CHAPTER 2

Localization of Lesions in the Nervous System

In Chapter 1, the fundamentals of organizing a diagnosis, neuroanatomy, and neurologic examination were described. In this chapter, the principles of lesion localization will be described using the anatomic information described in Chapter 1.

LOCALIZATION TO A REGION OF THE SPINAL CORD OR THE BRAIN

Upper Motor Neuron (UMN) and Lower Motor Neuron (LMN) Signs

Examination of the motor system should allow the clinician to localize the lesion to one of five levels of the spinal cord or to the brain (Figure 2-1 and Table 2-1).

Gait and postural reactions detect paresis or paralysis; spinal reflexes detect LMN abnormality. The thoracic and pelvic limbs should be classified as normal or as exhibiting LMN or UMN signs (see Table 1-3). Briefly, LMN signs are paresis or paralysis, a loss of reflexes, and a loss of muscle tone. UMN signs are a loss of voluntary motor activity (paresis or paralysis), an increase in muscle tone, and normal or exaggerated reflexes. Note that with both UMN and LMN signs, paresis or paralysis is the primary finding. The status of the reflexes distinguishes between the two.

The examiner can localize a lesion to a region of the spinal cord or the brain by using these findings and the material presented in Figure 2-1, which is an algorithm that explains the logic of the diagnosis. For example, paresis in the pelvic limbs with normal thoracic limbs indicates that the brain and spinal cord as far caudal as T2 are functioning. Therefore, a lesion is caudal to T2. To determine whether the lesion is in the T3-L3 or L4-S2 region, the reflexes of the pelvic limb must be evaluated. If they are normal or exaggerated, the L4-S2 segments must be functioning and the lesion is between T3 and L3. If the reflexes are decreased or absent, the lesion is in the L4-S2 segments. Paresis or paralysis of all four limbs indicates a lesion cranial to T3. Reflexes are tested in all four limbs. Normal or exaggerated reflexes of all limbs indicate a lesion cranial to C6. Other findings are used to localize the lesion further. With a lesion cranial to C6, one should examine the cranial nerves and mentation to rule out brain disease. The sensory examination is further reviewed for possible signs related to the neck (e.g., C1-5).

Using only the information related to LMN and UMN signs of the limbs, the examiner can localize the lesion to one of the following regions: (1) the brain, (2) C1-5, (3) C6-T2-brachial plexus (thoracic limb), (4) T3-L3, (5) L4-S2-lumbosacral plexus (pelvic limb), or (6) S3-Cd5.

LOCALIZATION TO A SEGMENTAL LEVEL OF THE SPINAL CORD

Lower Motor Neuron Signs

If LMN signs are present in the limbs, the examiner can localize the lesion further by identifying the affected muscles. Table 2-2 lists spinal cord segments (roots) and peripheral nerves for the most commonly tested reflexes.

The examiner can localize within two to four segments or to a peripheral nerve if LMN signs are present. Spinal cord segments do not correlate directly with vertebral levels. When the examiner has determined the spinal cord level, Figure 1-8 can be referred to for an estimation of the vertebral level. As a general rule, the sacral segments overlie L5. This can be remembered because S (for sacral) resembles the number 5.

Peripheral nerve lesions usually cause monoparesis (paresis of one limb) because the most common lesions are the result of injury to a limb or a specific nerve. Localization of lesions in monoparesis is reviewed in Chapter 5. The primary exception is generalized peripheral neuropathies, which affect all of the limbs. These conditions are discussed in Chapter 7.

Sensory Perception

Hyperesthesia (increased sensitivity to noxious stimuli) is a useful localizing sign and may be present with little or no motor deficit. The animal's limbs and trunk, especially the

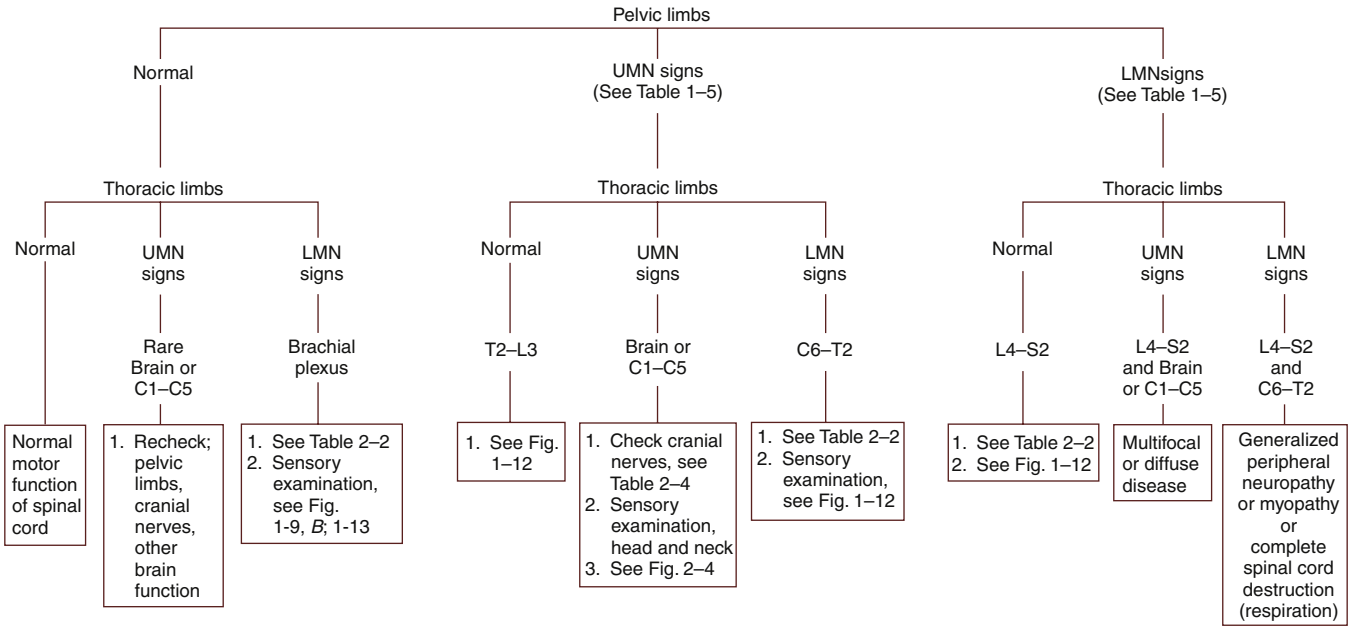

Figure 2-1 Localization of lesions based on motor function. *UMN,* Upper motor neuron; *LMN,* lower motor neuron; *C,* cervical; *T,* thoracic; *L,* lumbar; *S,* sacral spinal cord segments. (From Hoerlein BF: Canine neurology, ed 3, Philadelphia, 1978, WB Saunders.)

TABLE 2-1

Signs of Lesions in the Spinal Cord

Site of Lesion	Sign
Cd1-5	LMN—tail
S1-3	UMN—tail
Pelvic plexus	LMN—bladder
Pudendal nerve	LMN—anal sphincter, urethral external sphincter
L4-S2	UMN—tail
Lumbosacral plexus	LMN—hind limbs
	UMN or LMN—bladder, anal sphincter
T3-L3	UMN—hind limbs, bladder, anal sphincter
	LMN—segmental spinal muscles
C6-T2	UMN—hind limbs, bladder
Brachial plexus	LMN—forelimbs
C1-5 or brainstem	UMN—all four limbs, bladder

LMN, Lower motor neuron; *UMN,* upper motor neuron.

vertebral column, are palpated and manipulated while the examiner observes for signs of pain. Obvious reactions may include resistance to movement and tensing of the muscles. If the clinician places one hand on the animal's abdomen while squeezing each vertebral segment with the other hand, increased tension of the abdominal muscles may be felt as painful areas are palpated. The skin is pinched with a hemostat after palpation is completed. A fold of skin is grasped gently with the hemostat, and the skin is pinched lightly so that no significant behavioral reaction is elicited from normal areas. Pinching areas of hyperesthesia may elicit an exaggerated skin twitch or a behavioral response.

