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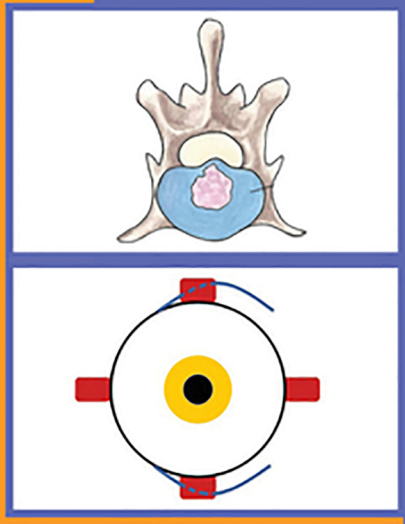
A Practical Approach to Neurology for the Small Animal Practitioner

Paul M. Freeman
Edward Ives



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'To all of my teachers, past and present, and to my mother Claire Ives,
the kindest teacher of all.'

Edward Ives
April 2020

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Preface

Neurology is still perceived by many students as one of the more difficult areas of veterinary medicine to understand. In clinical practice, neurological cases can also cause much anxiety, particularly in regard to clinical decision making when referral is not an option. Ed and I wanted to write a book for students and general practitioners that represents the way we feel neurology should be taught and understood, and one which would simplify a complex topic. There are many wonderful textbooks on the subject already in existence, and this text is in no way designed to compete with those excellent volumes, but rather we hope that it might prove to complement them as an approachable and useful companion for those looking for a better grasp of a complex subject. We have tried to design and produce a practical book full of hints and tips taken from our personal collective experience, which would be accessible as a quick reference guide for use in general practice, as well as hopefully being an easy-to-read textbook for final-year veterinary students. We have tried to include as many photographs and diagrams as possible to illustrate and simplify some of the more complex subject areas, and we are sure that the videos on the accompanying website will aid recognition of some of the less common clinical presentations. We have also included video clips of a normal neurological examination as a reference. This has been a labour of love for us and we are indebted to the many colleagues, both past and present, who have inspired, taught, and assisted us, and are still doing so. The book represents the product of many hours of study, conversation, observation, and clinical practice, and we hope that it may be used and enjoyed by many.

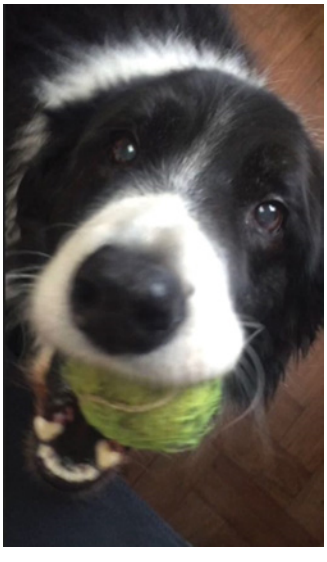
Paul M. Freeman and Edward Ives

Acknowledgements

The authors are Board-certified neurologists with a combined total of more than 30 years in general practice before entering full-time specialist practice. This has given us a great insight into the needs of general practitioners when it comes to dealing with neurological cases. We have both benefited from excellent teaching through the University of Cambridge, both for our veterinary degrees and later our residency training. We would like to acknowledge the support and advice of our current and past colleagues, as well as that of family and friends.

The original drawings for the book were created by Paul's son Jack Freeman, with digital images produced by Edward himself. We are grateful also to our clients, past and present, especially those of the Queen's Veterinary School Hospital, University of Cambridge, and Anderson Moores Veterinary Specialists, whose images and videos bring this text to life.

Finally, thanks to Tick, star of the neurological examination videos and Paul's long-time but now sadly deceased Border Collie, whose presence in the neurology office in Cambridge was a source of education and comfort for students and staff alike.



About the Companion Website

Don't forget to visit the companion website for this book:

www.wiley.com/go/freeman/neurology 

There you will find valuable material designed to enhance your learning, including:

- videos of the normal neurological examination and specific disease presentations

Scan this QR code to visit the companion website.

Chapter 1 'Is it Neurological?'

