signs plateau and improve by 1 year of age.

A wide-based stance, dysmetria, bunny-hopping gait, hindlimb bouncing while standing and a fine tremor are seen. Seizures, nystagmus, absent menace response, weakness, imbalance, stiff hindlimbs are also reported.

In this case: Given the young age, a congenital malformation of the CNS or viral inflammation were thought to be the most likely disease. A storage disease was possible as many induce ataxia and tremors in young animals. Congenital malformations may present as a static clinical course or a rapid decompensation.

CASE WORK-UP

The owner's chief concern was that the littermate be preserved in good health. To that end, the affected kitten was checked for infectious disease. Serological titres for FIP, FeLV and FIV were negative. Toxoplasma serology was also negative. Imaging of the brain and CSF collection had been discussed but was declined.

The affected kitten worsened in the next few days and was no longer able to walk without falling.

DIAGNOSIS

Globoid cell leukodystrophy (GCL) was found on postmortem.

This is a rare lysosomal storage disease caused by a deficiency in the enzyme galactocerebrosidase which leads to the accumulation of psychosine, a substance toxic to oligodendrocytes and Schwann cells of the CNS and PNS.

GCL has been reported in two DSH cats aged 3 and 4 months with progressively worsening hindlimb ataxia, paraplegia, loss of pain perception in the hindlimbs, and tremors of the thoracic limbs.

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49 Ataxia: vestibular ataxia

INITIAL PRESENTATION (VESTIBULAR DYSFUNCTION)

- Ataxia
- Falling, leaning, rolling or circling
- Head tilt, nystagmus or positional strabismus.

INTRODUCTION

Balance in a general sense is the ability to remain upright and so anything interfering with the mechanisms keeping an animal upright (strength, muscle tone, proprioception and coordination) could be said to affect balance.

In neurological terms, balance describes the control of posture exerted by the vestibular system. The receptors of the vestibular system are referred to as balance receptors and detect the position and movement of the head. This is termed 'special' proprioception. (see Chapter 57, p 234)

SIGNALMENT

An 8.5-year-old neutered female crossbred dog.

CASE PRESENTING SIGNS

Loss of balance and left head tilt.

CASE HISTORY

Signs commenced gradually 3 weeks prior to referral and had been slowly worsening since.

The dog veered to the left when walking. The left hind gave way a little.

CLINICAL EXAMINATION

An alert ambulatory dog with a left head tilt was observed to lean to the left and veer off to the left when walking. Hopping and proprioception were normal in all limbs. Decreased left facial sensation was detected. No abnormal nystagmus was found.

NEUROANATOMICAL LOCALIZATION

Leaning and falling to one side in the absence of weakness is suggestive of vestibular disease. Peripheral vestibular disease leaves the postural reactions intact as in this dog but when vestibular disease is present and worsening, one would expect to find spontaneous, i.e. abnormal, nystagmus on examination.

The absence of abnormal nystagmus in this dog and the abnormal facial sensation should ring warning bells; do not be fooled by the signalment. This is not a simple case of old dog idiopathic peripheral vestibular disease.

The abnormal facial sensation indicates either a left CN V lesion or, more rarely, a right cerebral lesion affecting the quintothalamic pathway.

No cerebral signs are present so, remembering occam's razor, this is probably a focal left CN V and left CN VIII lesion. These two nerves are anatomically close at the cerebellopartive angle.

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It could be that the central vestibular pathways in the left cerebellum have been affected. Central vestibular disease from cerebellar lesions sometimes, but not always, cause paradoxical signs.

No paradoxical signs are present in this dog as the head tilt and leaning are both to the left.

DIFFERENTIAL DIAGNOSIS

A focal lesion such as neoplasia, inflammation, or infarction.

The latter would not have a gradual onset with progressive worsening.

CASE WORK-UP

MRI found a large contrast-enhancing lesion with the characteristics of a meningioma or neuroma at the left cerebellopontine angle. The large size suggested the lesion had developed slowly (Fig. 49.1).

DIAGNOSIS

Neoplasia.

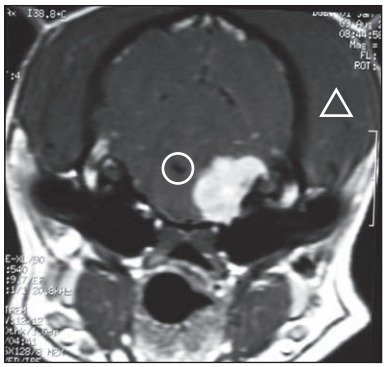


Figure 49.1 MRI T1WI + contrast. Compression of the fourth ventricle (circled) by the mass had not caused ventriculomegaly. No atrophy of the temporalis muscle despite CN V involvement (triangle).

TREATMENT

Prednisolone then euthanasia. Radiation therapy had been discussed with the owner.

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SECTION 7

LAMENESS

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50 Lameness – an introduction

Lameness is an abnormal gait caused by pain or mechanical dysfunction. The gait is predictably irregular (*cf.* ataxia). Proprioception pathways are not damaged so ataxia does not occur and proprioception testing is normal.

PRESENTING SIGNS

Lameness may be recognized by the following changes:

1. Decreased weight-bearing (limping)
   - Holding the limb off the ground
   - The unaffected limb placed further medially to support more of the animal's weight
   - Shortened stride length of the affected limb
   - Shifting body weight to relieve pressure on affected limb(s)
     - head bob. The head jerks upwards as the affected forelimb strikes the ground in an effort to limit weight being transferred to the painful forelimb
     - lowered head and arched back to shift the weight to the forelimbs from painful (or weak) hindlimbs.
2. Decreased flexion or extension of limbs
   - Shortened stride, shuffling gait, appear stiff/'stilted' or crouched.

Other signs of pain:

- A reduced activity level. Refusing to go for walks.
- Unwilling or unable to jump up or down
- Slow to rise from a recumbent position
- Depression, weight loss, abnormal elimination habits, poor grooming or aggression.

GRADING LAMENESS

Force plate analysis is an objective measure of weight-bearing. A subjective scale to score lameness is used during examination. Most scales assign 0 as normal, and 5/5 or 10/10 as non-weight-bearing lameness (Table 50.1).

CLINICAL TIP

A surprisingly large number of pet owners do not equate lameness with pain. Many believe that an old animal with a stiff gait, slowness to rise, and a reduced exercise tolerance is normal and do not request treatment.

Table 50.1 Grading lameness

Grade	Description of lameness	Weight-bearing at rest
0	Normal gait	Normal
1	Difficult to observe intermittent	Normal
2	Observable: minor constantly apparent	Normal
3	Obvious, constant	Normal
4	Severe, constant	Incomplete
5	Non-weight bearing	Absent

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51 Lameness: nerve root compression

INITIAL PRESENTATION

- Nerve root compression or infiltration causes pain, resulting in 'root signature' lameness in the innervated limb.
- Three cases of forelimb lameness are described.

SIGNALMENT 1

A 9-year-old neutered male Dalmatian.

CASE PRESENTING SIGNS

Neck pain and left fore lameness.

CASE HISTORY

The dog had been yelping intermittently for 1 month. Left forelimb lameness commenced 1 week prior to the referral. Although the dog was described as active, there had been no history of trauma.

CLINICAL EXAMINATION

A weight-bearing left forelimb lameness and a resistance to lateral neck flexion were found. No neurological deficits were present.

NEUROANATOMICAL LOCALIZATION

Nerve roots to the left forelimb, within the vertebral canal to account for the neck pain.

DIFFERENTIAL DIAGNOSIS

- Lateralized disc extrusion
- Nerve root tumour
- Meningioma
- Vertebral tumour
- Discospondylitis could collapse the disc space and compress a nerve root.

CASE WORK-UP

MRI was chosen to investigate the causes of neck pain because of the potential for nerve root compression. A lateralized extrusion of disc material into the intervertebral foramen is difficult to outline with myelography as the subarachnoid space is not sufficiently compressed. Rotated views may help, but the lesion may not be outlined even then.

MRI showed ventral compression of the cord over the C5–6 IVD space (Fig. 51.1).

CSF was not taken.

DIAGNOSIS

IVD extrusion was confirmed at surgery and the cord was decompressed. The lameness and neck pain resolved and the dog remained normal without pain relief.

Forelimb lameness may precede or occur simultaneously with the onset of neck pain caused by a lateralized disc extrusion. Signs may wax and wane and are generally of sudden onset. Progression to tetraparesis, or weakness of the forelimbs can occur.

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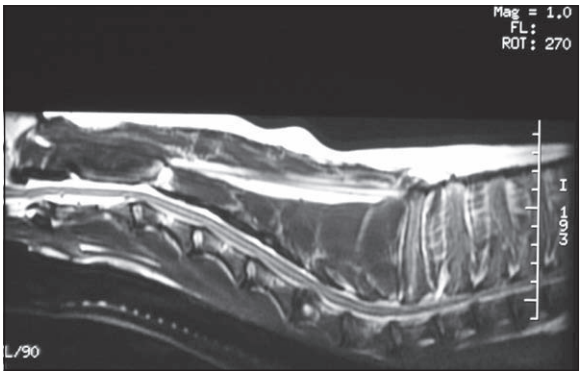


Figure 51.1 Sagittal T2WI. Compression of the cord at C5–6 IVD space.



Figure 51.2 Some normal male dogs do not lift a hindlimb to urinate.

Myelography shows the filling of the subarachnoid space with contrast agent. Dorsal and vertical nerve roots are enclosed by the subarachnoid space just to the level of formation of the spinal nerve. Lateral compression of the spinal nerve at the intervertebral foramen will go unnoticed by myelography. MRI is an ideal tool to detect this and other causes of neck pain. Transverse slices should be taken through each IVD space from C2–3 to C7–T1. Prognosis for relief from pain and restoration of strength is excellent after surgery.

SIGNALMENT 2

A 9-year-old male working cocker spaniel.

CASE PRESENTING SIGNS

Left hindlimb lameness and a reluctance or inability to jump into the car.

CASE HISTORY

One month's duration of difficulty getting into the car, reduced activity, and non-weight-bearing on the left hind when standing. A bunny-hopping, synchronous hindlimb gait occurred when the dog tried to run. Atrophy of the hindlimbs had been noted. The dog was slow to squat when defecating and had stopped lifting a hindlimb to urinate.

CLINICAL EXAMINATION

The dog was alert and ambulatory with decreased weight-bearing on the left hind when standing and a shortened stride of the left hind. The hindlimb gait was not ataxic but the altered stride length did give it an irregular look when viewed front on and from the rear but when viewed from the side the left hind lameness and right hind compensation was easier to see.

Atrophy of the muscles of the left hind was noted. Hopping and proprioception were normal. The patellar reflex was 3+ bilaterally and the withdrawal reflex was normal in all limbs. The anal tone was normal. Pain could be elicited by pressure over the LSJ.

Applying firm pressure to the lumbosacral joint while elevating the pubis elicited pain. Hip pain was found by forced extension of the hips. To differentiate the source of pain, avoid hip extension when applying pressure to the LSJ.

Decreased tarsal flexion during limb withdrawal (flexor reflex) indicates sciatic involvement. A lack of antagonism from sciatic deficits creates pseudo-hyperreflexia of the patellar reflexes.

NEUROANATOMICAL LOCALIZATION

A root signature lameness affecting lumbar nerve roots, cauda equina, and lumbosacral junction was suspected.

Weakness, lack of coordination, and pain can prevent an animal from lifting a hindlimb or squatting to urinate (Fig. 51.2). The owner will know what is normal for their pet but generally will not mention a change unless specifically asked.

Such animals tend to assume the squatting posture but having started to eliminate will rise up and walk along while finishing. Urination has a voluntary initiation and cessation. Urine retention may occur.

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DIFFERENTIAL DIAGNOSIS

Compression of a lumbar nerve root at the L7–S1 intervertebral site or at an intervertebral foramen can produce 'root signature' lameness. The ipsilateral hindlimb is held in partial flexion to avoid weight-bearing. Intermittent partial weight-bearing creates a '*stamping*' action.

Compression at L7–S1 by a combination of prolapsed disc, articular facet overgrowth, L7–S1 instability, ligament or joint capsule hypertrophy or stenosis of the cranial aperture of the S1 is common in middle to large breed adult male dogs. It presents with signs of pain or neurogenic paresis:

1. Pain
   - Slow to rise and to sit
   - Difficulty maintaining a squatting position to defecate (gets up and walks off while defecating)
   - Reluctant to jump in or out of vehicles
   - Reluctant to exercise
   - Uni- or bilateral (or shifting) hindlimb lameness. Permanent or intermittent.
2. Paresis
   - Compression of nerve roots supplying tail, anal sphincter, external urethral sphincter and sciatic nerve
   - Tail paresis or paralysis
   - Urinary and faecal 'overflow' incontinence
   - 'Knuckling' of paw
   - Licking, biting paws or tail (suspect paresthesia).

In this case: Compression of nerve roots by IVDD, articular facets, neoplasia (vertebral or cauda equina) or discospondylitis were considered.

CASE WORK-UP

Diagnosis requires demonstrating compression of the lumbosacral nerve roots. As these nerve roots arise from the lumbosacral intumescence imaging must include vertebrae L3–S1 and not just the L7–S1 disc space. One must always remember the neuroanatomic localization of the lesion when interpreting any imaging and not be beguiled by lesions in adjacent, irrelevant locations seen within the field of view.

The efficacy of myelography is hampered by the variable position of the dural sac within the spinal canal. In approximately 15–20% of dogs, the dural sac does not cross the L7–S1 disc space. Epidural leakage of contrast agent during a lumbar injection, and a dural sac that lies in the middle of the vertebral canal, or does not fill with contrast agent will also limit interpretation. Epidural placement of contrast has been used diagnostically by injecting contrast agent external to the dural sac between S3 and Cd1 vertebrae or at the LS space (epidurography). Filling may be incomplete due to epidural fat.

MRI has the advantage of transverse imaging through any level of the spine while producing excellent soft tissue detail. CT provides superior bone detail compared with MRI and better soft tissue contrast than radiographs.

Despite nerve root involvement abnormal spontaneous potentials may not be found on electromyography.

In this case: MRI demonstrated ventral compression of the cauda equina at the L7–S1 disc space.

DIAGNOSIS

Type II disc prolapse.

TREATMENT

If clinical signs have not resolved with strict rest and pain relief, surgery must be considered. In such cases it is generally more cost- and time-efficient to perform MRI initially, rather than radiography. All causes of localized lumbar pain can be assessed, including nerve root tumours (rare), discospondylitis (often younger dogs), vertebral body tumours (rare), DJD of the articular facets and paravertebral abscesses. Spondylosis is common at L7–S1 and its presence is not diagnostic of neural compression.

The degree of compression is not correlated with the severity of the signs or prognosis. Once again, treatment decisions are based on the severity of the clinical signs.

1. Rest, pain relief for the first episode of pain or intermittent pain. Some 50% had a good outcome within approximately 3 months.
2. Surgical decompression for recurrent or continuous pain or neurological deficits. Some 75% had a satisfactory outcome over a 3-year follow-up. In one study, 78% of affected working dogs returned to their normal activity level. Incontinence failed to resolve postoperatively in 55–87%.

Some sources report that the preoperative duration of neurological deficits such as incontinence negatively affects prognosis. Incontinence for more than 1 month before surgery is less likely to resolve after surgery.

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A L7–S1 dorsal laminectomy was performed in this case. The bulging anulus fibrosis was removed. The dog's gait and activity level returned to normal.

SIGNALMENT 3

An 11-year-old neutered female Briard dog.

CASE PRESENTING SIGNS

Neck pain and left forelimb weakness.

CASE HISTORY

There had been a sudden onset of neck pain and left forelimb lameness of 3 weeks' duration. The dog had progressively worsened and was presented with profound forelimb weakness.

Carprofen and tramadol were used to manage the pain.

CLINICAL EXAMINATION

The dog was alert and responsive with a non-ambulatory tetraparesis. When the body weight was supported, the forelimbs were seen to be weaker than the hind. Proprioception was tested by supporting all of the dog's body weight and was found to be decreased in all limbs, more so in the forelimbs. Spinal reflexes were intact in all limbs. Neck pain was elicited by gentle palpation of the neck muscles.

NEUROANATOMICAL LOCALIZATION

C1–5 spinal cord segments. Strength and proprioception were reduced in all four limbs, and the spinal reflexes were intact. UMN tetraparesis. Compression of a nerve root to the left forelimb could account for the initial left fore lameness.

DIFFERENTIAL DIAGNOSIS

- IVD extrusion
- Spinal cord tumour
- Vertebral body tumour
- Distension of the cord from within may also cause pain.

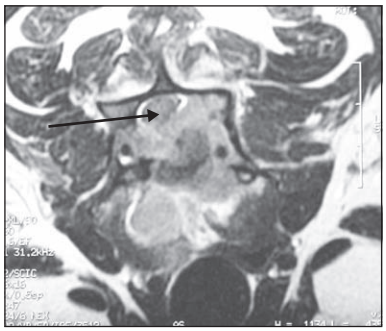


Figure 51.3 T1WI + contrast. Compression of the spinal cord (arrow) resulting in UMN tetraparesis.

The associated lameness which developed into forelimb weakness is an extra clue that the neck pain is of related origin and cervical in nature rather than cerebral. Also, cerebral lesions are not expected to cause such a profound change in the gait without some change in the mental state.

CASE WORK-UP

Myelography was discussed, but MRI was chosen to investigate a possibly lateralized or invasive lesion.

MRI showed a large tumour affecting the C5 vertebral body and surrounding tissue (Fig. 51.3).

DIAGNOSIS

Probably a sarcoma. The dog was euthanized.

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52 Lameness: nerve root tumours

INITIAL PRESENTATION

Tumours of the peripheral nerve sheath such as schwannomas are slow-growing and typically present as a chronic persistent forelimb lameness lasting months for which no orthopaedic explanation can be found. They are common in middle-aged dogs and more than 80% affect the brachial plexus or its nerve roots.

SIGNALMENT 1

A 9-year-old neutered female West Highland white terrier.

CASE PRESENTING SIGNS

Right fore lameness.

CASE HISTORY

There was a 6-week duration of right forelimb lameness. The limb had recently been giving way causing the dog to stumble when running or turning. There had been no history of trauma. The signs had not improved with NSAID therapy.

CLINICAL EXAMINATION

There was a generalized atrophy of the right forelimb. Hopping was decreased in the right fore and also in the right hind. Both the right fore and hind paws were quickly returned to position during proprioceptive testing but they were replaced in an abducted position which is abnormal. Placing in the right fore was slightly reduced. The right fore withdrawal reflex was reduced but pain perception in the limb was present. No Horner's syndrome was found. The cutaneous trunci reflex was normal bilaterally.

NEUROANATOMICAL LOCALIZATION

Nerve roots of the right brachial intumescence. The proprioceptive deficits of the right hind indicate a lesion within the vertebral canal.

DIFFERENTIAL DIAGNOSIS

The first sign was lameness, suggesting nerve root involvement, possibly compression by a lateralized IVD extrusion which had since worsened to cause right hind deficits. Peripheral nerve sheath tumours, meningioma, lymphoma were possible.

Early diagnosis of a nerve root tumour requires an index of suspicion. The caudal cervical nerve roots C6–8 are most commonly affected, weakening both flexion and extension. When the T1 and T2 nerve roots are involved ipsilateral Horner's syndrome and cutaneous trunci reflex loss occurs. Successful treatment requires forelimb amputation with clean tissue margins in the severed nerve roots. If the dog is an unsuitable candidate for a forelimb amputation then one could argue that further diagnostics are not warranted.

CASE WORK-UP

Electromyography is a useful tool. It detects an unstable muscle membrane caused by denervation and aids differentiation of orthopaedic from neurogenic causes of

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lameness. Lumbar CSF should be collected even in the absence of ipsilateral hindlimb signs. A compressive spinal cord lesion often results in elevated total protein levels and its presence would signal that invasion of the spinal canal has taken place. Enlargement of the intervertebral foramen by an encroaching nerve root tumour can be detected on non-contrast radiography. Myelography has been used to rule out extension of the tumour to the spinal canal. Oblique views are required. An intradural-extramedullary pattern ('golf tee') compression is seen. MRI and CT have replaced myelography to a large extent.

MRI has proved extremely useful in locating nerve root tumours. Most have some degree of contrast-enhancement. Multiple planes through both axillae and the spinal canal are required. Metastases are rare.

In this case the EMG showed spontaneous potentials present in the right forelimb indicating denervation rather than disuse atrophy was present.

Lumbar CSF: The total protein was elevated at 0.80 g/l. WBC = 7/mm^3. Cytology was normal.

MRI showed enlargement of the right-sided C7 nerve root. And ventral compression of the cord by an irregular mass at the level of the C6–7 IVD space (Figs 52.1 and 52.2).

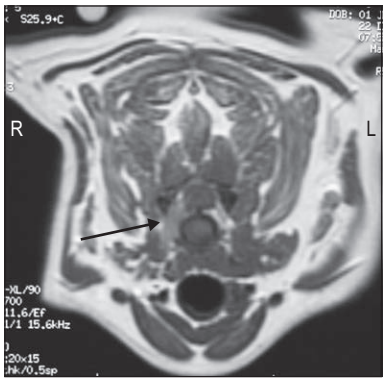


Figure 52.1 T1WI + contrast: thickened nerve root (arrow).

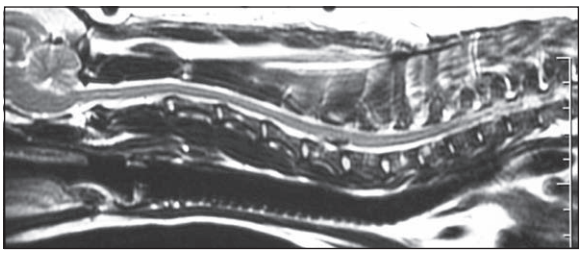


Figure 52.2 T2WI sagittal. There are eight cervical cord segments and nerve roots. C7 exits the canal at the C6–7 IV foramen.

DIAGNOSIS

Neoplasia: nerve root tumour.

Neurogenic atrophy, paresis and loss of sensation are not present during the initial period of lameness. Each muscle is supplied by several nerve roots. If only one nerve root is affected LMN signs will not be visible. However, invasion of one nerve root is sufficient to cause pain. Self-mutilation of the affected limb is uncommon.

As the tumour enlarges, more nerve roots of the brachial plexus are affected and a painful mass may be palpable in the axilla. LMN signs become obvious. Invasion of the spinal canal through the intervertebral foramen is the inevitable outcome resulting in ipsilateral hindlimb proprioceptive deficits and paresis. Compression of the brachial intumescence will eventually result in LMN weakness of the contralateral forelimb and bilateral hindlimb UMN weakness.

Less than 20% of peripheral nerve sheath tumours occur elsewhere. Their slow growth can go unnoticed until they compress the spinal cord.

Lymphoma of the brachial plexus occurs in the cat and has a rapid progression of signs from lameness to paresis and proprioceptive deficits within weeks rather than months. Lumbar CSF analysis is useful as it may detect malignant lymphocytes. Enlargement of the intervertebral foramen is not seen in this acute presentation.

TREATMENT

The dog was euthanized 2 months later.

SIGNALMENT 2

A 7-year-old neutered female crossbred dog.

CASE PRESENTING SIGNS

Right hindlimb lameness.

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CASE HISTORY

The dog suddenly began limping on the right hindlimb 4 months ago. Hip dysplasia was diagnosed and treated with NSAIDs and hydrotherapy. Some initial improvement occurred but the lameness worsened and became intermittently non-weight-bearing. No other problems were reported. The dog was still able to jump.

CLINICAL EXAMINATION

The bright, alert ambulatory dog had a right hindlimb weight-bearing lameness. At rest, the right hind would sometimes be held off the ground. When walking, the right hindlimb adducted, and the right hind claws were heard to intermittently scrape the ground. Right hind proprioception was reduced.

The right withdrawal reflex was weak with poor flexion of the stifle and tarsus. The hip flexed but was weak. The patellar reflex was normal/increased. Pain perception was present.

Anal tone was present and appeared to be normal. The dog appeared painful when the LSJ was pressed. No hip pain was found.

NEUROANATOMICAL LOCALIZATION

Right sciatic nerve or nerve roots.

DIFFERENTIAL DIAGNOSIS

- Nerve root tumour
- Compression by IVDD.

CASE WORK-UP

MRI showed enhancement of the right lateral cauda equina beginning at the level of the cranial end of the L5 vertebral body and running to the L6–7 right side foramina. There was no compression at the LSJ (Figs 52.3 and 52.4).

CSF analysis showed: an increase in total protein: 0.91 g/l. Increased WBC 16, with normal cytology. RBC 130 µl was thought to be iatrogenic during collection.

DIAGNOSIS

Suspected schwannoma rather than lymphoma as the latter would be expected to have a shorter clinical time course.

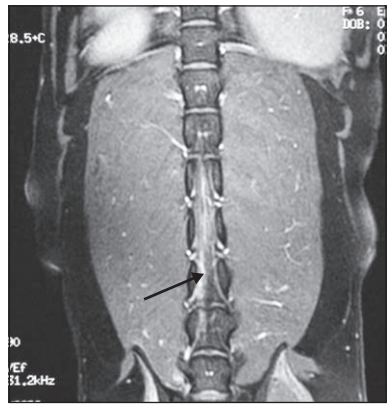


Figure 52.3 Dorsal plane. The signal from fat has been suppressed, allowing contrast enhancement of the nerve roots on the right to be visible (arrow).

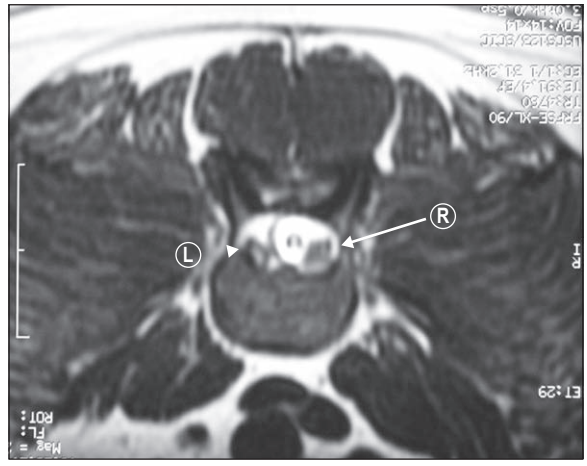


Figure 52.4 Transverse image. T2WI: the right-sided nerve (arrow) is enlarged compared with the left (arrowhead).

TREATMENT

The owner declined surgery due to the lack of a 100% guarantee the dog would not be worse postoperatively. The dog remained ambulatory and continent 7 months after the diagnosis. The right hind 'knuckled' over constantly. The dog received daily NSAID and did not appear to be in pain.

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53 Lameness: ischaemia

INTRODUCTION

Arterial thromboembolism produces limb ischaemia and either paresis or paralysis, depending on the degree of occlusion and amount of collateral circulation.

This is most common in the cat and may be the first sign of cardiac disease. More than 70% of cats with this condition have an underlying cardiomyopathy. It is rare in the dog and risk factors include cardiac disease, hyperadrenocorticism, disseminated intravascular coagulation (DIC), protein-losing nephropathy or enteropathy, heartworm, AIHA, neoplasia and sepsis.

A saddle thromboembolism (TE) forms at the aortic trifurcation in 90% of affected cats. Clinical signs result from ischaemia of the cauda equina and hindlimb muscles. The proximal limb may retain voluntary movement while the distal limb is paralysed, or vice versa. Tail function and anal tone may be intact. Trauma or neoplasia to the cauda equina tends not to be so selective. Muscles particularly the gastrocnemius and cranial tibial muscles are firm, painful and swollen.

Acute thrombosis also occurs in single limbs causing transient or permanent paresis/paralysis. Owners may report transient signs as lameness.

Acute and chronic onset of signs have been reported in dogs aged 5–10 years of age. Chronic onset (>24 hr) produces exercise intolerance and pain (restlessness, refusal to move, vocalization from palpation along the spine) for weeks to months prior to diagnosing the causative aortic thromboembolism. The femoral pulse was abnormal in all dogs. Minimal neurological deficits were present. Abdominal ultrasound was 100% successful in demonstrating the thromboembolism. Muscle enzymes were generally within normal limits (*cf*. acute onset <24 hr). Survival times ranged from 1 month to 2 years. Cavalier King Charles spaniels comprised the majority of dogs affected, although larger breeds were not spared.

The dog featured in Figure 53.1 presented with a 24-hour history of shifting hindlimb lameness followed by an arched back and then sudden paraplegia on the day of referral. Femoral pulses were absent bilaterally but the hind paws were warm until they became cold 2 hours later. The right quadriceps femoris muscle was firm and were painful when touched as were the lumbar epaxial muscles. Pain perception and spinal reflexes were absent in both hind limbs. The cause was thought to be a hypercoagulable state from a protein-losing glomerulonephropathy. The dog was euthanized as the prognosis for long-term normality was guarded and the pain was extreme.

SIGNALMENT

A 6-year-old neutered male domestic longhaired cat.

CASE PRESENTING SIGNS

Intermittent shifting lameness.

CASE HISTORY

Lethargy, inappetance and weight loss had been present for 2 months. At 6 weeks prior to referral, a 3-day duration of right hindlimb dragging occurred. Three weeks after this improvement, the left fore pad was swollen, had lost fur and was painful, and 48 hours later still, the hindlimbs were dragged on waking up from sleep. One week later, the hindlimbs were momentarily seen to be dragged and the right forelimb was lame and non-weight-bearing. This improvement was followed by the hindlimbs being dragged again which subsequently improved in the 4 days prior to referral.

There had been bowls of rat bait in the kitchen but it was not known if the cat had had access to any of it.

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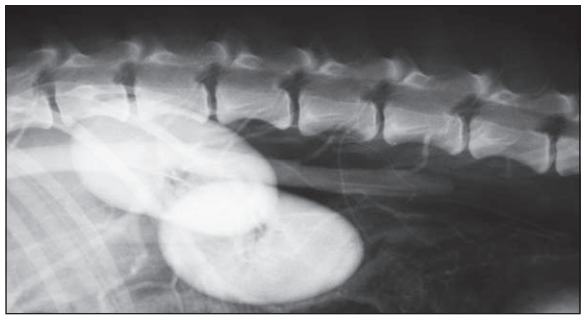


Figure 53.1 Non-selective angiography of a 9-year-old male collie cross with aortic thromboembolism. (photo courtesy of Dr Jacques Sottiaux).

CLINICAL EXAMINATION

The neurological examination found a very quiet but mentally alert and responsive cat with voluntary movement in both hindlimbs but more so in the right hind. The tail had voluntary movement and anal tone was present. The patellar reflex was normal bilaterally. The hindlimbs flexed by hip movement. The forelimb withdrawal reflex was normal. Cranial nerve and fundus examinations were normal. The gastrocnemius muscles were firm and felt enlarged but were not painful. The femoral pulses were poor/very difficult to feel but all paws felt warm and as warm as each other. The respiratory rate was 56 bpm at rest and there was a visible respiratory effort, HR = 140.

NEUROANATOMICAL LOCALIZATION

The lesion was localized to muscles and nerves distal to the stifle, secondary to poor perfusion.

DIFFERENTIAL DIAGNOSIS

The poor pulses, increased respiratory rate and episodic lameness/weakness were suggestive of a vascular cause; haemorrhage or infarction (emboli; hyperviscosity from polycythaemia or hypergammaglobulinemia).

The sudden onset of paralysis and pain in *arterial thromboembolism* mimics recent trauma. CK, AST, and ALT values were high; 22% of affected cats will have a femoral pulse on presentation, which can complicate diagnosis as the classic sign of cold extremities may not be present. Blood glucose concentration from the affected limb(s) can be compared with the non-clinical limbs (The results for the ischaemic limb should be less than that from the non-ischaemic limb).

Signs of thromboembolism

- Pain
- Pallor
- Pulses absent
- Hypothermia
- Paralysis.

Polycythaemia is an elevated red blood cell count. Dehydration produces a relative polycythaemia and signs are secondary to dehydration. Absolute polycythaemia is rare and most cases will present with neurological signs due to the hyperviscosity and resulting poor perfusion. Seizures, behavioural change, blindness, tremors and ataxia occur. Lameness and muscle pain may be the presenting signs (Table 53.1).

CASE WORK-UP

Bleeding times were normal PT 17 minutes (Normal = 15–23), APTT 89 minutes (Normal = 70–120), ruling out Vitamin K antagonism from rat bait.

In-house globulin and PCV were normal. Serum K was normal (*Note*: this can be raised from reperfusion injury). Total calcium was normal (*Note*: hypocalcaemia can cause muscle spasm; weakness and episodic signs). AST

Table 53.1 Differentiating relative and absolute polycythaemia

Relative polycythaemia	Absolute polycythaemia
Loss of plasma volume	EPO-independent proliferation
Splenic contraction (dogs)	Primary ('vera') Myeloproliferative disorder
	EPO-dependent proliferation
	Secondary
	Hypoxia (cardiopulmonary)
	Non-hypoxic (renal: neoplasia)
Normal total RBC mass	Increase in total RBC mass
Signs of dehydration, hyperviscosity, and the underlying disease	CNS signs. Muscle pain. Hypoxia: dyspnoea, cyanosis
PCV mildly increased: 55–65% (dog)	PCV 60–70%
Treatment: fluid therapy	Phlebotomy, leeches*, treat underlying mechanism

*On average over a 48 hr attachment, 1 leech decreased the PCV by 6%.

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1878 IU/l (15–45), ALT 451 IU/l (30–6), CK >9400 IU/l (0–400). Globulin level was normal. Haematology was normal. The rest of the biochemistry was normal. Glucose was 8.4 mmol/l and thought to be due to the stress response.

Cardiology consultation found hypertrophic cardiomyopathy (HCM) and pulmonary oedema.

DIAGNOSIS

HCM with thromboemboli.