The superficial pain examination should be performed in a caudal to cranial direction because areas caudal to a lesion usually have decreased sensory perception (see Figures 1-16 and 1-39). A level of normal or increased sensory perception can be ascertained by this method. If a spinal lesion is present, the sensory level should have the conformation of a dermatome (see Figure 1-12). Peripheral nerves have a different pattern of sensory distribution (see Figure 1-13).

The cutaneous trunci (panniculus) reflex is elicited with a hemostat in the same manner as that just described for detecting hyperesthesia. Cutaneous sensation enters the spinal cord at each segment (dermatomes) and ascends to the brachial plexus (C8-T1) to the lateral thoracic nerve, which innervates the cutaneous trunci (panniculus carnosus) muscle. Contraction of the cutaneous trunci muscle causes a skin twitch. A segmental lesion blocks the ascending afferent stimulus, abolishing the reflex. Pinching the skin in a caudal to cranial direction identifies the first level at which the reflex can be elicited. This segment is normal, and the lesion is one segment caudal to this level. The superficial pain pathways must be damaged to abolish the reflex. Normally the cutaneous trunci reflex is most apparent in the thoracolumbar (saddle) area. A minimal response is obtained from a stimulus applied to the sacral or caudal regions, and no response is obtained from a stimulus applied to the cervical region. Cervical spinal hyperesthesia is assessed by manipulation of the neck and deep palpation of the vertebrae. Although defining the location of the pain precisely may be difficult, determining whether it is in the cranial, middle, or caudal cervical segments is usually possible by performing palpation carefully and gently.

Hypesthesia (decreased sensory perception) and anesthesia (lack of sensory perception) also are good localizing signs. Single nerve root lesions usually do not produce a clinically detectable area of decreased sensation because of the overlapping pattern of cutaneous innervation (see Figure 1-12). Multiple nerve roots may be involved in some lesions, especially in the area of the cauda equina. Lesions of the spinal cord may result in decreased perception of pain caudal to the lesion. Determining the level of sensory loss was discussed earlier, and the prognostic implications of the loss of sensation are discussed later. A carefully performed sensory examination localizes the lesion to within three segments of the spinal cord

TABLE 2-2

Spinal Reflexes

Reflex	Muscle(s)	Peripheral Nerve	Segments*
Myotatic (stretch)	Biceps brachii	Musculocutaneous	(C6), C7-8, (T1)
	Triceps brachii	Radial	C7-8, T1, (T2)
	Extensor carpi radialis	Radial	C7-8, T1, (T2)
	Quadriceps	Femoral	(L3), L4-5, (L6)
	Cranial tibial	Peroneal (sciatic)	L6-7, S1
	Gastrocnemius	Tibial (sciatic)	L6-7, S1
Flexor (withdrawal)	Thoracic limb	Radial, ulnar, median, musculocutaneous	C6-T2
	Pelvic limb	Sciatic	L6-S1, (S2)
Cutaneous trunci	Cutaneous trunci	Lateral thoracic	C8, T1
Perineal	Anal sphincter	Pudendal	S1-2, (S3)

Modified with permission from Oliver JE Jr: Localization of lesions in the nervous system. In Hoerlein BF, editor: Canine neurology, ed 3, Philadelphia, 1978, WB Saunders.
*Parentheses indicate segments that sometimes contribute to a nerve.

TABLE 2-3

Signs of Lesions in the Brain and Peripheral Vestibular System

Lesion Site	Mental Status	Posture	Movement	Postural Reactions	Cranial Nerves
Cerebral cortex	Abnormal behavior, and mentation, seizures	Normal; head turned toward side of lesion	Gait normal to slight hemiparesis (contralateral)	Deficits (contralateral)	Normal (vision may be impaired contralateral side)
Diencephalon (thalamus and hypothalamus)	Abnormal behavior and mentation, endocrine and autonomic dysfunction	Normal	Gait normal to hemiparesis or tetraparesis	Deficits (contralateral)	CN II, abnormal pupillary light reflex
Brainstem (midbrain, pons, medulla)	Dullness, stupor, coma	Normal, turning, falling	Hemiparesis or tetraparesis, ataxia	Deficits (ipsilateral or contralateral)	CN III-XII
Vestibular, central (cranial medulla)	Normal to depressed	Head tilt, falling (usually toward side of lesion)	Ipsilateral hemiparesis, vestibular ataxia	Deficits usually ipsilateral	CN VIII, may involve CN V and VII; nystagmus
Vestibular, peripheral	Normal	Head tilt, circling, falling, rolling	No paresis, severe vestibular ataxia	Normal	CN VIII, sometimes CN VII; Horner's syndrome, nystagmus
Cerebellum	Normal	Wide-based stance or normal unless paradoxical vestibular disease is present, decerebellate posture	No paresis; severe cerebellar ataxia, dysmetria, and resting and intention tremors	No paresis; dysmetria present	Usually normal. May see depressed menace response nystagmus, or vestibular signs

or to a peripheral nerve. For additional details on pain, see Chapter 14.

LOCALIZATION IN THE BRAIN

If the lesion has been localized to the brain, the next step is to determine what part of the brain is involved. Localization to one of five regions of the brain or to the peripheral vestibular apparatus (labyrinth) is made on the basis of clinical signs (Table 2-3).

BRAINSTEM

For our purposes, the functional brainstem includes the midbrain, pons, and medulla oblongata. Lesions of the brainstem produce UMN signs in all four limbs (tetraparesis) or in the

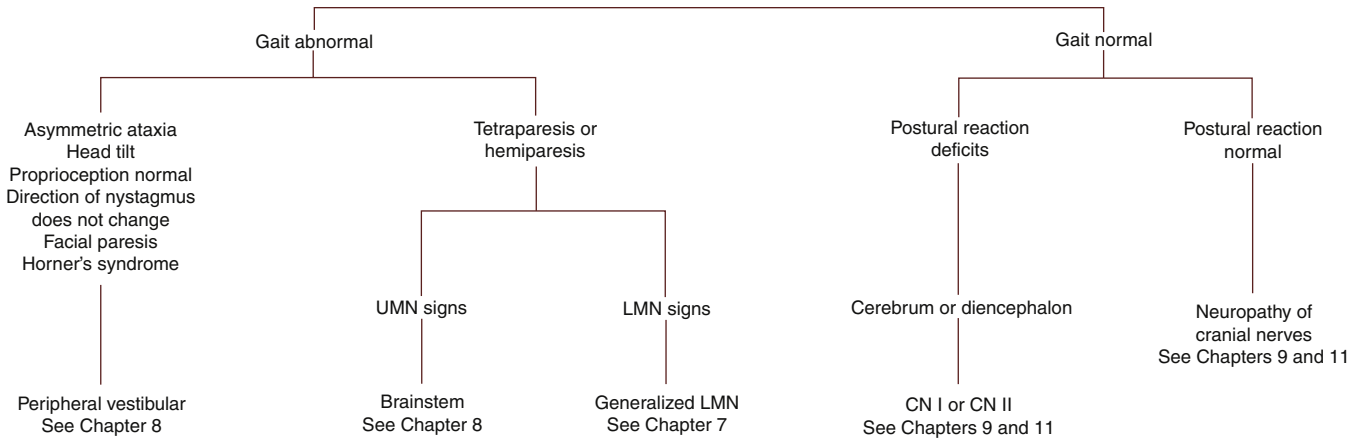

Figure 2-2 Localization of lesions causing cranial nerve signs.

thoracic and pelvic limbs on one side (hemiparesis). The paresis or paralysis produced by brainstem lesions is obvious both in the gait and in postural reactions. Cerebral or forebrain lesions affect postural reactions with minimal change in gait, although compulsive walking and circling may be seen. Brainstem or hindbrain lesions can cause abnormal posture resulting from vestibular involvement. Cranial nerve signs (CN III-XII) are present with larger or extensive brainstem lesions and provide important localizing signs (LMN or sensory) (Figure 2-2). The evaluation of cranial nerves is outlined in Table 2-4 (see Chapters 1, 9, 10, and 11).