Paul M. Freeman

The first step to any neurological evaluation of a veterinary patient, and probably the most common question we are asked by practitioners (in a variety of different ways), is whether the problem facing them is partially or completely neurological. In order to answer this question, it is important to understand the nature of the problem from the point of view of both the animal and the owner, and furthermore to understand what is meant by the question 'Is it neurological?'. For example, when presented with a dog that is showing signs of exercise intolerance, one possible reason may be a neuromuscular disorder, such as myasthenia gravis. However, this dog may present with a completely normal neurological examination, and the clue may be in the presenting clinical signs or medical work-up. Therefore, in such a case the initial answer may have to be 'It might be'. A dog whose problem is intermittent 'episodes' of abnormal behaviour may be having seizures, but might also be suffering from syncope, a compulsive behavioural disorder, or a movement disorder. All of these possibilities may in some way be classified as 'neurological' problems, but with very different aetiologies, treatment possibilities, and prognoses. The goal of this book is to allow practising vets to feel confident that they are approaching potentially neurological problems in a reasonable and evidence-supported way. The first step in this process is learning how to recognise when a particular presentation may be caused by a disease process somewhere within the central nervous system (CNS) or the peripheral nervous system (PNS).

1.1 What Is the Problem?

One of the key skills to be developed in general practice, as well as referral practice, is learning how to control the consultation in order to establish what the client's primary concern is, what they hope to achieve from their visit with you as the veterinarian, and how their expectations match up to their ability and/or

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willingness to afford and allow potential investigations and/or treatments to be performed. This is not an easy skill, especially for the GP vet who may have very limited time in which to carry out the initial consultation. It is, however, key to both ultimate client satisfaction and to providing the most effective service to the animal patient. Without establishing these essential facts, much time can be wasted. In the worst case the client's real concerns may not be addressed at all, meaning that they may leave the practice dissatisfied, perhaps with good reason.

Top tip: Always ask the client directly why they have come to see you today.

Don't make this mistake: Take care not to become side-tracked by a problem which may be very interesting to you, but is possibly chronic and completely unrelated to the reason for the visit!

1.2 Is this Problem Neurological?

There are many possible manifestations of neurological disease, some of which are much easier to recognise as neurological than others. There are also many non-neurological diseases which can mimic a problem involving the nervous system. In this section, we will look at the scope of neurological disease manifestations that the clinician may be presented with and we will aim to provide some clues as to the correct recognition of neurological disorders.

1.3 Neuroanatomy

When attempting to decide whether the problem is neurological, it is important to be aware of the different parts of the nervous system and how disease processes may affect them. Broadly we are concerned with the CNS (the brain and spinal cord) and the PNS (consisting of the peripheral nerves and muscles and the neuromuscular junctions between them). In the brain we can generally distinguish clinical signs referable to the forebrain, the brainstem, and the cerebellum.

The forebrain is known as the prosencephalon, and can be further divided into the telencephalon, which consists of the cerebral hemispheres, and diencephalon, containing the thalamus and the hypothalamus. The group of clinical signs which are commonly caused by lesions affecting the forebrain are sometimes referred to as a prosencephalic syndrome, and include behavioural change, central blindness, and seizure disorders in particular.

The brainstem consists of the midbrain (mesencephalon), the pons (ventral metencephalon), and the medulla (myelencephalon). Disease of the brainstem

also leads to characteristic signs, which can be used to anatomically localise the problem, including proprioceptive deficits and ataxia, sometimes clusters of cranial nerve signs, vestibular syndrome, and mentation change associated with dysfunction of the ascending reticular activating system.

The third major division of the brain is the cerebellum (dorsal metencephalon), and lesions in this region can lead to some of the most recognisable abnormalities in the neurological examination, including hypermetria and intention tremor.

The medulla is contiguous with the spinal cord, which can be divided into a series of segments from which a pair of spinal nerve roots arises, one pair for each segment. For neuroanatomical localisation purposes, the spinal cord segments are grouped together according to their motor function, and whether or not they contain the cell bodies of the nerves which directly supply the skeletal muscles of the limbs (known as lower motor neurons, LMNs). In this regard, the first five cervical spinal segments (C1–C5) contain only the so-called upper motor neurons (UMNs) which run from the gait-generating centres of the cerebral cortex and brainstem to the LMNs innervating the limbs and other structures. Spinal cord lesions affecting segments in this region lead to a characteristic set of neurological examination findings involving UMN effects in all four limbs.

The sixth, seventh, and eighth cervical segments, along with the first two thoracic segments, C6–T2, contain the LMN cell bodies supplying the thoracic limbs, as well as UMNs to the pelvic limbs; lesions here cause a so-called LMN paresis or plegia (paralysis) of thoracic limbs and an UMN paresis/plegia of the pelvic limbs.

Lesions in spinal segments caudal to the second thoracic segment will generally only affect the pelvic limbs, and the division between UMN and LMN here occurs between the third and fourth lumbar segments. Hence, lesions affecting the T3–L3 spinal cord segments lead to UMN paresis of pelvic limbs, whereas lesions caudal to L3 (L4–S3) cause LMN paresis/plegia of the pelvic limbs.