PROGNOSIS

Recovery: 50% of cats will regain some degree of limb function within 2 months. Monitor blood flow to the limb with a Doppler blood pressure monitor. Monitor return of sensation. A general rule of neurology is that deficits are reversed in the order of their occurrence, i.e. sensation is the last function to go and the first to return. Thromboemboli can spontaneously resolve. Surgical removal has a high associated fatality rate. Reperfusion injury occurs if the clot is removed surgically but not if it spontaneously resolves. Longevity: 3 months to 2.5 years. Aspirin does not prevent recurrence.

TREATMENT

Aspirin 325 mg; one-quarter of an aspirin every other day

Bumetanide 1 mg; half tablet BID

Diltiazem 10 mg; half a tablet TID.

The cat gradually showed improvement in his hindlimb function and femoral pulses. He could walk on all four limbs, trailing the left hind behind him but advancing it at the hip. There was decreased tone of the left tarsus and a knotted feel to the left gastrocnemius muscle persisted. Both hindlimbs had patellar reflexes. The right hind flexed when the metatarsals were stimulated but not when the toes were stimulated; the left hind flexed the hip and stifle but not the tarsus.

A month later, the cat vomited dark brown material thought to be digested blood. The aspirin was stopped and sucralfate and ranitidine was used as gastrointestinal protectants. The vomiting stopped. Eight months later the cat was walking normally and eating well and was not on any medication.

GASTROINTESTINAL ULCERATION

Gastrointestinal ulceration, presenting as depression, anorexia, vomiting and melaena, is a common side-effect of NSAID therapy, or the combination of NSAID and corticosteroids. It occurs in the gastric antrum and duodenum. Abdominal pain is uncommon.

Colonic ulcers also occur after glucocorticoid use and spinal surgery, causing abdominal pain, constipation and fever. Perforation is 100% fatal.

Risk factors

- The use of one or more NSAID with or without concomitant corticosteroid therapy
- High dose dexamethasone or methylprednisolone
- Spinal surgery (increased sympathetic tone, inhibition of colon motility and perfusion).

Treatment

1. Coat the ulcer
   - Liquid sucralfate 0.5–1 g PO q4–8 hr
   - There is no proven efficacy of inert adsorbents such as Kaopectate or barium sulphate contrast media.
2. Decrease acid production
   - H2-antagonist
     - Cimetidine 5–10 mg/kg PO, IM, IV q6–8 hr
     - Ranitidine 2 mg/kg PO, IV q8–12 hr (dog)
     - Ranitidine 2.5 mg/kg IV or 3.5 mg/kg PO q12 hr (cat).
   - Proton pump inhibitor
     - Omeprazole 0.7 mg/kg up to 20 mg PO q24 hr.
3. Resection of the ulcer if it perforates or uncontrolled haemorrhage is present.

Prophylaxis

1. Sucralfate (1 hr before meals) – may prevent stress-induced ulcers but not NSAID-induced ulcers
2. Misoprostol – a prostaglandin E2 analog – prevents NSAID-induced ulcers
3. H2-antagonist
4. The combination of misoprostol, cimetidine and omeprazole has not prevented gastric ulcers in dogs receiving high dose glucocorticoids.

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SECTION 8

POSTURE

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54 Posture – an introduction

Posture is a description of the relative placement, arrangement, or position of the animal's head, body and limbs with respect to gravity. The term *attitude* refers to the position of the eyes, head and limbs relative to the body. Some authors have used 'attitude' as a synonym for mental activity or behaviour. As all abnormal postures should be described in the course of an examination, this point of terminology is not hugely important.

Posture is controlled by:

- Muscle tone
- Strength
- Balance
- Coordination.

Posture is described by:

- Direction: to the left, to the right, ventrally, dorsally. The direction is ALWAYS given from the animal's perspective NOT the viewer's. 'To the left' refers to the animal's left side. When taking the history be sure that the owner is describing the abnormality in this manner
- Proximity to the medial plane: abducted (further from), adducted (closer to)
- Degree of limb extension: increased (hyper) or decreased (hypo) extension, plantigrade or palmigrade.

DESCRIPTION OF ABNORMAL POSTURE

Head

- *Tilt*: one ear is closer to the ground than the other, e.g. a right head tilt has the right ear closer to the ground.
- *Turn*: the head is level with the nose pointing caudally, turning as if to look behind. 'A left head turn', or 'the head turns to the left' describes the nose pointing to the left side of the animal (see also Fig. 54.1).
- The combination of a head tilt and a head turn has been called a *yaw*.
- Bob, nod, intention tremor (see p 244)
- *Head pressing* (*obstinate progression*): animals push themselves forward until they meet an obstacle then continue to push against it. It is a sign of cerebral disease.

Neck

- *Dorsiflexion*: 'stargazing', head looking upwards
- *Ventroflexion*: lowered head posture, head rests on sternum
- *Torticollis*: twisting of neck
- May be held in a stiff, rigid, straight, unmoving position, lowered or level with the thoracic spine.

Trunk

- *Lordosis*; ventrally arched back, 'dipped', a concave arch, hunched, 'tucked up'
- *Kyphosis*: dorsally arched back, a convex arch. The head is often lowered and the hindlimbs held further forward, closer to the centre of gravity. It is a sign of neck, back, or abdominal pain and is also seen with hindlimb or generalized weakness
- *Scoliosis*: lateral curve to the spine
- *Pleurothotonus*: tetanic bending of the body to one side
- *Truncal ataxia*; side to side oscillation, irregular unpredictable movement, tremor
- *Falling*: forward, backward, or to the one or the other side. It can result from weakness, loss of proprioception, loss of balance (uneven vestibular input), alterations in muscle tone or a combination of all these factors

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Figure 54.1 Aged Jack Russell terrier with a right cerebral tumour. Head turn to the right.



Figure 54.2 Leaning to the left induced by an ipsilateral pontine glioma.

- *Leaning*: the upright trunk is positioned lateral to its centre of gravity. A sign of uneven muscle tone, or balance loss (see Fig. 54.2).
- *Lurching*: is a sudden movement in one direction. More than a lean; not quite a fall. Falling to one side but not hitting the ground.

Limbs

- *Adduction* (mnemonic: dd, limb = d, limbs close together) limb closer to median plane than normal
- *Abduction*: limb further from median plane than normal
- Wide-based stance, 'splayed': limbs bilaterally abducted
- Hindlimbs held closer to forelimbs, shifting weight to forelimbs



Figure 54.3 Left plantigrade stance.

- Non-weight-bearing
- Weight-bearing on the dorsal surface of the paw: 'knuckling over'
- *Plantigrade*: weight-bearing on the phalanges and metacarpals or metatarsals rather than the digits. Palmigrade is sometimes used to refer to a plantigrade forelimb position (see Fig. 54.3).
- Increased extension: stiff, stilted, rigid, spastic, 'saw horse' (all limbs in extension)
- Decreased extension resulting in a joint being more ventral than normal, e.g. a 'dropped' hock
- Oscillating extension and flexion causing a bouncing, jerky coarse tremor.

Repetitive limb flexion in the conscious patient

- *Pain*: lameness. Common
- *Partial seizure*: typically short-lived before generalizing to other body parts
- *Tremor*: orthostatic tremor. Unusual
- *Dyskinesia*: breed-related. Rare.

SYNDROMES

A syndrome is a group of clinical signs resulting in a distinct clinical picture.

- *Decerebrate rigidity*: extension of all limbs and dorsiflexion of the head and neck
- *Decerebellate rigidity*: opisthotonus with thoracic limb extension, flexion of pelvic limbs (hips) up under the body
- *Opisthotonus*: extension of forelimbs and dorsiflexion of the head and neck
- *Schiff–Sherrington*: extensor hypertonia of the forelimbs with paraplegia
- *Adversive syndrome*: leaning, head turning, propulsive circling, or ocular deviation toward the side of the rostral thalamic lesion.

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55 Torticollis

INTRODUCTION

Scoliosis describes a lateral curvature of the spine. It may be congenital due to a wedge-shaped malformation of vertebral bodies (hemivertebrae). Acquired scoliosis occurs secondary to syringomyelia or hydromyelia from unequal tone in or sensory input from the spinal musculature.

Kyphosis: The dorsal deviation of the thoracic vertebral column is seen as an arching of the back. It commonly accompanies back and abdominal pain. Vertebral malalignment from hemivertebrae or vertebral fracture or luxation are less frequent causes.

Lordosis is a downward curvature of the lumbar spine with exaggeration of the normal dip between the scapulae and the hip (dipped back, swayback). It can be due to hemivertebrae or vertebral fracture/luxation.

Torticollis, also known as 'wry neck', results from an involuntary unilateral contraction of the neck muscles causing torsion (twisting) of the neck. Head rotation (head tilt) with *laterocollis* (turn to one side), *retrocollis* (dorsiflexion), but usually not *antecollis* (ventroflexion) is seen. It may be secondary to an acquired cervical spine or spinal cord lesion, or muscle spasm from local or higher CNS origin.

SIGNALMENT

A 9-year-old male Maltese terrier.

CASE PRESENTING SIGNS

A twisted neck and inability to walk.

CASE HISTORY

The dog had tried to jump onto a chair to steal a hamburger but was restrained by a short lead and fell backwards, hitting the ground. The dog had remained immobile in a twisted position since the accident 2 hours prior to referral.

CLINICAL EXAMINATION

The dog was obtunded and in lateral recumbency with an almost 90° turn of his head and neck to the right. The left eye was visible and had a menace response, a normal direct PLR in a medium sized pupil. The left fundus was normal. There was no strabismus or spontaneous nystagmus in the left eye. The right eye was hidden by the abnormal posture (Fig. 55.1).

NEUROANATOMICAL DIAGNOSIS

The lesion was initially localized to the neck. Vestibular disease can cause a head tilt. Cerebral disease can cause a head turn. A combination of vertebral trauma, vestibular and cerebral lesions was a possibility given that trauma was the cause.

DIFFERENTIAL DIAGNOSIS

A fundamental question is: did the signs result from the fall or induce the fall? If the latter, then many other differential diagnoses needed to be considered. In this case, the dog was historically normal until directly after the trauma caused by restricted movement.

There was a good chance that C1–2 subluxation was present following neck trauma.

Small or toy breed dogs with a malformation of the dens or the supporting apical, alar, and transverse

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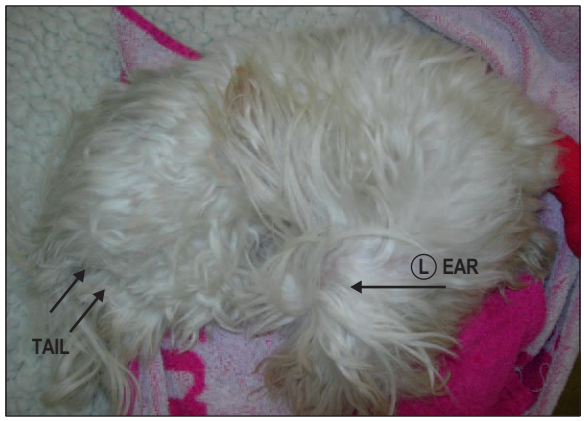


Figure 55.1 Torticollis. Left ear (arrow). Tail (double arrows).

ligaments are predisposed to developing C1–2 subluxation after minor trauma. Hypoplasia of the dens is the most common malformation but a simple absence of the dens does not guarantee subluxation is present.

Animals of any age may be affected but 80% of clinical cases appear in dogs under 2 years of age. Signs may be intermittent. The majority have gait deficits (84%) while 61% have neck pain. Paresis and ataxia are more common presentations than tetraplegia. Such instability is manifest by a widened space between the dorsal arches of C1 and C2. Slight ventroflexion of the neck makes this more pronounced at the substantial risk of worsening the spinal cord compression.

The wings of the atlas may obscure the location of the dens, so a slightly rotated position may overcome this. Open-mouth views cause dorsiflexion of the neck during positioning and should be avoided in animals with clinical signs.

Trauma to the cervical spine in an animal with a normal dens and associated ligaments usually results in a fracture of the dens from the body of the axis.

The prognosis is determined by the degree of neurological deficit and ranges from good to fair to guarded accordingly.

CASE WORK-UP

Conscious cervical radiographs were taken but were not diagnostically useful due to the torticollis.

The following day the dog was more alert. Non-contrast lateral and VD cervical radiographs taken under general anaesthesia were normal. The slightly flexed lateral view of the neck did not show C1–C2 subluxation (Fig. 55.2).

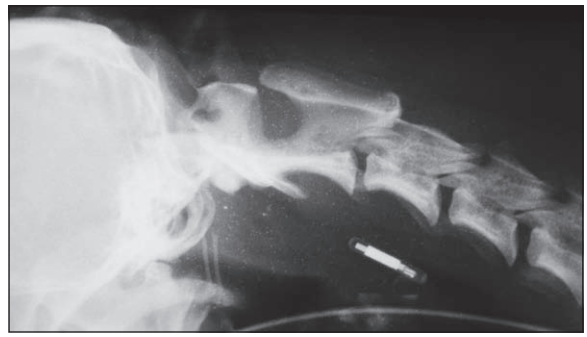


Figure 55.2 Flexed lateral view C1–C2.

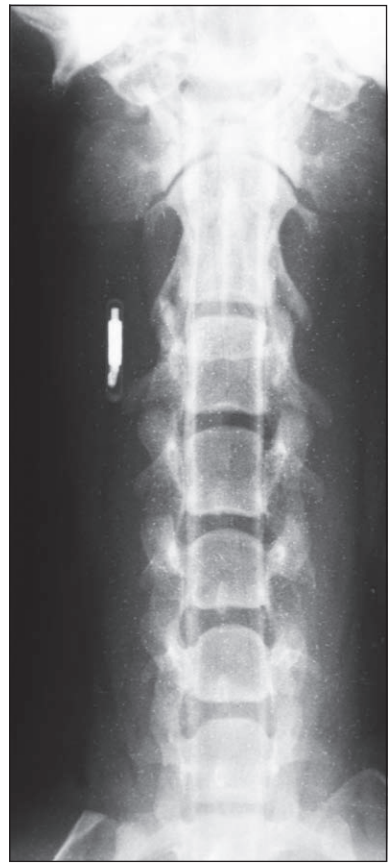


Figure 55.3 VD myelogram of the cervical cord.

CSF was collected at the cisterna magna without incident and a myelogram was done to look for traumatic extrusion of IV discs. No compression of the cervical cord was found on lateral, VD or oblique views (Fig. 55.3).

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The CSF was pink and opaque. CSF results showed RBC 3211/mm^3, WBC 47/mm^3 and TP 1.23 g/l. No RBC phagocytosis was found in the CSF, indicating this was a recent bleed into the subarachnoid space. Iatrogenic haemorrhage also increases the RBC count of the CSF. However, in most cases the blood appears as wisps of haemorrhage swirling within clear CSF or as frank haemorrhage. During collection Iatrogenic haemorrhage of 3211 RBC/mm^3 would not be expected to increase the total protein to this degree. For these reasons the abnormal CSF values were thought to be due to trauma.

DIAGNOSIS

Two days after the trauma torticollis to the right, absent placing in all limbs, absent right menace response, and spontaneous vertical nystagmus changing intermittently to horizontal nystagmus fast phase to the right were found. These findings indicated central vestibular disease. The direction of the horizontal nystagmus was opposite to that expected (paradoxical). The right menace deficit indicated that this paradoxical central vestibular lesion involved the cerebellum. A right cerebellopontine lesion was suspected, probably due to a developing oedema secondary to the head trauma.

MRI of the head was offered but declined.

FOLLOW-UP

Voluntary movement improved and the torticollis resolved within 8 days. Right-sided placing deficits were noted but these subsequently resolved.

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56 Collapse

INTRODUCTION

Sudden onset of *collapse* is a common presenting sign in small animal practice but not all such animals are afflicted with neurological disease. Physical examination, haematology and biochemistry are invaluable in distinguishing neurological lesions from other causes of weakness such as fever, sepsis or hypovolaemia. Always try to ascertain the level of mental awareness of the animal during and after the collapse. Systemically unwell animals are depressed (obtunded), which mimics cerebral disease. Syncopal animals lose consciousness but return to normal mentation almost immediately, unlike seizure activity, which usually leaves the animal more lethargic than normal for a variable period of time lasting minutes to hours. A severely depressed mentation (stupor) or a persistent lack of response to stimuli (coma) signal dysfunction of the brainstem or cerebrum. Stupor and coma occur in metabolic or structural encephalopathies. Signs of vestibular disease causing a loss of balance and subsequent collapse are usually strikingly obvious on examination. An animal that has acutely lost balance is mentally alert but usually pays less attention to the owner, as it struggles to regain its footing.

Collapse may be sustained (recumbency), or may occur episodically, with spontaneous resolution.

CLINICAL TIP

Never accept the owner's interpretation of the collapse. Always request a description.

Causes of collapse

1. Syncope (fainting, see Table 56.1) is a temporary loss of consciousness due to an inadequate supply of oxygen or glucose to the brain from:
   a. Reduced cardiac output
      - Decreased left ventricular output: cardiomyopathies, aortic stenosis or shock
      - Reduced left ventricular filling: cardiac tamponade or hypovolaemia
      - Reduced right heart function: pulmonary embolism or pulmonary hypertension
      - Arrhythmias: bradycardia (sick sinus syndrome or A-V block) or tachycardia (supraventricular or ventricular).
   b. Hypotension
      - Decreased systemic vascular resistance (fever or sepsis)
      - Reflex hypotension (vasovagal).
   c. Anoxia
      - Anaemia
      - Hypovolaemia
      - Poor perfusion from hyperviscosity.
2. Endocrine disorders
   - Hypoglycaemia
   - Tetany/hypocalcaemia
   - Pheochromocytoma
   - Hypoadrenocorticism
3. Neurological disorders
   - Seizures
   - Central or peripheral vestibular disease
   - Weakness
   - Increases in intracranial pressure
   - Narcolepsy/cataplexy
   - Dystonia, e.g. myotonia

The history and physical examination localized the cause of collapse in the following dogs of identical age and breed.

SIGNALMENT 1

A 7-year-old neutered male boxer dog.

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Table 56.1 Distinguishing seizures from syncope

	Syncope	Seizure
Associated activity	May or may not be associated with exercise, feeding, Valsalva manoeuvre	Often at rest
Duration of collapse	<1 minute	>1 minute
Convulsive movements	Rare	Common
Urine incontinence	Unusual	Common
Return of mental alertness	Within seconds	Usually quiet, lethargic for minutes to hours
Mucous membranes	Pale	Cyanosis or normal

CASE PRESENTING SIGNS

A 3-month history of episodic collapse and unresponsiveness.

CASE HISTORY

The dog had been diagnosed with pituitary-dependent hyperadrenocorticism (PDH) 3 months prior to referral when the collapsing episodes had also been reported. The collapsing episodes all appeared to be the same and consist of the dog sinking down to the floor into sternal recumbency, where it stayed motionless for a few minutes in a mentally unresponsive state. The dog then lifted its head and rose to a standing position. He was described as being a bit lethargic after the event and the dog rested for several hours before returning to normal. The events occur at any time of the day but had not occurred when out exercising. There was no history of exercise intolerance although the dog did collapse on two occasions when let into the garden to urinate and defecate. Treatment was trilostane for hyperadrenocorticism.

CLINICAL EXAMINATION

Examination was made difficult by the dog biting the neurologist. Once muzzled, the dog persisted in trying to bite which was apparently out of character. The dog was otherwise neurologically normal but was pot-bellied, had patchy alopecia and panted a lot.

NEUROANATOMICAL DIAGNOSIS

The events may be either syncope or seizure activity. Raised intracranial pressure can result in collapse. Seizures are often followed by a period of slightly altered mentation. The intermittent nature of the collapse with an altered mentation, along with a return to normal, was suggestive of cerebral involvement. It is common for animals to be fearful in a consulting room so the act of biting the vet should not immediately be ascribed to intracranial disease.

DIFFERENTIAL DIAGNOSIS

Intracranial disease causing collapse or seizures: pituitary tumour or glioma.

CASE WORK-UP

Haematology and biochemistry, cardiac ultrasound, blood pressure measurement and thoracic radiographs did not reveal a cause for the collapse. ACTH stimulation indicated good control of the PDH.

MRI of the brain found a contrast-enhancing smooth-bordered mass 16 mm tall in the region of the pituitary. The mass was hyperintense on T2 and isointense on T1 images (Figs 56.1 and 56.2).

DIAGNOSIS

Pituitary macroadenoma.

Pituitary tumours

ACTH-producing adenomas are the most common canine pituitary tumours. The presenting signs reflect ACTH secretion (hyperadrenocorticism) and the space-occupying effects of the mass (obtunded, altered behaviour, seizures, blindness, anisocoria and ataxia). The normal height of the canine pituitary gland is 2.1–6 mm. The height of a pituitary tumour, causing neurological signs or not, overlaps the normal range. The smaller the tumour, the better the prognosis following radiation therapy, although others say the height of the tumour does not impact on survival. The growth rate of canine pituitary tumours is unknown.

Pituitary tumours in cats are uncommon. Functional anterior pituitary tumours secrete excessive growth hormone or ACTH. Insulin-resistant diabetes is the most common presentation of acromegaly in cats. Polyuria, polydipsia, polyphagia and weight *gain* had been present

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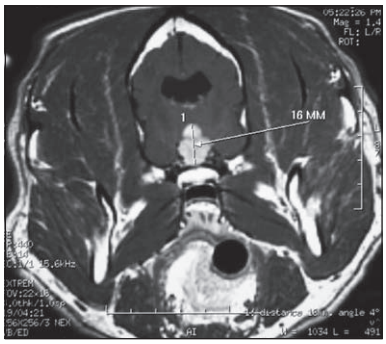


Figure 56.1 Transverse T1WI+C. Pituitary tumour with contrast-enhancement.

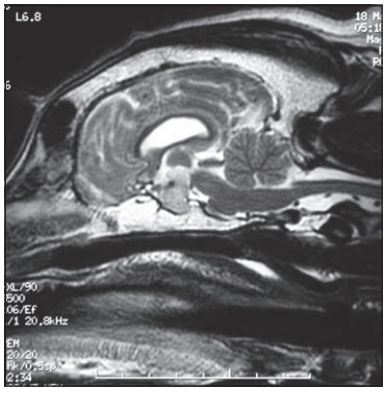


Figure 56.2 Sagittal T2WI. Pituitary tumour compressing the thalamus ventrally.

for 2–42 months prior to diagnosis in one study of 17 cats. The vast majority of acromegalic cats are middle-aged to older castrated males. CNS signs from a space-occupying macroadenoma occurred later and were infrequent in comparison. Lethargy, impaired vision, dilated pupils, reduced PLR, circling and vocalizing were reported. A plantigrade hindlimb stance occurred secondary to chronically inadequate glycaemic control.

Radiation therapy has had mixed success at reducing hormonal (GH) secretion. CNS signs improve during or soon after radiation therapy. Tumour height pre-treatment has been measured at 3.1–12.6 mm.

PROGNOSIS

Rapid enlargement of an ACTH-secreting pituitary tumour can occur following treatment for hyperadrenocorticism. The bilateral destruction of the adrenal glands removes the excess circulating cortisol which had provided a negative feedback to the hypothalamic production of corticotrophin-releasing hormone (CRH). The resultant increase in CRH stimulates the growth of the pituitary adenoma. The neurological signs and skin hyperpigmentation which follow expansion of the pituitary tumour in this manner are referred to as *Nelson's syndrome*.

TREATMENT

The owners' declined to treat the dog's brain tumour with radiation therapy. The dog was euthanized after it bit the owner the following month.

SIGNALMENT 2

A 7-year-old neutered male boxer dog.

CASE PRESENTING SIGNS

Episodic collapse.

CASE HISTORY

Three episodes of collapse had been witnessed. These occurred at seemingly random times of the day and had commenced 2 weeks prior to presentation. Neck pain was first reported a few days prior to referral. The dog was described as being otherwise normal.

CLINICAL EXAMINATION

Neck pain was detected on lateral neck flexion but the dog would still spontaneously move its neck in all directions. The dog was neurologically normal but a little quiet.

NEUROANATOMICAL LOCALIZATION

The cause of collapse could be:

1. Cardiac disease
2. Cerebral disease
3. Cervical cord disease
4. Systemic illness.

The neck pain and collapsing episodes both occurred in a similar timeframe so could result from the same underlying problem. Neck pain may be the first sign of intracranial disease. If this is a possibility, as judged from the

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history, then MRI should be performed rather than myelography.

DIFFERENTIAL DIAGNOSIS

- Intracranial haemorrhage
- Neoplasia.

CASE WORK-UP

The animal's general health was assessed with haematology, biochemistry and an ACTH stimulation test. These were all normal.

Cardiac work-up (thoracic radiographs, ECG and cardiac ultrasound) was also normal.

Cerebral disease was then considered the most likely localization due to the intermittent signs. Neck pain alone does not cause an animal to collapse. Cervical cord disease significant enough to result in collapse would also produce tetraparesis and ataxia.

The dog then collapsed three times in 1 day and became obtunded. Increased intracranial pressure from a space-occupying lesion was suspected. Dexamethasone 2 mg/kg was given IV and the dog's mentally state improved dramatically.

In the following few days prior to MRI the dog had a generalized tonic-clonic seizure confirming the suspicion of a cerebral lesion. Phenobarbitone and prednisolone were commenced. The dog remained mental bright and no further neck pain was noted.

DIAGNOSIS

MRI of the brain found an ovoid mass in the right parietal lobe. T1 hypointensity and T2/FLAIR hyperintensity suggested a fluid-like consistency. Contrast enhancement of the rim was present. Peri-lesional oedema was present which partially compressed the right lateral ventricle. A cystic glioma was suspected, based on the MRI appearance and the signalment of the dog (Fig. 56.3).

Surgical and radiation therapy were offered but declined.

TREATMENT

The dog was euthanized.

Postmortem examination found a soft gelatinous cystic mass within the cerebral parenchyma. Histopathology diagnosed a cystic oligodendroglioma.

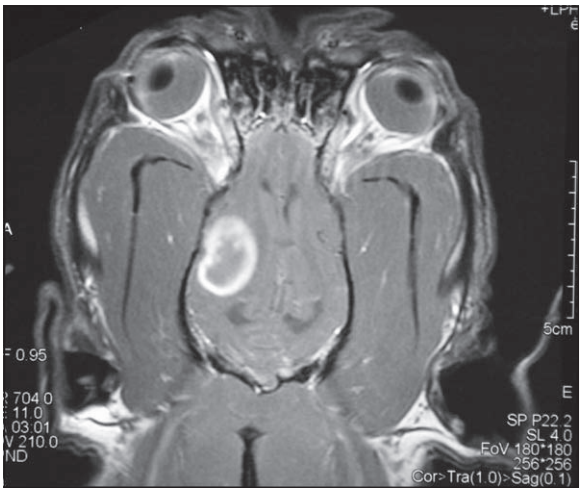


Figure 56.3 Dorsal T1WI+ contrast. Ring-enhancing glioma.

SIGNALMENT 3

A 7-year-old female boxer dog.

CASE PRESENTING SIGNS

Neck pain and collapse.

CASE HISTORY

The dog had a 6-week history of restlessness, intermittent yelping and holding the neck rigidly. There had been a recent onset of collapse and being unable to stand.

CLINICAL EXAMINATION

Clinical examinations showed a quiet but alert dog with non-ambulatory tetraparesis. The forelimbs were weaker then the hindlimbs, and the left-sided limbs were the weakest (Fig. 56.4). Proprioception was reduced in all limbs. Spinal reflexes were normal. Neck pain was found. CNN were normal.

NEUROANATOMICAL LOCALIZATION

C1–C5 spinal cord segments. Neck pain suggested involvement of the intervertebral disc, vertebrae, meninges or nerve roots or possibly cerebral disease.

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Figure 56.4 UMN tetraparesis, weaker in the forelimbs than the hind.

DIFFERENTIAL DIAGNOSIS

- IVDD
- Vertebral tumour
- Extradural or intradural spinal tumour or a space-occupying lesion within the cerebrum.

CASE WORK-UP

MRI showed a left ventrolateral extradural contrast-enhancing mass at the level of the C2–C3 disc space. The mass was isointense on T1WI. Location and contrast-enhancement suggested either a meningioma or a nerve root tumour.

Meningioma is the most common canine spinal cord tumour, 68% occurring in the cervical cord with most at the level of, or cranial to, the C3 vertebra. Signs are slowly progressive and include mild to moderate neck pain, paresis and ataxia. Neoplastic cells are not found in the CSF. The dorsal and transverse planes on MRI best show the gradual expansion of the subarachnoid space cranial and caudal to the lesion which is similar to the 'golf tee' sign on myelography. MRI contrast-enhancement and a broad-based dural margin (tapering to a dural tail, i.e. meningeal enhancement beyond the tumour) aid diagnosis. Surgical removal alone creates a prolonged remission from 4 months to 4 years.

DIAGNOSIS

Nerve root tumour.

FOLLOW-UP

The owners refused surgical exploration due to the poor prognosis for recovery.

Postmortem examination found an intradural malignant peripheral nerve sheath tumour with severe compression of the spinal cord and hydromyelia.

Spinal cord tumours

Like every other lesion in neurology, the location of a spinal cord tumour defines the presentation.

The onset of signs may be slowly progressive or of sudden onset. Progression is influenced by the rate of tumour growth but also by any secondary changes the tumour induces such as oedema or a pathological fracture. In summary, tumours can mimic other causes of spinal cord lesions and their presence should not be discounted by the apparent general health of the animal or its relative youth.

Classifying the tumour's relationship to the meninges by myelography or other imaging aids differential diagnosis.

- Extradural: peripheral to the dura mater. Compresses the cord. Most common. Often causes pain
- Intradural, extramedullary: within the subarachnoid space. Compresses the cord. Common. Can cause pain
- Intramedullary: within the spinal cord parenchyma. Least common. Generally not painful.

Clinical onset is usually fastest with intramedullary lesions and slowest with intradural lesions. Variation prevents prognosis being assigned solely by the speed of onset.

Extradural

1. Vertebral body tumours: primary tumours are common in the dog (osteo-, chondro-, fibro-, haemangiosarcoma and myeloma), but rare in the cat.
2. Metastatic neoplasia is unusual in dogs. Lymphosarcoma is the most common spinal tumour in cats.

Intradural

1. Nerve sheath tumours are slow-growing. They most commonly affect the brachial plexus and signs are of slow onset. If occurring in a nerve root supplying epaxial muscles signs begin acutely once the cord is compressed.

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2. Meningiomas are slow-growing. They often affect the cervical cord in dogs. They are rare in cats. Treatment is by surgical removal.
3. Nephroblastoma occurs at T10–L2 in young dogs aged 6–12 months. Affected animals are often German shepherd dogs or retrievers.

Intramedullary

1. Primary glial tumours are rare.
2. Metastatic tumours are rare: haemangiosarcoma, lymphosarcoma, malignant melanoma, mammary carcinoma in dogs.

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SECTION 9

BALANCE

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57 Balance – an introduction

Balance is maintained by equal input from the vestibular receptors found bilaterally in the inner ears (see Fig. 57.1). The receptors detect a change in head position and the impulses generated are transmitted into the brainstem via the vestibular branch of the vestibulocochlear nerve (CN VIII). Damage to the receptors, or the vestibular nerve before its entry into the medulla oblongata, results in a clinical syndrome known as *peripheral vestibular disease*.

The vestibular nerve enters the rostral medulla and terminates in the ipsilateral vestibular nuclei, with a few fibres directly entering the cerebellum via the caudal cerebellar peduncle. From the vestibular nuclei, the information is relayed to the:

1. *Cerebellum*, via the caudal cerebellar peduncle.
   Axons from the vestibular nuclei project to the flocculonodular lobe and the fastigial nucleus of the cerebellum. Lesions in this location give rise to the syndrome known as paradoxical vestibular disease.
2. *Spinal cord*, via the vestibulospinal tract.
   The vestibulospinal tract originates in the lateral vestibular nucleus and projects caudally to interneurons which connect with ventral horn cells throughout the entire spinal cord. The vestibulospinal tract facilitates ipsilateral extensors, and inhibits ipsilateral flexors and contralateral extensors with the net effect of providing ipsilateral extensor tone. A lesion in this tract or the lateral vestibular nucleus, *or the* ipsilateral vestibular receptor creates the opposite effect: the contralateral extensor tone increases, causing the animal to lean and fall to the side of the lesion.
3. *Cranial nerve nuclei III, IV, VI*, via the medial longitudinal fasciculus (MLF).
   The medial vestibular nucleus gives rise to the MLF, which projects rostrally in the brainstem to the cranial nerve nuclei III, IV, and VI. This coordinates eye movement with the position of the head and gives rise to the vestibulo-ocular reflex (VOR). Uneven input occurring in unilateral vestibular disease gives rise to spontaneous abnormal nystagmus. Bilateral destruction of the vestibular receptors from a congenital defect or a drug-induced degeneration removes the afferent stimulus and no nystagmus, physiological or pathological, can be generated. Destruction of the MLF within the brainstem is accompanied by coma. Absence of the VOR in this case signals a grave prognosis for recovery.

   The MLF also projects caudally to the cervical and cranial thoracic spinal cord and has the same function as the vestibulospinal tract.
4. *Reticular formation within the brainstem*.
   The vestibular nuclei project to the emetic centre within the brainstem which explains vomiting occurring with an acute loss of balance.
5. *Thalamus and then the sensory cortex,* via the medial lemniscus.
   Lesions of the thalamus and sensory cortex do not result in signs of vestibular disease.

   Vertigo is the hallucination of movement. It is not known if animals experience this sensation of dizziness.

Damage to the vestibular projections (pathways) within the CNS gives rise to a clinical syndrome called *central vestibular disease*.

PRESENTING SIGNS

The vestibular system provides the major sensory contribution to the orientation of the eyes, trunk and limbs relative to the position or movement of the head. Intact vision and normal proprioception from joints and skin enable the animal to compensate a little for vestibular dysfunction. Blindfolding an animal with vestibular disease worsens the loss of balance.