Cranial nerve dysfunction with brainstem disease is ipsilateral to the lesion, whereas motor dysfunction may be ipsilateral or contralateral, depending on the level and the pathways involved. The animal's mental status may be altered, especially in lesions of the midbrain and the pons, which disrupt the ascending reticular activating system. Signs vary from dullness to coma (see Chapter 12).

Diencephalon

Diencephalic lesions (lesions of the thalamus or hypothalamus) may produce UMN signs in all four limbs (tetraparesis) or in the thoracic and pelvic limbs on one side (hemiparesis), depending on the symmetry of the lesion. The gait is not severely affected (similar to cerebral lesions), but postural reaction deficits may be present. The animal may circle in either direction depending upon lesion symmetry. CN II (optic) may be affected in diencephalic lesions with evidence of visual and pupillary light reflex abnormalities. Space-occupying lesions (e.g., tumors, abscesses) of the diencephalon also may affect CN III, IV, and VI depending on lesion extent (see Table 2-3). Cranial nerve signs are ipsilateral to the lesion, whereas postural reaction deficits can be ipsilateral or contralateral to the lesion. It is not uncommon for signs of diencephalic lesions to be vague and may include only mentation changes.

The most characteristic signs of diencephalic lesions are related to abnormal function of the hypothalamus and its connections with the pituitary gland. The hypothalamus is the control center for the autonomic nervous system and most of the endocrine system. If the hypothalamus is not affected, distinguishing diencephalic from cerebral lesions is difficult.

All sensory pathways of the body, with the exception of those serving olfaction, relay in the thalamus (diencephalon) en route to the cerebral cortex. Clinical signs of lesions in these systems in the diencephalon usually are not localizing. A rare generalized hyperesthesia has been described as a result of an abnormality in the relay nuclei of the pain pathways. Large lesions in the diencephalon may produce alterations in the level of consciousness (stupor, coma) because of interference with the ascending reticular activating system (see Chapter 12).

Vestibular System

Vestibular signs may be the result of central (brainstem) or peripheral (labyrinth) disease. Distinguishing central disease from peripheral disease is important because of the differences in treatment and prognosis. General signs of vestibular disease include falling, rolling, head tilting, circling, nystagmus, positional strabismus (deviation of one eye in certain positions of the head), and asymmetric ataxia (Figures 2-3 and 2-4). Vestibular ataxia is observed with other signs of vestibular dysfunction. Pathologic nystagmus (jerk nystagmus) seen with vestibular dysfunction can be spontaneous (at rest) or induced with change in head position. Jerk nystagmus consists of a slow phase that is followed by a fast phase. The direction of the fast phase of nystagmus is noted and recorded as rotary, horizontal, and vertical. A reliable technique to elicit a pathologic nystagmus is to decompensate the animal (if small) by rapidly flipping it on its back or in larger animals by rapidly elevating the head. Congenital nystagmus (pendular nystagmus) and strabismus occur in some breeds of exotic cats as a result of an anomaly in routing of the visual pathway from the retina to the contralateral visual cortex but cause no visual impairment.

Peripheral lesions involve the labyrinth within the petrosal bone. Middle-ear lesions (bulla ossea) may produce a head tilt with no other signs, presumably through pressure changes on the windows of the inner ear. Horizontal or rotatory nystagmus may be seen occasionally. Inner-ear disease, which actually involves the receptors and the vestibular nerve, usually produces one or more of the signs listed earlier in addition to the head tilt. In either case, the head tilt is ipsilateral to the lesion. Horner's syndrome (miosis, ptosis, enophthalmos) of the ipsilateral eye may be present with either middle- or inner-ear disease in the dog and cat because the sympathetic nerves pass through the middle ear in proximity to the petrosal bone. The facial nerve (CN VII) may be affected in inner-ear disease as it courses through the petrosal bone in contact with the vestibulocochlear nerve (CN VIII). The primary characteristics of peripheral vestibular disease are an asymmetric ataxia without deficits in postural reactions and a horizontal, or rotatory, nystagmus that usually maintains a constant direction with different head positions. The quick (jerk) phase of the nystagmus is away from the side of the lesion.

Any signs of brainstem disease in association with vestibular signs indicate that central involvement is present. The

TABLE **2-4**

Cranial Nerves

Number and Name	Origin or Termination in Brain	Course	Function	Test	Normal Response	Abnormal Response	Occurrence
CN I olfactory	Pyriform cortex	Nasal mucosa, cribriform plate, olfactory bulbs, olfactory tract, olfactory stria, pyriform cortex	Sense of smell	Smelling of nonirritating volatile substances (food)	Behavioral reaction; aversion or interest	No reaction	Rare: nasal tumors and infections (evaluation difficult)
CN II optic	Lateral geniculate nucleus (vision), pretectal nucleus (pupillary reflex)	Retina, optic nerve, optic chiasma optic tract, lateral geniculate nucleus, optic radiation, visual cortex, optic tract, pretectal nucleus, parasympathetic nucleus of CN III, oculomotor nerve	Vision, pupillary light reflexes	Menace response, obstacle test and behavior, placing reaction, following movement, pupillary light reflex, ophthalmoscopy	Blinks, avoids obstacles and responds to visual cues, placing good, follows objects, pupillary light reflexes present, retina normal	No blink, poor avoidance of obstacles, no visual placing, direct pupillary light reflex absent, retina or optic disk may be abnormal	Optic neuritis, neoplasia, orbital trauma, orbital mass
CN III oculomotor	Midbrain, tegmentum (level of rostral colliculus)	Nucleus ventral to mesencephalic aqueduct, exits ventral to midbrain between cerebral peduncles, runs in cavernous and CN VI, exits orbital fissure	Constriction of pupil; ciliary muscle for accommodation reaction of lens; extraocular muscles: dorsal, ventral, and medical rectus and ventral oblique	Pupillary size, pupillary light reflex, eye position, eye movements, physiologic nystagmus	Pupils symmetric, pupils constrict to light, eyes centered in palpebral fissure, eyes move in all directions	Mydriasis, ipsilateral, no direct pupillary reflex, ventrolateral strabismus, no movement except laterally (CN VI)	Orbital lesions, tentorial herniation, midbrain lesion
CN IV trochlear	Midbrain, tegmentum (level of caudal colliculus)	Nucleus ventral to mesencephalic aqueduct, exits dorsal to tectum, caudal to caudal colliculus, contralateral to origin, courses along ridge of petrosal bone, follows course of CN III	Dorsal oblique muscle, rotates dorsal portion of eye medioventrally	Eye position, eye movements, physiologic nystagmus	Eye centered in palpebral fissure, eyes move in all directions	Normal; rotation may be detected in animal with elliptical pupil or by position of vessels	Rare, difficult to evaluate; reported in polioencephalomalacia of cattle, but eyes move
CN V trigeminal ophthalmic, maxillary, and mandibular nerves	Motor nucleus: Pons Sensory nucleus: Pons, medulla, C1 spinal cord segment	Motor: Pons, exits at cerebellopontine angle, trigeminal canal of petrosal bone, oval foramen, mandibular nerve Sensory: Same except trigeminal ganglion in trigeminal canal; ophthalmic, maxillary, and mandibular nerves	Motor: Muscles of mastication Sensory: Face rostral to ears	Motor: Ability To close mouth, jaw tone Sensory: Palpebral reflex, pinch face, touch nasal mucosa	Closed mouth, good jaw tone; no atrophy of temporal or masseter muscles; palpebral reflex present; behavioral response to noxious stimulus	Jaw hangs open (bilateral), poor jaw tone, atrophy, loss of palpebral reflex or behavioral response to noxious stimulus (check all three branches)	Idiopathic mandibular paralysis, trigeminal neuritis, cerebellopontine angle tumors,nerve sheath tumors, rabies, trauma