As well as the descending motor tracts within the spinal cord, there are of course ascending sensory tracts including those carrying proprioceptive information from the limbs and trunk; therefore, a spinal cord disorder will usually lead to variable degrees of ataxia and proprioceptive dysfunction, as well as the paresis associated with the loss of motor function.

The final part of the nervous system within which we can make an anatomical localisation consists of the peripheral nerves, neuromuscular junctions, and muscles. Lesions affecting this neuromuscular system tend to lead to more obvious weakness and LMN paresis/plegia affecting all limbs. Therefore, syndromes such as weakness, stiffness, exercise intolerance, and collapse may all arise as a result of disease affecting this PNS.

The neurological examination and neuroanatomical localisation of specific lesions will be explored and explained in more detail in Chapters 3 and 4, but an understanding of the anatomical structures involved in neurological disorders is

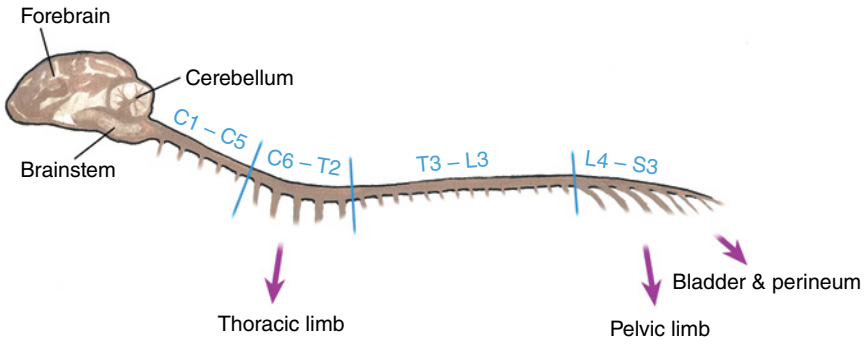

Figure 1.1 Neuroanatomical regions. The aim of the neurological examination is to localise the site of a lesion into the brain, the spinal cord, or peripheral (neuromuscular) regions. Functionally, the brain is separated into the forebrain, brainstem, and cerebellum; the spinal cord into sections containing the segments 1st cervical – 5th cervical (C1–C5), 6th cervical – 2nd thoracic (C6–T2), 3rd thoracic – 3rd lumbar (T3–L3), and 4th lumbar – sacral (L4–S3); and the peripheral nervous system consists of peripheral nerves, with the neuromuscular junctions and muscles also considered as a potential neuroanatomical localisation.

an essential part of the ability to recognise when one is facing a neurological problem (see Figure 1.1).

1.4 Manifestations of Diseases of the Nervous System

Disorders that affect different parts of the nervous system may manifest themselves in a wide variety of ways. In this section, we will briefly look at each of these, with the most important and more common problems being examined in greater detail in Chapter 6. The majority of nervous system disease will result in one or more of the following clinical presentations, some of which can be more easily defined and recognised than others. Where relevant, non-neurological disorders that can mimic or also result in these clinical presentations will also be mentioned:

- seizures
- collapse
- movement disorder/dyskinesia
- mentation change
- behaviour change
- blindness
- deafness
- tremor
- paresis/plegia
- ataxia

- abnormality of head position (head tilt/turn)
- abnormality of eye position or movements
- hypaesthesia
- lameness
- pain
- disorders of micturition/urination.

1.4.1 Seizures

For the purposes of this text, an epileptic seizure will be defined as an acute onset of excessive and hypersynchronous brain activity that results in transient visible motor activity; this description does not allow for so-called 'absence seizures', where a person may become transiently 'absent', since no motor activity is seen in such a seizure. However, such seizures, although they may occur in cats and dogs, are very difficult to diagnose without the help of an electroencephalogram (EEG).

It is rare that an affected animal will seizure during the consultation, so usually we rely on owner description and often video evidence when trying to decide if a problem is truly an epileptic seizure or some kind of seizure mimic. A generalised seizure is normally easily recognisable (see Video 1); the seizure may begin with a focal contraction of, for example, the facial muscles, but this rapidly spreads to cause loss of conscious state and tonic contraction of all the anti-gravity muscles leading to recumbency. There follows a period of tonic and clonic muscle activity causing running-like movements, and there may be vocalising, hypersalivation, jaw champing, and often urination or defecation. The tonic-clonic movements gradually subside and the animal will frequently adopt paddling movements as consciousness returns and attempts are made to stand. There will usually then follow a post-ictal period, which may last for minutes or hours, when the animal may show varying levels of altered behaviour, circling, ataxia, blindness, and other neurological abnormalities.