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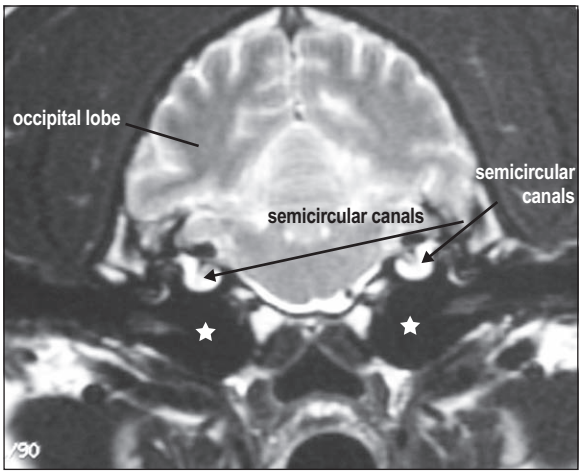


Figure 57.1 Brain T2WI. Semicircular canals visible on T2 as 'rubber duck' shaped outlines (arrows). Air-filled tympanic bullae (stars).

Vestibular dysfunction occurs unilaterally or asymmetrically in almost all cases of disease. The aim of examination is to determine which side is affected and, second, if the dysfunction is peripheral (outside the CNS) or central (within the CNS) in nature. Peripherally-located disease processes usually have a better prognosis than centrally located lesions.

Vestibular projections control eye movement and muscle tone. Dysfunction of the vestibular system, peripherally or centrally, results in alteration of these two aspects.

Ataxia

Unilateral loss of vestibular input reduces the ipsilateral extensor tone in the limbs. The animal therefore leans, falls, and may roll toward the side of the lesion. Lack of ipsilateral extensor tone in the axial muscles may cause the trunk to flex, with the concavity ipsilateral to the lesion. The combination of the two contributes to the animal circling towards the side of the lesion. The circles are typically of small radius and the gait is quite fast (*cf.* cerebral disease)

The rolling will continue unless the animal is physically restrained or sedated. The latter may be required in the acute phase of vestibular disease to prevent injury to the animal. The cage floor and walls should be well-padded. Pay particular care when handling a recumbent animal because they can suddenly turn, and bring the head down against the ground with some force. Enlist help when moving such an animal. Never ever leave such an animal unattended on a table (to state the obvious).

CLINICAL TIP

Animals with cerebral lesion propulsively walk, continuing even when an object is in the way. It is described as 'aimless', as to the outside observer there does not appear to be a stimulus or motivation for the action. The animal tends to circle towards the side of the cerebral lesion. No ataxia is noted. The circling can be of varying diameter and the gait appears unhurried.

Head tilt

The head tilts towards the side of the peripheral vestibular lesion but may paradoxically point to the contralateral side in some cases of central vestibular disease.

Nystagmus

The three semicircular canals contain receptors which detect accelerating or decelerating rotation of the head in any plane. Rotation of the head generates increased activity in one receptor and decreased activity in the paired contralateral receptor. This difference in activity creates uneven vestibular input between the left and right sides which generates (physiological) nystagmus when the head is rotated.

The vestibular neurons are tonically active at rest. A unilateral vestibular lesion results in uneven sensory input at rest and a spontaneous nystagmus occurs. Nystagmus is abnormal when present in a motionless head.

Nystagmus is an involuntary rhythmic to and fro movement of the eyes. The oscillation may be smooth and equal in either direction (pendular). This is commonly seen in the Siamese cat and lacks clinical importance.

A slow drift of the eyes alternating with a fast saccade correcting the direction of the gaze is known as jerk nystagmus. The quick (jerk) phase may be horizontal, rotatory, or vertical. The fast phase is easiest to notice and its direction is opposite to the side of the lesion in peripheral vestibular disease and in most cases of central vestibular disease.

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Strabismus

Extension of the neck by elevating the nose stimulates the vestibular receptors and impulses project to the extraocular muscles via the MLF. Normally the eyes remain centered in the palpebral fissures. A unilateral vestibular lesion can produce an ipsilateral ventral strabismus when the neck is dorsiflexed. This is called positional strabismus and indicates abnormal sensory input rather than an abnormal efferent supply to the extraocular muscles. (see page 26)

Central vestibular disease has the additional signs of ipsilateral hemiparesis and ipsilateral proprioceptive deficits due to damage of adjacent descending motor tracts and ascending proprioceptive tracts within the brainstem. If the vestibular pathways within the cerebellum are affected, an ipsilateral menace deficit, ipsilateral hypermetria, with preservation of strength and general proprioception may be seen.

Cerebellovestibular lesions can present with some or all of the signs of vestibular dysfunction. In addition, some of the signs may appear, paradoxically, on the opposite side to other signs of vestibular dysfunction. This is called paradoxical vestibular disease. To the uninitiated, this may appear as multifocal disease when in fact the lesion has a precise anatomic location. Paradoxical vestibular disease only occurs with cerebellar lesions and therefore is always classed as central vestibular disease. The lesion location in paradoxical vestibular disease cannot be judged from the direction of the head tilt, or from the direction of the spontaneous nystagmus; it is ipsilateral to any gait deficits present (hypermetria, CP deficits or hemiparesis).

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58 Loss of balance

INITIAL PRESENTATION

Leaning, falling, rolling, circling, ataxia, head tilt, nystagmus.

INTRODUCTION

Sudden onset of idiopathic peripheral vestibular disease is very common in the older dog. Vomiting may immediately precede the loss of balance. The animal leans and falls to one side and as a result is usually recumbent. The dog appears to struggle to remain upright and pays little attention to the owner while doing so. This dramatic presentation is often mistaken for a seizure, especially if the animal has also urinated. Examination in the acute phase always detects a head tilt and spontaneous jerk nystagmus. The head and eyebrows may also twitch in synchrony with the nystagmus. The animal is alert. Postural testing to determine proprioception and strength is almost impossible in the first 24–48 hr due to the extreme ataxia. Two people are required to support the larger dog on a non-slip surface while hopping and 'knuckling' is attempted. Neurological signs correspond to a unilateral peripheral vestibular lesion. The age and speed of onset are highly suggestive of the diagnosis.

Signs do not worsen with time. Improvement is slow and is expected within a few days. Ataxia resolves first, followed by cessation of the spontaneous nystagmus. Ambulation without assistance may take 2–3 weeks but the prognosis for return to normal function is excellent. A residual head tilt may persist for months. Recurrence has been reported. Treatment is purely supportive.

Idiopathic peripheral vestibular disease also occurs in cats of any age, not specifically the geriatric animal. Onset and improvement follow a similar pattern to that of the dog. In a group of 75 affected cats, approximately half presented with a head tilt, ataxia and no spontaneous nystagmus. Six cats had bilateral peripheral vestibular disease.

CLINICAL TIP

If the presentation has a progressive onset, or an unusual constellation of presenting signs, consider aetiologies other than idiopathic and recommend further investigation.

SIGNALMENT 1

An 8-year-old male Labrador dog.

CASE PRESENTING SIGNS

Head tilt and loss of balance.

CASE HISTORY

A left head tilt and balance loss began 1 month ago and had not improved. The dog was unsteady when jumping off furniture and had fallen over.

The dog was unable to localize the direction of sound. Mild otitis externa had been an intermittent problem over the years.

CLINICAL EXAMINATION

The dog was alert, ambulatory and had a normal gait. Hopping and proprioception were also normal. A spontaneous rotatory nystagmus with the fast phase to the right, slight positional ventral strabismus of the left eye and a left head tilt were present (Fig. 58.1). The left upper lip appeared to droop, owing to the left head tilt.

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Figure 58.1 Left head tilt (no head turn present).

Facial muscle function was normal bilaterally. Facial sensation was present bilaterally.

Table 58.1 Differentiation of central and peripheral vestibular disease

	Peripheral	Central
Nystagmus (type)	Horizontal Rotatory	Horizontal Rotatory Vertical May be positional May change direction
Nystagmus (direction)	Fast phase directed away from the side of the lesion Direction does not alter with a change in head position	Fast phase directed away from OR towards the side of the lesion Direction may alter with change in head position
Proprioceptive deficits	Absent	Ipsilateral to the lesion
Weakness	Absent	Ipsilateral to the lesion

NEUROANATOMICAL DIAGNOSIS

The lesion was localized to the left peripheral vestibular apparatus – the inner ear to affect both the cochlear duct and semicircular canals given the inability to localize sound as well as the loss of balance or the surface of the brainstem where the left CN VIII enters (Table 58.1).

DIFFERENTIAL DIAGNOSIS

- The lack of improvement after a month, the inability to localize sound and the continuing presence of spontaneous nystagmus ruled out idiopathic vestibular disease
- Neuroma
- Meningioma
- Otogenic abscess.

Ototoxicity was not considered given lack of exposure to drugs.

Ototoxicity affects both hearing and balance receptors. Aminoglycosides can damage the hair cells of the peripheral balance and hearing receptors. Streptomycin and gentamicin have their greatest effects on the vestibular receptor whereas kanamycin, tobramycin, and neomycin cause more damage to the auditory receptor. Furosemide can cause ototoxicity in the cat with high IV doses. Combining furosemide with aminoglycosides increases the risk of ototoxicity.

Metronidazole toxicity occurs within 2 weeks of commencing the antibiotic. Anorexia and intermittent vomiting for 24 hr may precede the development of a generalized ataxia and vertical positional nystagmus. The ataxia may be asymmetric and can progressively worsen. Seizures have been reported in addition to the central vestibular signs.

The dose rates implicated ranged from 67–129 mg/kg/day in the dog. Recovery usually occurs within 1–2 weeks and relies on removal of the drug and supportive care. Complete return to normal can take weeks to months. The recommended dose rate to avoid neurological signs is 30 mg/kg/day given in divided doses. Susceptibility varies between animals. Acute high doses or chronic moderate to high dose therapy may increase the risk.

CASE WORK-UP

MRI showed a contrast enhancing mass on the periphery of the brainstem at the entrance of the CN VIII.

BAER could have been used to investigate the possible hearing loss.

CSF analysis showed RBC 2016 µl; WBC 6 µl; TP 1.54 g/l. Cytology detected mostly mature neutrophils with occasional small/medium lymphocytes and monocytes. These results indicated necrosis and a space-occupying mass. No evidence of extension of infection from the middle ear was seen.

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DIAGNOSIS

The size of the mass suggests it has been slow growing as a mass of that size suddenly appearing in the cerebellopontine angle would produce greater deficits of balance and mentation.

The differentials based on location and response to contrast agent were meningioma or neuroma.

TREATMENT

- Radiation therapy
- Prednisolone.

CLINICAL TIP

Peripheral vestibular disease usually carries a better prognosis for recovery than central vestibular disease because of the anatomical site of the lesion.

SIGNALMENT 2

A 5-year-old male Cavalier King Charles spaniel.

CASE PRESENTING SIGNS

Head tilt, balance loss, and pain on opening the mouth.

CASE HISTORY

The dog had had a chronic bilateral otitis externa for months. He had become reluctant to fetch toys, and would not allow the owner to open his mouth. Hard dog biscuits were refused but the dog would eat soft canned food. A right head tilt and loss of balance had commenced during the week prior to referral.

CLINICAL EXAMINATION

The dog was alert and ambulatory with a right head tilt. Spontaneous rotatory nystagmus with a fast phase to the left was present. Right facial weakness and left facial paralysis were noted. The left lip appeared to have contracted. The dog struggled and refused to allow its mouth to be opened. Proprioception and hopping were normal in all limbs. Tear production was normal bilaterally. No neck pain was detected.

NEUROANATOMICAL DIAGNOSIS

- Right peripheral vestibular apparatus
- Right facial nerve

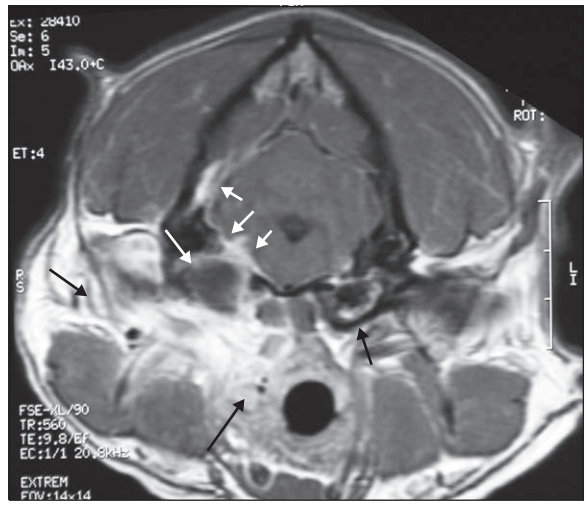


Figure 58.2 T1WI+Contrast-enhancement of tissue within skull and around TMJ.

- Left facial nerve chronic (muscle contraction had occurred).

DIFFERENTIAL DIAGNOSIS

Otitis media-interna on the right. Idiopathic facial paralysis can be unilateral or bilateral. The left facial paralysis may have resulted from left otitis media or could be idiopathic. Osteomyelitis of the tympanic bulla with inflammatory tissue around the temporomandibular joint (TMJ) may link the CNN deficits with the reluctance to open the mouth.

CASE WORK-UP

MRI showed severe inflammation around the bulla and TMJ extending into the calvarium (Fig. 58.2).

CSF analysis showed RBC 10 µl; WBC 1 µl; TP 0.29 g/l. Cytology: normal.

DIAGNOSIS

Right inner and middle ear otitis/osteomyelitis. Inflammation of the masticatory muscle secondary to ear disease.

Meningitis of the brainstem.

TREATMENT

Treatment consisted of surgical debridement of the bulla and antibiotics. Resolution of the neurological signs occurred.

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59 Truncal ataxia

INTRODUCTION

Inflammatory CNS disease is a very common cause of neurological signs in small animals, more so in the young to middle-aged. Peripheral leukocytes invade the brain, cord and meninges in response to tissue injury. In the majority of cases, the aetiology remains elusive and the disease is described in histopathological terms. Vasculitis may clinically mimic CNS inflammation.

Involvement of other systems (skin, lung, GIT, eye, haematopoietic system or blood vessels) and structures adjacent to the CNS (sinus, ear, nose, pharynx or intervertebral disc), the possibility of haematogenous spread (bite wounds, dental disease or endocarditis), predisposing conditions (immune-compromised, young, FeLV, FIV or CDV) and exposure (rabies, fungal, tick-borne pathogens or parasites) should be considered.

Clinical diagnosis of infectious disease requires:

- Clinical signs referable to the agent
- Serological evidence of exposure to the agent
- Exclusion of other causes
- Demonstration of the agent or a response to treatment.

A recent or active infection is suggested by:

- The presence of IgM
- An increasing antibody titre over 2–3 weeks
- Seroconversion from negative to positive.

Presenting signs

- Meningitis: spinal pain, blepharospasm
- Encephalitis. A variety of signs which may not reflect all the sites of disease which may be focal or multifocal. Balance loss is common
- Acute or chronic onset/progression
- Fever and neutrophilia are not consistently present.

The presentations of infectious and non-infectious meningoencephalitis are similar. Certain patterns can be recognized but it is a good idea to approach every case as potentially infectious. Be aware of the endemic parasites, vector-borne infections and fungal diseases in your area. This will increase the chances of a timely diagnosis.

SIGNALMENT 1

A 4-year-old neutered female West Highland white terrier.

CASE HISTORY

Hindlimb ataxia was first noted 6 months prior to referral. A high serum titre for *Neospora* was found (>1600) and the dog improved after receiving clindamycin for 3 weeks. The problem recurred 1 month after treatment ceased and a 10-day course of clindamycin improved the dog again. Two months later, hindlimb ataxia recurred again and the dog improved on 14 days of clindamycin. The *Neospora* serum titre at the time of referral was unchanged at >1600. The dog improved to normal on clindamycin after each recurrence of signs but had not improved with treatment for the current return of hindlimb ataxia.

CLINICAL EXAMINATION

The dog was alert with a truncal ataxia at rest and a hindlimb ataxia when walking. Hopping was normal on the left side but slow to initiate movement on the right. Placing was slow in the right fore and hind.

NEUROANATOMICAL LOCALIZATION

The lesion was localized to the cerebellum to account for the truncal ataxia, and either the right brainstem or cervical cord, or left cerebrum, to account for the placing deficits.

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CEREBELLAR TREMOR

At rest, the body may sway from side to side, forward to backward, up and down with irregular amplitude and frequency creating a 'jerky', 'coarse' movement known as truncal ataxia. A fine head tremor is commonly seen and worsens with movement, particularly at the end of a goal-directed action (intention tremor) such as reaching for food or sniffing an object. This can occur with lesions of the cerebellum or spinocerebellar tracts.

DIFFERENTIAL DIAGNOSIS

Inflammatory disease was suspected in this small breed young dog. Neosporosis was considered likely given the response to treatment and the high serology titre.

CASE WORK-UP

CSF analysis showed WBC 2 μl. Total protein was increased at 0.58 g/l. Cytology was normal with small lymphocytes and monocytes/macrophages.

MRI showed ventriculomegaly and atrophy of the cerebellum.

DIAGNOSIS

Increased total protein in the CSF is not specific for any particular pathology. The lack of pleocytosis does not rule out encephalitis.

Neosporosis was suspected.

FOLLOW-UP

The dog improved on clindamycin and trimethoprim/sulphonamides, which were continued for 3 months. At the check-up 1 month later, the owners reported that there was occasional hindlimb ataxia at home. Hopping and proprioception were normal on examination. No truncal ataxia was present. The antibiotics were stopped and the dog remained normal.

SIGNALMENT 2

A 7-year-old neutered male West Highland white terrier.

CASE PRESENTING SIGNS

Abnormal hindlimb gait and ataxia.

CASE HISTORY

Six to eight weeks prior to referral the dog had been unable to go up stairs, had collapsed when getting out of his bed and had swayed and 'looked drunk' when standing. He had fallen off the ramp leading up the steps. The gait looked more normal when the dog was moving fast than when staying still. The dog 'wobbled' when eating, and fell over when lifting a hindlimb to urinate.

Crepitus in both hips was found and radiographs revealed bilateral hip dysplasia. Glucosamine, chondroitin and carprofen were commenced but did not improve the dog's movement, which had worsened in the 4 weeks prior to referral.

CLINICAL EXAMINATION

The dog was quiet but alert and responsive to the environment. Cranial nerve examination was normal. Truncal ataxia was obvious with the dog lurching and falling to the right. There was hindlimb ataxia with a wide hindlimb stance at rest. There was also hindlimb paresis and reduced placing response in all limbs, which was worse on the right side. Spinal reflexes were increased in the hind and normal in the fore limbs. No spinal pain was found.

NEUROANATOMICAL LOCALIZATION

The lesion was localized to the cerebellum, to account for the truncal ataxia and falling, and the brainstem or cervical spinal cord to account for the proprioceptive deficits and weakness. Hindlimb ataxia and weakness may result from an additional lesion in the T3–L3 spinal cord segments.

DIFFERENTIAL DIAGNOSIS

A focal lesion within the cerebellum and adjacent brainstem with a progressive course could be neoplastic, such as a choroid plexus papilloma, meningioma, ependymoma, or inflammatory disease such as GME or *Neospora* infection. Globoid cell leukodystrophy can present with hindlimb ataxia and cerebellar signs in the West Highland white terrier but the onset of signs occurs in younger animals. Cerebellar abiotrophy also occurs in younger animals.

CASE WORK-UP

Haematology, biochemistry and urine analysis were normal.

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Table 59.1 Protozoal infections of the CNS

Organism	Neurological signs	Other signs	Dx	Tx
Toxoplasmosis (*T. gondii*)	CNS, muscle	Liver, lung, skeletal muscle, pancreas, eye	IgM >1 : 64 IgG ×4 increase Antibody and PCR on CSF	Clindamycin HCl Trimethoprim- sulphonamide Azithromycin
Canine neosporosis (*N. caninum*)	Neuromuscular, encephalitis	Heart, skin, lung, liver	Antibody; serology, CSF PCR	Trimethoprim- sulfadiazine, pyrimethamine, clindamycin
Canine leishmaniasis *(Leishmania* spp*)*	Uncommon Neck pain, weak	Skin, renal, joints, viscera, eye	Demonstrate amastigotes, serology, PCR	Antimony Allopurinol Amphotericin B

MRI showed meningeal enhancement.

CSF analysis showed neutrophil pleocytosis with an elevated total protein: RBC 0 µl (normal = 0), WBC 728/µl (normal <5), total protein 2.85 g/l (normal <0.25 g/l). Cytology showed degenerate neutrophils (80%), with small numbers of macrophages/monocytes and well differentiated small lymphocytes.

CSF culture (bacterial) was negative.

Toxoplasma IgG titre 100, indicating exposure to the organism, IgM titre <20.

Neospora caninum serology (IFAT) was positive at >1 : 1600.

DIAGNOSIS

Meningoencephalitis. Empirical treatment for protozoal infection was commenced with clindamycin 15 mg/kg BID and trimethoprim/sulphonamide 16 mg/kg PO BID. Prednisolone was used at 0.25 mg/kg BID in an attempt to limit inflammation and signs which gave a 2-day improvement before signs worsened.

FOLLOW-UP

At 5 days after presentation, the dog was unable to stand, was mentally dull, and turned to the right. At 9 days after presentation, hindlimb paralysis occurred.

The dog was euthanized.

Postmortem examination found protozoal tachyzoites in some of the inflammatory lesions within the cerebrum, pons, medulla and cerebellum. Neosporosis was diagnosed.

Protozoal infection

Toxoplasma gondii tissue cysts form in the CNS, muscle and viscera and may persist for the life of the host. Immune suppression causes a recrudescence. Toxoplasmosis is associated with canine distemper, FeLV, FIV, and FIP viral infections. Signs result from host cell death during replication and following inflammation. Serological diagnosis is difficult. A positive response to treatment aids diagnosis.

Neosporosis has only been reported in dogs. The young tend to have progressive paraparesis/polymyositis. The hindlimb extensor rigidity does not resolve. Older dogs have CNS signs which may be acute or chronic (Table 59.1). Prognosis is guarded but not hopeless.

Leishmania spp are transmitted by a sand fly vector. Neurological signs are rare and may result from vasculitis.

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60 Ventroflexion of the neck

INITIAL PRESENTATION

Head droop, head drop, 'dropped head', chin on or close to sternum

INTRODUCTION

The major antigravity support for the head is supplied by two groups of muscles. The major and minor rhomboids attach to the dorsal spinous processes of the C4–T4 vertebrae medially while the levator scapulae and serratus ventralis insert on the lateral processes of the cervical vertebrae and the ribs.

Cervical ventroflexion occurs in cats but not dogs as a sign of muscle weakness. The cat remains able to see despite the dramatic posture. Other signs such as generalized weakness, resting the head on the ground, and a stiff forelimb gait are noted. Winging of the scapulae in which the dorsal scapulae slightly protrude during weight-bearing is another sign of muscle weakness (Fig. 60.1).

Observation differentiates it from:

- A lowered head posture secondary to neck pain or when shifting the centre of gravity forward in paraparesis or severe hindlimb lameness.
- Hypnic jerk which is the sudden drop and recovery of head position when falling asleep.
- Tetraplegia resulting in lateral recumbancy and inability to lift the head from the floor.
- A generalized sudden loss of muscle tone in syncope and narcolepsy-cataplexy.

SIGNALMENT

A 6-year-old neutered female domestic shorthaired cat.

CASE PRESENTING SIGNS

Decreased activity.

CASE HISTORY

There was a 2–3 week history of waxing and waning changes described as a change in personality. The cat lay on top of the shed in the rain, would not mix with the other cats and did not run away. Food was prehended but then dropped. The cat's head 'fell forwards' and the animal dribbled saliva. No vomiting or regurgitation was reported.

CLINICAL EXAMINATION

Clinical examination showed an alert cat with slight neck ventroflexion and a slight right head tilt/turn. She walked without ataxia but her forelimbs became stiff and she stopped walking and lay down, sometimes also lowering her head. Hopping was normal. Proprioception was normal. Muscle tone was normal. Jaw tone and the blink reflex were both decreased. Spinal reflexes were normal. Schirmer Tear Test was 0 mm in the right eye.

NEUROANATOMICAL DIAGNOSIS

The lesion was localized to the LMN. Neuromuscular junction, myopathy, nerve roots were possible sites. The behavioural change was caused by the cat being too weak to move.

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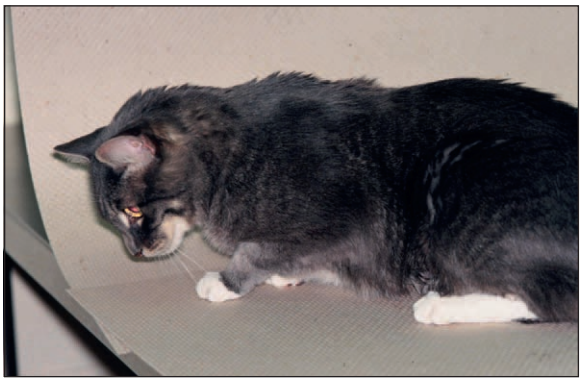


Figure 60.1 Ventroflexion in a hypokalaemic cat.

DIFFERENTIAL DIAGNOSIS

Neck ventroflexion has been reported in:

1. Hypokalemic polymyopathy
   - hereditary in the Burmese cat
   - acquired from diuretics, renal dysfunction, chronic vomiting/diarrhoea and vegetarian diets
   - serum potassium <3.5 mEq/l.
2. Hyperthyroidism
3. Myasthenia gravis
4. Myopathies: immune-mediated; hereditary, hypernatremic or inflammatory
5. Polyneuropathy
6. Ammonium chloride toxicity
7. Hypocalcaemia
8. Portosystemic encephalopathy
9. Thiamine deficiency
10. Organophosphate toxicity
11. Ethylene glycol intoxication.

CASE WORK-UP

Haematology and biochemistry, including electrolytes and muscle enzymes, were normal. An ACTH stimulation test was normal. Toxoplasma serology IgM <20; IgG <50, i.e. negative. Thoracic radiographs were normal. Acetylcholine receptor antibody level was negative. Tensilon test was negative. Motor NCVs were normal. EMG was normal. Muscle biopsies: no abnormalities found in either cranial tibial or triceps muscles

DIAGNOSIS

Diagnosis of autoimmune polymyositis is aided by muscle enzyme levels which commonly elevate after exercise and EMG which helps map the distribution of involvement for biopsy purposes. Multiple muscle biopsies are required to ensure a diagnostic sample is obtained. Joint taps and CSF analysis are also recommended to rule out other causes of pain and lameness. Thoracic radiographs check for megaoesophagus, secondary pneumonia, and neoplasia. Haematology, biochemistry and urine analysis are used to find clues of other organ involvement. Cats aged 6 months to 14 years may present with a sudden onset weakness secondary to an acquired idiopathic polymyositis. Neck ventroflexion occurs. Muscle pain may be present. Spontaneous recovery can occur in approximately 30% of affected cats.

The diagnosis in this cat was uncertain. Polyradiculoneuritis, antibody-negative acquired myasthenia gravis, or polymyositis were considered.

TREATMENT

Prednisolone 1–2 mg/kg PO q12 hr is given as treatment of autoimmune polymyositis once samples have been taken. Prognosis for recovery is good but like any immune-mediated disease, relapse during the reduction of the prednisolone dose is possible. Monitoring the fall in muscle enzyme levels and improvement in exercise ability suggests the best time to begin the tapering process.

The cat initially improved while cage rested but returned 2 weeks later in a weaker state. 0.25 mg/kg prednisolone BID was commenced and the cat was normal 8 weeks later.

FOLLOW-UP

The prednisolone dose was tapered and stopped and the cat remained normal. The improvement may have been spontaneous.

Head tremor (bobbing, nodding)

Head tremors may occur in isolation or in the context of a generalized tremor. The character may be jerky, disorganized, and worsen with goal-directed movement (intention). Regular movement in the horizontal plane is termed 'no-no'. Movement in the vertical plane is described as 'yes-yes'. The pathological significance of the direction is unknown in animals.

Animals with bilateral vestibular dysfunction have a low crouching stance, are ataxic, fall to either side and often move the head in wide sweeping arcs side to side. Concomitant signs indicate the origin of the head movements.

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Table 60.1 Direction of isolated benign head tremor in dogs

'No-no'	'Yes-yes'
Bulldog, boxer	Bulldog, boxer, Doberman pinscher, Shetland sheepdog

Head nodding in hypokalaemic cats reflects the muscle weakness which gives rise to neck ventroflexion. Serum hypokalaemia <3.2 mEq/l and increased serum creatine kinase aids diagnosis.

Head bobbing when walking is a compensatory movement of lameness to reduce weight-bearing on the affected limb.

Isolated intermittent head nodding has been reported in dogs (Table 60.1). The cause is not known but it appears to be benign. It does not interfere with normal activity and consciousness is unimpaired throughout. Therapeutic response to anticonvulsants is variable.

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SECTION 10

PALPATION

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61 Muscle tone – an introduction

Tone is the resistance of a muscle to stretch. It is generated by reflex circuits of muscle spindles and Golgi tendon organs projecting onto motor neurons. The motor neurons in turn receive input from the UMN.

There are conduction delays in the feedback loops regulating muscle length and this gives rise to a small oscillation called *physiologic tremor*. The tremor worsens with any change to the feedback loop or the UMN influences upon it.

If the motor nerve is damaged, the muscle loses tone and becomes flaccid. If inhibition to the motor neurons is removed, muscle tone increases (*hypertonia*, spasticity).

There are both inhibitory and facilitatory UMN pathways descending through the brainstem and cord to influence the LMN. Lesions of descending UMN pathways produce the clinical picture of 'release', i.e. removal of inhibition to the extensor muscles.

In humans, the loss of descending inhibition from the basal ganglia creates a rigid, unchanging hypertonia, whereas loss of other descending UMN inhibition results in a spastic, varying hypertonia. The terms rigid and spastic do not carry such localizing value in veterinary neurology.

Interneurons within the spinal cord are inhibitory to motor neurons limiting their duration, intensity and distribution of discharge. A sudden severe thoracolumbar spinal cord lesion in the dog can remove this inhibition to the forelimbs, resulting in the *Schiff–Sherrington syndrome* (Table 61.1).

Do not confuse this with a severe C1–C5 spinal cord lesion (Table 61.2).

CLINICAL TIP

Schiff–Sherrington syndrome signifies severe spinal cord dysfunction but does not imply its irreversibility. Prognosis is assessed as in other cases of paralysis.

Schiff–Sherrington syndrome usually disappears 10–14 days after onset to be replaced by the usual signs of T3–L3 paraplegia: normal to increased spinal reflexes and muscle tone.

The release of inhibitory neurotransmitters from spinal cord interneurons is prevented by the tetanus toxin tetanospasmin. Similar clinical signs of tetanic muscle spasm occur in strychnine poisoning which also acts on spinal interneurons.

Acute spinal lesions in vertebrates cause a period of spinal shock in which spinal reflexes caudal to the lesion are profoundly depressed resulting in areflexia and atonia. The duration is proportionate to the degree the gait is controlled by the cerebrum. Spinal shock may be present for 1–2 hours in the dog and cat. If areflexia is found in an acutely paralysed animal recheck reflexes frequently thereafter. Hyperreflexia and hypertonia may develop. Spinal shock is rarely encountered in small animal patients.

PRESENTING SIGNS

Tremor is an involuntary rhythmic oscillation of a body part. Oscillations are of large (coarse) or small (fine) amplitude.

Clonus is the involuntary alternate contraction and relaxation of skeletal muscle occurring in rapid succession. It is a sign of UMN dysfunction and is commonly seen during seizures.

Myoclonus is a sudden, brief involuntary movement of a body part from an abrupt muscle contraction. It is also described as quick, jerky and shock-like. Such movements may be rhythmic (implying repetition) or arrhythmic. Focal, multifocal or generalized myoclonus occurs either spontaneously, in reaction to a stimulus, or following voluntary action. The trigger for myoclonus originates in the CNS or PNS.

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Table 61.1 Signs of Schiff–Sherrington syndrome

Forelimbs	Hindlimbs
Rigid extension: hypertonia	Hypotonia
Normal reflexes	Normal reflexes
Voluntary movement present: stiff gait	Paraplegia
Normal proprioception	*
Pain perception present	Pain perception absent

*Proprioception testing is fruitless in a paralysed animal because the animal is unable to move its limb into the correct position. A spinal cord lesion producing paraplegia will also damage the ascending proprioceptive pathways. Lastly, *if* pain (sensory) perception is absent, proprioceptive testing will also be absent.

Table 61.2 Signs associated with a C1–5 cervical cord lesion

Forelimbs	Hindlimbs
Hypertonia	Hypertonia
Increased spinal reflexes	Increased spinal reflexes
Paralysis	Paralysis
Pain perception present	Pain perception present

Jerk: sudden reflex or involuntary movement.

Startle: myoclonus in response to an unexpected stimulus.

Hyperekplexia: a pathologically exaggerated startle response. Inherited syndromes are reported in the human associated with the inhibitory glycine receptor gene.

Twitch is the term for a brief contraction of skeletal muscle in response to a neuronal impulse.

Fasciculation is the involuntary, rapid, arrhythmic contraction of small numbers of muscle fibres within a muscle, insufficient to generate tension and move the joint. It is visible to an observer and may be induced by tapping a partially denervated muscle belly. The spontaneous discharge may arise from anywhere along the LMN. It occurs in diseases of nerve, muscle, or less commonly, spinal cord grey matter. It rarely occurs secondary to UMN pathology. It is a feature of thyrotoxicosis, tetany, anaesthetic muscle relaxants and acetylcholinesterase inhibitors.

Fibrillation is an electrophysiological hallmark of denervation produced by spontaneous contraction of a single muscle fibre. It is too small to be noticed on physical examination.

Myokymia is evocatively described as 'worms under the skin'. This involuntary, undulating, wave-like rippling movement within a muscle represents spontaneously discharging groups of motor units caused by motor nerve hyperexcitability. It is a rare clinical finding and occurs with other neurological signs.

Spasm is a sudden involuntary contraction of a muscle or group of muscles which may be associated with pain from nerve compression or meningitis. Cramp is another term for a painful spasm. It can occur spontaneously or be triggered by light touch.

Tonic: a contraction characterized by continuous tension (*cf*. clonic).

Tetany: intermittent tonic muscle contraction as occurs, for example in hypocalcaemia.