Continued

TABLE 2-4

Cranial Nerves—cont'd

Number and Name	Origin or Termination in Brain	Course	Function	Test	Normal Response	Abnormal Response	Occurrence
CN VI abducent	Medulla (rostral and dorsal)	Medulla, lateral to pyramid,	Lateral rectus and retractor bulbi muscles, lateral movement of eye, retraction of globe exits orbital fissure	Eye position, eye movements	Eye moves laterally and retracts corneal reflex	Medial strabismus, lack of lateral eye movements or retraction of globe	Orbital trauma, orbital mass, brainstem disease
CN VII facial	Medulla (rostral and ventrolateral)	Motor: Axons leave nucleus, loop around abducent nucleus, and exit ventrolateral medulla ventral to CN VIII to internal acoustic meatus, facial canal in petrosal bone, and stylomastoid foramen to muscles of facial expression Taste: Solitary tract and nucleus, medulla follows course of trigeminal nerve Sensory: Geniculate ganglion and branches from vagus nerve	Muscles of facial expression and taste, rostral two thirds of tongue, and cutaneous sensation of inner surface of pinna	Facial symmetry, palpebral reflex, ear movements Taste: Atropine applied to rostral two thirds of tongue with cotton swabs Sensory: Touch inner surface of pinna	Face symmetric; normal movements of lips, ears, eyelids; palpebral reflex present; ears move in response to stimulation Taste: Aversive reaction immediately Sensory: Behavioral and ear twitch response	Asymmetry of face, ptosis, lip drops, deviation of nasal philtrum, palpebral reflex absent (check CN V), ears do not move Taste: No reaction until mouth is closed and material reaches caudal portion of tongue Sensory: No behavioral or ear twitch response	Idiopathic facial paralysis, polyneuropathies, inner ear infections, brainstem lesions
CN VIII vestibulocochlear	Vestibular nuclei medulla; cochlear nuclei medulla; cerebellomedullary angle, medulla	Inner ear, petrosal bone, internal acoustic meatus to medulla (nestibular nuclei)	Equilibrium, hearing	Vestibular: Posture and gait, eye movements, rotatory and caloric tests Hearing: Startle reaction, electrophysiology (EEG alerting, brainstem-evoked response)	Vestibular: Normal posture and gait, oculocephalic reflex, normal, brief postrotatory nystagmus and caloric-induced nystagmus Hearing: Startled reaction to handclap, evoked response present	Vestibular: Head tilt, head twist, circling, nystagmus, prolonged or absent postrotatory nystagmus, abnormal or absent caloric response Hearing: Poor startle reaction, no evoked response	Otitis media and otitis interna, idiopathic vestibular disease, polyneuropathy, brainstem disease

CN IX glosso-pharyngeal	Medulla (caudal)	Sensory: Solitary tract and nucleus Motor: Parasympathetic, ambiguus nucleus, exit together along lateral surface of medulla, exit through jugular foramen	Sensory and motor to pharynx and palate, parasympathetic to zygomatic and parotid salivary glands (in CN V); sensory to carotid body and sinus	Gag reflex	Swallowing	Poor gag reflex, dysphagia	Rare; common in rabies; brainstem disease
CN X vagus	Medulla (caudal)	Same as CN IX	Sensory and motor to pharynx and larynx, thoracic and abdominal viscera	Gag reflex, laryngeal reflex, slap test, oculocardiac reflex	Swallowing, coughing, bradycardia	Poor gag reflex, dysphagia, inspiratory dyspnea, no abduction of laryngeal folds, regurgitation	Rare, except in laryngeal paralysis; polyneuropathy
CN XI accessory	Medulla (caudal) and cervical spinal cord	Ambiguus nucleus of medulla and cervical gray matter, axons run rostrally from cervical cord to join cranial roots, exit jugular foramen	Trapezius and parts of sternocephalicus and brachiocephalicus muscles	Palpate for atrophy of muscles; EMG	Normal muscles	Atrophied muscles, denervation	Rare
CN XII hypoglossal	Medulla (caudal)	Axons exit medulla lateral to pyramid, hypoglossal canal to tongue	Movements of tongue	Protrusion of tongue (wet nose), retraction of tongue	Tongue protrudes symmetrically and can lick in both directions, strong withdrawal of tongue	Tongue deviates to side of lesion, atrophy, weak withdrawal	Brainstem disease, polyneuropathy

CN, Cranial nerve; *EEG,* electroencephalography; *EMG,* electromyography.

most important differentiating feature is a deficit in postural reactions. Peripheral vestibular disease does not cause paresis or loss of proprioception, whereas central disease frequently does (due to involvement of sensory and motor long tracts in the brainstem). Postural reactions must be evaluated critically because an animal with peripheral vestibular disease has deficits in equilibrium, which make the performance of tests such as hopping awkward. An evaluation of proprioceptive positioning is an excellent method for discrimination. Alterations in mental status or deficits in CN V and CN VII also are indicative of central vestibular disease (see Figure 2-3); however, some polyneuropathies may affect the cranial nerves, including CN V, VII, and VIII.

Lesions near the caudal cerebellar peduncle may produce what has been called a paradoxical vestibular syndrome. The signs are usually similar to those of central vestibular disease except that the direction of the head tilt is contralateral to the side of the lesion.[6] Additional signs of cerebellar disease, such as dysmetria and ataxia, may be seen.

Bilateral vestibular disease, which can be peripheral or central, produces a more symmetric ataxia. The animal walks with the limbs flexed and spread apart to maintain balance. The head often sways with wide excursions from side to side. No nystagmus is present, and vestibular eye movements are usually absent.[7]

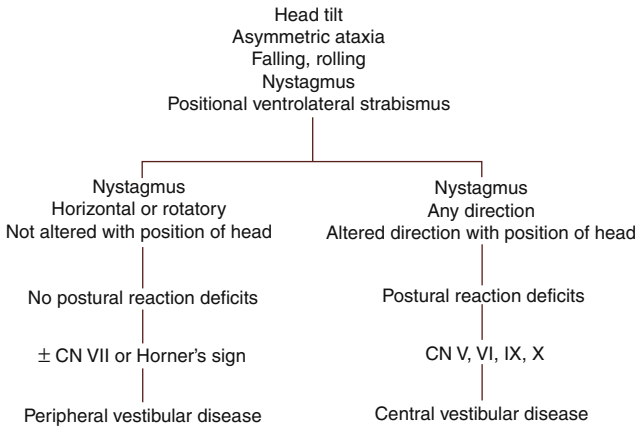

Figure 2-3 Algorithm for differentiating central and peripheral vestibular diseases.