Focal seizures may involve simple focal muscle twitching, commonly of the orofacial muscles (see Video 2), but may also include more complex patterns of movement and/or behaviour – leading to apparent loss of consciousness and awareness. Such activity can be very difficult to distinguish from obsessive behavioural abnormalities and movement disorders for instance, but there are some key indicators which may be helpful in recognising when a problem is a true focal epileptic seizure (see below). A specific form of focal seizure is the myoclonic seizure, characterised by jerky twitches resembling the dog being fearful of a sudden stimulus, and seen especially in the genetic storage disease of Miniature Wire-haired Dachshunds, Beagles, and Bassett Hounds known as Lafora Disease (see Video 3).

Epilepsy implies a tendency for seizures to recur and can be further classified as 'reactive/metabolic', when there is an extracranial cause, 'structural', when there is an abnormality of brain structure such as an inflammatory or neoplastic lesion, and 'idiopathic', when brain structure is normal.

It is important to try to ascertain whether the abnormal event described by the owner or captured on video is an epileptic seizure or not. This is because the occurrence of true seizures implies a possible prosencephalic or forebrain lesion and dictates a certain path of investigation. The initiation of antiepileptic medication is always a subject for discussion and consideration, since all antiepileptic drugs (AEDs) can have undesirable side-effects, and their use in non-seizure disorders is usually contraindicated.

The description above contains some clues to the features of epileptic seizures that can be used in order to ascertain whether the event described is truly an epileptic seizure or represents a 'seizure mimic'. These include:

1. *Presence of post-ictal signs*. This is common and can include ataxia, blindness, polyphagia, or a temporary character change such as becoming aggressive.
2. *Presence of autonomic signs during the event*. In itself this is not necessarily pathognomonic for an epileptic seizure, but involuntary salivation, urination, and/or defecation is more common in seizure events than syncope, and is rarely observed for movement disorders.
3. *Absence of consciousness during the event*. It can be difficult to be sure about this, especially in mild focal seizures. However, the ability to rouse an animal from the behaviour, as is the case for idiopathic head-bobbing for instance, is an indication that the event is probably not a seizure.
4. *Presence of some form of tonic muscular activity, twitching, or stiffness*.

There are many other causes of paroxysmal transient events, or 'seizure mimics', and these should always be considered when trying to decide whether or not we are dealing with an epileptic seizure. These include:

- *Syncope, which may have a cardiovascular or respiratory cause*. Most cases of syncope result in a flaccid collapse, but this is again not always the case, and there is a significant 'grey zone' of signs seen with seizures and syncope which can make them difficult to distinguish. This is also the case in human medicine, with a significant percentage of people being diagnosed with epilepsy when in fact they are suffering syncopal attacks.
- *Dyskinesia or movement disorder*. These paroxysmal disorders of movement are being increasingly described, often with specific breed associations. Examples include the epileptoid cramping syndrome of Border Terriers (which has been associated with a gluten sensitivity and recently renamed paroxysmal gluten-sensitive dyskinesia, see Video 4) and episodic falling in Cavalier King Charles Spaniels (CKCSs), which has a known genetic mutation associated with it. Although these syndromes are fairly well described, the fact that the CKCS also suffers with idiopathic epilepsy can make this an even more complicated picture in this breed.

- *Behavioural events*. These are often described as obsessive compulsive or OCD behaviours, such as some cases of so-called 'fly-catching'. The latter can also be a focal seizure, and has even been associated with floating bodies within the anterior chamber of the eye, or gastrointestinal pain (see Video 5).
- *Transient vestibular events*. Perhaps associated with transient ischaemic attacks or TIAs.
- *Narcolepsy/cataplexy*. This is a rare condition with a suspected genetic basis in certain breeds, but can look very much like the so-called 'drop seizures' described in humans (see Video 6).
- *Idiopathic generalised tremor syndrome* or so-called 'little white shaker disease' (see Video 7). This is an inflammatory condition of the CNS leading to a generalised tremor. The tremor can wax and wane, worsens with excitement, and is potentially confusable with an animal suffering with a prolonged seizure or status epilepticus, such as may be seen following exposure to certain toxic substances (e.g. aflatoxins or mouldy food).
- *Episodic pain*, such as that associated with some cervical intervertebral disc herniations, may cause episodic, short-lived behavioural and physical changes. The French Bulldog in particular seems prone to such manifestations (see Video 8).
- *Episodic collapse*, which may have a neuromuscular, metabolic, or orthopaedic cause; if recovery is rapid; such events may be mistakenly diagnosed as seizures.
- *Rapid eye movement (*REM*) sleep disorder*. This is a condition where affected dogs may suffer violent episodes of paddling and limb movement during periods of REM sleep (see Video 9). It has recently been shown to have a higher incidence in dogs recovering from tetanus (Shea et al. 2018).