Tetanus: sustained muscle contraction, e.g. *C. tetani* intoxication (tetanus), strychnine poisoning.

Spastic, spasticity: abnormally high muscle tone which resists passive movement but which can be altered by the speed and degree of joint movement imposed. It may suddenly give way, like the closing of a clasp knife.

Rigid, rigidity: unchanging hypertonia with a constant resistance to flexion ('lead pipe rigidity').

Hypertonic, hypertonia: exaggeration of normal muscle tone manifest as resistance to passive movement. It is a sign of UMN pathology (release of inhibition).

Tic: A tic is an involuntary, abrupt, arrhythmic, repetitive movement of varying intensity involving discrete muscle groups. It may, confusingly, be referred to as a spasm.

CLINICAL TIP

Increased muscle tone (spasticity) is seen in the gait as stiffness.

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Stiffness is defined as a lack of flexibility, being incapable of bending and occurs with:

- Joint pathology restricting movement
- UMN spinal cord lesions increasing extensor muscle tone
- Muscle fibrosis restricting limb movement
- Continuous motor unit activity within a muscle.

Dyskinesia can be used as a general term to describe excessive involuntary movements. In humans it implies dysfunction of the basal ganglia. Repetitive episodic excessive involuntary movements involving the whole body or individual muscle groups occur.

This is a poorly defined area in veterinary neurology. Behavioural abnormalities, stereotypic actions, benign head nodding and action-induced dystonias are abnormal repetitive movements occurring in apparently conscious animals. Rare reports of writhing movements in animals exist.

The following descriptive terms are included here to aid communication with the internet-informed owner:

- *Athetosis*: slow, undulating, purposeless writhing movements most evident in the distal limbs
- *Ballism, ballismus*: rapid large amplitude, erratic, violent, flinging or flailing of limbs
- *Chorea*: a fidgety appearance due to random unpredictable brisk abrupt, or slower, flowing, involuntary movements that shift randomly from one part of the body to another. This is called *choreoathetosis* when combined with slow writhing movements.

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62 Tremor

INTRODUCTION

Tremor is an involuntary rhythmic oscillation of a body part. Oscillations are of large (coarse) or small (fine) amplitude. They may occur when the animal is recumbent, weight-bearing at rest or during voluntary movement. Tremors often worsen with stressful or exciting stimuli such as play and disappear with sleep. The description of a tremor aids diagnosis.

Shake, quiver and shiver are synonyms.

SIGNALMENT

A 2-year-old neutered female Bichon Frise dog.

CASE PRESENTING SIGNS

Generalized tremor.

CASE HISTORY

A sudden onset of generalized tremor which was present at rest and which worsened when the dog was excited or stressed. The dog was lethargic.

CLINICAL EXAMINATION

Clinical examination revealed a fine generalized tremor of the head, trunk and limbs but sparing the eyeballs. There was no intention tremor. The dog was ambulatory and had normal proprioception and hopping responses. Cranial nerve function was normal.

NEUROANATOMICAL LOCALIZATION

A generalized tremor syndrome was diagnosed. No evidence of cerebellar disease was present.

DIFFERENTIAL DIAGNOSIS

Causes of tremor

- Weakness
- Pain
- Hypothermia
- Metabolic derangement: hypoglycaemia, hypocalcaemia, pheochromocytoma or hyperthyroidism
- Intoxication:
  - metaldehyde: slug and snail bait
  - methylxanthines: chocolate, caffeine
  - macadamia nuts
  - mycotoxin: mouldy food
  - organophosphates, pyrethrins: flea treatments
- Dysmyelination or hypomyelination of the CNS.

CASE WORK-UP

Haematology and biochemistry were normal. MRI of the brain was normal. CSF was normal. *Neospora* and *Toxoplasma* titres were negative.

DIAGNOSIS

Idiopathic tremor syndrome was diagnosed in this case.

The condition is commonly referred to as:

- *Little*: <15 kg
- *White*: approximately 50% have a white coat colour
- *Shaker*: generalized fine tremor of the head and body, including the extraocular muscles
- *Dogs*: not reported in the cat.

The main presenting sign is a generalized tremor appearing with voluntary movement, worsening with excitement, stress or exercise and usually disappearing with

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sleep. A fine tremor of the eyeballs may occur in any direction in an irregular waveform (*cf*. vestibular dysfunction). The age of onset is usually less than 5 years.

CLINICAL TIP

Nystagmus is defined as an involuntary oscillation of the eyeballs in a jerk or pendular waveform. It is classified according to direction i.e. horizontal, vertical, rotatory.

Normal activity and vision are not impeded. Hypermetria, truncal ataxia and seizures occur infrequently. Signs may progress or remain static.

Investigations rule out other causes. Most cases respond to a combination of prednisolone and diazepam within a few days. If not, the diagnosis should be reconsidered or the doses increased. Relapse is possible but treatment remains effective for subsequent episodes.

A generalized tremor occurs in central axonopathy of Scottish terriers which develop severe ataxia and tremors at 10–12 weeks of age. Weakness was reported in one. It is thought to be progressive.

TREATMENT

1 mg/kg prednisolone BID and 0.2 mg/kg diazepam TID reduced the tremor and the dog became more active as a result. A month after commencing treatment the doses were reduced and removed over a 1-month period.

Tremor in the Great Dane dog has three important differential diagnoses. Historical and physical factors indicate the diagnosis in most cases.

1. *Inherited myopathy (central core) of Great Danes* presents as exercise intolerance, muscle wasting and exercise-induced tremors commencing at less than 1 year of age in both sexes (Fig. 62.1).
2. Cervical cord compression (*Wobbler syndrome*) produces proprioceptive deficits and ataxia.
3. *Orthostatic tremor syndrome* has been reported in Great Danes of either sex at 18 months of age. A constant tremor of all limbs and the trunk begins a few seconds after standing up and develops into a 'marching up and down' appearance caused by the constant shifting of weight from one limb to another. The tremors disappear in the swing phase of walking, when leaning against a support and when lying down. They worsen with increased weight-bearing such as in forelimbs when leaning forward to eat, and in hindlimbs when assuming a sitting position. Limb tremors were induced by pressing against the ventral surface of the paws to mimic weight-bearing in the recumbent dog. Proprioception and strength are normal. Intermittent facial tremors may occur. No underlying pathology could be found in the CNS or PNS. EMG performed in the conscious weight-bearing dog found continuous discharges of 13–16 Hz in both extensor and flexor muscles. Auscultation of the affected limbs detected a low-pitched repetitive throbbing/thumping sound, akin to a distant helicopter, which corresponded to the muscle tremors. Phenobarbitone had some success in reducing the severity of the tremors. Effective serum levels were similar to those used for seizure control.



Figure 62.1 Muscle atrophy in inherited myopathy (central core) of Great Danes.

Interesting fact: Humans with primary orthostatic tremor display a characteristic tremor of the patellae known as knee bobbing, juddering or quivering.

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63 Tetanus

INTRODUCTION

Tetanus is the disease produced by the *Clostridium tetani* toxin tetanospasmin within the CNS where it prevents inhibitory interneurons from releasing glycine and GABA. Uninhibited continuous muscular contraction results in either a generalized or focal form of the disease. The toxin load and possibly species susceptibility determines which will develop and the rate of progression of signs.

C. tetani is ubiquitous and survives in the environment for months to years as spores. Inoculation of tissue by a penetrating wound creates the anaerobic environment required for conversion to the toxin-producing vegetative form of *C. tetani*.

Stick injuries, teething, nail bed infections, bites, and penetrating wounds which quickly heal may go unnoticed by the owner. Snake bite, large burns, and iatrogenic causes such as inadequate sterilization of surgical equipment, poor wound management, IM injections and surgical contamination with ingesta have also been reported as risk factors.

SIGNALMENT

A 7-year-old neutered female English springer spaniel.

CASE PRESENTING SIGNS

Exophthalmos.

CASE HISTORY

Exophthalmos was initially noted, followed 2 days later by the ears appearing more upright and the nostrils flared. The dog seemed stiff, made odd noises when eating and drinking and made a different noise when panting. A claw had been torn 1 week before signs commenced.

CLINICAL EXAMINATION

Clinical signs typically commence 3–18 days after inoculation. Mild generalized tetanus commonly presents as a stiff gait and a rigid stance often described as a 'saw horse' posture. The tail may be erect or elevated. Skeletal muscle of the head is generally affected at the same time or soon after, and trismus ('lockjaw'), retracted lips, erect ears, enophthalmos and protrusion of the third eyelid are easily noticed (Fig. 63.1). Flickering of the third eyelid is produced by intermittent spasm of the retractor bulbi muscles which creates momentary enophthalmos and a passive protrusion of the third eyelid. Laryngeal spasm, dysphagia, constipation and urine retention are possible.

CLINICAL TIP

Animals do not smile but the expression *risus sardonicus* persists in veterinary terminology to describe the facial spasm of tetanus. Whether animals are sardonic or wry is open to owner interpretation.

Severe generalized tetanus results in recumbency and opisthotonus. All muscle groups are affected but extensor tone predominates. Ventilation may be impaired. Stimuli such as light, noise and touch can increase muscle spasticity further.

Focal tetanus presents as rigidity in the inoculated limb (Fig. 63.2). It may remain localized to that limb or spread to the contralateral limb and then to the CNS. Focal tetanus is more common in cats than dogs. Recognition of signs is difficult in the slowly progressive generalized form presenting as stiffness.

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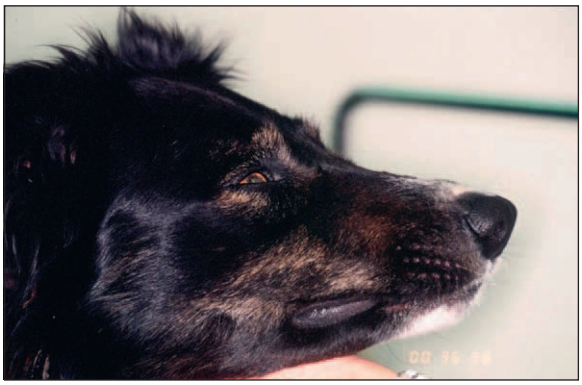


Figure 63.1 Generalized tetanus: retraction of ears and lips, small palpebral fissures.



Figure 63.2 Focal tetanus in the right hind limb.

Examination of this case detected a stiff gait, ears retracted higher on head, bilateral exophthalmos and less flaccid lips than expected for a spaniel. The tongue was prominent between the mandibles ventrally. There was harsh panting. The temperature was 40.3°C. She could open her mouth and could blink. There were no myotonic dimples. The gait did not improve with exercise. Strength and proprioception were normal. Muscle tone was increased in all limbs.

NEUROANATOMICAL DIAGNOSIS

A generalized increase in muscle tone gave rise to the clinical signs. Continuous motor unit activity within a muscle is generated by electrically unstable muscle membranes (e.g. myotonia) or may be neural in origin (e.g. tetanus).



Figure 63.3 Right exophthalmos secondary to a retrobulbar mass. More sclera is visible on the right.

Exophthalmos is a more forward positioning of the eyeball in the socket (*cf.* enophthalmos). It is a characteristic sign of orbital disease (Fig. 63.3). An increased volume within the retrobulbar space pushes the globe rostrally (Fig. 63.4). The axis of gaze may be deviated by retrobulbar masses and cause strabismus (see Fig. 63.5). Exaggerated exophthalmos is called *proptosis*, although the terms may be used synonymously.

Ocular asymmetries can give the impression of exophthalmos (pseudoproptosis)

1. Globe enlargement
   - Glaucoma
   - Maximally dilated and fixed pupils give the appearance of an enlarged globe, e.g. iris atrophy, CN III paralysis.
2. Asymmetry of the lid fissures
   - Widened palpebral fissure (CN VII): highly visible sclera
   - Narrowed palpebral fissure (ptosis) gives the impression of enophthalmos and hence contralateral exophthalmos
   - Macroblepharon: excessively long lids leading to exposure of the sclera.
3. Enophthalmos can create a false impression of contralateral proptosis.
4. Asymmetry of the bony orbits.

In this case the exophthalmos was caused by increased muscle tone pushing the eye forward.

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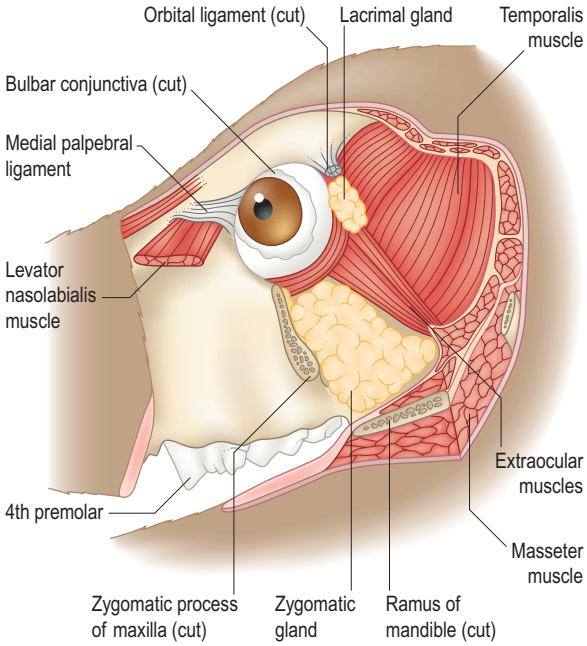


Figure 63.4 Anatomy of the retrobulbar area.



Figure 63.5 Greyhound hit by a (blue) car. Fractured frontal process of left frontal bone, causing left exophthalmos and a slight lateral strabismus.



Figure 63.6 Myotonia secondary to hyperadrenocorticism. Note excessive bruising and increased tone in hindlimbs creating a wide-based stance.

DIFFERENTIAL DIAGNOSIS

- Tetanus
- Myotonia
- Hypocalcaemia.

Muscle membrane excitability is altered by abnormal ion channel numbers or function. In *myotonia* this results in delayed muscle relaxation and manifests clinically as stiffness in the gait and stance, difficulty rising, muscle hypertrophy, dysphagia, regurgitation, a high-pitched bark, stridor, tongue protrusion and drooling. The stiff stilt-like movement complicates climbing stairs or stepping off a kerb and the animal may fall as a result. Muscle stiffness improves with exercise, and may worsen with a sudden change in posture. Opening the mouth may be difficult. It is not a painful condition (*cf.* tetanus). Clinical examination detects myotonic dimples which are depressions remaining in muscle bellies after percussion.

Inherited, congenital forms of myotonia exist in the Chow Chow and miniature Schnauzer breeds of dog. Domestic kittens may be affected. Signs worsen with cold temperatures. Acquired myotonia is a side-effect of iatrogenic and naturally-occurring hyperadrenocorticism. A stiff gait may be the presenting complaint rather than polyuria and polydipsia (Fig. 63.6).

CASE WORK-UP

Haematology and electrolytes were normal. Creatine kinase was slightly elevated at 700 U/l and probably resulted from increased muscular contraction. The lack of a prolonged history including exercise intolerance ruled out muscular dystrophies.

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DIAGNOSIS

A diagnosis of tetanus was based on clinical signs. Isolation of the bacteria from the wound is rarely successful. Measurement of serum antibody to tetanus toxin has been used. Electromyography could be helpful in diagnosis of focal tetanus.

TREATMENT

Treatment for tetanus was commenced.

1. Tetanus antitoxin to neutralize circulating toxin.
   - Tetanospasmin can spread to other motor neuron endplates via the bloodstream
   - No effect on toxin within nervous tissue
   - Equine anti-tetanus serum IM or IV
   - Test dose given SQ. If no adverse reaction after 15–30 minutes, the IV dose is given
   - Dose chosen empirically but does not exceed 20,000 U IV, IM, or SQ
   - Dose is based on the amount of toxin to be neutralized rather than the patient's body weight.
2. Antibiotics to eliminate *C. tetani* and prevent more toxin formation.
   - Metronidazole 10–15 mg/kg IV q8 hr
   - For 2 weeks or until clinically recovered.
3. Wound debridement to remove the anaerobic environment.
   - Eliminate necrotic tissue.
4. Sedatives to reduce sensitivity to external stimuli and lessen muscle spasms.
   - Acepromazine 0.1–0.2 mg/kg IM q6 hr
   - Chlorpromazine 0.5 mg/kg IV q8 hr
   - Diazepam 0.5–1.0 mg/kg IV as needed.
5. Adequate nutrition, bladder and bowel evacuation, prevention of pressure sores.
   - Continuous muscle contraction raises body temperature and uses calories
   - Sphincter hypertonus may prevent urine expression. Consider catheterization
   - Limit handling by synchronizing treatments of the patient
   - Avoid noise and movement.

This case was treated with metronidazole 200 mg TID and diazepam 5 mg TID PO for 2 weeks.

PROGNOSIS

Prognosis depends on the extent and severity of the spasticity. Respiratory failure may occur within 5 days. Improvement may be noticed within 1 week. Signs can persist for months. Return to normal is possible.

FOLLOW-UP

The dog improved and was normal within 2 months.

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64 Hypocalcaemia

INTRODUCTION

Clinical signs of hypocalcaemia reflect calcium's importance to the electrical stability of excitable tissue. Spasms, cramp and tetany are commonly observed. Handling and excitement typically induce or worsen the signs. Onset is sudden and dramatic and may follow a period of non-specific illness comprising lameness, stiffness, pain, nervousness, panting, lethargy, weakness, inappetance and fever. Total serum calcium may not reflect the low ionized calcium and the latter should always be specifically checked. The neuromuscular signs may be episodic despite a persistent hypocalcaemia. Diagnosis: ionized serum calcium <1.0 mmol/l.

The muscle spasms are often painful (cramp). Rigid abdominal muscles and pain induced by palpation of the spinal epaxial muscles may be confused with spinal pain from IVDD, discospondylitis, etc. Pancreatitis may be an equally erroneous conclusion.

A stiff, stilted hunched gait, with fasciculation and trembling occurs (Fig. 64.1).

Ear and facial twitching may occur. Elevated third eyelids are reported in the cat. Intense facial rubbing occurs in cats and is seen in over 50% of dogs. Intense biting or licking of the paws is another manifestation of what may be a paraesthesia. Humans report numbness and tingling of the fingers, feet and the perioral area.

Seizures are commonly reported, and witnessed at the veterinary practice, while those caused by idiopathic epilepsy or intracranial lesions have usually resolved before the animal is presented.

Muffled heart sounds, weak pulses and paroxysmal tachyarrhythmias are commonly detected on physical examination.

SIGNALMENT

A 5-year-old neutered female terrier dog.

CASE PRESENTING SIGNS

Neck pain.

CASE HISTORY

The dog had been quiet and depressed for months and was inappetant. Vomiting had been intermittent. Painful episodes had occurred in the week prior to presentation. NSAID administration had not improved the dog's condition. The dog had been referred for treatment of suspected IUD disease.

CLINICAL EXAMINATION

The dog was depressed and lethargic. Examination found shaking, trembling, a stiff neck, tetraparesis, lateral recumbancy and increased muscle tone in all limbs. Meleana was present. Heart rate and body temperature were both low.

NEUROANATOMICAL LOCALIZATION

Neck pain and stiffness suggested meningitis. A generalized stiffness and tremor may have been a response to spinal pain. Bradycardia, vomiting, melena and weakness suggested a systemic involvement.

DIFFERENTIAL DIAGNOSIS

- Hypocalcaemia
- Tetanus
- Meningitis
- Discospondylitis.

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Figure 64.1 Hypocalcaemic muscle pain. Arched back, low neck carriage, wide hindlimb stance.

CASE WORK-UP

Bleeding and clotting times were normal.

Hypocalcaemia and hypokalaemia were found on biochemistry.

DIAGNOSIS

Common causes for hypocalcaemia:

- Primary hypoparathyroidism, which is life-long.
- Post-parturient eclampsia (puerperal tetany), which is transient.
- Post-thyroidectomy in cats, which is also transient.

In this case, the parathyroid hormone (PTH) level was 18.0 pg/ml (10–60) and the total calcium level was 1.28 mmol/l (2.3–3.0).

The diagnosis was hypocalcaemia secondary to primary hypoparathyroidism.

TREATMENT

Treatment is dictated by the severity of the signs but as tetany is a common presentation, prompt use of IV calcium gluconate is indicated in most cases.

Emergency: tetany

- Intravenous 10% calcium gluconate 0.5–1.5 ml/kg (or 5–15 mg/kg) given slowly over 10–30 minutes
- Dose and frequency of use is determined by recurrence of the signs
- May be used as an infusion or as bolus. Do not add to bicarbonate-containing solutions
- Discontinue if bradycardia occurs
- Hyperthermia will resolve once muscle contractions cease
- Limit stimulation of the patient.

Stabilization phase: hypocalcaemia with mild persistent or intermittent signs

- 10% calcium gluconate diluted 1:1 in saline and given SQ q6–8 hr
- Maintain serum calcium levels just below normal for 2 days before reducing the SQ calcium gluconate dose. Withdraw after another 2–3 days of satisfactory serum calcium levels
- Commence oral calcium carbonate: dog 1–4 g/day. Cat 0.5–1 g/day
- Commence oral Vitamin D: 1,25-dihydroxyvitamin D (Calcitriol) has an onset of action of 1–4 days, and a half-life of less than 24 hours. It does not require activation in the kidney.

Long-term prevention of return of clinical signs

- Oral calcium carbonate tablets: may be able to taper and stop after several months
- Vitamin D is the mainstay of treatment. It stimulates GI absorption and bone resorption of calcium.

Monitoring is crucial for successful management. Hypercalcaemia must be avoided. The animal should be hospitalized during stabilization. Check the ionized calcium level at least monthly in the stabilized pet.

In this case IV calcium gluconate followed by oral vitamin D and oral calcium gluconate was used to control the clinical signs. The calcium gluconate was eventually stopped and the dog was maintained on oral vitamin D.

Yelping and head shaking commenced when the ionized calcium dropped from 1.04 to 0.81 mmol/l (N = 1.12–1.4). This resolved with an increase in the vitamin D dose.

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65 Trismus

INITIAL PRESENTATION

This condition is characterized by an inability to open the mouth or catch balls and carry sticks. Swelling or atrophy of masticatory muscles, a stiff jaw, swollen head and pain on opening the mouth may be seen.

INTRODUCTION

Trismus is a forced closure of the mouth. It may be referred to as 'lockjaw'.

It results from increased muscle tone:

- Seizures
- Tetanus, strychnine poisoning
- Hypocalcaemic tetany
- Myotonia.

or masticatory muscle contracture:

- Inflammation, e.g. masticatory muscle myositis (MMM)
- Fibrosis, e.g. end-stage MMM, muscular dystrophy.

Pain on opening the mouth mimics trismus but represents an unwillingness to open the mouth rather than a physical inability to do so. Pain on opening the mouth suggests a lesion of the retrobulbar area, temporomandibular joint, ear canal, parotid gland, pharynx or masticatory muscles. Any structure that moves or is compressed during jaw movement has the potential to cause pain. An animal with oral pain may attempt prehension but drop the object.

Source of pain

1. Dental: trauma, foreign bodies
2. Masticatory muscle: spasm; inflammation- immune-mediated; polymyositis
3. TMJ: craniomandibular osteopathy
4. Retrobulbar: abscess, inflammation
5. Tympanic bulla: osteomyelitis; tumour (e.g. multilobular osteochondrosarcoma)
6. Caudal pharyngeal lesion
7. Neck pain: usually able to differentiate from jaw pain by keeping the neck still while manipulating the jaws.

SIGNALMENT

A 3-year-old neutered male German shepherd dog.

CASE PRESENTING SIGNS

Unable to prehend objects.

CASE HISTORY

The owners thought the dog's head had looked bony for a few months. Three weeks prior to referral the dog was unable to prehend balls and sticks. The dog had a good appetite but could not chew hard, dry food. There was no exercise intolerance. The referring vet was unable to open the mouth fully.

CLINICAL EXAMINATION

Clinical examination showed profound atrophy of the masseter and temporalis muscles bilaterally. The mouth could not be fully opened (trismus) (Fig. 65.1). Bilateral enophthalmos was present. Sensation to the face was intact.

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Figure 65.1 Trismus under general anaesthesia.

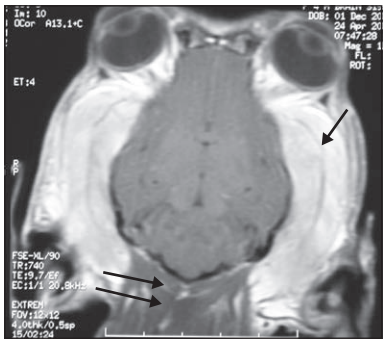


Figure 65.2 T1WI+contrast. MMM presenting as exophthalmos, trismus, and increased head size. Enhancement of muscle (arrow). Normal muscle (double arrows).

NEUROANATOMICAL DIAGNOSIS

Restriction of jaw movement indicated increased muscle tone or muscle inflammation, neoplasia, fibrosis or an abnormality of the temporomandibular joint (TMJ).

DIFFERENTIAL DIAGNOSIS

- Masticatory muscle myositis (MMM)
- TMJ disease with secondary (disuse) atrophy of the masticatory muscles.

Masticatory muscle applies pressure to the retrobulbar area. Its atrophy can lead to enophthalmos. Masticatory muscle myositis is also known as eosinophilic myositis due to the presence of peripheral eosinophilia. It is common in adult German shepherd dogs. The degree of elevation of CK does not reflect the severity of the clinical signs.

Increased jaw tone from tetanus occurs in the generalized form of the disease. Focal tetanus affects limbs.

CASE WORK-UP

- Haematology: mild inflammatory reaction with moderate eosinophilia
- Muscle enzymes: AST = 62 IU/l (N), ALT = 47 IU/l (N), CK = 469 IU/l (raised)
- General anaesthesia was performed. The mouth could not be opened more than 3 cm
- TMJ radiographs were normal
- Electromyography (EMG) found small areas of spontaneous fibrillation in the temporalis muscle
- A muscle biopsy was taken from the left temporalis muscle for diagnosis/prognosis. The biopsy found marked endomysial lymphohistiocytic inflammation, with some necrotic muscle fibres. Prominent fibrosis was seen
- The serum 2M antibody level was positive at 1:500 (>1:100 is consistent with a diagnosis of MMM) (Fig. 65.2).

DIAGNOSIS

Masticatory muscle myositis.

TREATMENT

Prednisolone 1 mg/kg BID PO was commenced.

FOLLOW-UP

Recheck appointment 3 weeks later found a lethargic, depressed polyuric and polydipsic dog with more masticatory muscle loss. The eyes were very enophthalmic and the third eyelids covered the pupils obscuring vision causing the dog to bump into objects. Trismus had not improved. The jaws could not be separated more than 2–3 cm. The owner could not tolerate the polyuria and had the dog euthanized.

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66 Bilateral atrophy of the masticatory muscles

INTRODUCTION

Atrophy of the masticatory muscles (MM) allows the skull shape to be more obvious and owners often report a 'lump' which is a newly palpable or visible occipital protuberance. Loss of MM leads to enophthalmos. Depending on the cause of the MM atrophy, a dropped jaw or trismus may be seen.

SIGNALMENT

An 8-year-old male golden retriever dog.

CASE PRESENTING SIGNS

Generalized muscle loss, especially on the head.

CASE HISTORY

Diarrhoea, fever and weight loss were noted 1 month prior to referral. The diarrhoea stopped but inappetance, lethargy, fever and weight loss continued. Muscle loss was generalized but particularly prominent on the head which prompted referral. Blood results were abnormal. They showed a normocytic normochromic non-regenerative anaemia, thrombocytopenia, neutropenia, relative lymphocytosis with pleomorphic lymphocytes and occasional atypical large lymphocytes. Alb 23 g/l, ALKP 306 μ/l, ALT 53 μ/l, AST 130 μ/l, CK 259 μ/l, pre-prandial bile acids 25.

Prednisolone 25 mg BID was then given for 2 weeks as the referring vet was concerned about MMM despite a low 2M antibody of <1 : 100 (>1 : 100 is consistent with a diagnosis of MMM). The muscle loss did not improve and the dog was referred for EMG.

CLINICAL EXAMINATION

Clinical examination revealed a mentally depressed dog with petechia of the conjunctiva and retina bilaterally. Mucous membranes were pale. The temperature was 39.7°C. There was a generalized muscle loss, including masticatory muscles bilaterally (Fig. 66.1). Jaw tone was good. The dog had normal facial muscle function. Sensation to the face was present. No neurological deficits were found.

NEUROANATOMICAL LOCALIZATION

Bilateral atrophy of the masticatory muscles results from:

Bilateral trigeminal neuropathy

The masticatory muscles (temporalis, masseter, pterygoideus, digastricus muscles) are innervated by the mandibular branch of the trigeminal nerve (CN V). Damage to a motor nerve typically results in the clinical signs of atrophy, flaccidity and weakness of the innervated muscle. Bilateral involvement of the mandibular branch of the trigeminal nerve causes the jaw to partially or fully hang open. The jaw can easily be closed by the examiner but drops open again when unsupported. The sensory branch of the mandibular nerve is unaffected in idiopathic mandibular neuropathy.

Myopathy: focal or generalized

The masticatory muscles contain the 2M fibre type which can lead to a specific attack by the immune system resulting in masticatory muscle myositis. Generalized myopathies can also affect the 2M fibres. Trismus is more common than a dropped jaw.

Weight loss

Cachexia from systemic illness.

In this case the lesion was localized to the hemopoietic system. The weight loss was thought to be the cause of the masticatory muscle loss as no jaw weakness or sensory deficits to the face were found. A dropped jaw and bilateral atrophy of the masticatory muscles

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Figure 66.1 Profound atrophy of the masticatory muscles, pale gums and sclera.

resulting from aleukemic myelomonocytic leukaemia has been reported in the dog.

DIFFERENTIAL DIAGNOSIS

Neoplasia.

CASE WORK-UP

APTT 108 seconds, PT 12 seconds. Repeated blood samples showed severe non-regenerative anaemia PCV 0.17, Hb 5.6, RBC 2.32, leukocytosis WBC 131.7, absent neutrophils 0×10^9, lymphocytosis 27.66×10^9, thrombocytopaenia 17 and high numbers of undifferentiated cells 104×10^9.

Leukaemia was diagnosed.

OUTCOME

The dog collapsed and died within a few hours of arrival.

CLINICAL TIPS

- Do not ignore other body systems.
- Do not perform tests if you plan to ignore the results.

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67 Unilateral atrophy of the masticatory muscle

INITIAL PRESENTATION

Asymmetry of head.

SIGNALMENT

A 6-year-old neutered female Labrador retriever dog.

CASE PRESENTING SIGNS

Skin wounds to the right face, altered facial symmetry.

CASE HISTORY

The dog was initially presented for a right upper lip wound and abrasions to the right zygomatic area. One week later the right face suddenly 'caved in'. The dog wasn't catching thrown items as skilfully as usual.

CLINICAL EXAMINATION

Clinical examination showed atrophy of the right temporalis and masseter muscles. The jaw could be voluntarily closed by the dog. Sensation to the right side of the face (canthus, cornea, nostril, upper and lower lips) was absent. Right enophthalmos was present but no anisocoria was noted in either a light or darkened room. Schirmer Tear Test (STT) was 0 mm/minute on the right and 9 mm/minute on the left. The wound on the right upper lip pierced the lip and the hole corresponded to the position of the canine tooth. Observation found normal voluntary movement of the eyelids, nostrils and ears bilaterally. The rest of the neurological examination was normal (Fig. 67.1).

NEUROANATOMICAL LOCALIZATION

The lesion was localized to the right trigeminal nerve (CN V), affecting all three branches as indicated by lack of sensation to the eye (ophthalmic branch), eyelids (ophthalmic/maxillary branches), upper lip (maxillary branch), lower lip (mandibular branch), and atrophy of masticatory muscles (mandibular branch) on the right side.

Enophthalmos can result from masticatory muscle atrophy or from Horner's syndrome. No miosis was present in this dog so the former is the most likely cause.

Dry eye (STT = 0 mm/minute) can result from trigeminal nerve lesions.

When facial muscles are paralysed (CN VII) the upper lip is flaccid and can become trapped between teeth when eating. The dog typically drops food rather than bite through its own lip. The lip wound in this dog has resulted from lack of sensation to the face and resulting self-trauma.

Lack of proprioceptive deficits or paresis in the limbs indicates that the CN V lesion may be peripheral to the brainstem, rather than affecting the nucleus within the brainstem. Compression of the brainstem may be present but clinical signs were not detected.

DIFFERENTIAL DIAGNOSIS

- Nerve sheath tumour of the trigeminal nerve
- Lymphoma
- Meningioma.

Unilateral masticatory muscle atrophy often results from a trigeminal neuropathy which is generally neoplastic.

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Figure 67.1 Facial wounds (arrow), atrophy of the right temporalis and masseter muscles.

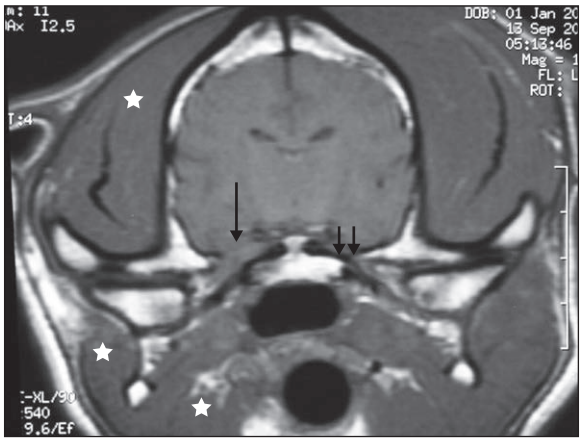


Figure 67.2 T1WI enlarged right trigeminal nerve (arrow). Oval foramen smaller on the normal side (double arrows). Atrophy of masticatory muscles (stars).

Trigeminal nerve sheath tumours present as unilateral trigeminal motor and sensory dysfunction. Affected animals may rub the ipsilateral face early or late in the development of signs. The tumours are slow-growing and eventually compress the brainstem at which time euthanasia is performed. Surgical removal has been performed by few veterinary neurosurgeons. Potential morbidity from a surgical attempt should be weighed against prolonged survival times (0.5–2 years) in the untreated dog. A lobulated or solitary extra-axial mass with displacement of brainstem is found on MRI. The oval foramen may be enlarged.