Cerebellum

The cerebellum coordinates movements. It controls the rate and range of movements without actually initiating motor activity. Cerebellar lesions may be unilateral or bilateral, depending on cause. Characteristic signs include spastic ataxia, wide-based stance, dysmetria, intention tremor, and no obvious signs of weakness. Cerebellar ataxia is often characterized by dysmetria, which denotes stride lengths that are too short (hypometria) or too long (hypermetria). Head movement abnormalities differentiates cerebellar lesions from spinocerebellar tract lesions, which may produce similar signs in the limbs. For example, head dysmetria usually is recognized as a severe head drop when the head is elevated and suddenly released. Intention tremors are uncoordinated movements that become much worse as the animal initiates an activity, such as eating or drinking (see Figure 2-4).[8] The animal may stick its nose too far into its water dish when drinking or may even hit the edge of the dish.

Nystagmus may occur in cerebellar disease but is usually more of a tremor of the globe than the slow-quick (jerk) movements associated with vestibular disease. Cerebellar nystagmus is most pronounced as the animal shifts its gaze and fixates on a new field (an intention tremor). A down-beat vertical nystagmus may be seen upon dorsal extension of the head.

Acute injury to the cerebellum can cause a decerebellate posture, typically extensor hypertonus in the thoracic limbs, flexion in the pelvic limbs, and opisthotonos.[6] Isolated cerebellar trauma is unusual because of the protected location of the cerebellum. These signs are most pronounced when combined with brainstem lesions at the level of the midbrain or the pons.

Lesions of the flocculonodular lobes of the cerebellum produce signs similar to those of vestibular disease, including loss of equilibrium, nystagmus, and tendency to fall (see Chapter 8).

Diffuse cerebellar lesions may cause the menace response to be absent with vision remaining normal.

Cerebrum

Cerebral lesions (including the cerebral hemispheres and basal nuclei) usually cause alterations in behavior or mental status, seizures, loss of vision with intact pupillary light reflex, contralateral decrease in facial sensation, and mild contralateral hemiparesis and deficits in postural reactions.[9] Only one or two of these signs may be present because the cerebrum is a relatively large structure with well-localized functional areas. Signs are generally contralateral to the lesion.

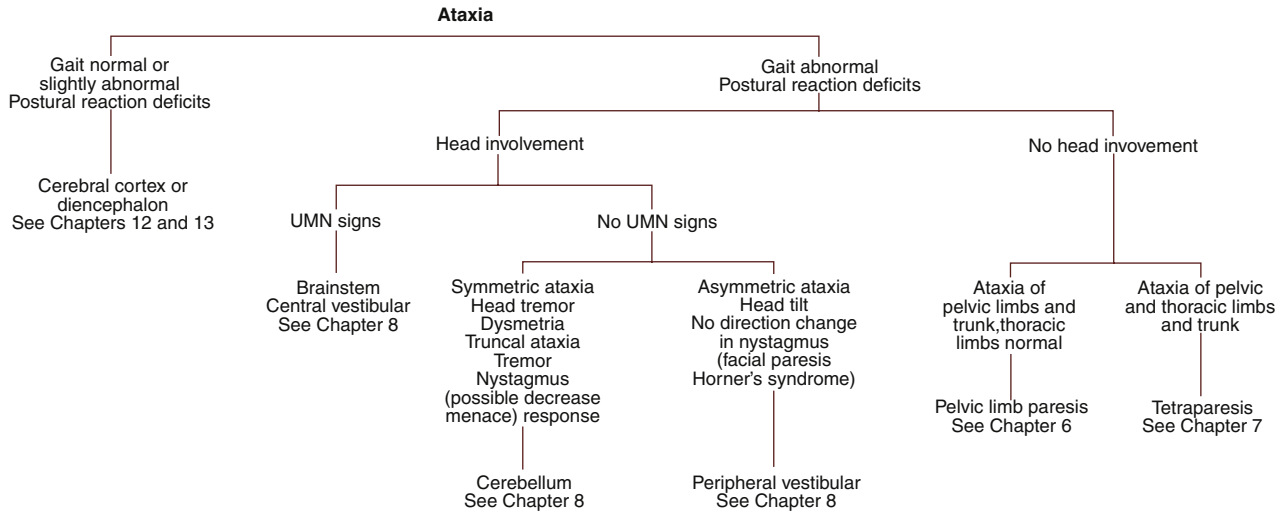

Figure 2-4 Algorithm for the diagnosis of ataxia based on gait, head involvement, and motor function of the limbs.

Behavioral changes usually reflect a lesion of the limbic system or the frontal or temporal lobes of the cortex. Frontal-lobe lesions often cause a disinhibition that results in excessive pacing. Compulsive pacing may continue until the animal walks into a corner and stands with its head pressed against the obstruction. If the lesion is unilateral or asymmetric, the animal may circle. Circling in an animal with a cerebral lesion is usually to the same side as the lesion. The animal's movement tends to be in large circles. The gait is reasonably normal, although obstacles may not be perceived. Circling is not a localizing sign because it can be caused by lesions in the forebrain, brainstem, and vestibular system.

Dullness, stupor, and coma represent decreasing levels of consciousness caused by a separation of the cerebral cortex from the ascending reticular activating system of the brainstem. Mentation abnormalities are usually more severe with brainstem lesions and diffuse cerebrocortical disease (see Chapter 12). Conscious visual perception requires intact visual pathways to the occipital lobes of the cerebral cortex. Occipital cortical lesions cause blindness with intact pupillary reflexes (see Chapter 11).

The sensorimotor cortex is important for voluntary motor activity but is not necessary for relatively normal gait and posture. Animals with lesions in this area can stand, walk, and run with minimal deficits. The animal's ability for fine discrimination is lost, however, and it is unable to avoid obstacles smoothly or to perform fine maneuvers, such as walking on the steps of a ladder. Markedly abnormal postural reactions are found.

Localization to one of the five regions of the brain is usually adequate for a clinical diagnosis. Cranial nerve signs provide positive evidence for precise localization within the brainstem. Clinical signs referable to several parts of the nervous system indicate diffuse or multifocal disease, such as infection, metabolic disorder, or malignant neoplasia (see Chapter 15).

CASE STUDIES

The following case studies use the information presented in chapters 1 and 2. The reader is encouraged to review the case, localize the lesion(s), and develop a diagnostic plan. Case summaries are listed at the end of the case studies section.

Key: *0*, Absent; *+1*, decreased; *+2*, normal; *+3*, exaggerated; *+4*, very exaggerated or clonus: *PL*, pelvic limb; *TL*, thoracic limb; *NE*, not evaluated.

CASE STUDY 2-1 *TURBO*

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▪ Signalment

German shepherd dog, male, 8 years old

▪ History

CC: seizures and paraparesis

Cluster seizures for 1 year. Treated with phenobarbital and potassium bromide. One month ago had a seizure and developed paraparesis immediately on recovery. Paraparesis was progressive and somewhat responsive to corticosteroid therapy.