Top tip: Seizures can have multiple causes but also have multiple mimics.

Careful observation of video footage, together with accurate history taking, is key to defining whether the presenting problem is truly a seizure.

Ultimately, the only sure way to define a seizure (currently categorised as a Tier 3 level of diagnostic confidence for idiopathic epilepsy) is by making EEG recordings of abnormal brain activity during the abnormal event. Even in specialist referral practice this is rarely an option, and therefore we are usually working at a lower level of confidence.

1.4.2 Collapse

Collapse implies a loss of extensor motor tone, and may be either intermittent or permanent. When an owner describes a problem of collapsing, usually they are referring to episodic collapse, since a permanent collapse is immediately obvious!

Differential diagnoses for intermittent collapse include seizures, syncope, narcolepsy/cataplexy, and metabolic disorders (such as hypoglycaemia). More permanent collapse may be caused by musculoskeletal and orthopaedic problems, inflammatory conditions such as polyarthritis or polymyositis, as well as medical conditions involving weakness, anaemia, or pain. Neurological causes of loss of extensor muscle tone or strength may include brain, spinal cord, and PNS disorders. The neurological examination is therefore key to identifying, firstly, whether the problem is neurological, and then obtaining a correct neuroanatomical localisation.

Care must be taken in interpretation of the findings of the neurological examination in cases of collapse; an animal which is suffering from shock, trauma, or acute pain may well show apparent neurological deficits without any neurological disease or pathology. Examples include animals in shock which apparently have poor proprioception and spinal reflexes, lack of menace response, and even absent deep pain response. Once the shock and pain has been appropriately managed, the neurological abnormalities may disappear.

Top tip: Be careful with interpretation of the neurological examination in severely shocked or traumatised animals.

Don't make this mistake: Diagnosing severe spinal trauma due to apparent loss of deep pain perception post-trauma, especially in cats following a road traffic accident. Once stabilised, the neurological examination may change, indicating a less severe injury.

1.4.3 Movement Disorders

Movement disorders are an increasingly recognised group of conditions which affect many dog breeds. These conditions involve abnormal muscular movements, occasionally episodes of collapse, but no change of consciousness or autonomic signs that may be seen in focal epileptic seizure events (see Video 10). Well-documented examples include episodic falling in CKCSs ('Collapsing Cavaliers'), paroxysmal gluten-sensitive dyskinesia of Border Terriers (epileptoid cramping syndrome or 'Spikes disease', see Video 4), 'Scotty cramp' in Scottish Terriers and 'dancing Dobermann' disease.

Some of these conditions have a known genetic cause, and a specific test may be available (e.g. episodic falling in Cavaliers), but for many there is no specific diagnostic test. Dancing Dobermann disease is suspected to be a polyneuropathy, Spike's disease has been shown to be caused by a hypersensitivity to gluten in the diet, and it may be that some so-called movement disorders are in fact focal seizures.

In all of these conditions, the neurological examination should be normal, and in general all investigations are likely to be unremarkable (with the exception of specific genetic tests for example). The diagnosis is often one of exclusion and pattern recognition, and for many of the conditions there is no specific treatment available.

Further discussion of movement disorders and how they may present in practice can be found in Section 6.2 'Movement Disorders'.

1.4.4 Mentation and Behaviour

Observation and recognition of changes in either behaviour or mentation can be crucial in recognising that one is dealing with a neurological problem. Mentation concerns the level of general alertness and is sometimes referred to as the *level* of mentation (as opposed to the *quality* of mentation, which may be better be understood as behaviour). Mentation levels can be categorised as:

1. *Normal*
2. *Obtundation*. A generalised reduction in level of alertness or interaction with the environment; an animal which appears to show an absence of the usual anxiety and stress associated with a visit to the veterinarian, a lack of normal interaction with the owner, a 'can't be bothered' or 'couldn't care less' attitude. There can clearly be a marked difference between mild and severe obtundation, and milder levels may be variously described as lethargy or depression, although these terms should be used with caution in animal species.
3. *Stupor*. This implies a level of mentation in which the animal is basically asleep or semi-asleep, and only arousable by a noxious stimulus.
4. *Coma*. An animal which is not rousable even by a noxious stimulus.