CASE WORK-UP

MRI of the head showed enlargement and contrast enhancement of the right trigeminal nerve. Atrophy of the ipsilateral masticatory muscles was evident. Denervation atrophy results in fatty replacement and increased signal intensity on T1 and T2 (Fig. 67.2).

CSF was normal.

DIAGNOSIS

Nerve sheath tumour of the trigeminal nerve.

TREATMENT

Surgical resection of the non-healing penetrating wound of the top lip was discussed. As the wound was not infected, the owner elected to continue bathing it in salt water. The eye was lubricated frequently to treat dry eye and help prevent corneal ulceration. When evidence of brainstem compression begins, such signs may improve on prednisolone therapy.

FOLLOW-UP

The dog was euthanized 13 months later because of worsening ataxia. Histopathology confirmed the diagnosis of a trigeminal nerve sheath tumour.

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SECTION 11

PAIN

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68 Pain – an introduction

Almost all pet owners equate pain with a poor quality of life. It is important to recognize the presence of pain in a disease process and to put it into perspective for the owner by discussing definitive and palliative treatment and the duration and severity of pain expected.

The sensation of pain is a subjective interpretation of a noxious stimulus which is transmitted to the cerebral cortex via sensory nerves, dorsal spinal roots, spinal tracts and thalamic nuclei (Fig. 68.1).

The presence of pain in an animal is inferred by the animal's altered behaviour, posture or gait.

PRESENTING SIGNS

- Altered behavioural patterns: patients may hide, become aggressive, reclusive or lethargic, 'act like an old dog', become restless or agitated and have a decreased appetite
- Vocalizes: spontaneously or triggered by certain movements
- Looks or bites at the source of pain
- Increased sympathetic drive: increased heart rate, dilated pupils
- Reduced movement: decreased activity, will not greet the owner, stays in bed, will not jump into a car, lame, does not shake body or head fully, decreased grooming
- Altered posture: shifts position to limit weight-bearing, restricts head movement, arches back, lowers head, guards or becomes tense/rigid.

CLINICAL TIP

Cats purr even when in pain.

Humans can describe the quality of pain they experience and this gives rise to a lot of jargon.

It is difficult to judge what an animal is experiencing when it scratches, chews, self-mutilates, or twitches the skin. The assumption is that sensation is present but altered and possibly unpleasant. Once a causative lesion has been found in such an animal the human experience of that lesion is assigned, rightly or wrongly, to the animal. This is how we presume animals experience paraesthesias. In this case using jargon can help in finding the correct treatment.

DEFINITIONS

Noxious: Potentially damaging to tissue.

Paraesthesia: Spontaneous abnormal sensation, 'tingling, prickling, crawling'. Not pleasant but not painful.

Dysaesthesia: An elicited or spontaneously occurring unpleasant, abnormal sensation, often with a 'burning or electrical' quality.

Allodynia: Pain evoked by light mechanical stimuli (touch or light pressure) which normally do not elicit pain.

Hyperalgesia: An exaggerated perception of pain evoked by a noxious stimulus.

Hyperpathia: An unpleasant sensation. 'Burning' pain provoked by noxious or non-noxious stimuli. The pain spreads beyond the site of the stimulus and may intensify with repeated stimulation and may continue after the stimulus has ceased. The sensory threshold required to elicit pain increases (*cf*. allodynia).

Hypoalgesia: Diminished pain perception in response to a normally painful stimulus.

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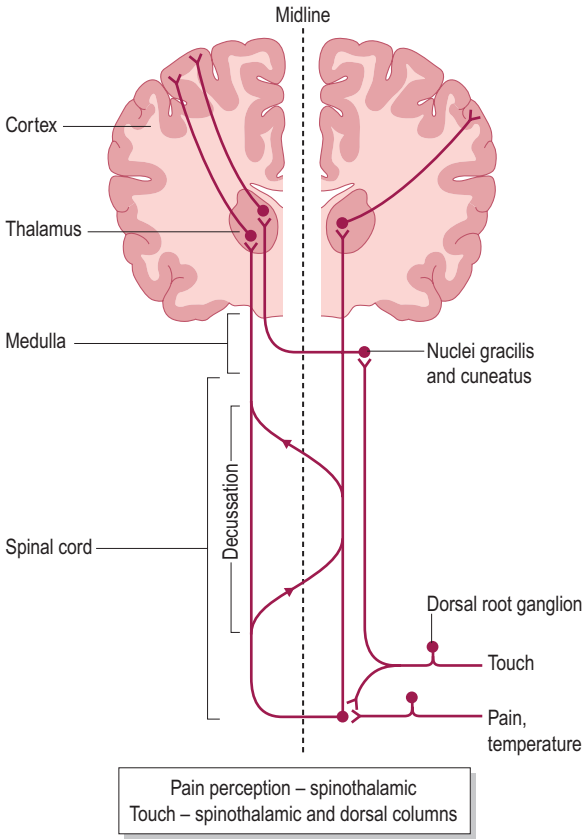


Figure 68.1 Anatomic pathways for pain perception.

Analgesia: Complete loss of pain sensation.

Hypoaesthesia (hypaesthesia): Decreased sensory perception in any modality. Frequently used as a synonym for hypoalgesia.

Anaesthesia: Total sensory loss. May be regional or global (general anaesthesia). May only involve some modalities (e.g. temperature, pain: analgesia).

EXAMINATION

Stimuli not normally associated with a painful reaction are used in examination, e.g. flexing and extending the neck, pressure over the lumbosacral joint, passive range of motion of a joint. When such a stimulus causes pain it is noted on the examination form as a significant finding.

Equally, when a noxious stimulus is applied to a body part, the lack of a painful response from the animal is significant. Bear in mind that pain is a subjective response and individual variation exists. Firm pressure applied by a haemostat on the nail bed should elicit a painful response even in stoical animals. No normal conscious animal tolerates the sharp end of a pen poked into its nasal mucosa.

There is a trend to try and separate the presence of deep pain from the presence of superficial pain. Pinching the skin vs. traumatizing the periosteum are the two tests reported. The pragmatic approach is to find out if an animal feels any kind of pain or not. If it does, then the prognosis for recovery from the causative lesion is generally held to be better than if it was absent.

DIFFERENTIATING REFLEX ACTION FROM A PAIN RESPONSE

There is a mistaken belief that the cutaneous trunci reflex is synonymous with the presence of superficial pain. This is an excellent moment to remember that a spinal REFLEX involves only a sensory arc, a motor arc, and the spinal synapses joining them. There is NO cerebral interpretation required. Pain, on the other hand, is a subjective interpretation. It requires mental capacity. It depends on a behavioural response for an observer to know that it exists.

A noxious stimulus enters the spinal cord via the dorsal root. This sensory impulse can enter a reflex arc and produce an automatic, reflex action, e.g. the withdrawal or flexor reflex. This is an excellent evolutionary development as it removes the limb from noxious stimuli without the organism having to mentally register what has taken place. It is fast, automatic, and involuntary (Fig. 68.2).

The noxious sensory impulse can also travel up the spinal cord to the cerebrum and be registered as a painful experience.

This is where the confusion for many lies. The reflex occurs almost simultaneously with the painful response in a normal animal, but what is forgotten is that these two results are different phenomena. They are usually simultaneous in the normal animal, but in neurological disease it is vital to recognize each action as depending on different anatomical pathways.

CLINICAL TIP

The withdrawal (flexor) reflex is not the same as the ability to perceive pain.

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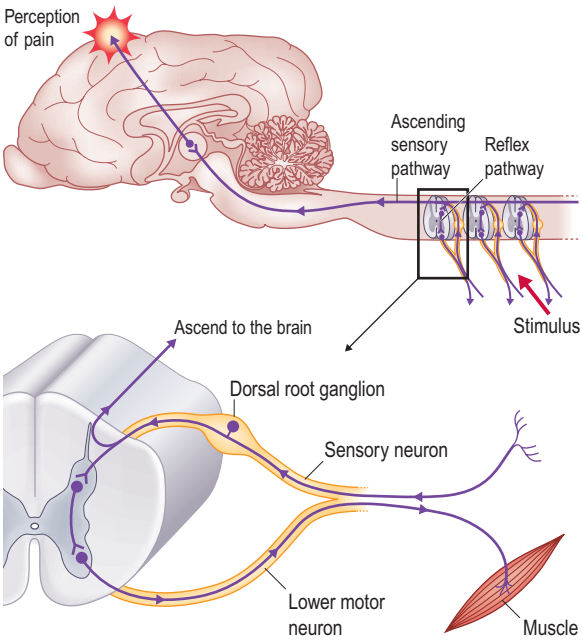


Figure 68.2 Pain perception and the reflex arc.

It is very important to make a 'big song-and-dance' over this point of neuroanatomy. If a dachshund has pain perception in its paralysed limb(s) it means that some ascending information can pass though the damaged area of spinal cord and thus the prognosis for return of motor function is favourable. If the vet mistakes a reflex action for the perception of pain, then they will be unable to offer an accurate prognosis and they will be unable to monitor the progression of disease. The animal's treatment suffers as a result.

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69 Neck pain

INITIAL PRESENTATION

Presenting signs may include: low head carriage, not lowering the head to eat from the floor, shaking, trembling, neck spasms which may create a ventral head bob, episodic circling (small radius) and moving the neck and trunk as one unit.

INTRODUCTION

Common causes of neck pain include intervertebral disc disease, meningitis and syringomyelia. All may present acutely. Disc disease is more likely than the others to give rise to ataxia and paresis (see Fig. 69.1). The pain may appear episodic in disc disease and syringomyelia. Meningitis may be multi-focal, and while signs may appear worse on some days compared with others, they are never entirely absent. Haemorrhage into the subarachnoid space causes spinal pain.

Focal lesions of the canine cerebrum may present as neck pain. In the absence of other signs of cerebral disease such as seizures and behaviour change, this can create a diagnostic dilemma. Consider the age and breed of dog and comb the history for clues which may suggest either cerebral disease or cervical spine or cord lesions. Consider imaging techniques which will encompass all realistic differential diagnoses. Remember that hypoventilation increases ICP which could result in herniation followed rapidly by death during anaesthesia of patients with brain tumours.

SIGNALMENT

A 6-year-old male Great Dane dog.

CASE PRESENTING SIGNS

Neck pain.

CASE HISTORY

The dog had surgery for gastric dilation a few weeks prior to developing neck pain.

CLINICAL EXAMINATION

The dog was unable to rise from lateral recumbancy without assistance but when standing was ambulatory with a shortened fore and hindlimb stride length. No neurological deficits were present. Neck pain was severe.

NEUROANATOMICAL LOCALIZATION

Vertebrae. Meninges. Nerve roots.

DIFFERENTIAL DIAGNOSIS

- Discospondylitis
- Meningitis
- Neoplasia of the spine.

Discospondylitis is the infection of the disc space and contiguous vertebral endplates. Bacteria gain access via the bloodstream (dental disease, endocarditis, urogenital, skin or respiratory tract infection) or by extension (migrating grass awn, direct trauma – pharyngeal stick injury). Fungal infection has been reported.

Young to middle-aged large-to-giant breed male dogs are affected most commonly. Some 18-28% of

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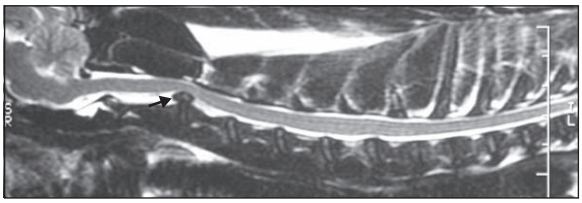


Figure 69.1 T2WI. C2–3 IVD extrusion in a miniature dachshund with neck pain (arrow).

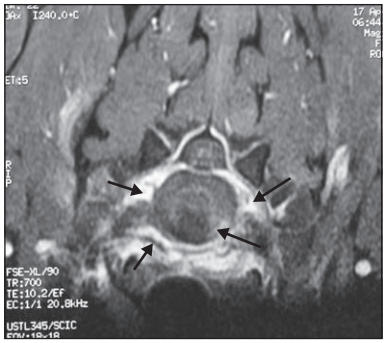


Figure 69.2 T1WI+C. Discospondylitis.

dogs have multiple areas affected, with the highest incidence in the lumbar region, especially L7–S1, followed by the thoracic and cervical spines. Extension to the vertebral canal and meninges is rare. Discospondylitis has a similar presentation in cats.

The chief presenting sign is spinal pain, with or without fever. A stiff, stilted hindlimb gait and hindlimb lameness give the appearance of hindlimb weakness. Signs occur suddenly or develop over several weeks. Chronic discospondylitis may develop if the spinal pain responds to NSAID and no further diagnostic investigations are performed.

CASE WORK-UP

MRI showed contrast-enhancement of endplates, nerve roots, longus colli muscles (arrows) at C7–T1 (Fig. 69.2).

The MRI appearance of discospondylitis includes decreased T1 and increased T2 signal intensity of the intervertebral disc, adjacent vertebral endplates, and soft tissue ventral to the vertebral bodies, with non-uniform contrast enhancement of the affected tissue. Such changes precede the development of radiographic abnormalities.

Table 69.1 Radiographic diagnosis of discospondylitis

Early changes	Chronic changes
Irregular lysis of vertebral endplates	Widening of the IVD space
Collapse of IVD space	Bony proliferation – vertebral osteophytes
	Fusion of adjacent bodies
	Endplate sclerosis

CLINICAL TIP

Collect CSF while the animal is under anaesthesia for imaging. Sterile meningitis also presents as spinal pain with or without fever in young larger breed dogs.

DIAGNOSIS

Discospondylitis. Recent gastric surgery may have caused a bacteraemia.

Diagnosis of discospondylitis can be difficult if the radiographic signs are not present and MRI is unavailable. Consider the following:

1. Haematology: may be normal
2. Radiograph the whole spine looking for endplate lysis
3. Blood and urine cultures are 30–75% positive
4. Consider CSF tap and joint taps to rule out meningitis and polyarthritis
5. Consider MRI if radiographs are normal: MRI is sensitive and specific for spinal inflammation (Table 69.1).

CLINICAL TIP

Pain may be present 2–6 weeks before the radiographic changes appear.

Discospondylitis should be differentiated from:

1. Neoplasia
   - Less likely to affect a disc space
   - Can affect dorsal laminae.
2. Collapsed vertebral fracture
   - Loss of continuity of vertebral borders
   - Separation/avulsion of articular facets
   - Change is size and shape of vertebral body or arch.
3. Spondylosis
   - No destruction of endplates
   - Smooth proliferation.

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TREATMENT

In the absence of a positive blood or urine culture, commence treatment for a suspected *Staphylococcus* spp infection with a first generation cephalosporin. Analgesics are required initially for at least 1–2 weeks. Resolution of signs requires 6–14 weeks of antibiotic therapy which is continued for a further 2 weeks after resolution.

Re-radiograph 0.5–3 months after the diagnosis. Radiographic improvement appears as vertebral fusion and resolution of bony lysis. MRI improvement appears as resolution of contrast-enhancement. A failure to respond may be due to a fungal organism, a foreign body, *Brucella canis* infection, soft tissue abscess or resistant bacteria. Consider surgical biopsies if the animal is not responding to treatment.

In this case, opiates, cephalexin and metronidazole did not improve the dog within 24 hours and the owner requested the animal be euthanized rather than continue.

PROGNOSIS

- Bacteria – good
- Resistant bacteria – guarded
- Fungal – bad.

The empirical time frame for the use of antibiotics does not apply to each case but this does not imply a poor prognosis.

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70 Meningitis of large breed dogs

INITIAL PRESENTATION

Acute onset of neck pain, fever, lethargy, reluctance to walk, and a stiff gait.

SIGNALMENT

A 1-year, 3-month-old male golden retriever dog.

CASE PRESENTING SIGNS

Pain, fever, inappetance and a shuffling gait.

CASE HISTORY

The dog became quiet, anorexic and appeared to be stiff 3 days prior to referral. The dog struggled to squat to defecate, looked like it was 'walking on egg shells' and was unable to turn in tight circles. The dog shifted its weight from limb to limb and was panting. There was a fever of 40.8°C. Meloxicam and buprenorphine were given.

The dog had been lame, depressed and febrile for a few days, 5 months earlier. This had resolved with meloxicam. Six weeks prior to referral, the dog was lethargic, inappetant and febrile for 1 week. Thoracolumbar pain was detected at this time. Meloxicam was given.

CLINICAL EXAMINATION

Clinical examinations showed a quiet, mentally responsive dog which could walk unaided but which had an arched back, shuffling hindlimb gait with small steps taken but no ataxia. There was a lowered head position, normal proprioception and normal CNN tests. Spinal reflexes were normal. No joint swelling was found. The temperature was 39.3°C.

NEUROANATOMICAL LOCALIZATION

Pain leading to altered posture and gait.

Sources of pain in neurological disease

- Nerve: nerve root entrapment/compression, transection, inflammation or infiltration
- Meninges: meningitis, vasculitis or stretch/tension
- Destruction of central spinal cord: syrinx formation
- IV disc: rupture of annulus fibrosis or discospondylitis
- Periosteum: vertebral tumours or osteomyelitis
- Spinal ligaments: dorsal longitudinal ligament or joint capsules/synovium of the articular facets
- Muscle: sublumbar muscle/retroperitoneal space disease, migrating plant awns or muscle spasm
- Skin: infected dermoid cyst
- Thalamus syndrome (rare).

DIFFERENTIAL DIAGNOSIS

- Meningitis and/or polyarthropathy
- Discospondylitis
- Haemorrhage into the subarachnoid space
- Vasculitis
- Fever resulting from inflammatory, neoplastic or immune-mediated tissue destruction
- Polyarthritis-meningitis syndrome is relatively common and affected animals present with pyrexia, stiffness and neck pain.

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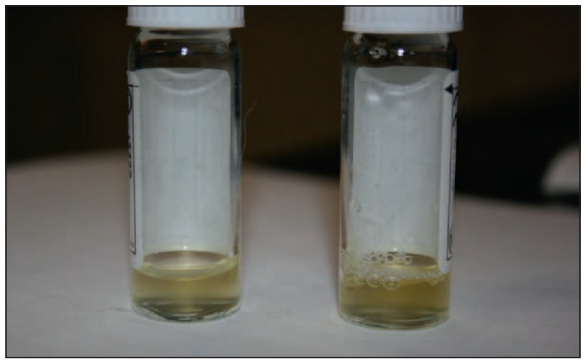


Figure 70.1 Xanthochromic CSF.

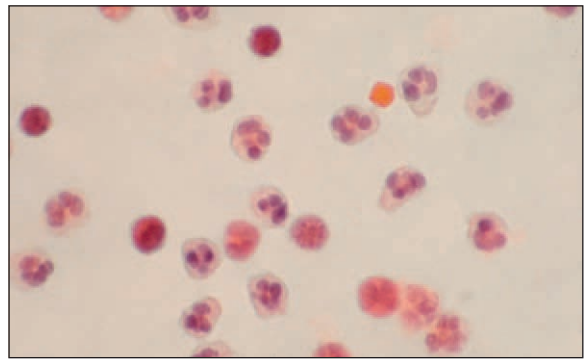


Figure 70.2 Neutrophilic CSF.

CASE WORK-UP

Haematology showed a mature neutrophilia. Biochemistry was normal.

Lumbar CSF had a xanthochromic appearance, which can be due to high cell counts or a high total protein level (Fig. 70.1). The cells consisted of mainly mature neutrophils (73%) with a few lymphocytes (5%) and monocytes (22%).

Cisterna magna CSF looked cloudy and contained: RBC 13 µl, WBC 858 µl. Total protein 0.72 g/l. Cells consisted of mature neutrophils (81%) with some lymphocytes (3%) and monocytes (16%) (Fig. 70.2).

Joint taps: cellularity was negligible.

Non-contrast radiographs of the spine in lateral and VD views were normal.

Serology, urine culture and blood culture were all cancelled as the financial limit for the client had been exceeded.

DIAGNOSIS

The diagnosis in this case was made, as in many cases, on the basis of probability. It is most likely that all three, episodes of pain and fever were caused by the same disease. Discospondylitis should have shown radiographic changes during this period. As no lesions were seen, this diagnosis was eliminated. CSF was then collected and the gross appearance was highly suggestive of meningitis as the results proved.

Neutrophilic meningitis signals tissue damage and is a common finding in steroid-responsive meningitis of larger breed dogs. Bacterial meningitis, on the other hand, is rare in small animals. *Tick-borne intracellular bacterial infection* does not occur in the UK, as yet (Table 70.1).

Ehrlichiosis presentation is similar to Rocky Mountain spotted fever (RMSF) in the acute phase. Thrombocytopenia (with defective platelet function), leukopenia and anaemia follow. Weight-loss can be severe. Haemorrhage has a varied pattern of occurrence. Neurological signs of meningoencephalitis, lameness, and pain occur. Dramatic improvement occurs within 1–2 days of commencing treatment. Long-term sub-clinical infection can cause disease years later.

Rickettsia induce endothelial damage and vasculitis. Diffuse pain suggestive of meningitis, polyarthritis, or polymyositis may be the first sign of disease. Systemic signs are various and include fever, anorexia, vomiting and diarrhoea as well as haemorrhage and oedema. Clinical improvement is seen within 24 hours of commencing antibiotics.

Concurrent infection with *Ehrlichia*, *Rickettsia*, *Babesia* and *Bartonella* spp occurs. Screen for all likely pathogens and choose a broad spectrum treatment.

The diagnosis in this case was steroid responsive meningitis-arteritis (SRMA).

SRMA is a common immune-mediated disease and should be suspected in the young, adult, medium-to-large breed dogs with an acute onset of neck pain, fever, lethargy, a reluctance to walk and a stiff gait.

CSF analysis shows non-degenerate neutrophilic pleocytosis with normal to raised total protein levels. There may be a peripheral leukocytosis, with or without a left shift. High CSF and serum IgA concentrations are found.

Signs may wax and wane if left untreated but such patients are at risk of developing neurological deficits

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Table 70.1 Tick-borne infection of the CNS

Organism	Neurological signs	Other signs	Dx	Tx
Canine ehrlichiosis (*Ehrlichia* spp)	CNS/meningeal inflammation or haemorrhage	Thrombocytopenia Leukopenia Anaemia Polyarthritis Vasculitis	Morulae Antibodies PCR	Tetracycline Doxycycline
Canine RMSF (*R. rickettsii*)	Meninges, brain, spinal cord	Thrombocytopenia Leukopenia then leukocytosis Mild anaemia Vasculitis	Indirect IFA ≥×4 increase, 2–3 weeks apart PCR	Tetracycline Doxycycline Enrofloxacin
Canine bartonellosis (*Bartonella* spp)	Meningoencephalitis, meningomyelitis	Lame, bone pain, heart, skin, eye, joints Thrombocytopenia Anaemia	Antibodies PCR	Fluoroquinolone Azithromycin Doxycycline

(spinal or multifocal). There is no improvement with antibiotics. A dramatic improvement, often within 24 hours, is seen with corticosteroids.

Beagles (Beagle pain syndrome), Bernese mountain dogs, boxers, Akitas and German shorthaired pointers have a high incidence of SRMA.

Dogs with immune-mediated polyarthropathy (IMP) were found to have SRMA, and vice versa. Both contribute to the animal's painful condition. Joints are rarely swollen and pain may not be present on joint manipulation. Specific limb lameness is not noted. This was thought to be a specific syndrome in Akita and Bernese mountain dogs but has been seen in other medium-to-large breed dogs under 3 years old. Males are more frequently affected. Joint taps contain non-degenerative neutrophils, or lymphocytes, macrophages and neutrophils.

Treatment is the same for both IMP and SRMA. Treatment commences with prednisolone but relapses on medication or when tapering the dose indicates the need for a second agent. There are no controlled studies to say that one agent is better than another. Choice is made by availability of the agent, convenience of the treatment schedule and severity of the side-effects. Serum acute phase protein levels may be useful in future in the monitoring of SRMA.

TREATMENT

The dog improved on 1 mg/kg prednisolone BID which was continued for 2 months before being gradually withdrawn.

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71 Retroperitoneal disease

INITIAL PRESENTATION

Lumbar pain, pyrexia, lethargy, anorexia and abdominal pain.

INTRODUCTION

The retroperitoneum is the potential space between the ventral fascia of the sublumbar muscles and the peritoneum. Its lateral extent borders the lumbar epaxial muscles. It communicates with the mediastinum and the pelvic space.

Non-absorbable suture material use during ovariohysterectomy, penetrating wounds, kidney, ureter, or urethral trauma or migrating grass awns may all cause retroperitonitis. A fluctuating abscess or firm swelling with or without a draining sinus in the dorsal lumbar region may occur concurrently or some time after development of spinal pain.

Migrating plant awns may cause an initial cough weeks or months before back pain commences.

Migration theories

1. Inhaled and migrates along the diaphragmatic crus to lodge in sublumbar muscles
2. Swallowed and passes through caudal duodenum to ascend in the mesoduodenum to the lumbar vertebrae
3. Penetrates the terminal oesophagus, passing through the crus of the diaphragm to rest ventral to the body of T13 or L1.

SIGNALMENT

A 4-year-old neutered female springer spaniel dog.

CASE HISTORY

Four months prior to referral, the dog became lethargic and painful. She would not jump into the car and yelped when picked up. This persisted. The following month, the dog developed an arched back and fever (39.8°C). The lumbar muscles were felt to be very tense ('rock hard'). The owner thought the dog was putting on weight as the waist was wider than normal. Five days prior to referral, the dog was again pyrexic (39.9°C) and bilateral swelling of the lumbar muscles was found with serosanguineous fluid discharging from the right lumbar area. Synulox was given for 4 days prior to bacteriology culture and sensitivity of the suspected left lumbar abscess.

CLINICAL EXAMINATION

The lumbar muscles felt firm to touch. No neurological deficits were present. Temperature was normal.

NEUROANATOMICAL LOCALIZATION

Lumbar muscles.

DIFFERENTIAL DIAGNOSIS

- Foreign body
- Bite wounds
- Injection abscess
- Discospondylitis with soft tissue abscessation.

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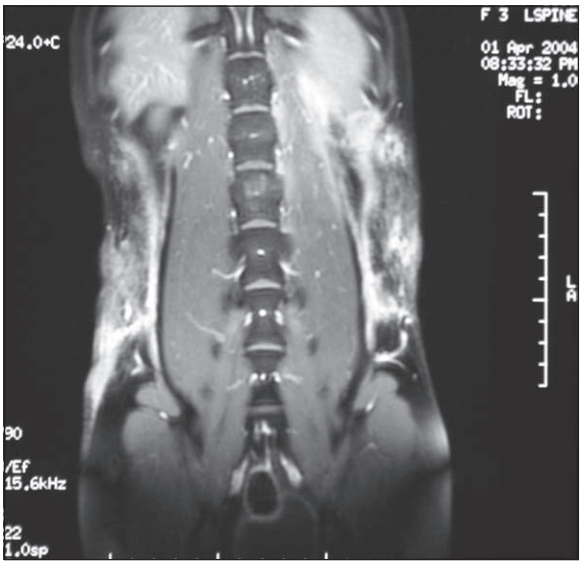


Figure 71.1 Dorsal T1WI+C (fat saturation) of lumbar spine. Bilateral subcutaneous areas of mottled hyperintensity post-contrast with enhancement of the left lumbar muscle.

CASE WORK-UP

Haematology showed a mature neutrophilia and monocytosis.

MRI showed spondylitis of the L4 vertebra with sublumbar inflammation and abcessation (Fig. 71.1).

In cases of migrating foreign bodies radiography can show mild to moderate ventral periosteal reactions of the T13–L4 vertebrae which may not become visible until several weeks after the onset of back pain signs. Sublumbar muscles are enlarged and diffusely outlined. Osteomyelitis, periostitis and discospondylitis are frequent complications of grass awn migration.

DIAGNOSIS

Migrating plant material, possibly inhaled.

TREATMENT

Synulox was continued for 8 weeks, then withdrawn. No recurrence of pain occurred. Had signs recurred, surgical exploration to remove foreign bodies would have been performed.

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72 Scratching

INTRODUCTION

Biting, scratching, chewing and auto-amputation of digits are rare but can occur with sensory neuropathies (Fig. 72.1). Cauda equina compression in dogs may result in self-mutilation of the perineum, tail or pelvic limbs.

Scratching or rubbing the face is a sign of irritation or pain. The trigeminal nerve is sensory to the face. The facial nerve conveys sensation from the concave surface of the pinna, vertical ear canal and middle ear.

Hypocalcaemia commonly presents with frantic face rubbing.

Trigeminal neuromas are thought to create a paraesthesia of the ipsilateral side of the head which leads to scratching at the face. This generally follows development of ipsilateral atrophy of the masticatory muscles so the diagnosis is self-evident.

Face rubbing is occasionally noted in dogs with facial paralysis. It is not the reason for referral. It may be related to pseudo-ptyalism.

Self-induced excoriations of the head and neck are seen in a small number of cats treated with methimazole or carbimazole within the first 3 months of treatment. Drug withdrawal and glucocorticoids are required.

Episodic pawing at one or both sides of the mouth, with exaggerated licking and chewing movements has been reported in cats, predominantly Burmese. Facial and tongue mutilation may occur. Predisposing factors seem to be dental (periodontal disease, erupting teeth or routine dental treatment) or living in a multi-cat household. No neurological deficits are found. The cause is not known.

SIGNALMENT 1

A 12-year-old neutered male domestic shorthaired cat.

CASE PRESENTING SIGNS

Rubbing the face.

CASE HISTORY

The cat had refused to eat or drink for a month, brusquely veering away from the food. At this time, the cat began wiping the left side of its face with a forepaw. Various investigations had been performed to find a source of oral, dental or TMJ pain to no avail. The cat then began having short episodes of sitting still and staring into space. The cat slept deeply and needed shaking to arouse it. The jaw was seen to hang open and the tongue stuck out and made multiple licking movements.

CLINICAL EXAMINATION

The cat was alert, friendly, ambulatory and had a normal gait, postural reactions and spinal reflexes. The left menace response was present. The left pupil was enlarged and lacked direct and indirect PLR. The left palpebral reflex was absent. Eye movement was normal. The jaw hung partially open and had decreased tone. The lower jaw had intact pain perception. Atrophy of the left masseter muscle could be palpated. The tongue had movement but protruded more than normal. A raspy noise from the upper respiratory tract was heard (Fig. 72.2).

NEUROANATOMICAL LOCALIZATION

Left CN III because of the mydriasis and lack of pupil constriction. Left CN V because of the lack of sensation to the face, atrophy of masticatory muscles and poor jaw tone. The mandibular branch of CN V was affected because it carries the motor fibres to muscles of mastica-

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Figure 72.1 A 4-month-old female Border collie presented for investigation of an arched back and knuckled over hind paws. She had intact patellar reflexes but absent withdrawal reflexes in the hindlimbs. Pain perception was absent in the hind paws and reduced in the forelimbs. The dog later chewed off one of its toes and was euthanized.

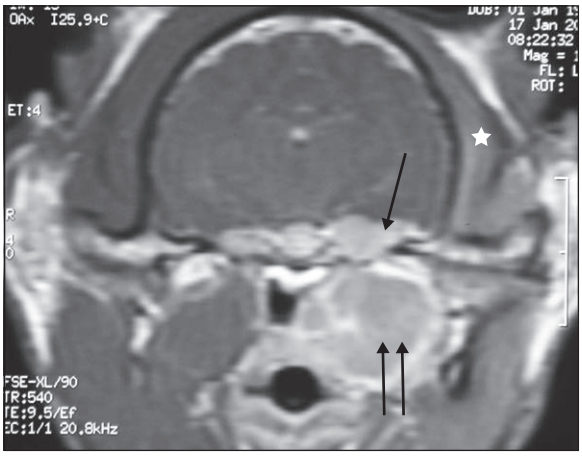


Figure 72.3 T1WI+C: large mass surrounding the left tympanic bulla (double arrows) and invading the floor of the skull (single arrow). Atrophy of ipsilateral masticatory muscles (star).

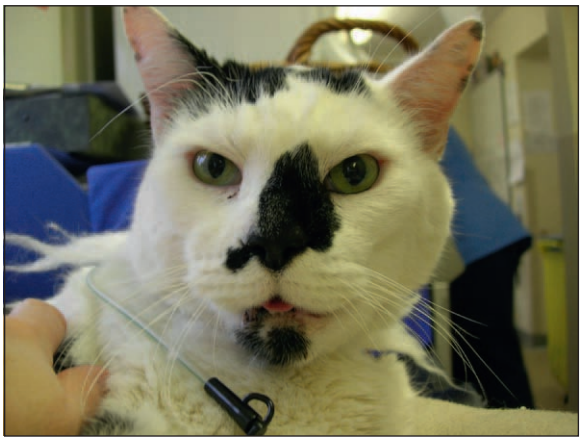


Figure 72.2 Dropped jaw, visible tongue, anisocoria (L>R).

tion. It is also sensory to the lower jaw, but this function remained intact. The raspy URT noise could have been due to laryngeal paralysis.

CN III and CN V lie close together within the cavernous sinus. An invasive skull lesion in the region of the oval foramen and therefore the TMJ was suspected as the cat first presented with a reluctance to eat and rubbing the face, suggesting pain and a possible paraesthesia. No history of ear disease was present so osteomyelitis of the bulla and an otogenic brainstem abscess were not thought to be a realistic differential. CN X departs the skull via the tympano-occipital fissure.

DIFFERENTIAL DIAGNOSIS

- Neoplasia of the skull, centred on the left tympanic bulla region
- Lymphoma of CNN
- Meningioma on the left side of the brainstem.

CASE WORK-UP

MRI images were obtained (Fig. 72.3).

CSF was normal with RBC 0 µl; WBC 2 µl; TP 0.20 g/l. Cytology detected low cellularity with scattered small medium lymphocytes and monocytes only.