▪ Physical Examination

Normal except for neurologic signs

▪ Neurologic Examination

Mental status: alert and responsive
Posture: normal
Gait: marked paraparesis with ataxia; occasionally crosses front feet
Palpation: negative; no perispinal pain noted

Postural reactions

Left	Reactions	Right
	Proprio (positioning) ceptive	
+1	PL	+1
+2	TL	+2
+2	Wheelbarrowing	+2
	Hopping	
+1	PL	+2
+2	TL	+2–+2
	Extensor postural	
NE	thrust	NE
NE	Hemistand-hemiwalk	NE
+1–+2	Tonic neck	+1–+2
	Placing, tactile	
NE	PL	NE
NE	TL	NE
	Placing, visual	
NE	PL	NE
NE	TL	NE

Spinal reflexes

Left	Reflex, Spinal Segment	Right
	Quadriceps	
+3	L4-6	+2–+3
	Extensor carpi radialis	
+2	C7-T1	+2
	Triceps	
+2	C7-T1	+2
	Flexion–PL	
+2	L5-S1	+2
	Flexion–TL	
+2	C6-T1	+2
Absent	Crossed extensor	Absent
	Perineal	
+2	S1-2	+2

Cranial nerves: normal
Sensory evaluation

1. Hyperesthesia: none
2. Superficial pain perception: normal
3. Deep pain perception: NE

Complete the sections below before reviewing the case summary.

Assessment (lesion localization and estimation of prognosis)
Diagnostic plan

▪ Rule-outs

1.
2.
3.
4.

CASE STUDY 2-2 *BIRK*

veterinaryneurologycases.com

▪ Signalment
Weimaraner, male castrated, 6 years old

▪ History
CC: Lameness—left pelvic limb; circles to right, leans to right, and has right head tilt
Referring veterinarian had diagnosed severe degenerative joint disease of the left coxofemoral joint. The dog seemed to respond to corticosteroids and nonsteroidal antiinflammatory drugs. The head tilt and circling have been present for several weeks and are slowly getting worse. Dog has been referred for total hip replacement surgery.

▪ Physical Examination
Pain and crepitus in right coxofemoral joint

▪ Neurologic Examination
Mental status: Alert and responsive
Posture: Right head tilt
Gait: Circles and falls to the right; no obvious paresis at gait
Palpation: Pain and crepitus in right coxofemoral joint

Postural reactions

Left	Reactions	Right
	Proprioceptive positioning	
+2	PL	+2
+2	TL	+2
+2	Wheelbarrowing	+2
	Hopping	
+2	PL	+1
+2	TL	+1–+2
	Extensor postural	
NE	thrust	NE
+2	Hemistand-hemiwalk	+1–+2
+2	Tonic neck	+2
	Placing, tactile	
NE	PL	NE
NE	TL	NE
	Placing, visual	
NE	PL	NE
NE	TL	NE

Spinal reflexes

Left	Reflex, Spinal Segment	Right
	Quadriceps	
+2	L4-6	+2–+3
	Extensor carpi radialis	
+2	C7-T1	+2
	Triceps	
+2	C7-T1	+2
	Flexion-PL	
+2	L5-S1	+2
	Flexion-TL	
+2	C6-T1	+2
Absent	Crossed extensor Perineal	Absent
+2	S1-2	+2

Cranial nerves: normal
Sensory evaluation
1. Hyperesthesia: none
2. Superficial pain perception: normal
3. Deep pain perception: NE

Complete the sections below before reviewing the case summary.

Assessment (lesion localization and estimation of prognosis)
Diagnostic plan

▪ Rule-outs
1.
2.
3.
4.

CASE STUDY 2-3 *DOSSIE BOY*

veterinaryneurologycases.com

▪ Signalment
Mixed breed canine, male, 5-6 years old,

▪ History
CC: comatose condition
Obtained as stray 1 year ago Rabies vaccination unknown. One month ago, coughing and microfilaria were found on blood smear. Referring veterinarian gave 0.5 mL levamisole SC. That evening, dog was acutely recumbent and may have had a seizure. Since then, dog has been in comatose state. Dog has been given atropine, antibiotics, and fluids prior to admission.

▪ Physical Examination
See neurologic examination (performed about 3 days post onset of signs)

▪ Neurologic Examination*
Mental status: Comatose and cannot be aroused
Posture: At times demonstrates opisthotonos and extension of the thoracic limbs
Gait: Severe tetraplegia, limbs are hypotonic
Palpation: Negative

Continued

CASE STUDY 2-3 *DOSSIE BOY*—cont'd

Postural reactions

Left	Reactions	Right
	Proprioceptive positioning	
0	PL	0
0	TL	0
0	Wheelbarrowing	0
	Hopping	
0	PL	0
0	TL	0
	Extensor postural	
0	thrust	0
0	Hemistand-hemiwalk	0
NE	Tonic neck	NE
	Placing, tactile	
NE	PL	NE
NE	TL	NE
	Placing, visual	
NE	PL	NE
NE	TL	NE

Spinal reflexes

Left	Reflex, Spinal Segment	Right
	Quadriceps	
+2–+3	L4-6	+2–+3
	Extensor carpi radialis	
NE	C7-T1	NE
	Triceps	
NE	C7-T1	NE
	Flexion-PL	
+2	L5-S1	+2
	Flexion-TL	
+2	C6-T1	+2
Present	Perineal crossed extensor	Present
+2	S1-2	+2

*Dog is comatose and cannot be roused. Severe tetraplegia. Limbs are hypotonic. Sometimes demonstrates decerebrate posture. Patellar reflexes are +2-+3. Bilateral miosis with response to light, 0 menace, horizontal nystagmus that changes direction. Response to noxious stimuli is reduced.

Cranial nerves

1. No menace response in either eye
2. Horizontal jerk nystagmus that changes direction depending on which side dog is lying (lying on right side, jerk phase is to the right; lying on left side, jerk phase is to the left)
3. Normal (+2) palpebral reflexes (CN V and VII)
4. Decreased facial sensation (CN V)
5. Bilateral miosis with pupillary constriction to strong light

Sensory evaluation

1. Hyperesthesia: none
2. Superficial pain perception: Difficult to assess; marked decrease response to noxious stimuli
3. Deep pain perception: present but hard to elicit cerebral response

Complete the sections below before reviewing the case summary.

Assessment (anatomic diagnosis and estimation of prognosis)
Diagnostic plan

▪ Rule-outs

1.
2.
3.
4.

CASE STUDY 2-4 *BRANDY* veterinaryneurologycases.com

▪ Signalment

Malamute-shepherd cross, female spayed approximately 7-8 years old

▪ History

Since a puppy, dog has been clumsy. The signs are not progressive. Dog has been treated by several veterinarians for coxofemoral degenerative joint disease. Owner wants to know what is causing the gait problem in her dog. Local veterinarian sends owner-provided video tape for consultation.

▪ Physical Examination

See neurologic examination

▪ Neurologic Examination

1. Mental status: normal
2. Posture: base wide; no head tilt or circling
3. Gait: generalized ataxia, truncal ataxia, intention tremors, hypermetria, no paresis
4. Palpation: NE

Postural reactions: not provided
Spinal reflexes: not provided
Cranial nerves: NE
Sensory evaluation: NE

Complete the sections below before reviewing the case summary.

Assessment (anatomic diagnosis and estimation of prognosis)
Diagnostic plan

▪ Rule-outs

1.
2.
3.
4.

CASE STUDY 2-5 *RUDY* veterinaryneurologycases.com

▪ Signalment
Canine, Labrador retriever, male castrated, 2 years old

▪ History
Acute onset of clinical signs. Became base wide in pelvic limbs and falling when walking. Taken to RDVM that morning and four injections of dexamethasone SP were administered. Spinal, radiographs were taken but no lesions found. There has been no change in neurologic status over the past 3 days. Dog is not painful and the bladder is easily expressed. Dog has difficulty moving his tail.