A change in the level of mentation frequently implies neurological disease; stupor and coma are most commonly seen in moderate to severe brainstem disorders where the ascending reticular activating system is affected. This causes a reduction in sensory input to the cerebral cortex. Forebrain disease may also cause altered mentation, and in particular pituitary macroadenomas can present with obtundation as the only neurological sign.

Focal cerebrocortical lesions more rarely present with obviously altered mentation although larger lesions may, including compulsive pacing or circling behaviour. Animals with generalised encephalopathies, such as those seen in portosystemic shunting for instance, often show an altered level of mentation, sometimes intermittently. Another important observation is that raised intracranial pressure (ICP) can affect mentation, and serial observation and monitoring of mentation level in animals with suspected head trauma or with other reasons for potentially raised ICP (usually as part of the modified Glasgow Coma Scale) is extremely important in allowing recognition of a deteriorating situation/rising ICP.

Top tip: Altered mentation may be the only sign of severe intracranial pathology.

It is also important to be aware that altered mentation can occur as a result of non-neurological disease. Animals which have significant medical disorders,

pyrexia, anaemia, or pain may in some situations show moderate to severe obtundation without any primary neurological disease. As already mentioned, animals which have suffered significant trauma or stress, for example after a road traffic accident, may also show an apparently reduced level of mentation. In these situations, mentation may be expected to improve if the underlying problems are corrected, and this should allow a reduced suspicion of the presence of a primary neurological disorder.

Changes in behaviour or the quality of mentation usually imply involvement of the cerebral cortex. Behavioural changes can be caused by many things, including ageing, stress, change of environment or home situation, metabolic disease, chronic pain, dietary change, etc. Therefore, taking a careful clinical history is crucial in cases where a change of behaviour forms part of the presenting complaint. If a more subtle behavioural change is reported during the history-taking related to the investigation of a problem for which altered behaviour was not immediately apparent, then this may raise the level of suspicion for a possible intracranial disease.

Altered behaviour is less commonly the primary clinical sign reported for neurological disease, possibly due to our relative lack of sensitivity as veterinarians and owners in being able to identify more subtle changes. However, there are occasions when an owner will have recognised a loss of learned behaviour, or a change in demeanour or willingness to play in their pet, and this may be significant. As already stated, neurological disease leading to behavioural change usually implies a prosencephalic lesion and, in such situations, careful performance and interpretation of the neurological examination may identify other abnormalities consistent with a lesion affecting the cerebral cortex.

1.4.5 Blindness

An acute onset of blindness is usually noticed by pet owners, since it may be extremely disorientating for the affected animal, and may lead to behavioural changes as well as more obvious signs such as bumping into objects. It is often the case that vision is lost progressively rather than acutely, but in these situations owners frequently do not notice the more subtle changes brought about in their pet by a reduction in the quality of their vision, and it is only when the problem has progressed to complete or near-complete blindness that the owner becomes aware. When presented with an animal with reduced vision, it is therefore important to question the owner carefully in order to elucidate whether the visual loss may in fact have occurred progressively rather than acutely.

A blind cat or dog may be reluctant to move around, become ‘clingy’ or fearful in situations where they were previously relaxed and confident, and may even develop obsessive or phobic behaviours. For neuroanatomical evaluation, the important thing to establish is whether the blindness appears peripheral or central. Central blindness implies dysfunction of the central visual pathways and/or visual cortex; this is usually rare in the absence of other signs of intracranial

disease. More commonly, vision may be lost in one eye as a result of a lesion affecting the contralateral visual cortex, but as vision remains normal in the other eye, the owner either may not be aware of the vision loss or may have noticed the animal bumping into objects on only one side. The menace response will be absent in the affected eye, but the pupillary light reflex (PLR) (as well as palpebral reflex and other cranial nerves) are likely to be normal. Use of the so-called 'cotton-ball' test (see Chapter 3) may also help to identify visual loss in one eye.

In general, animals that present as completely or almost completely blind with no other neurological abnormalities, will have lesions affecting either the optic nerves or the eyes themselves. In the case of ocular disease, if there are no grossly obvious abnormalities such as cataracts, then the retina is the region most likely to be affected. A fundic examination should be performed and may reveal evidence of retinal disease or detachment. In the absence of an obvious cause for sudden blindness, then the two conditions which must be considered are sudden acquired retinal degeneration syndrome (SARDS) and optic nerve disease; optic nerve disease can have a number of possible causes (see Section 6.4 'Blindness').