DIAGNOSIS

Histopathology confirmed the suspected diagnosis of a sarcoma. A highly infiltrative round cell sarcoma was found (Figs 72.4, 72.5).

Scratching at the neck is a common sign of syringomyelia in Cavalier King Charles spaniels, especially when walking on a lead or with exertion, barking or excitement.

Dilation of the central canal is termed hydromyelia. Channel formation lacking ependymal lining within the spinal cord is known as syringomyelia. Syringohydromyelia indicates communication between the two. Both occur in similar situations so differentiating them is not thought to be clinically important.

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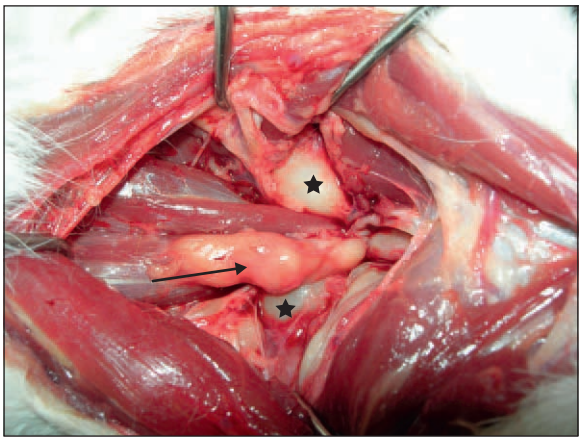


Figure 72.4 Postmortem. Tumour (arrow) visible adjacent to tympanic bulla (star).

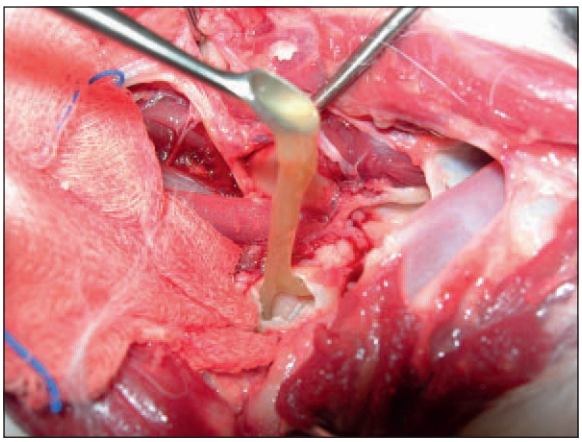


Figure 72.5 Postmortem. Gelatinous material within left bulla.

The condition occurs congenitally with or without concomitant malformations of the brain (hydrocephalus or Chiari-like malformation) or spinal cord (spinal dysraphism). Acquired forms associated with spinal trauma, IV disc herniation, neoplasia and inflammation have been reported.

Clinical signs comprise torticollis, scoliosis, spinal pain, ataxia and weakness and may be slowly progressive over weeks or years or acute in onset. It may be the cause of delayed worsening postoperatively or after trauma.

SIGNALMENT 2

A 4-year-old male Cavalier King Charles spaniel.

CASE PRESENTING SIGNS

Scratching the neck.

CASE HISTORY

For 6 months the dog had been scratching its neck when walked on a lead or if the left side of the neck was touched. The dog did not scratch if only wearing the collar, or if walked on a harness and lead; 2–3 times in the last 4 years the dog had yelped. The frequency of yelping for no apparent reason increased to once a week.

CLINICAL EXAMINATION

Lightly stroking the left side of the neck induced scratching movements in the ipsilateral hindlimb. No neurological deficits were noted.

NEUROANATOMICAL LOCALIZATION

The syndrome of scratching Cavalier King Charles spaniels is well known now and it is difficult not to become tunnel-visioned when presented with the breed. The lesion was localized to the cervical cord, with syringomyelia suspected as the source of paraesthesia.

DIFFERENTIAL DIAGNOSIS

Dermatological causes must be considered. Always ask if the animal bites, scratches or chews other parts of the body or has a history of otitis externa. If neck pain is more prominent, always consider IVDD and meningitis and plan investigations accordingly. Hypocalcaemia can cause muscle spasms and facial pruritus.

CASE WORK-UP

CSF was normal: RBC 19 µl, WBC 2 µl. Total protein 0.25 g/l. Cytology was normal, consisting of few monocytes and small to medium lymphocytes.

MRI: syringomyelia was detected (Figs 72.6 and 72.7) with caudal descent of the cerebellum into the foramen magnum.

DIAGNOSIS

Chiari-like malformation and syringomyelia (CM/SM).

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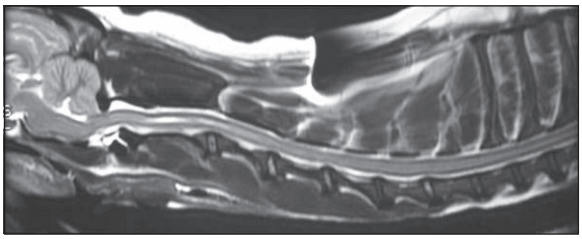


Figure 72.6 Sagittal T2WI. Large syrinx within spinal cord and caudal descent of the cerebellum.

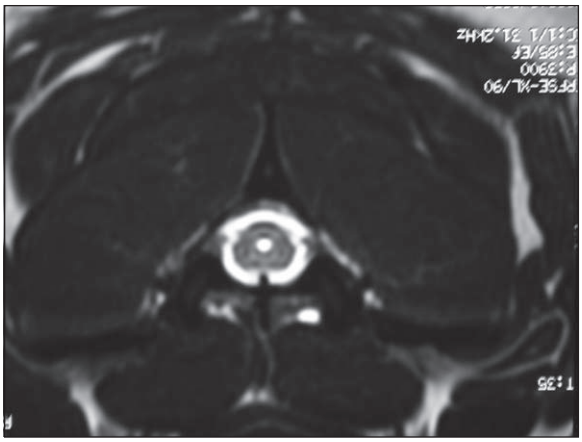


Figure 72.7 Transverse T2WI. Syrinx visible within the cord at the level of the axis.

TREATMENT

Treatment of syringomyelia commences with the least invasive option of pain relief but may culminate in surgery for those with worsening signs or unremitting pain. Postoperative improvement was seen in 80% of cases but 47% went on to deteriorate again within 2.3 years. Another surgical study of four Cavalier King Charles spaniels found no improvement in clinical signs or reduction of the syrinx by 3 months postoperatively. It is recommended that surgery is a last resort in Cavalier King Charles spaniels with unremitting pain.

Treatment of pain

Treatment in this case consisted of a combination of carprofen 2 mg/kg SID and gabapentin at 10–20 mg/kg TID. The yelping decreased in frequency but the scratching continued with the same stimuli (Table 72.1).

Table 72.1 Options for pain relief

Drug	Mechanism	Use
Morphine, methadone, fentanyl	Pure μ-receptor agonist opioid	Moderate to severe, acute pain Nociceptive and neuropathic pain
Buprenorphine	Partial μ-agonist/κ-antagonist opioid	*Note*: ceiling effect
Butorphanol	Mixed κ-agonist/μ-antagonist opioid	Visceral pain *Note*: ceiling effect
NSAIDs	Inhibit cyclooxygenase isoenzymes. Role in the CNS?	Mild to moderate. Bone and joint pain *Note*: ceiling effect. Avoid use in renal, GIT, hepatic, coagulation disorders and shock.
Metomindine, xylazine	α2-agonists	Sedation and analgesia
Ketamine	NMDA-receptor antagonist	Hyperalgesia
Anticonvulsants Phenytoin Carbamazepine Gabapentin valproate	Doses required are not necessarily the same as the anticonvulsant doses	Neuropathic pain, central pain syndromes
Tricyclic antidepressants; amitriptyline	Blockade of serotonin and norepinephrine reuptake.	Nociceptive pain Neuropathic pain
Corticosteroids	Anti-inflammatory	Inflammation, nerve compression, meningeal irritation
Carbamazepine Lamotrigine Mexiletine Tricyclic antidepressants; amitriptyline Gabapentin pregabalin	Na channels	Spontaneous pain Paraesthesias, dysaesthesias Neuropathic pain

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SECTION 12

URINARY INCONTINENCE

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73 Urinary incontinence – an introduction

Urination becomes a voluntary action in the young animal. It is governed by interactions of the CNS and PNS, many of them reflex. Cerebral influences inhibit or initiate urination. The pontine micturition centre is the major coordinator of the storage and voiding phases. The spinal cord transmits sensory and motor impulses. The pelvic, hypogastric and pudendal nerves directly innervate the smooth and skeletal muscle of the bladder and sphincter. Fast conduction through the tight connections between bladder muscle fibres facilitates bladder contraction (Fig. 73.1).

Incontinence is the lack of voluntary control of urination. Neurological causes rarely affect the bladder and sphincter alone. Other abnormalities are found in the history or on examination. Urological disease must always be considered as a cause or a sequela.

CLINICAL TIP

Grade 4 and 5 spinal cord lesions, sacral fractures, tail pull injury and cerebral disease are common causes of urinary incontinence.

PRESENTING SIGNS

No attempt to urinate, leaks, dribbles urine, strains to urinate and increased urgency to urinate.

TREATMENT

1. Identify the underlying cause. Urological vs. neurological.
2. Treat the underlying neurological lesion.
3. Treat urinary tract infections.
4. Express and empty the bladder every 6–8 hours. If this is not possible, perform intermittent clean catheterization. Place indwelling catheters in female dogs.
5. Reduce sphincter tone if necessary:
   - Phenoxybenzamine (smooth muscle) 2.5–5 mg q12–24 hr (cat) and 5–20 mg q12–24 hr (dog). The effects take 3–4 days to start.
   - Prazosin (smooth muscle). There is a risk of hypotension. Give half the calculated dose and observe for syncope, seizures or collapse.
   - Diazepam (striated muscle) 1–2.5 mg q8 hr (cat) and 2–10 mg q6–8 hr (dog).
   - Dantrolene (striated muscle) 0.5–2 mg/kg PO q8 hr (cat) and 1–5 mg/kg PO q8 hr (dog).
6. If detrusor atony secondary to distension is present, place an indwelling urinary catheter on a closed collection system to ensure the bladder is kept small at all times. Identify and treat urinary tract infections. Use bethanechol to stimulate bladder contraction.
   - Bethanechol chloride 2.5–5 mg PO q8 hr (cat), 5 mg PO q8 hr (small/medium dog) and 10–15 mg PO q8 hr (large dog). Effects commence in the first week of treatment.
   - Treatment of acute bladder distension is usually successful but takes 1–2 weeks. The indwelling catheter is removed after 10–14 days and urination is observed.
   - Treatment of chronic bladder distension is made difficult by inflammation and fibrosis of the muscular wall.
   - If UMN paralysis of the bladder is present bethanechol can be used after the sphincter tone has been reduced.
7. Detrusor hyperreflexia may be decreased by:
   - Propantheline bromide 0.25–0.5 mg/kg PO q8–12 hr.
   - Imipramine 5–15 mg PO q12 hr (dog) and 2.5–5 mg PO q12 hr (cat).

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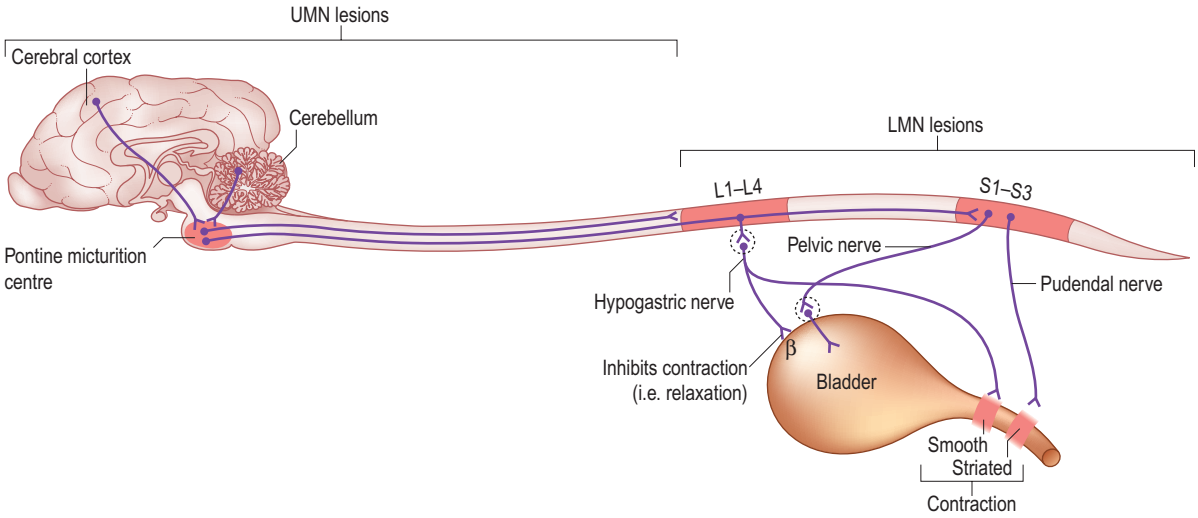


Figure 73.1 Control of urination.

8. Reflex dyssynergia requires a decreased urethral tone.
9. Sphincter incompetence is common in middle-aged to older spayed female dogs. It is not a primarily neurological disease.
   - Phenylpropanolamine: 1.5–2.0 mg/kg PO q8 hr.
   - Hormone replacement using diethylstilboestrol.

CLINICAL TIP

There may be resistance to bladder expression with sacral cord lesions. It is generated by the internal urinary sphincter innervated by the hypogastric nerve originating from the lumbar cord.

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74 No attempt to urinate

INTRODUCTION

No attempt to urinate results in urine retention. Overflow of urine may occur but the bladder does not fully empty. In time, a sacral reflex may initiate urination involuntarily but emptying will be incomplete and poorly coordinated with sphincter relaxation. Normal residual volume post-urination is usually less than 10 ml. Affected animals have palpably distended bladders. Ease of expression depends on the sphincter tone. Causes include:

- Cannot perceive bladder distension: lesions of the cerebrum, spinal cord, pelvic nerve or sacral segments (S1–3)
- Does not initiate voluntary urination: cerebral lesion
- Cannot contract bladder: lesion of the detrusor muscle, pelvic nerve, sacral cord, or dysautonomia
- Can perceive bladder distension and can contract bladder wall but is recumbent and cannot assume posture, so will not urinate.

CLINICAL TIP

Rule out anuria or bladder rupture.

SIGNALMENT

An 8-year-old neutered female domestic shorthaired cat.

CASE PRESENTING SIGNS

Hindlimb weakness.

CASE HISTORY

There was a 1 month history of hindlimb paresis and ataxia which had started gradually, first affecting the left hindlimb. The cat was unable to get in and out of the cat flap or jump on to furniture. If jumping off furniture, the hindlimbs would collapse on landing. Recently, the cat stayed in its basket and no urination had been noticed.

CLINICAL EXAMINATION

The left hindlimb lacked all voluntary motor ability but there was a small amount of voluntary movement in the right hind. Hindlimb proprioception was absent bilaterally. Spinal reflexes were normal to increased in the hindlimbs. Muscle tone was normal in all limbs. The bladder was enlarged and difficult to express. No spinal pain was found.

NEUROANATOMICAL LOCALIZATION

The T3–L3 spinal cord segments.

DIFFERENTIAL DIAGNOSIS

- Neoplasia
- IVD disease
- Myelitis.

CASE WORK-UP

Myelography showed ventral extradural compression at the level of the L2–3 IV space.

CSF was normal.

DIAGNOSIS

A left-sided hemilaminectomy at L2–3 allowed removal of a large amount of calcified disc material the nature of which was confirmed by histopathology.

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TREATMENT

Urinary incontinence persisted postoperatively. Diazepam and phenoxybenzamine made it easier to express the bladder, but the cat was unable to voluntarily void. Detrusor atony was suspected so an indwelling urinary catheter on a closed system was placed and the cat was monitored closely upon its removal 10 days later. The cat became able to void small amount of urine but the bladder remained enlarged and required expression. She was readmitted and placed on another closed system and indwelling urinary catheter for another week while receiving diazepam, phenoxybenzamine and bethanechol.

A degree of voluntary movement returned to both hindlimbs within 2 weeks. The cat was able to urinate normally soon after removing the catheter the second time.

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75 Leaks, dribbles urine

INTRODUCTION

Leaking or dribbling urine is a passive action and urine is found where the animal has been sitting or lying and may occur as the animal walks about. It occurs with:

- Weak external sphincter due to a pudendal nerve lesion or sacral cord lesion (S1–3)
- Weak bladder neck and smooth muscle of the urethra (internal sphincter) due to a lesion of the hypogastric nerve or lumbar cord (cat: L2–5, dog: L1–4).
- Urine retention with overflow due to LMN or UMN bladder paralysis
- 32% of dogs that had recovered pain perception and ambulation following a grade 5 IVDD-induced T3–L3 spinal cord injury had urinary incontinence, infrequently dribbling urine in the house. All had mild faecal incontinence.

SIGNALMENT

A 9-year-old neutered male Maine coon cat.

CASE PRESENTING SIGNS

Tail paralysis, faecal and urinary incontinence and a bilateral plantigrade stance.

CASE HISTORY

A low tail carriage and an abnormal hindlimb stance described as 'rabbit-like' developed suddenly 2 weeks prior to presentation. Faecal and urinary incontinence commenced with urine and faeces leaking or dropping out where the cat lay or as the cat moved about the house. The owner did not think the cat was in pain as the appetite remained normal and the pet had not become reclusive.

CLINICAL EXAMINATION

The cat was alert and ambulatory. A bilateral plantigrade stance of the hindlimbs was present with the left slight worse than the right hindlimb. The tail hung flaccidly and lacked all voluntary movement (Fig. 75.1). Anal tone was significantly reduced. The cat did not detect noxious stimulation of the tail or the anus (Fig. 75.2). The bladder was enlarged and there was little resistance to expression. Faeces could be digitally removed from the conscious cat. The withdrawal reflex was present in all limbs and seemed normal. The patellar reflex was 2–3+ bilaterally. No spinal pain was detected.

NEUROANATOMICAL LOCALIZATION

The lesion was localized to the caudal nerves of the tail and sacral nerves S1–3 or the associated spinal cord segments of the cauda equina.

This would account for the paralysis of the tail, anal sphincter, urethral sphincter and the inability of the bladder to contract. Distension of the bladder by urine retention can separate the tight conjunctions between the detrusor muscle fibres which would also interfere with synchronous bladder contraction. Extension of the tarsus is mediated by the sciatic nerve whose chief components are the L7–S1 segments and nerves. Plantigrade stance of the hindlimbs may also result from tarsal injury or gastrocnemius rupture but neither was found on palpation.

DIFFERENTIAL DIAGNOSIS

- Trauma: A sudden onset of neurological signs is often blamed on trauma, especially in cats with access to the outdoors. Spinal fractures are uniformly painful. Survey radiographs sent by the referring veterinarian were normal.

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Figure 75.1 Plantigrade stance, flaccid tail.

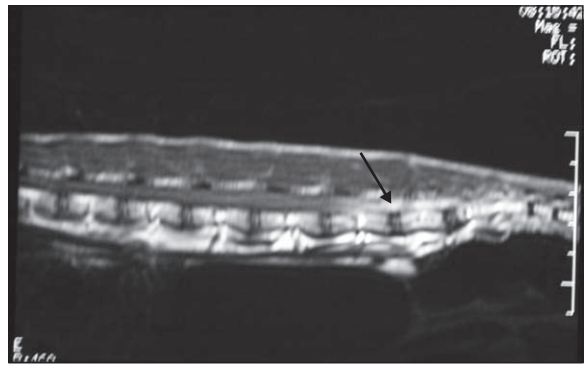


Figure 75.3 T1WI+contrast sagittal, lumbar spine. Enhancement of cauda equina (arrow).



Figure 75.2 Lack of pain perception in the tail.



Figure 75.4 Distortion of the cauda equina.

- Vascular: Infarction of the lumbosacral intumescence can produce a sudden onset of LMN signs and would not be expected to cause spinal pain. Spinal infarction may produce strikingly unilateral deficits but the small size of the sacral segments and oedema associated with infarction would render signs bilateral.
- Intervertebral disc extrusion at L7–S1 was a possibility.
- Neoplasia: neoplasia or inflammation of the cauda equina and spinal tumours are all generally painful conditions.

CASE WORK-UP

MRI showed a contrast-enhancing lesion within the spinal canal at the level of the L6–S1 vertebrae (Fig. 75.3).

Only a few drops of lumbar CSF could be collected. Cytology was judged the most important component to assess as lymphoma, a differential diagnosis, may shed neoplastic cells into the CSF. Surgical exploration was recommended but refused. Prednisolone was commenced to treat possible lymphoma. Sensation returned to the right side of the anus the following day. The cat also became intensely painful and was restless and attacked his hindquarters. Methadone IM was required every 4 hours for pain relief. CSF cytology was normal.

DIAGNOSIS

Euthanasia was performed due to concerns for resolution of the pain and incontinence.

At postmortem examination no extradural surgically removable lesion was present. The cauda equina appeared discoloured through the dural sac. Lymphoma of the cauda equina was found (Fig. 75.4).

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MCQs

1. **A dog was left at the practice with a short note from the client saying the dog had fallen down the stairs early that morning and had head trauma. Physical examination is normal. Your priority is to:**
   - **(a)** Anaesthetize the dog and radiograph the head for possible fractures
   - **(b)** Inject the dog with dexamethasone to treat increased intracranial pressure
   - **(c)** Call the owner and clarify what happened
   - **(d)** Dispense phenobarbitone
   - **(e)** Take blood tests

2. **A 2-year-old German shepherd dog has been having clusters of 3–4 generalized seizures. Treatment consists of rectal diazepam administered by the owners when the first seizure occurs, and a twice-daily regime of 60 mg phenobarbitone. Seizure activity was first noticed 1 year ago. The dog's behaviour in the inter-ictal period has always been normal. You want to improve the seizure control. What information do you need to aid rational treatment decisions?**
   - **(a)** Length of the inter-ictal period
   - **(b)** Serum phenobarbitone level
   - **(c)** Liver function tests
   - **(d)** Repeat neurological examination results
   - **(e)** All of the above

3. **Cervical fractures most commonly affect the axis. Animals typically present with neck pain. Which combination of signs might be seen if the fracture site becomes unstable and compresses the spinal cord?**
   - **(a)** Paraplegia, urinary retention, increased muscle tone
   - **(b)** Non-ambulatory tetraparesis, increased muscle tone and normal spinal reflexes in all limbs
   - **(c)** Tetraplegia, hypoventilation, loss of all spinal reflexes
   - **(d)** Non-ambulatory tetraparesis, respiratory paralysis, loss of pain perception
   - **(e)** Paraplegia, urinary and faecal incontinence, tail paralysis

4. **The menace response is absent in the left eye. Where is the lesion?**
   - **(a)** Right facial nerve
   - **(b)** Right trigeminal nerve
   - **(c)** Left facial nerve
   - **(d)** Left oculomotor nerve
   - **(e)** Left cerebrum

5. **One month ago a 10-year-old male domestic shorthaired cat began having episodes of sitting very still, twitching its face and growling. The events last about 10 seconds and are followed by hours of pacing and circling. Upon questioning, the owner says that the cat has not used the cat flap for the last 3 months and will sit facing into corners for long periods. Examination finds that the cat is unresponsive to the environment and relentlessly but slowly circles to the right. You decide to:**
   - **(a)** Do a routine dental as that is probably why the face is twitching
   - **(b)** Dispense diazepam as it is sure to be idiopathic epilepsy
   - **(c)** Recommend ear cleaning as the cat appears to have vestibular disease
   - **(d)** Discuss imaging the brain as it appears to be cerebral disease
   - **(e)** Treat with flea control products as an infestation could make the cat restless

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6. A 6-month-old collie dog was hit by a car. The hindlimbs are paralysed, have 3+ patellar reflexes and withdrawal reflexes bilaterally. Perception of pain is absent when the hindlimbs are tested. The dog struggles to sit, but can use both forelimbs. Where is the lesion?

(a) C1–5 spinal cord segments
(b) C6–T2 spinal cord segments
(c) T3–L3 spinal cord segments
(d) L4–S1 spinal cord segments
(e) S1–3 spinal cord segments

7. Lateral spinal radiographs of the same 6-month-old collie dog detect an undisplaced oblique fracture of T13 vertebral body. The cutaneous trunci reflex is absent caudal to the level of T9 vertebral body. Which statement is true?

(a) Undisplaced fractures of the vertebral body are stable. The dog should respond well to cage rest and an external splint
(b) The articular facets appear to be fractured at T12–T13. The fracture site is potentially unstable. The dog has a good prognosis as long as the site is surgically stabilized
(c) MRI of the cord is required to look for herniated disc material
(d) Ascending myelomalacia has probably occurred and the prognosis is poor
(e) The cutaneous trunci reflex is absent because the brachial plexus is avulsed bilaterally

8. A 9-year-old male Staffordshire bull terrier became lethargic 6 months ago and aggressive 2 weeks ago. The dog is impossible to handle but could be observed safely. When indoors the animal just lay on the exam room floor. When taken outside he appeared to be blind and showed little interest in the surroundings and would sink to the ground and lie in sternal recumbency. The gait was normal. Pupils were large, symmetric and unchanging in light and darkness. PLR were absent bilaterally. The facial, jaw and tongue muscles appeared normal in movement and symmetry. There is one lesion … where?

(a) The optic chiasm
(b) Right cerebrum
(c) Left cerebrum
(d) Optic radiation bilaterally
(e) The lateral geniculate nuclei bilaterally

9. A 12-year-old male domestic shorthaired cat is presented because the owner says the cat is no longer sitting in the usual places. In the last 2 weeks, he has been found lying in the empty bath and also in the owner's wardrobe. Examination finds a mentally dull cat with a normal gait and decreased placing responses in the left fore and hindlimbs. Hopping was normal. Spinal reflexes were normal. The menace response was absent bilaterally. PLR were normal. Where is the lesion?

(a) Bilateral optic nerves
(b) Right cerebrum
(c) Left medulla oblongata
(d) Bilateral CN III
(e) Left cerebellum

10. A 6-year-old male Dalmatian ran into a tombstone while playing. He continued to walk but had an arched back for the next 24 hours before developing hindlimb paralysis. You localize the lesion, ponder the differential diagnoses and commence investigation. Lumbar CSF analysis detects 30 mg/dl total protein and no WBCs or RBCs. Myelography shows ventral extradural compression at the T12–13 IV space. The VD view is normal. This corresponds with the anatomic localization. What neurological abnormalities do you find on examination?

(a) Forelimb weakness, flaccid hindlimbs and absent cutaneous trunci reflex
(b) Hypertonia and hyper-reflexia of the hindlimbs. Normal forelimb proprioception
(c) Increased withdrawal reflex in the hindlimbs, absent right patellar reflex and flaccid anal tone
(d) Absent patellar reflex, absent withdrawal reflex and normal pain perception
(e) Torticollis, tetraparesis and 3+ spinal reflexes in all limbs

11. The local hunt kennels called to say that half the dogs have collapsed and are vomiting and the others look weak. All dogs were recently fed rotting meat. Examination of the

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worst affected dog reveals non-ambulatory tetraparesis, an absent gag reflex, decreased blink reflex, flaccid areflexic limbs and non-productive attempts to bark. Pain perception appears to be normal. The lesion is localized to:

(a) The medulla oblongata, to explain the loss of gag reflex and laryngeal paralysis
(b) Autonomic nervous system to account for the weakness and regurgitation
(c) C6–T2 and L4–S1 spinal cord segments to account for the atonia and areflexia
(d) The neuromuscular junction to account for the widespread LMN dysfunction
(e) Spinal cord interneurons to account for widespread LMN dysfunction

12. The ventricular system is lined by ependyma which can become inflamed in cases of FIP. Blockage of the mesencephalic aqueduct results in:

(a) Dilation of the fourth ventricle
(b) Dilation of the quadrigeminal cistern
(c) Dilation of the cisterna magna
(d) Dilation of the lateral ventricles
(e) Dilation of the central canal

13. A left trigeminal nerve lesion can result in:

(a) Left ptosis
(b) Paralysis of the left upper lip
(c) An absent menace response on the left
(d) An absent left blink reflex
(e) Atrophy of the left side of the tongue

14. Anisocoria. The left pupil is mydriatic. The right pupil is mid-sized. Light is shone in the left eye and only the right pupil constricts. Light is shone in the right eye and only the right pupil constricts. The animal is comatose, so the menace response cannot be assessed. There is one lesion … where is it?

(a) Left optic chiasm to explain the left-sided mydriasis
(b) The right ophthalmic nerve to explain the left-sided mydriasis
(c) The left oculomotor nerve to explain the lack of left pupil constriction
(d) The left oculosympathetic supply to explain the lack of left pupil constriction
(e) The midbrain to explain the lack of left pupil constriction

15. A young dog slips its collar, runs onto the road and is hit by a car. Happily the dog survives, but immediately develops a non-weight-bearing lameness of the left forelimb. A fractured radius is diagnosed radiographically. A week post-surgery, the left fore is still unable to move or support weight and the dorsal surface of the paw rests upon the ground. The left pupil is miotic in light and darkness and has a normal PLR. The cutaneous trunci reflex is absent on the left side. Which of the following sentences best summarizes the clinical assessment?

(a) The limb is non-weight-bearing because the fracture site is infected and is causing the dog pain
(b) The radial nerve was bruised and is expected to recover
(c) Head trauma has caused Horner's syndrome. The limb is not infected but the dog requires more pain relief and an MRI of the head
(d) The radial nerve was pierced by the internal fixation which accounts for the pain and all the neurological deficits. The dog requires additional post-surgery radiographs of the limb
(e) The left brachial plexus has been damaged. This accounts for the left fore paralysis, and ipsilateral LMN reflex changes and Horner's syndrome

16. Common causes of back pain include all of the following:

(a) Immune-mediated meningitis, IVD extrusion and discospondylitis
(b) Spinal cord infarction, polyneuropathy and degenerative myelopathy
(c) Thiamine deficiency, degenerative myelopathy and cerebral disease
(d) Schiff–Sherrington syndrome, spinal cord infarction and borborygmus
(e) Hyponatremia, hypoglycaemia hypokalaemia

17. A 5-year-old cat suddenly fell off the back of the armchair and is presented in a distressed state as an emergency 15 minutes later. The animal is ataxic and almost unable to walk as it repeatedly falls to the left. A left head tilt and spontaneous nystagmus, with a horizontal fast phase to the right are noted.

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Based on this preliminary information, where is the lesion?

(a) The left cervical cord to account for the falling to the left
(b) The left cerebellum to account for the paradoxical direction of the spontaneous nystagmus
(c) The left vestibular apparatus to account for all the signs
(d) The right vestibular apparatus to account for all the signs
(e) The heart to account for the collapse and distressed state

18. The same cat is examined the following day and found to have a normal placing response in all limbs. When the body is supported, the cat is able to hop normally on each limb. The lesion is localized to the:

(a) Right cerebellum because cerebellar lesions do not cause weakness
(b) Left peripheral vestibular apparatus because strength and proprioception are normal
(c) Left brainstem lesion as indicated by the horizontal nature of the nystagmus
(d) Left cochlear duct to account for the loss of balance
(e) Left pons to account for the left-sided balance loss

19. An 8-year-old retired greyhound was let out into the garden this morning. Ten minutes later the owner found the dog unable to stand. Half an hour after this occurred the dog is examined. The findings include: non-ambulatory tetraparesis, decreased hopping response in all limbs but more so on the left, absent proprioception in the left fore and hind limbs and decreased proprioception in the right fore and hind limbs. Spinal reflexes are slightly increased in all limbs. Muscle tone is normal in all limbs. The dog is mentally alert, has normal cranial nerve function and does not have any spinal pain. Where is the lesion?