▪ Physical Examination
No systemic signs noted

▪ Neurologic Examination
Mental status: alert and responsive
Posture: normal; see gait
Gait: paraparesis with signs more severe in the right pelvic limb. Dog dribbles urine and does not move his tail.
4. *Palpation:* normal

Postural reactions

Left	Reactions	Right
	Proprioceptive positioning	
+2	PL	0
+2	TL	+2
	Wheelbarrowing	
	Hopping	
+1	PL	0
+2	TL	+2
	Extensor postural	
NE	thrust	NE
	Hemistand-hemiwalk	
NE	Tonic neck	NE
	Placing, tactile	
NE	PL	NE
NE	TL	NE
	Placing, visual	
NE	PL	NE
NE	TL	NE

Spinal reflexes

Left	Reflex, Spinal Segment	Right
	Quadriceps	
+2	L4-6	0
	Extensor carpi radialis	
+2	C7-T1	+2
	Triceps	
+2	C7-T1	+2
	Flexion-PL	
+2	L5-S1	0
	Flexion-TL	
+2	C6-T1	+2
	Crossed extensor	
0	Perineal	0
+1	S1-2	0

Cranial nerves: normal
Sensory evaluation
1. Hyperesthesia: none
2. Superficial pain perception: normal from all areas except for localized area of hypalgesia in caudal lumbar area on the right side
3. Deep pain perception: normal

Complete the sections below before reviewing the case summary.

Assessment (anatomic diagnosis and estimation of prognosis)
Diagnostic plan

▪ Rule-outs
1.
2.
3.
4.

CASE STUDY 2-6 *DUSTY* veterinaryneurologycases.com

▪ Signalment
Whippet, female, 8 years old.

▪ History
3 months ago, acute onset of paraplegia. Dog was running and went around a farm building. Dog cried out and when owner found Dusty, she was paralyzed in her pelvic limbs and in considerable pain. She was seen by local veterinarian who provided symptomatic therapy. Signs are not progressive and dog is not in pain. Dusty can urinate and defecate but has no cerebral control over either. Owner is seeking second opinion and prognosis.

▪ Physical Examination
No systemic signs noted

▪ Neurologic Examination
Mental status: normal
Posture: normal
Gait: paraplegia
Palpation: generalized atrophy of lumbar and pelvic limb muscles

CASE STUDY 2-6 *DUSTY*—cont'd

Postural reactions

Left	Reactions	Right
	Proprioceptive positioning	
0	PL	0
+2	TL	+2
	Wheelbarrowing	
	Hopping	NE
0	PL	0
+2	TL	+2
0	Extensor postural thrust	0
NE	Hemistand-hemiwalk	NE
NE	Tonic neck	NE
	Placing, tactile	
NE	PL	NE
NE	TL	NE
	Placing, visual	
NE	PL	NE
NE	TL	NE

Spinal reflexes

Left	Reflex, Spinal Segment	Right
	Quadriceps	
+4	L4-6	+4
	Extensor carpi radialis	
+2	C7-T1	+2
	Triceps	
+2	C7-T1	+2
	Flexion-PL	
	Quadriceps	
+2	L5-S1	+2
	Flexion-TL	
+2	C6-T1	+2
Brisk	Crossed extensor (PL)	Brisk
	Perineal	
+2	S1-2	+2

Cranial nerves: normal
Sensory evaluation

1. Hyperesthesia: none
2. Superficial pain perception: poor caudal to T_{13}; the cutaneous trunci reflex is absent behind caudal to T_{13} vertebrae
3. Deep pain perception: good

Complete the sections below before reviewing the case summary.

Assessment (anatomic diagnosis and estimation of prognosis)
Diagnostic plan

▪ Rule-outs

1.
2.
3.
4.

CASE STUDY 2-7 *ANGEL*

veterinaryneurologycases.com

▪ Signalment
Feline, DSH, FS, 1 year old

▪ History
Cat was left in garage for 3 days while owners were out of town. When owners returned home, they noted ataxia, left head tilt, and salivation. Another cat at home is fine.

▪ Physical Examination
T 103° F, dehydrated 8% and tense abdomen on palpation

▪ Neurologic Examination
Observation
Mental status: alert; constantly vocalizes
Posture: Left head tilt and circles to the left; crouched posture and very reluctant to walk. Tends to swing head in wide excursions from side to side. Occasionally circles to the right.
Gait: asymmetric ataxia; no paresis detected
Palpation: normal

Postural reactions

Left	Reactions	Right
	Proprioceptive positioning	
+2	PL	+2
+2	TL	+2
+2	Wheelbarrowing	+2
	Hopping	
+2	PL	+2
+2	TL	+2
+2	Extensor postural thrust	+2
NE	Hemistand-hemiwalk	NE
NE	Tonic neck	NE
	Placing, tactile	
NE	PL	NE
NE	TL	NE
	Placing, visual	
NE	PL	NE
NE	TL	NE

Spinal reflexes: not examined
Cranial nerves

1. Menace: decreased in right eye; normal in left eye
2. Left pupil is constricted; slight protrusion of left membrana nictitans
3. Normal pupillary light reflexes
4. Normal palpebral reflexes
5. No spontaneous nystagmus
6. Normal facial sensation

Continued

CASE STUDY 2-7 *ANGEL*—cont'd

Sensory evaluation
1. Hyperesthesia: none
2. Superficial pain perception: normal
3. Deep pain perception: normal

Complete the sections below before reviewing the case summary.

Assessment (anatomic diagnosis and estimation of prognosis)
Diagnostic plan

▪ Rule-outs
1.
2.
3.
4.

CASE STUDY 2-8 *CHLOE*

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▪ Signalment
Golden retriever, female, 20 months old

▪ History
Dog presented with severe tetraparesis. Clinical signs developed at 10:00 AM on July 10. Dog became weak in all four legs and over a period of a few hours, she lost the ability to stand and walk. She has been in excellent health and Chloe is primarily an indoor dog that lives in a rural area and spends time outdoors unsupervised. She was eating fine until 2 days ago. Dog gags, coughs, and retches when eating and drinking. Several small "seed" ticks have been noted on the dog. She was in estrus 6 months ago. No treatment has been given. Dog was observed eating a dead rabbit about 2 days before onset of clinical signs. Another dog with her has developed similar but less severe clinical signs.

▪ Physical Examination Findings

Vital signs:	T: 101.5° F Pulse: 116 RR: 32 Wt: 28.4 kg.
General appearance	Dog is recumbent and unable to stand. She is very depressed.
Integument	Several small ticks are present. No engorged ticks are found.
Musculoskeletal	Mild muscle atrophy present in limbs. No hyperesthesia is noted.
Circulatory	No abnormalities noted.
Respiratory	Rapid shallow respiration noted.
Digestive	Abdomen is distended, nonpainful, and firm feces are present on rectal palpation.
Genitourinary	Bladder is distended with urine and easily expressed
Eyes	Left pupil is normal to dilated. Right pupil is small and third eyelid is prolapsed.
Ears	Normal
Nervous	See neurologic examination
Lymph nodes	Normal
Mucous membranes	Normal

▪ Neurologic Examination
Mental status: The dog is alert and responsive to her name. She has very shallow respirations.
Posture: No head tilt is noted. Dog is recumbent.
Gait: Dog is tetraplegic. She can wag her tail voluntarily and she can lift her head. Voluntary motor movements in the limbs are weak.
Palpation: Muscle tone is reduced in all limbs.