SARDS is a syndrome that usually presents as an apparent acute onset of complete blindness, although careful history taking may reveal evidence that vision has deteriorated over a period of weeks. There is frequently an accompanying history of polyphagia, polydipsia, and polyuria and sometimes behavioural changes. Menace responses are absent bilaterally, and PLRs are reduced although may still be present to some degree. Routine biochemistry can reveal increased alkaline phosphatase and cholesterol, and there is often a high suspicion of hyperadrenocorticism although this is rarely confirmed. Despite the problem being associated with retinal degeneration of an unknown cause, fundic examination is usually normal at least in the early stages. Diagnosis can be made with a high degree of certainty from the history, physical examination, and biochemistry findings, but confirmation requires the presence of a bilaterally abnormal electroretinogram. The prognosis for return of sight is poor, although other systemic signs may improve over time.

The optic nerves can be affected by neoplastic disease that either infiltrates the nerves, such as lymphoma, or that compresses the optic chiasm, such as pituitary macroadenomas. Inflammatory disease affecting the optic nerves is more common and can be either confined to the optic nerves as the condition 'optic neuritis', or present as part of a more generalised inflammatory condition of the CNS (such as meningoencephalitis of unknown origin, MUO). In optic neuritis cases, it is sometimes possible to visualise swollen optic nerve heads during the fundoscopic evaluation. Other neurological abnormalities may be observed in cases with MUO, such as postural reaction deficits or other cranial nerve abnormalities. Cerebrospinal fluid analysis frequently confirms the presence of inflammation.

Top tip: Acute blindness with reduced or absent PLRs and no other physical or neurological abnormalities usually indicates either SARDS or optic neuritis. In both cases, the prognosis is guarded but diagnosis is worth pursuing because some cases of optic neuritis are responsive to immunosuppressive therapy.

1.4.6 Deafness

Deafness is a rare presentation in isolation, but owners may report behavioural changes which could imply that deafness or reduced hearing may be present. Bilateral hearing loss may be very significant to the lifestyle and behaviour of individual animals, and its effects should not be underestimated. Unilateral deafness is more challenging to detect and may be more debilitating than is currently thought as a result of an inability to localise the source of sounds. Deafness is a difficult neurological deficit to confirm clinically, but careful testing may lead to a high index of suspicion. As with other conditions, careful clinical history taking may provide some clues as to the origin of the deafness. Physical examination should obviously include an examination of the external ear canals. Neurological examination may reveal other abnormalities which can give clues as to the origin of the deafness (e.g. vestibular signs, facial nerve paralysis, and Horner syndrome may all be associated with otitis media/interna).

Confirmation of deafness requires electrodiagnostic evaluation through the recording of brainstem auditory evoked responses (BAER). Deafness may be either conductive, associated with abnormalities of conduction of sound waves to the receptors in the inner ear (e.g. accumulation of debris in the external ear canal or fluid accumulation in the tympanic bulla), or sensorineural, associated with a problem in the cochlea of the inner ear or the cochlear nerve itself.

1.4.7 Tremor

Tremor associated with neurological disease can be broadly divided into head tremor and whole-body tremor. Head tremor is most commonly associated with cerebellar disease (see Video 11), when it may be permanent, but tremor often becomes more severe when an affected animal attempts to perform some action involving the head, such as eating or drinking (termed an 'intention tremor'). The tremor may be very mild and subtle, and occasionally has not been appreciated by the owner. If a head tremor is observed in the consultation, then a full neurological examination should be performed, particularly to look for other signs consistent with cerebellar disease.

A slower head 'tremor' known as idiopathic head bobbing is occasionally seen, particularly in young Boxers and English Bulldogs (see Video 12). The cause is unknown, the problem is intermittent, and is generally self-limiting. All investigative and diagnostic tests tend to be normal in affected dogs, and the head-bobbing movements may be vertical ('Yes') or horizontal ('No'). Affected dogs can usually be distracted during an episode; when distracted the head

bobbing will cease, and this can be used to distinguish this paroxysmal occurrence from a focal seizure.