(a) T3–L3 spinal cord segments, worse on the left
(b) Right cerebrum
(c) Left brainstem
(d) Left cerebellum
(e) C1–C5 spinal cord segments, worse on the left

20. Dry eye can occur secondary to:

(a) Horner's syndrome
(b) Otitis interna
(c) Facial nerve paralysis
(d) Oculomotor nerve paralysis
(e) Glossopharyngeal nerve paralysis

21. The oculomotor nerve innervates the:

(a) Lacrimal gland
(b) Pupil dilator muscle
(c) Pupil constrictor muscle
(d) The third eyelid
(e) All of the above

22. Horner's syndrome presents with:

(a) Miosis, ptosis and dry eye
(b) Miosis, wider palpebral fissure and reddened conjunctiva
(c) Miosis, ventrolateral strabismus and lack of direct PLR
(d) Mydriasis, ptosis and reddened conjunctiva
(e) Miosis, ptosis and enophthalmos

23. Which of the following signs has a prognostic value?

(a) The presence or absence of pain perception
(b) The direction of a head tilt
(c) Schiff–Sherrington sign
(d) The Babinski reflex
(e) The biceps reflex

24. Brain death is suspected in an animal lacking:

(a) Voluntary movement in all limbs
(b) Withdrawal reflexes in all limbs
(c) Pupillary light reflexes
(d) Vision
(e) Consciousness and all CNN reflexes

25. An 8-year-old boxer dog suddenly developed a right-sided head tilt. When walking around the house the dog keeps to the left, leaning on walls and occasionally staggering to the left but never falling. Examination detects a left-sided hemiparesis. Spinal reflexes are normal in all limbs. Proprioception is reduced in the left hindlimb. Fundic examination detects a spontaneous rotatory nystagmus with the fast jerk phase to the right. The dog is generally very well, but developed otitis externa of the right ear

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1 week ago. Where is the lesion and what could it be caused by?
- **(a)** Right-sided peripheral vestibular disease. Possibly 'old dog' idiopathic vestibular disease
- **(b)** Right-sided peripheral vestibular disease. Possibly otitis interna
- **(c)** Right-sided central vestibular disease. Possibly a brainstem abscess
- **(d)** Left-sided central vestibular disease. Possibly a cerebellopontine angle tumour
- **(e)** Bilateral peripheral vestibular disease. Possibly otitis interna

26. Circling is a sign of:
- **(a)** Settling down to sleep in a basket
- **(b)** Cerebral disease
- **(c)** Vestibular disease
- **(d)** Some lesions of the cerebellum
- **(e)** All of the above

27. Which of the following groups of cranial nerves exit the skull through the orbital foramen?
- **(a)** Optic, oculomotor and trochlear
- **(b)** Oculomotor, trochlear and abducent
- **(c)** Oculomotor, orbital and trochlear
- **(d)** Facial only
- **(e)** Oculomotor, abducent and facial

28. Which of the following is not a sign of facial nerve paralysis?
- **(a)** Lack of jaw tone
- **(b)** Lack of a blink reflex
- **(c)** Lack of a menace response
- **(d)** Slightly wider palpebral fissure
- **(e)** Dry eye

29. Which of the following is not a sign of peripheral vestibular disease?
- **(a)** Head tilt
- **(b)** Vertical nystagmus
- **(c)** Horizontal nystagmus
- **(d)** Rotatory nystagmus
- **(e)** Strabismus

30. Which signs do not indicate vestibular dysfunction?
- **(a)** Circling
- **(b)** Falling
- **(c)** Leaning
- **(d)** Seizuring
- **(e)** Rolling

31. Mydriasis occurs in:
- **(a)** Dysautonomia
- **(b)** Horner's syndrome
- **(c)** Otitis media-interna
- **(d)** Diffuse cerebral disease
- **(e)** Myasthenia gravis

32. Flaccid facial muscles may occur in
- **(a)** Trigeminal nerve paralysis
- **(b)** Facial nerve paralysis
- **(c)** Tetanus
- **(d)** Metronidazole toxicity
- **(e)** Otitis externa

33. CSF analysis is useful in cases of:
- **(a)** Fever of unknown origin
- **(b)** Chronic cases of otitis media-interna which develop neurological signs
- **(c)** Stiff, stilted gait with limited neck movement
- **(d)** Sudden onset paraplegia in a cat
- **(e)** All of the above

34. Absence of a withdrawal reflex indicates:
- **(a)** Lack of proprioception
- **(b)** A sciatic nerve lesion
- **(c)** Lack of pain perception
- **(d)** Paralysis of the limb
- **(e)** Radial nerve paralysis

35. Which of the following groups of signs are common in acute thoracolumbar disc extrusions?
- **(a)** Paraplegia, spinal pain and a flaccid anus
- **(b)** Paraplegia, increased patellar reflexes and absent hindlimb withdrawal reflexes
- **(c)** Non-ambulatory paraparesis, back pain and flaccid hindlimbs
- **(d)** An arched back, vomiting and cranial abdominal pain
- **(e)** Paraparesis, decreased proprioception and increased hindlimb spinal reflexes

36. Cauda equina syndrome in small animals produces:
- **(a)** Cranial nerve dysfunction
- **(b)** LMN deficits
- **(c)** UMN deficits

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(d) Generalized tetraparesis
(e) All of the above

37. The first priority when presented with an animal that may have neurological disease is:
(a) To take blood tests
(b) To schedule imaging
(c) To choose between MRI or myelography
(d) To decide if it has neurological disease or not
(e) To run a series of DNA tests for that breed

38. A dog walks into a bar. Why?
(a) Cerebral disease
(b) Retinal detachment
(c) Pituitary tumour pressing on the optic chiasm
(d) Optic neuritis
(e) All of the above

39. Sensation to the face is mediated by which nerve?
(a) Ophthalmic
(b) Facial
(c) Glossopharyngeal
(d) Trigeminal
(e) Vagus

40. The cat became quite contrary. It attacked the dog and ignored the canary. The final straw came, when it forgot its own name, and sat facing walls simply staring. Localize the lesion:
(a) UMN lesion
(b) LMN lesion
(c) Brainstem lesion
(d) Cerebellar lesion
(e) Cerebral lesion

41. Which disorder does not interfere with the neuromuscular junction?
(a) Tick paralysis
(b) Botulism
(c) Myasthenia gravis
(d) Polyradiculoneuritis
(e) Snake envenomation

42. Focal myasthenia gravis may cause weakness in all of the following, except for one … which is it?
(a) Gag reflex, blink reflex and laryngeal muscles
(b) Menace response, lip retraction and gag reflex
(c) Laryngeal muscles, gag reflex and oesophagus
(d) Pupil constriction, blink reflex and lacrimation
(e) Muscles of facial expression, laryngeal muscles and oesophagus

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MCQs – Answers

1. **(c)** Call the owner and clarify what happened
2. **(e)** All of the above
3. **(b)** Non-ambulatory tetraparesis, increased muscle tone and normal spinal reflexes in all limbs. Compression of the cord at the level of C2 vertebra would produce UMN deficits in all limbs. The presence of normal spinal reflexes and increased muscle tone indicate that the reflex arcs are intact, and the weakness is due to a UMN lesion. An animal that retains some voluntary movement from a C1–5 spinal cord lesion will have the ability to perceive pain. Respiratory paralysis and loss of pain perception indicate severe damage to the cervical cord and the animal would be paralysed or possibly dead.
4. **(c)** Left facial nerve. The menace response requires the perception of the visual stimulus and a blink. The menace response is absent because the animal is unable to blink (CN VII)
5. **(d)** Discuss imaging the brain as it could be cerebral disease. Altered mentation, altered behaviour, seizures and circling to the right indicate right cerebral disease
6. **(c)** T3–L3 spinal cord segments
7. **(d)** Ascending myelomalacia has probably occurred and the prognosis is poor. The fracture site indicates the site of one lesion. The lack of cutaneous trunci reflex indicates a more rostral lesion. The sudden onset paraplegia with loss of pain perception (grade 5 spinal cord lesion) secondary to a spinal fracture carries a very poor prognosis for recovery
8. **(a)** The optic chiasm. A lesion at the optic chiasm would cause blindness and lack of PLR. The aggression and lethargy are caused by the lesion extending into the thalamus dorsally
9. **(b)** Right cerebrum. Behaviour change, left-sided proprioceptive deficits. The absent menace response can be ascribed to the obtunded mental state
10. **(b)** Hypertonia and hyperreflexia of the hindlimbs. Normal forelimb proprioception. Normal forelimb function. UMN paraplegia
11. **(d)** The neuromuscular junction to account for the widespread LMN dysfunction. Generalized LMN disease is present.
12. **(d)** Dilation of the lateral ventricles. The mesencephalic aqueduct is in the midbrain.
13. **(d)** An absent left blink reflex. The blink reflex requires intact sensation, mediated by CN V and intact movement of the eyelids, mediated by CN VII
14. **(e)** The midbrain to explain the lack of left pupil constriction and coma. The left pupil is mydriatic and unable to constrict due to a left-sided CN III lesion. The coma indicates a lesion within the ARAS. A lesion of the left CN III nucleus within the midbrain would account for both signs. Compression of CN III nerve can also result from brain herniation
15. **(e)** The left brachial plexus has been damaged. This accounts for the left fore paralysis, and ipsilateral LMN reflex changes and Horner's syndrome. Radial nerve paralysis prevents extension of the limb and weight-bearing.
16. **(a)** Immune-mediated meningitis, IVD extrusion and discospondylitis. Cerebral disease is reported as causing neck pain. Schiff–Sherrington syndrome is a localizing sign only
17. **(c)** The left vestibular apparatus to account for all the signs. The signs all indicate left-sided vestibular involvement

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18. **(b)** Left peripheral vestibular apparatus because strength and proprioception are normal. Peripheral vestibular disease does not affect the general proprioception or the strength
19. **(e)** C1–C5 spinal cord segments, worse on the left. UMN to all limbs worse on the left
20. **(c)** Facial nerve paralysis. The lacrimal gland is innervated by CN VII. CN VII passes through the middle, not the inner ear
21. **(c)** Pupil constrictor muscle. The parasympathetic branch of CN III innervates the iris constrictor
22. **(e)** Miosis, ptosis and enophthalmos
23. **(a)** The presence or absence of pain perception. The ability to perceive pain indicates some remaining spinal cord integrity in the paraplegic patient. Lack of a biceps reflex may be a normal finding
24. **(e)** Consciousness and all CNN reflexes
25. **(d)** Left-sided central vestibular disease. Possibly a cerebellopontine angle tumour. The left-sided hemiparesis and proprioceptive deficit indicate the central origin of the vestibular signs. The head tilt is paradoxical in that it is directed in an opposite direction to that of the lesion. The paresis and proprioceptive deficits are ipsilateral to the side of the lesion
26. **(e)** All of the above. Obviously a history and examination will differentiate the possibilities from each other
27. **(b)** Oculomotor, trochlear and abducent
28. **(a)** Lack of jaw tone. Jaw tone is mediated by CN V
29. **(b)** Vertical nystagmus. *Be careful*. Rotatory nystagmus can look like vertical nystagmus if the patient is not examined carefully
30. **(d)** Seizuring. Seizures indicate cerebral dysfunction. *Be careful*. The sudden onset of vestibular signs cause animals to struggle to remain upright and they tend not to pay much attention to the owners while doing so. This results in vestibular disease being mistaken for seizure activity
31. **(a)** Dysautonomia. Parasympathetic dysfunction
32. **(b)** Facial nerve paralysis. CN VII innervates muscles of facial expression
33. **(e)** All of the above
34. **(b)** A sciatic nerve lesion
35. **(e)** Paraparesis, decreased proprioception and increased hindlimb spinal reflexes. T3–L3 lesions produce UMN signs of weakness, normal to increased spinal reflexes and muscle tone. Decreased proprioception is usually the first sign of spinal cord compromise
36. **(b)** LMN deficits
37. **(d)** To decide if it has neurological disease or not. The first step is to find the body system responsible for the presenting complaint. Take a history and perform an examination. Localize the lesion, whether it is to the nervous system or another body system. If the nervous system, is involved, specify the anatomic sites.
38. **(e)** All of the above. All of the options can result in blindness
39. **(d)** Trigeminal
40. **(e)** Cerebral lesion. Cerebral lesions can result in a behaviour change
41. **(d)** Polyradiculoneuritis. Polyradiculoneuritis, as the name suggests, affects the nerve roots
42. **(d)** Pupil constriction, and lacrimation are unaffected

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1 APPENDIX Abbreviations

Ab: antibody
Ach: acetylcholine
AchR: acetylcholine receptor
ARAS: ascending reticular activating system
BAEP: brainstem audioevoked potential
BAER: brainstem audioevoked response
BBB: bloodbrain barrier
CAT: computerized axial tomography
CN: cranial nerve
CNN: cranial nerves, assigned Roman numerals from I to XII, hence CN V, etc.
CNS: central nervous system
CP: conscious proprioception
CPP: choroid plexus papilloma
CSF: cerebrospinal fluid
CT: computerized tomography
CTZ: chemoreceptor trigger zone
dB: decibel
DIC: disseminated intravascular coagulation
EMG: electromyography
FCE: fibrocartilaginous thromboembolism
FLAIR: fluid-attenuated inversion recovery
GME: granulomatous meningoencephalitis
GRE: gradient echo
HBC: hit by car
HL: hind limb
ICP: intracranial pressure
MG: myasthenia gravis
MLF: medial longitudinal fasciculus
MMM: masticatory muscle myositis
MRI: magnetic resonance imaging
NCV: nerve conduction velocity
OCD: obsessive compulsive disorder
$PaCO_2$: partial pressure of carbon dioxide dissolved in arterial blood
PaO_2: partial pressure of oxygen dissolved in arterial blood
PNS: peripheral nervous system
PRN: acute idiopathic polyradiculoneuritis
PU, PD, PP: polyuria, polydipsia, polyphagia
REM: rapid eye movement
RMSF: Rocky Mountain spotted fever
RNS: repetitive nerve stimulation
RTA: road traffic accident
SLUD: salivation, lacrimation, urination, diarrhoea
STIR: short-tau inversion recovery
STT: Schirmer Tear Test
T13: depending on the context, either the 13th thoracic vertebra or spinal cord segment
T1WI: T1-weighted image
T2WI: T2-weighted image
VOR: vestibulo-ocular reflex

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2 APPENDIX Cerebrospinal fluid and the ventricular system

The central nervous system is created from a tube which expands. The centre of the tube remains hollow, is lined by ependymal cells and contains CSF. The central canal of the spinal cord is barely visible on MRI and contains a small amount of CSF. Expansion of the central canal is called hydromyelia. Formation of CSF-filled spaces without an ependymal lining in the spinal cord is referred to as syringomyelia, and the fluid-filled structure is called a syrinx. Both hydro- and syringomyelia are pathological processes (Fig. 72.6, Fig. 72.7).

The hollow CSF-filled chambers of the brain are called ventricles. A curved, banana-shaped lateral ventricle lies within each cerebral hemisphere. Each connects to the ring-shaped third ventricle by an interventricular foramen. The third ventricle lies on the midline and encircles the thalamic adhesion. A narrow passageway called the mesencephalic aqueduct passes caudally from the third ventricle to the fourth ventricle. The fourth ventricle is the last dilation of the ventricular system and its flattened structure lies dorsally within the medulla oblongata and is enclosed by a thin roof plate called the medullary velum. In normal circumstances, CSF exits the ventricular system, passing into the subarachnoid space via the lateral apertures, one on either side of the fourth ventricle. The central canal communicates with the fourth ventricle but CSF only enters the central canal in any great volume under pathological circumstances (Fig. A2.1).

CSF enters the subarachnoid space and travels over the surface of the brain and spinal cord. Flow is directed caudally, hence the collection of CSF caudal to the site of a lesion. CSF is absorbed chiefly through one-way valves called arachnoid villi composed of arachnoid granulations protruding into the venous sinuses. The flow of CSF is thought to be due to pulsations of intracranial arteries. CSF flow is also enhanced by compressing the jugular veins in the Queckenstedt manoeuvre.

QUECKENSTEDT MANOEUVRE

Compression of both jugular veins decreases venous return from the brain, thus increasing the blood volume within the skull. This increase in intracranial content is compensated by CSF leaving the cranial vault thus raising the pressure in the spinal subarachnoid space.

Queckenstedt noticed that the lumbar CSF pressure was slow to rise in cases of spinal cord compression. The increased CSF pressure could not be transmitted past the site of subarachnoid obstruction. The diagnostic utility of the Queckenstedt test, or jugular compression test, has been replaced by imaging techniques. It remains a useful method for increasing CSF flow during lumbar puncture.

Enlargements of the subarachnoid space are called cisterns. The cisterna magna lies between the caudal surface of the cerebellum and the dorsal surface of the medulla oblongata and is the site of CSF collection. The quadrigeminal cistern lies dorsal to the tectum of the midbrain. The term quadrigeminal refers to the paired rostral and caudal colliculi of the tectum.

The subarachnoid space extends to surround the optic nerves, the spinal roots and blood vessels entering the CNS (Fig. A2.2). CSF analysis is therefore a useful tool in the diagnosis of optic neuritis, polyradiculoneuritis and vasculitis.

CSF production (Table A2.1) is constant and determined by the osmotic pressure of the blood. This can be manipulated in treatment of increased ICP and hydrocephalus.

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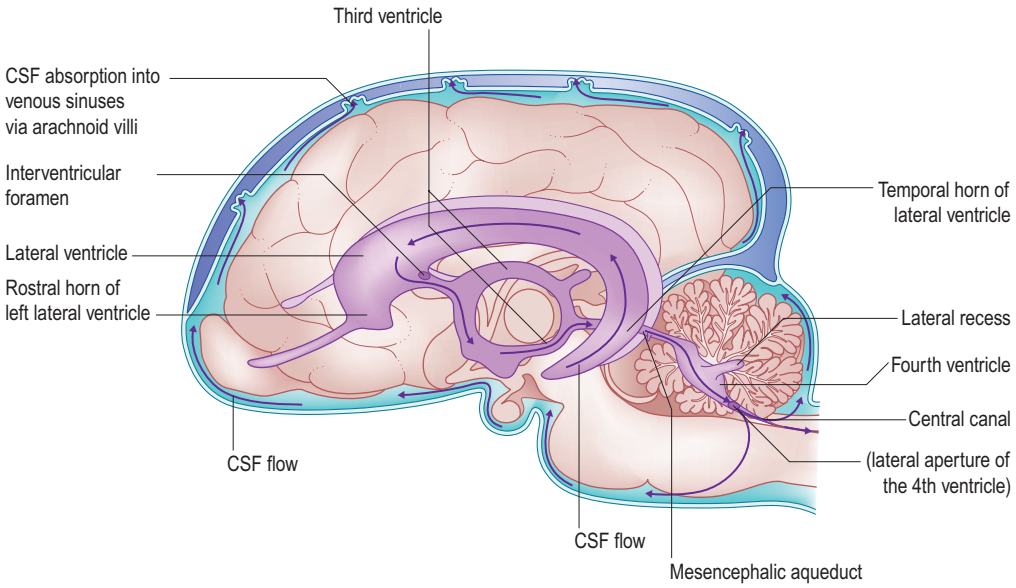


Figure A2.1 The ventricular system.

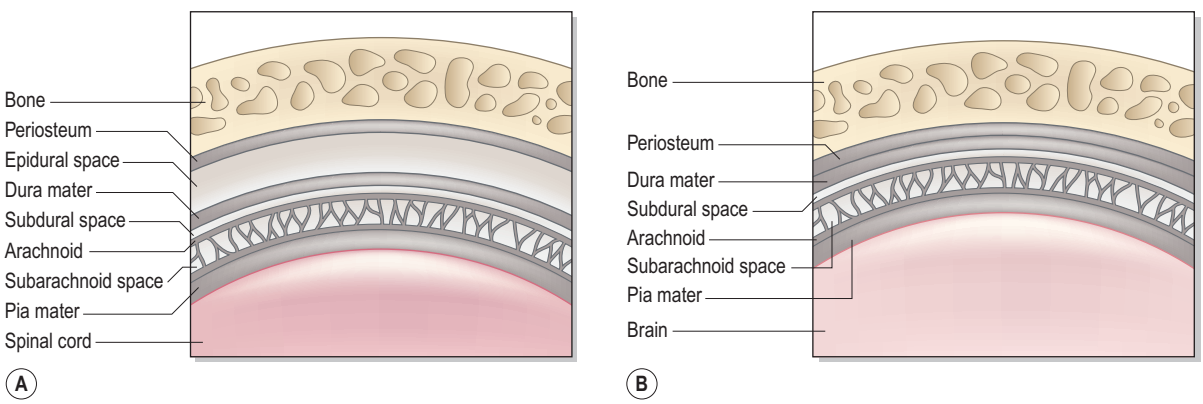


Figure A2.2 Relationship of the meninges to the CNS and surrounding bone of the (A) vertebrae and (B) skull.

Table A2.1 CSF dynamics

	Formation	Movement	Absorption
Site	Choroid plexus in all ventricles (major) Ependymal surface Blood vessels of the pia and arachnoid	Lateral ventricle to 3rd ventricle to aqueduct to 4th ventricle to subarachnoid space	Arachnoid villi in the venous sinuses (major) Transependymal, with increased ventricular pressure (minor)
Influenced by	Osmotic pressure of blood Independent of blood pressure	Pulsation of blood vessels, obstruction of pathway, increased ICP	ICP CSF pressure exceeds venous pressure (normal occurrence)
Dysfunction	Excess formation by choroid plexus tumours	Obstruction of pathway causes hydrocephalus	Rare. Absorptive capacity is 4 times greater than the productive capacity

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3 APPENDIX Collection of CSF

INDICATIONS FOR CSF COLLECTION

1. Spinal pain
2. Fever of unknown origin
3. CNS lesions, especially if MRI is normal
4. Inflammation of nerve roots, optic nerve, meninges, CNS or blood vessels
5. Periventricular or meningeal tumour
6. Suspected brachial plexus tumour (collect from lumbar site)
7. Prior to injecting contrast agent in myelography.

INCREASED RISK ASSOCIATED WITH CSF COLLECTION

1. Bleeding disorder
2. Increased intracranial pressure
3. Unsuitable candidate for general anaesthesia
4. Cervical instability, e.g. atlantoaxial subluxation
5. Lack of experience with the technique.

TECHNIQUE FOR CSF COLLECTION

1. Choose the site for collection
   - Caudal to the lesion.
   - The site closest to the lesion.
   - Both cisterna magna and lumbar punctures. Useful for inflammatory disease.
     - Cisterna magna at the atlanto-occipital space (Fig. A3.1).
     - Lumbosacral ventral subarachnoid space at the vertebral level of L4–5 or L5–6 (Fig. A3.2).
   - The skin is clipped and cleaned as if for surgery. A fenestrated drape may be used to cover the site and landmarks are palpated through this.
2. Insert the CSF needle
   - 40 mm length CSF needles are long enough for the cisterna magna in all but large breed dogs.
   - 65–90 mm CSF needles are required for lumbar taps in obese small dogs and larger dogs
   - Use the smaller 22-gauge needle for cisterna magna and lumbar taps in small dogs, cats, kittens and puppies. A 21-gauge needle can be used in most dogs for cisterna magna taps. The small gauge slows CSF flow. The clinician must wait and check for CSF flow before inserting the needle further.
3. Advance the needle
   - The skin offers the most resistance to advancement of the needle. Either cut the skin with a scalpel blade and insert through this small gap, or, tent the skin and push the needle sharply through the gathered tissue. Relax the skin and realign the needle with the landmarks for insertion.
   - Some clinicians advance the needle with the stylet in place. Others remove the stylet.
   - Flexion of the neck tenses fascial planes and a 'pop' may or may not be felt as the needle advances through layers of tissue.
   - The atlanto-occipital membrane occasionally feels firm to touch like inserting a needle into orange peel. Other times there is no resistance whatsoever. Do not rely on any particular sensation to signal the arrival of the tip of the needle into the cisterna magna. The distance between the skin and the subarachnoid space in kittens and puppies is frighteningly small.
   - Insertion of the needle through the cauda equina usually results in brief muscle twitching of the tail and hindlimbs. Ask the assistant holding the hindlimbs to watch out for this.
4. Reposition the needle if necessary.
   - If the needle hits bone keep on the midline, walking the needle forward or back in small increments.
   - If CSF does not flow, remove the needle from the animal. Reinsert the stylet and check for block-

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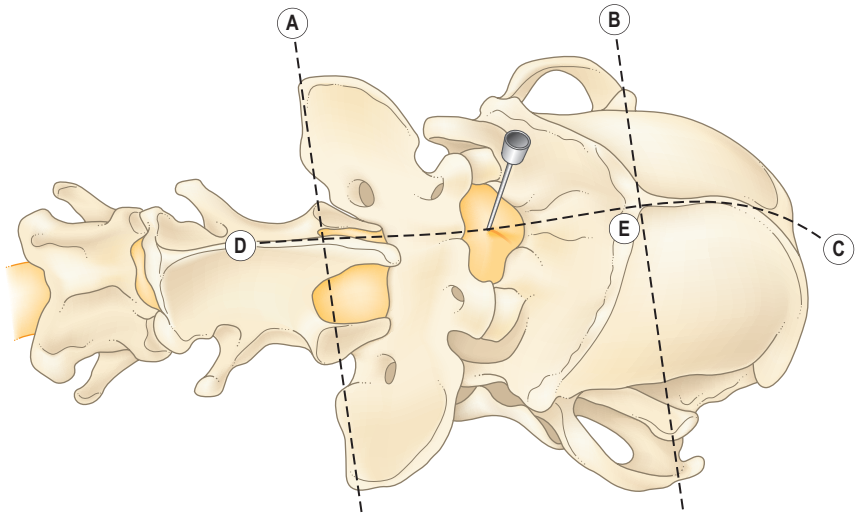


Figure A3.1 Collection of CSF from the cisterna magna. Insert the CSF needle halfway between line A and line B, on the midline (line C). To find the midline, palpate and align the dorsal spine of C2 (marked D) and the occipital protuberance (marked E).

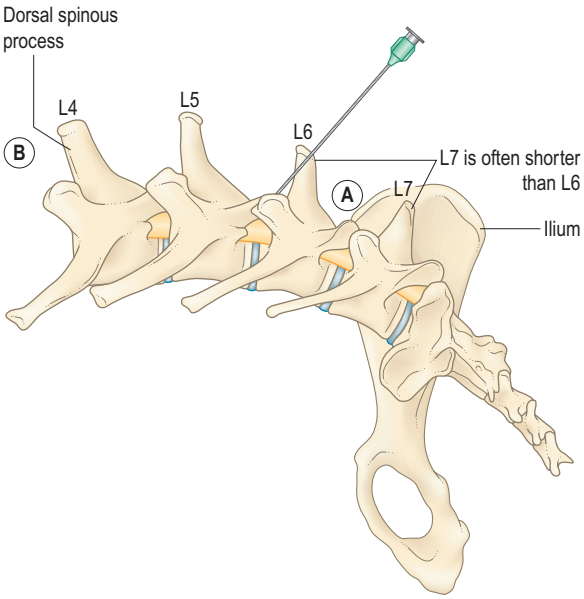


Figure A3.2 Collection of CSF by lumbar puncture. Flex the lower spine. Palpate the cranial border of the wings of the ilium (marked A), and locate the midline (marked B). Locate the dorsal spinous process of L6 (normally taller than that of L7). Insert the needle between L5 and L6 on the midline. Keep in the midline and adjust the angle of entry if required. A muscle twitch of the hindlimbs and tail indicates entry through the cauda equina.

ages. If tissue is found, smear and stain the material. Change needle or reinsert with the stylet in place. *The most common cause of lack of CSF flow is poor positioning*. Ensure the assistant is properly briefed on positioning the animal before the needle is inserted.

5. Collect CSF
   - CSF is constantly produced. Collect as much as required for a diagnosis; 1–2 ml is usually adequate.
   - Allow the CSF to drop into the collection container. Do not use a syringe, as this results in iatrogenic haemorrhage.
6. Submit for analysis
   - The protein content of CSF is low and EDTA is not required to stop the sample from clotting.
   - If iatrogenic haemorrhage results in clotting, another sample is required.
   - If only a small volume can be collected, decide on the diagnostic priority.
     - Cell count or cytology is most useful in suspected meningitis, lymphoma, or fungal infection.
     - The protein level for compressive lesions such as extension of a nerve root tumour into the spinal canal.
     - Culture for suspected bacterial meningitis.

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4 APPENDIX CSF analysis

Table A4.1 shows CSF dynamics.

CELL COUNT AND CYTOLOGY

Normal

- WBC <0–5/μl
- RBC 0/μl
- Cytology: lymphocytes and monocytoid cells.

Abnormal

- RBCs: phagocytosis of RBCs is a sign of previous haemorrhage.
- WBCs: an increased cell count is called pleocytosis. It is categorized by the total cell number:
  - Mild: 6–25/μl
  - Moderate: 26–100/μl.
  - Markedly elevated: >100/μl.

It is also categorized by the predominant cell type: neutrophilic, lymphocytic, mononuclear, eosinophilic, histiocytic, or mixed. There is no standard definition of these terms. One author defined a category as having ≥60% of one cell type, or that the percentage of one cell type was ≥40% of the total with no other cell type ≥33% of the total number of WBC. Mixed pleocytosis was defined as having no cell type ≥40% of the total WBC count, or as having two cell types each >33% of the total. WBCs enter the CSF with iatrogenic haemorrhage. The number is generally insignificant and it cannot be accurately predicted by the ratio of WBC to RBC in peripheral blood.

TOTAL PROTEIN LEVEL

Normal: Cisterna magna <25 mg/dl. Lumbar <35–45 mg/dl.

Albuminocytologic dissociation refers to an elevation of total protein level in the presence of a normal WBC count.

IgG INDEX

$$\text{IgG index} = \frac{\text{CSF IgG: serum IgG}}{\text{CSF albumin: serum albumin}}.$$

- <0.5: no evidence of intrathecal IgG production
- 0.5–0.75: intrathecal IgG production possibly raised
- >0.75: intrathecal IgG production is raised.

CSF PRESSURE

Measuring CSF pressure is of little diagnostic use as it varies so widely in the normal animal over a 24 hour period.

Table A4.1 CSF dynamics

	Formation	Movement	Absorption
Site	Choroid plexus in all ventricles (major) Ependymal surface Blood vessels of the pia and arachnoid	Lateral ventricle to 3rd ventricle to aqueduct to 4th ventricle to subarachnoid space	Arachnoid villi in the venous sinuses (major) Transependymal, with increased ventricular pressure (minor)
Influenced by	Osmotic pressure of blood Independent of blood pressure	Pulsation of blood vessels, obstruction of pathway, increased ICP	ICP CSF pressure exceeds venous pressure (normal occurrence)
Dysfunction	Excess formation by choroid plexus tumours	Obstruction of pathway causes hydrocephalus	Rare. Absorptive capacity is 4 times greater than the productive capacity

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5 APPENDIX Imaging the nervous system

Interpreting images of the CNS and its bony protection requires symmetry. General anaesthesia is necessary to achieve this. Survey radiographs of trauma patients are a satisfactory compromise in the emergency setting. Relaxation of the spinal muscles during anaesthesia or sedation increases the risk of subluxation and spinal cord injury in the unstable spine. Imaging the brain may need to be delayed while signs of intracranial dysfunction are stabilised.

RADIOGRAPHY

Key points: High mA, low KvP, grid, collimation and symmetry.

MYELOGRAPHY

Myelography is a contrast study of the spinal cord in which iodinated contrast material is injected into the subarachnoid space. Needle placement is the same as for collecting spinal fluid and is made closest to the site of the lesion. A lumbar tap at the L5–6 intervertebral space is appropriate for thoracic and lumbar lesions. If the site of interest lies in the cauda equina, injection of contrast at the cisterna magna will avoid injection artefacts in the area of interest. Cervical cord lesions are outlined by injections at the cisterna magna. If cervical instability is present a lumbar tap avoids the need to flex the neck.

The volume of contrast to inject varies based on the area to be outlined; 0.3 ml/kg–0.5 ml/kg is usually adequate. Obese animals may need slightly less: choose the dose based on the optimum body weight of the breed.

The injection should be done in a calm unhurried manner, with constant gentle pressure being applied. Some clinicians like to insert the syringe directly into the hub of the spinal needle, while others, those with shaky hands, might prefer to prefill a small extension attached to the syringe of contrast agent, and insert that instead.

After cisternal injection, the animal should be placed in sternal recumbancy with the head elevated to allow the contrast agent to flow caudally. If spinal instability is suspected, keep the neck in a neutral position and tilt the table top to achieve the same result. The animal is not repositioned after a lumbar injection, and remains in lateral recumbancy for the first post-injection radiographs to be taken.

Ventrodorsal images of the neck require the endotracheal tube to be removed. The ET tube creates linear artefacts that interfere with interpretation (Figs A5.1–A5.4).

Oblique views are useful for determining the circumferential location of lesions.

Seizures are reasonably common after injections of contrast agent at the cisterna magna. They are short-lived and occur during anaesthetic recovery. Treatment with IV diazepam usually suffices and the animals do not go on to develop chronic seizures. Paresis and ataxia may worsen after myelography. This is usually, but not always transitory.

Myelography is a difficult technique. It should not be attempted unless the clinician can deal with the aftermath of diagnosis, non-diagnosis and side-effects. This is only a brief outline of the technique. Other imaging techniques may be more appropriate.

MAGNETIC RESONANCE IMAGING (MRI)

Magnetic resonance produces digital images based on the differing mobility and quantity of hydrogen nuclei in tissues. Radiation is not used.

The animal is placed on a moveable gantry with the body part under investigation positioned on or in a coil. The gantry moves the animal into the tube or ring-shaped magnet. The magnet aligns the freely-moveable hydrogen ions of the body. Energy at radio frequency is

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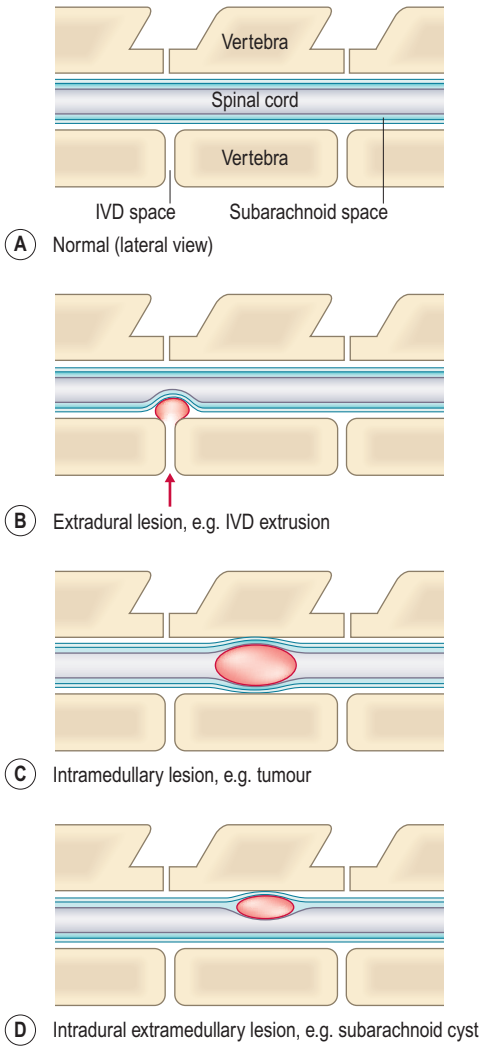


Figure A5.1 Myelographic patterns. Lateral view. (A) Normal; (B) Extradural, e.g. IVDD; (C) Intramedullary, e.g. tumour; (D) Intradural-extramedullary, e.g. subarachnoid cyst.



Figure A5.2 Lateral myelogram. Extradural compression of the cord, ventrally, over C6–7 IV space. Calcified disc material (arrow).

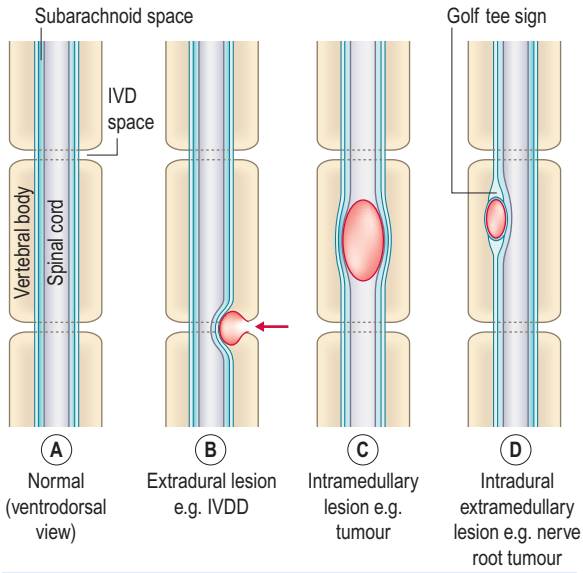


Figure A5.3 Myelographic patterns. Ventrodorsal view. (A) Normal; (B) Extradural, e.g. IVDD; (C) Intramedullary, e.g. tumour. A midline extradural lesion may have this appearance on VD view. (D) Intradural-extramedullary, e.g. nerve root tumour.

applied via a coil to the body part. The hydrogen ions are displaced to different degrees in different tissues. When the radiofrequency is turned off, the hydrogen ions relax back to the original aligned position. This releases energy which is detected and converted to an image.

The magnet strength remains constant but by altering the duration and timing of radio frequency application different image sequences are obtained which highlight tissue contrast in different ways.

Paramagnetic intravenous contrast agent, a chelate of gadolinium is used. It alters the relaxation of hydrogen ions and hence their appearance on MRI. Gadolinium can be seen in areas of damaged blood–brain barrier (pathology), absent BBB (pituitary, choroids plexus) or an incomplete BBB (neovascularization). Blood vessels may be seen. Some normal structures enhance. Some abnormal tissue will not.

Terminology

The appearance of tissue is described by its relative signal intensity. Soft tissue can vary in appearance in different

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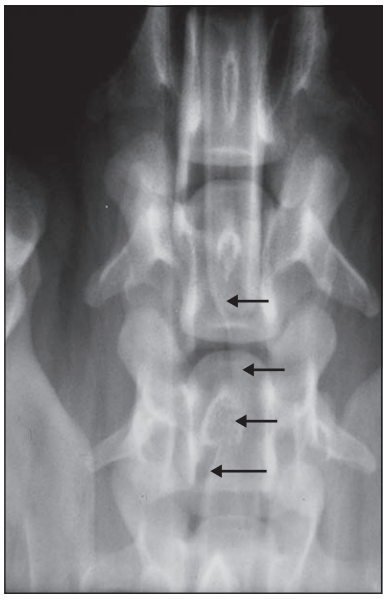


Figure A5.4 VD cervical myelogram. Right-sided extradural compression (arrows). IVDD.

sequences, and this sequence must be stated to clarify the comparison.

Hyperintense: e.g. the mass is hyperintense on *T2-weighted images* (*T2WI*).

Isointense: e.g. the mass is isointense to grey matter on *T1-weighted images* (*T1WI*).

Hypointense: e.g. the lesion is hypointense to grey matter on T1WI.

Heterogenous intensity: the tissue appearance varies throughout the structure.

Mnemonic for bright (hyperintense) on T1WI:

Fat – fat
Boys – blood
Mainly – melanin
Prefer – proteinaceous fluid
Chips – contrast agent.

Mnemonic for bright (hyperintense) on T2WI

T – tumour
W – water
O – oedema
FAT – fat.

GRE (gradient echo/T2) sequences detect lack of homogeneity in brain tissue and are sensitive for detecting haemorrhage. 'Black' hypointense areas appear within brain tissue.

Mnemonic for hypointensity on gradient echo images

Many – mineralization
Go – gas
Forth – fibrous tissue
In – iron deposits
Haste – haemorrhage.

CLINICAL TIP

The appearance of haemorrhage varies with time.

MRI is preferred for imaging of intracranial structures due to the soft tissue contrast, resolution within the caudal fossa, multiplanar imaging and absence of radiation. It is an ideal technique for investigating neck pain due to the cross-sectional images through the intervertebral foramen, sagittal views through the spinal cord and the potential to extend imaging to the cerebrum. It requires general anaesthesia to prevent movement artefacts (Fig. A5.5).

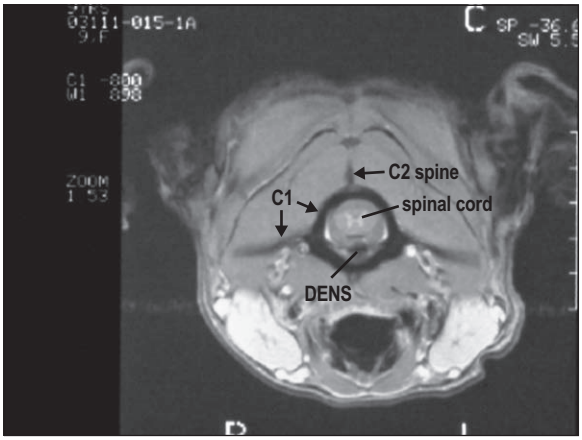


Figure A5.5 T1WI transverse image of C1. The dorsal spine of C2 and the dens are visible. Excellent grey and white matter differentiation.

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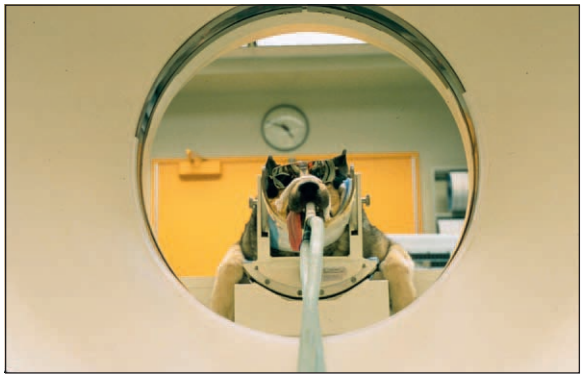


Figure A5.6 Dog positioned in CT machine.

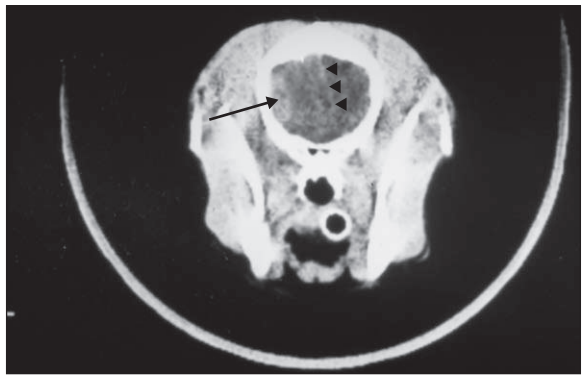


Figure A5.7 CT of brain haemorrhage (arrow). Midline shift (arrowheads).

COMPUTED (AXIAL) TOMOGRAPHY (CT)

CT generates images from a highly collimated X-ray beam rotating around the patient. This produces cross-sectional images similar to conventional radiography. Information is obtained in one plane. Reformatting from the original data provides other planes of view but with poorer resolution (Fig. A5.6).

CT gives excellent bone detail. Different tissue densities are compared using a grey scale of Hounsfield units (HU) (Table A5.1).

The contrast between tissues can be manipulated by altering 'windows' and 'levels' and by adding IV iodinated contrast agents. A region of interest can be examined and the HU measured pre- and post-contrast. Post-contrast enhancement >10 HU is abnormal (Fig. A5.7).

Terminology

Hyperdense: whiter, more attenuating, e.g. mineralization is hyperdense to brain parenchyma.

Isodense: equal in grey scale, similar in attenuation, e.g. the tumour is isodense to brain on pre-contrast images.

Hypodense: darker, less attenuating, e.g. oedema is hypodense to grey matter.

Table A5.1 CT grey scale in Hounsfield units (HU)

Substance	Attenuation (HU)
Air	–1000
Fat	–80
Water	0
CSF	0–15
Cerebral oedema	10–20
Brain white matter	25–35
Brain grey matter	35–50
Tumours	15–45
Flowing blood	20–50
Haemorrhage	0–80
Bone	>500
Dense metal	+1000

ULTRASONOGRAPHY

Ultrasonography through fontanelles may detect hydrocephalus. There is a poor correlation between ventricular size and clinical signs. MRI is preferred for intracranial anatomy and soft tissue resolution.

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6 APPENDIX Neuropathology

Clinical neurology has an undeserved reputation among students and general practitioners as simply being a delay to the inevitable delivery of the patient to the postmortem room.

In truth, a well-timed history and neurological examination alone, with no further investigation and no other expense, can sift the salvageable from the hopeless and prevent unnecessary euthanasia.

There will be occasions, despite one's best efforts, when euthanasia of a neurological case is required. Histopathology of the nervous system is very important. A definitive diagnosis is a teaching aid for the practitioner, an advancement of knowledge for the treatment of future patients and for advice to Breed Societies and most of all, a solace for the pet owner who has a final answer.

Asking a grieving owner for permission to perform a postmortem examination requires tact. Broach the subject after euthanasia has been discussed. Several conversational gambits have been helpful:

- Test results point to a few reasons why your pet died/isn't expected to recover. It would be immensely helpful to know precisely what has happened. It would help treatment of other pets and may lead to better outcomes in the future.
- Many owners telephone a few weeks later and ask for more information about why their pet died. Taking samples now will help answer that question.
- It is only by learning more from such cases that diagnosis and treatment improves.
- Looking at tissue samples is one of the best ways to learn about disease. In most cases, it is not possible to take samples of the nervous system while the animal is living.
- If the cause of death is thought to be infectious/toxic/inherited, this may have relevance for other animals you own.

REMOVAL OF THE BRAIN

This should be performed as soon as possible after death to preserve cell morphology and for ease of removal. If there is a delay, place the body in the fridge, not the freezer. Freezing and thawing damages cells. If delay is unavoidable and there isn't a fridge big enough and the head cannot be decapitated, then placing the animal in a freezer is better than allowing liquefaction at room temperature (Fig. A6.1).

1. Incise skin dorsally from the nose to the wings of the atlas, exposing the masticatory muscles and the neck.
2. Remove muscle from the skull bilaterally by incising the temporalis muscles at the midline and reflecting them laterally. This muscle is so thick that to gain good skull exposure often requires incising the muscle to allow folding back over the zygomatic arch.
3. Expose the occipital bone by severing all muscle attachments and reflecting muscle caudally (Fig. A6.2).
4. Ventroflex the neck and severe the cord between the occipital condyles and the atlas (Fig. A6.3).
5. An oscillating saw is required to enter the skull of larger dogs. Rongeurs may be used in cats or small dogs.
6. Cut across the frontal sinus. This exposes the inner calvarium which is then cut (Fig. A6.4).
7. On each side, make a cut cranial to caudal from the frontal sinus to the foramen magnum (Fig. A6.5).
8. Take care sawing through the thin temporal bone, the inner lamina of the frontal bone and the occipital bone caudally to avoid incising the brain.
9. Prise off the skull. Use a lever or grab the skull with rongeurs between the frontal bones and pull the top of the skull dorsally and caudally. Ideally the saw will have removed all obstruction to removal.

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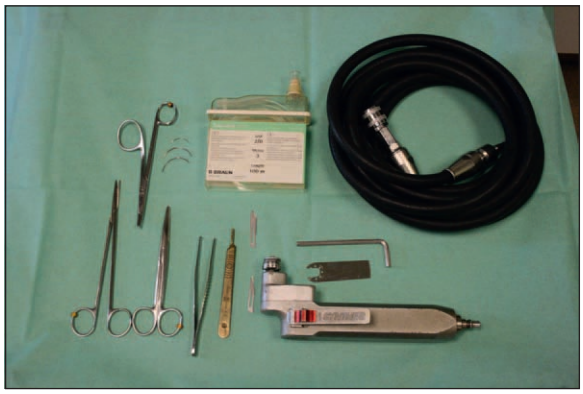


Figure A6.1 Equipment for postmortem examination. Hacksaws from the hardware store can be used but take 'forever' to breach the skull.

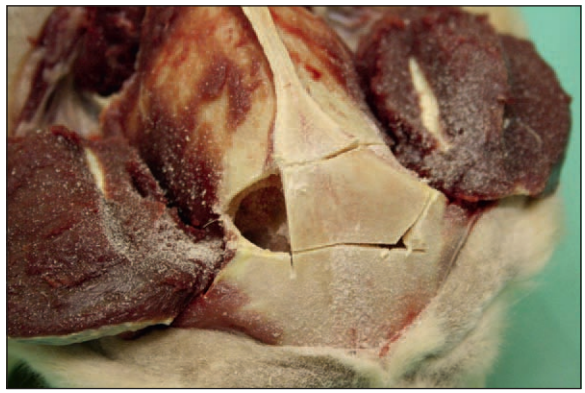


Figure A6.4 Entering the frontal sinus.

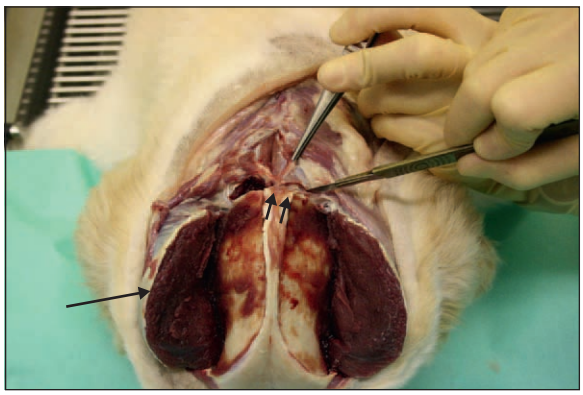


Figure A6.2 Temporalis reflected bilaterally (arrow). Removing muscle from occipital bone (double arrows).

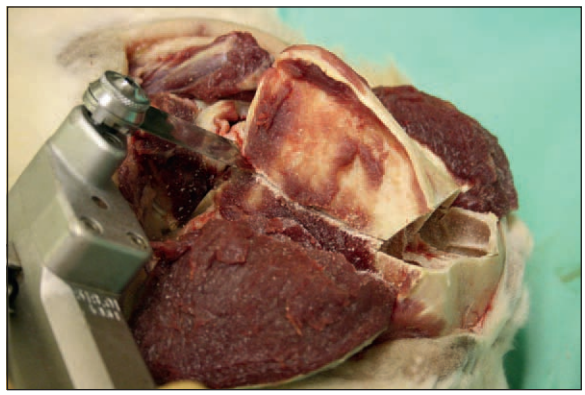


Figure A6.5 Incising the skull.

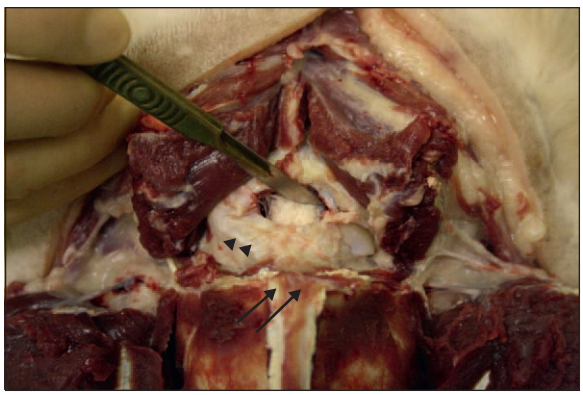


Figure A6.3 Occipital bone (double arrows). Condyles (arrowheads). Cutting the spinal cord.

The bony tentorium cerebelli will need gentle retraction to free it from attachments (Figs A6.6–A6.8).

10. Examine the inner surface of the skull. Place to one side. Examine the appearance of the meninges and then incise them, exposing the brain.
11. Ventroflex the neck so that the brain starts to fall out of the skull. (It will not actually do so at this stage because of the cranial nerves.) Using rat tooth forceps grab the meninges around the cord and gently pull the brain forward to expose the ventral cranial nerves (Fig. A6.9).
12. Severe the nerves close to the foramen. The olfactory bulbs should be severed as close to the ethmoid bone as possible. The blunt end of a scalpel blade holder serves this purpose (Fig. A6.10).

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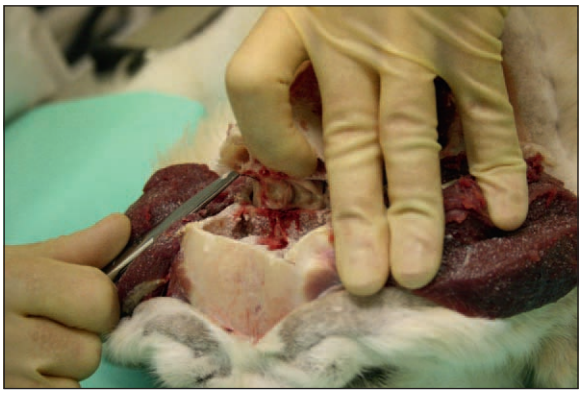


Figure A6.6 Lifting off the skull.

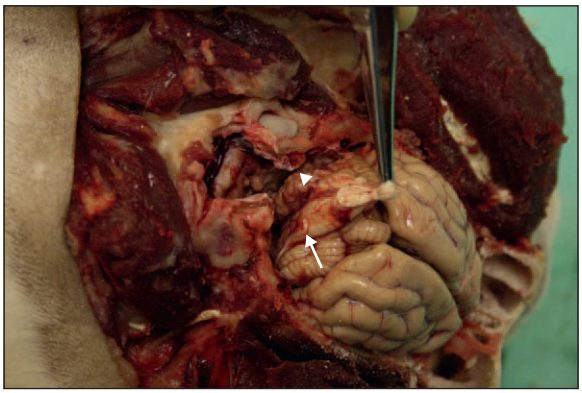


Figure A6.9 Retracting brain to gain access to the cranial nerves (arrow). Meningioma visible (arrowhead).

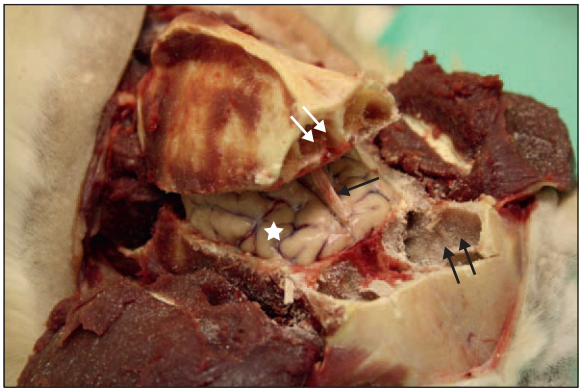


Figure A6.7 Falx cerebri (single arrow). Frontal sinus (double arrows). Brain (star).

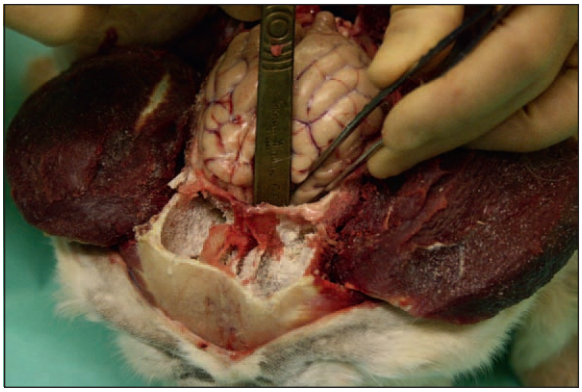


Figure A6.10 Severing the olfactory bulbs.

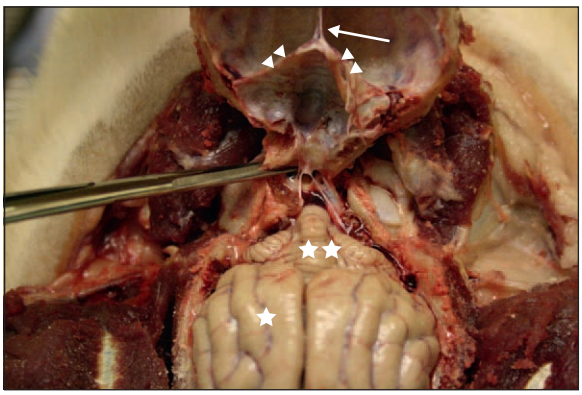


Figure A6.8 Cutting the final attachments. Falx cerebri (arrow). Tentorium cerebelli (arrowheads). Cerebrum (one star) Cerebellum (two stars).

13. The brain should gently fall into your hand. After briefly marvelling at the complexity of life look at the surfaces of the brain for discoloration and symmetry. Avoid touching the brain excessively (do not hand it around for examination, for instance). Gentle palpation may reveal firmness, undulation and yielding. Do not cut into the brain looking for pathology; this is done after fixation.
14. Immerse the brain in a large container filled with 10% buffered formalin. There should be at least 10 times the volume of formalin as brain tissue. All brain tissue should be covered by the fixative.
15. Seal and label the container. The brain should fix for 1 week. It can then be removed from the formalin and placed inside several sealable plastic bags. This is then wrapped in a padded protective layer prior to being sent to the neuropathologist.

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16. If the whole body is to be sent to the pathologist, make sure the history and results of any diagnostic tests accompany the animal. It is an excellent idea to telephone the pathologist ahead of time to discuss the case and the estimated time of arrival of the animal.
17. It is extremely important that the body be disposed of according to the owner's wishes, e.g. individual cremation with or without return of the ashes. Fill out all the necessary paperwork or instruct the pathologists to do so.

Cosmetic removal of the brain

This is possible. Do not shave any fur off the head. Once the brain is removed, replace the top of the calvarium. Consider padding the empty brain case with cotton wool to approximate the normal skull size. Suture the masticatory muscles together across the midline. Suture subcutaneous tissue. Clean and blow-dry the fur. The eyes, if not removed for examination, are sunken. Consider gluing the eyelids shut.

REMOVAL OF THE CORD

1. Incise the skin along the midline.
2. Reflect all tissue from the dorsal laminae of the vertebral column.
3. Using an oscillating saw or rongeurs perform a dorsal laminectomy.
4. Grasp the dura and gently retract the spinal cord out of the canal, cutting the spinal roots close to the intervertebral foramina. This ensures the dorsal root ganglion is also collected.
5. Cut the dura along the length of the cord to improve fixation by the 10% buffered formalin.
6. Immerse in 10× the volume.
7. Examine the vertebral canal for fractures, masses and IV disc disease.

INTERPRETING THE NEUROPATHOLOGY REPORT

Basic neuroanatomy (see Figs A6.11–A6.14).

Basic terminology

CNS

Neuron

- Chromatolysis is a response to injury and is characterized by cell swelling and lysis of Nissl substance causing a loss of basophilia.
- Axonal reaction is a regenerative change within the neuron following loss or damage to its axon.
- Neuronophagia refers to phagocytosis of a necrotic neuron.

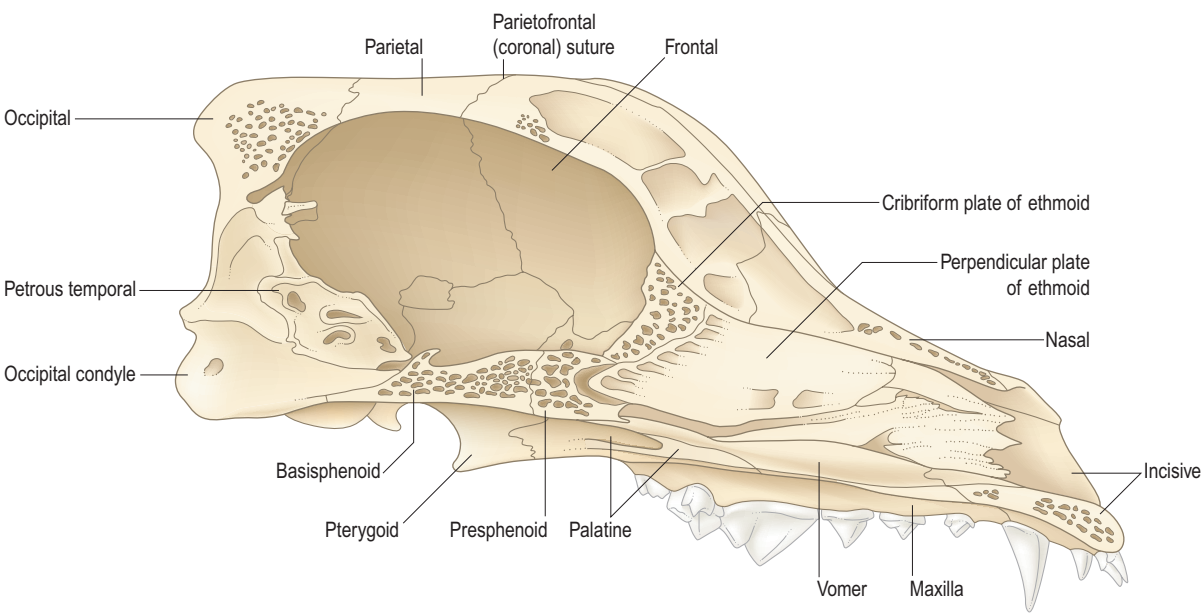


Figure A6.11 Bones of the skull.

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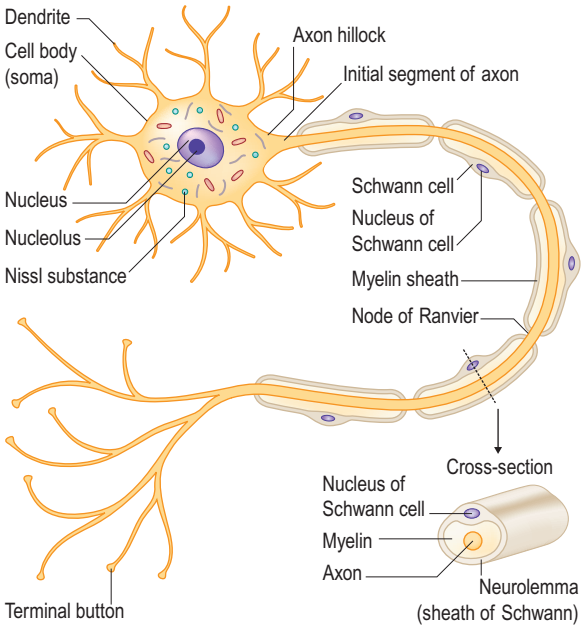


Figure A6.12 PNS: components of a myelinated axon.

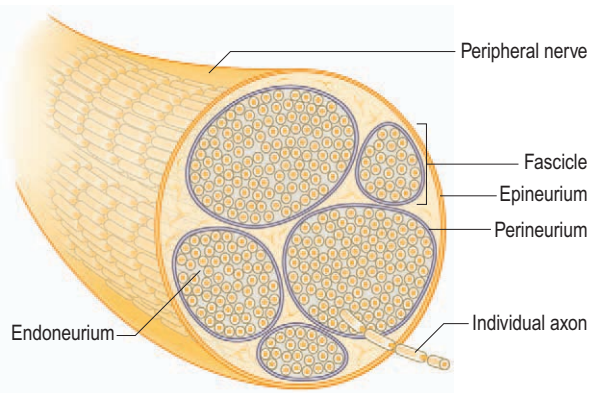


Figure A6.13 PNS: components of a peripheral nerve.

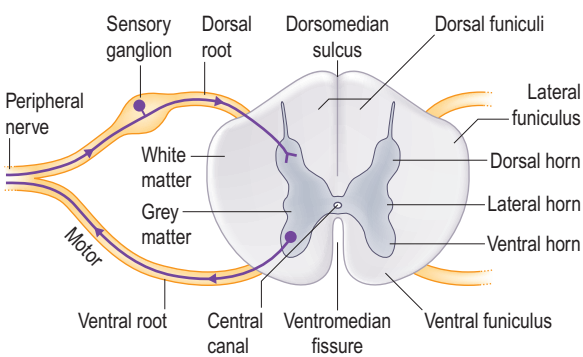


Figure A6.14 Cross-section of the spinal cord.

- Nissl bodies/granules/aggregations/substance are basophilic rough endoplasmic reticulum and polyribosomes in the cytoplasm.
- Dying-back refers to degeneration of the distal part of an axon due to failure of axonal transport.
- Spheroids are focal swellings of the distal axon within the CNS.
- Siderosis refers to necrotic neurons covered by calcium and iron salts.
- Internode is the segment of axon covered by myelin.
- Node of Ranvier is the space separating the internodes.
- Demyelination is the loss of normal myelin.
- Lysosomes are cytoplasmic membrane-bound organelles functioning in waste-disposal.

Neuroglia

- Satellitosis is the term for the proliferation of satellite cells around the neuron body following injury.
- Gliosis is proliferation, hypertrophy, or both, of glial cells (astrocytes and microglial cells) as a non-specific reaction to injury.
  - Astrocytes
    - Star-like cell
    - Astroblasts are immature astrocytes
    - Astrocytosis is proliferation of astrocytes
    - Astrogliosis refers to increased number of fibrous processes
    - Gemistocytes are large, plump reactive astrocytes formed in response to injury
    - Glia limitans is the network of astrocytes that covers the CNS, lying adjacent to the ependyma, pia mater and perivascular space.
  - Oligodendrocytes
    - Cell with few branches
    - Found in grey and white matter. Source of CNS myelin.
  - Microglial cells
    - Microgliosis is the proliferation of microglial cells
    - Found in grey and white matter.

General

- Leuko-: prefix denoting an association with the CNS white matter.
- Polio-: prefix denoting an association with the CNS grey matter.
- -itis: suffix denoting inflammation, e.g. mening*itis*, inflammation of the meninges.
- Ischaemia refers to a deficiency of the blood supply to a body part due to obstruction of circulation by a thrombus, embolus or vasoconstriction.

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- Infarction is tissue necrosis secondary to ischaemia.
- Malacia is pathological softening or softness.
- Embolus is a fragment of foreign material travelling within the vascular system from one site to another.
- Embolism is the sudden blockage of an artery by an embolus.
- Thrombus refers to an aggregation of blood products within the cardiovascular system forming a clot. A blood clot.
- CNS macrophages are transformed microglia, blood monocytes or blood neutrophils.
- Status spongiosis is a sponge-like vacuolation of nervous tissue.
- Pseudorosettes are cells arranged radially around blood vessels.
- Hallmarks of CNS inflammation are perivascular cuffing, gliosis, neuronal satellitosis and neuronophagia.
- Perivascular cuffing is the accumulation of leukocytes around a blood vessel.
- Angiocentric refers to the pathological process centred around blood vessels.
- Syncytial refers to the merging of cells to form a multinucleate cellular mass.
- Virchow–Robin space (perivascular space) is the space between glia limitans and the blood vessel.
- Anaplasia is characterized by a loss of differentiation of cells. Characteristic of neoplasia.
- Malignant refers to the tendency to worsen and end in death.
- Benign means non-recurrent. The chance of recovery is favourable.
- Interstitial refers to occupying the intervening spaces or crevices of a tissue.

PNS

Neuron

- Wagnerian degeneration describes neurologists at the Oktoberfest.
- Wallerian degeneration means fragmentation and phagocytosis of the nerve segment and myelin distal to the site of nerve transaction.
- Neurapraxia is a functional, not structural, disturbance of nerve conduction due to injury.
- Axonotmesis is axonal damage which spares the endoneurial and Schwann cell scaffolding enabling subsequent axon regeneration. Wallerian degeneration occurs.
- Neurotmesis is the traumatic sectioning of all or most nerve structures. Wallerian degeneration occurs.
- Fascicle is a bundle of nerve fibres.
- Epineurium is the connective tissue surrounding multiple nerve fascicles.
- Perineurium is the connective tissue surrounding individual nerve fibres.
- Endoneurium is the connective tissue between the individual nerve fibres.

Myelin

- Blebbing, ballooning, bubbling refers to focal separation and distension of peripheral myelin.
- Schwann cells encircle peripheral nerve axons thus forming their myelin sheath.

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7 APPENDIX Referral of a patient

WHEN TO REFER

- When you are unsure which organ system is involved
- When you recognize neurological deficits but cannot interpret the significance of the clinical findings
- When you can localize the lesion but you are unsure of the possible diagnoses and so are unable to make a diagnostic plan and offer a prognosis with and without various treatment options
- When you are concerned that you may have missed other subtle but important deficits
- When you desire a second opinion on the clinical variation in the case presentation
- When you lack equipment for a diagnosis or when you lack the time to perform or ability to interpret results
- When you are unsure of the treatment options
- When you are unable to perform the surgery or anaesthesia required
- When you lack expertise in monitoring the response to the treatment.

WHEN NOT TO REFER

- When the owner has no means or inclination to pay for the referral
- When the animal has not yet been examined by the general practitioner.

WHEN REFERRAL IS NOT AN OPTION

When you are unsure of the organ system involved or signs, such as inactivity, weakness, collapse, exercise intolerance or pain can be caused by dysfunction of various body systems such as the cardiac, endocrine, haemopoietic or orthopaedic systems, not just the nervous system, you should:

- Scour the history and re-question the owner. Ask specific questions. Clarify
- Repeat the physical examination
- Make a problem list
- Next to each problem, list which organ system could be involved
- List organs systems in order of probability of involvement
- Investigate the organ or body system
  - listed most frequently
  - easiest or cheapest to eliminate from the list
  - with most life-threatening illnesses/greatest morbidity/mortality
  - which have possibly treatable disease.

If you lack expertise with diagnosis:

- Some specialists can offer advice over the phone based on the primary veterinarian's examination findings but this is fraught with danger. If the examination misses deficits or if the history is inadequate it will not be possible to give an accurate assessment. Most of the diagnosis relies on the history and physical examination.

If you lack equipment or expertise at performing tests:

- Sometimes tests are not required. Check if the possible test outcomes would change any treatment plan, e.g. unilateral facial paralysis with no other signs, and no history of ear disease or head pain does not warrant skull radiographs and MRI of the head would be of little benefit to the animal.
- Non-contrast spinal radiographs are useful in cases of spinal pain to check for vertebral tumours, discospondylitis, but not for IVDD. If IVDD is severe enough to require surgery then contrast study or MRI is required. If it is not bad enough to require surgery then plain radiographs will not alter treatment. Sometimes it is not possible to differentiate infection, tumour and disc disease from the history, signalment

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and examination, in which case non-contrast radiographs are useful.
- CSF taps require practice. Know when to stop inserting the needle.

IF YOU ARE UNABLE TO TREAT

- A percentage of paralysed animals will recover with rest. Consider carts for mobility of paraplegics.
- Euthanasia for unremitting pain or poor quality of life remains a realistic option.

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