Whole-body tremor is an uncommon presentation, but when seen can be severe and debilitating. The two most common forms of whole-body tremor are that associated with toxin ingestion (particularly tremorgenic mycotoxins found in mouldy food, metaldehyde, chocolate, and some prescription medications) and idiopathic generalised tremor syndrome (see Section 6.2 'Movement Disorders' and Video 7). Tremors resulting from toxicity usually affect the whole body, can be severe and unremitting, do not subside with recumbency, and may occasionally progress to generalised seizures. This is a neurological emergency and requires emergency treatment to prevent hyperthermia and hypoglycaemia. Diagnosis generally relies on thorough history taking and the clinical presentation.

Another type of tremor that may be encountered is orthostatic tremor. This type of tremor predominately affects the limbs in large and giant breed dogs (particularly Great Danes) and is present only when the limbs are weight-bearing (see Video 13). The condition is benign and there is no treatment. Occasionally, a limb tremor may be associated with orthopaedic disease, especially if there is significant muscle atrophy or pain. It can also occur in some older dogs, apparently as part of an ageing process, and this is termed an 'essential' or 'senile' tremor (see Video 14).

There are a few conditions affecting the myelination of axons which present as whole-body tremors in young puppies around 6–8 weeks of age. Breeds that are typically affected include the English Springer Spaniel, Chow, Samoyed, Weimaraner, and Dalmatian.

Top tip: The major differential diagnoses for an acute onset of generalised tremor in an adult dog are toxin ingestion and idiopathic generalised tremor syndrome ('little white shaker' disease); in the former the tremor is persistent and severe, in the latter it may wax and wane and disappears when the dog is at rest.

1.4.8 Paresis/Plegia

Paresis can be defined as a reduced ability to initiate or maintain motor activity. It may manifest as a reduced ability to support weight (LMN paresis) or a reduced ability to generate gait (UMN paresis). Plegia is defined as an absence of voluntary movement (i.e. paralysis). A plegic animal usually indicates that severe CNS injury has occurred, either at the level of the brainstem or neck for tetraplegia or caudal to the T2 spinal segment for paraplegia. Paraplegia, involving only the pelvic limbs, is far more common than tetraplegia, partly because any lesion in the CNS that is capable of causing tetraplegia may be accompanied by other severe effects which may include respiratory failure and death. When an animal is presented in such a condition, particularly if there is a history of possible or

definite trauma, great care should be taken when moving the patient in case there is instability of the vertebral column and the potential for further spinal cord damage.

In an animal that is plegic (and only in these animals), it is necessary to include the testing of so-called 'deep pain perception' (DPP), which is the ability of the animal to recognise a noxious stimulus applied to the deep structures of the foot or toe (see Chapter 3). The loss of DPP is a poor prognostic indicator in many situations, since the sensory fibres that convey this information are carried in many tracts which are located deep within the spinal cord. Their loss may therefore indicate spinal cord damage at the deepest level. However, DPP is *not* likely to be lost in animals which have less severe spinal cord injury and remain paretic; testing for DPP therefore need not be performed in such animals.

The neurological examination is critical to being able to localise the likely anatomical location of any lesion causing paresis or plegia. This particularly includes an understanding of the spinal reflexes. It is still a common mistake to interpret the presence of a normal pedal withdrawal reflex as an indicator that DPP is present, and this can lead to overly optimistic prognosis being given to owners of animals with severe spinal cord injury (see Chapter 3 and Video 15).

Don't make this mistake: In paralysed animals, make sure you understand the difference between testing spinal reflexes and testing for deep pain perception (see Chapter 3).

Disorders affecting the PNS can present as a relatively acute onset of tetraplegia or severe tetraparesis. In this situation, the differentiation from a spinal cord lesion can usually be made by the fact that the neurological deficits cannot be explained by a single, focal CNS lesion as the spinal reflexes are reduced in all limbs. The major differentials for acute onset severe tetraparesis/tetraplegia localising to the PNS are acute canine polyradiculoneuritis, fulminant myasthenia gravis, and botulism.

Milder forms of paresis may be much more difficult to identify and a degree of paresis affecting one or more limbs may be the only sign of a neurological problem. Close observation of gait, including retrospective and slow-motion video analysis, may be helpful in identifying a reduction in the quality of movement. UMN paresis occurs when the control or initiation of movement is affected by a lesion that is affecting either the gait-generating regions of the cerebral cortex or brainstem or the spinal cord pathways containing the UMNs descending to synapse with the LMNs in the brachial or lumbosacral plexi. UMN paresis may lead to a long-strided gait, toe dragging, and postural reaction deficits, but the affected limbs retain normal spinal reflexes and are not weak. LMN paresis, by contrast, causes a paresis like that seen in PNS diseases, characterised by weakness, an inability to support weight, and a short-strided, choppy gait. For