Postural reactions

Left	Reactions	Right
	Proprioceptive positioning	
0	PL	0
0	TL	0
	Wheelbarrowing	
	Hopping	
0	PL	0
0	TL	0
0	Extensor postural thrust	0
NE	Hemistand-hemiwalk	NE
NE	Tonic neck	NE
	Placing, tactile	
NE	PL	NE
NE	TL	NE
	Placing, visual	
NE	PL	NE
NE	TL	NE

Spinal reflexes

Left	Reflex, Spinal Segment	Right
	Quadriceps	
0–+1/2	L4-6	0
	Extensor carpi radialis	
0	C7-T1	0
	Triceps	
0	C7-T1	0
	Flexion-PL	
+1/2	L5-S1	+1/2
	Flexion-TL	
+1/2	C6-T1	+1/2
None	Crossed extensor	None
	Perineal	
+2	S1-2	+2

Cranial nerves
1. Menace: slightly delayed in left eye and normal in right eye.
2. Pupils: Left pupil is dilated compared to right.

Continued

CASE STUDY 2-8 *CHLOE*—cont'd

3. Pupillary light reflexes: decreased in left eye but normal in right eye.
4. Palpebral: normal
5. Facial sensation: normal
6. Gag reflex: decreased
7. Voice: decreased

Sensation: location
1. Hyperesthesia: none
2. Superficial pain perception: +2
3. Deep pain perception: +2

Complete the sections below before reviewing the case summary.

Assessment (anatomic diagnosis and estimation of prognosis)
Diagnostic plan

▪ Rule-outs
1.
2.
3.
4.

CASE STUDY 2-9 *DUFFY*

 veterinaryneurologycases.com

▪ Signalment
DSH cat, spayed female, 7 years old

▪ History
Owner found cat dragging its right pelvic limbs. Cat is inside and owner denies any possibility of trauma. Signs are not progressive and cat is normal in all respects.

▪ Physical Examination
Nothing abnormal except monoparesis

▪ Neurologic Examination
Mental status: alert and responsive
Posture: normal
Gait: paralysis of right pelvic limb
Palpation: painful in right caudal thigh

Postural reactions

Left	Reactions	Right
	Proprioceptive positioning	
+2	PL	0
+2	TL	+2
	Wheelbarrowing	
	Hopping	
+2	PL	0
+2	TL	+2
+2	Extensor postural thrust	0
NE	Hemistand-hemiwalk	NE
NE	Tonic neck	NE
	Placing, tactile	
NE	PL	NE
NE	TL	NE
	Placing, visual	
NE	PL	NE
NE	TL	NE

Spinal reflexes

Left	Reflex, Spinal Segment Quadriceps	Right
+2	L4-6 Extensor carpi radialis	+2
NE	C7-T1 Triceps	NE
NE	C7-T1 Flexion-PL	NE
+2	L5-S1 Flexion-TL	0
+2	C6-T1	+2
Absent	Crossed extensor Perineal	Absent
+2	S1-2	+2

Cranial nerves: normal
Sensory evaluation
1. Hyperesthesia: very painful in caudal thigh region
2. Superficial and deep pain perception: cat does not perceive noxious stimuli from the right pelvic paw (tibial and peroneal nerves). Deep pain is perceived in the distribution of the superficial saphenous nerve. Normal perception of noxious stimuli from the tail and others of the body.

Complete the sections below before reviewing the case summary.

Assessment (anatomic diagnosis and estimation of prognosis)
Diagnostic plan

▪ Rule-outs
1.
2.
3.
4.

CASE STUDY 2-10 *BRITTANY* veterinaryneurologycases.com

▪ Signalment
Brittany spaniel dog, female spayed, 2 years old

▪ History
Presented a few hours after being injured by an automobile. At the time of admission, the dog was non ambulatory in the pelvic limbs. Depression, bilateral epistaxis, and conjunctival hemorrhage in the right eye were noted. One hour after admission, the dog was noted to fall on the right thoracic and pelvic limbs and tended to circle to the left. Thoracic and abdominal radiographs were negative.

▪ Physical Examination: see neurologic examination

▪ Neurologic Examination
Examination performed the following morning and after dog was stable and improving.
Mental status: Dog is alert and responsive. She explores her environment.
Posture: No head tilt is present.
Gait: Dog tends to aimlessly walk in wide circles to the left and sometimes to the right. No paresis or knuckling of the paws are noted.
Palpation: normal

Postural reactions

Left	Reactions	Right
	Proprioceptive positioning	
+2	PL	+1
+2	TL	+1
+2	Wheelbarrowing	+1
	Hopping	
+2	PL	+1
+2	TL	+1
+2	Extensor postural thrust	+1
+2	Hemistand-hemiwalk	+1
NE	Tonic neck	NE
	Placing, tactile	
NE	PL	NE
NE	TL	NE
	Placing, visual	
NE	PL	NE
NE	TL	NE

Spinal reflexes

Left	Reflex, Spinal Segment	Right Quadriceps
+2	L4-6 Extensor carpi radialis	+2–+3
+2	C7-T1 Triceps	+2
+2	C7-T1 Flexion-PL	+2
+2	L5-S1 Flexion-TL	+2
+2	C6-T1	+2
Absent	Crossed extensor Perineal	Absent
+2	S1-2	+2

Cranial nerves
1. Decreased menace response in right eye but normal palpebral reflex is present
2. Decreased perception of noxious stimuli to right side of face
3. PLRs are normal and pupils are normal size and equal

Sensory evaluation
1. Hyperesthesia: none
2. Superficial pain perception: decreased right side of face
3. Deep pain perception: normal

Complete the sections below before reviewing the case summary.

Assessment (anatomic diagnosis and estimation of prognosis)
Diagnostic plan

▪ Rule-outs
1.
2.
3.
4.

CASE STUDY 2-11 *LINUS* veterinaryneurologycases.com

▪ Signalment
Golden retriever, male, 9 years old

▪ History
Owner reports progressive difficulty eating and drinking for several weeks. Dog has trouble closing its mouth and is becoming more depressed and lethargic. There is recent weight loss and partial anorexia. Owner reports no gait abnormalities.

▪ Physical Examination: see neurologic examination

▪ Neurologic Examination
Mental status: subdued
Posture: normal
Gait: normal
Palpation: atrophy of left temporalis muscle

CASE STUDY 2-11 *LINUS*—cont'd

Postural reactions: normal

Spinal reflexes: normal

Cranial nerves
1. Olfaction is decreased on right side compared to the left side
2. Difficulty closing mouth; atrophy of left temporalis muscle
3. Palpebral reflex: absent right eye; normal left eye
4. Menace response: normal left eye; right eye: no blink but dog moves head to avoid hand
5. Facial sensation: decreased on the right side and mandible but present on left side

Sensation: location
1. Hyperesthesia: none
2. Superficial pain perception: decreased right facial area
3. Deep pain perception: NE

Complete the sections below before reviewing the case summary.

Assessment (anatomic diagnosis and estimation of prognosis)
Diagnostic plan

▪ Rule-outs
1.
2.
3.
4.

CASE SUMMARIES

CASE STUDY 2-1 *ASSESSMENT*

▪ Lesion Localization (anatomic diagnosis and estimation of prognosis)
1. The neurologic examination is consistent with a bilateral lesion T3-L3. One cannot exclude a caudal cervical lesion.
2. The seizures are most likely due to a forebrain lesion.

▪ Diagnostic plan
The history supports a progressive myelopathy. Given the history, categories of disease to consider are degenerative, neoplastic, inflammatory/infectious.

Rule-outs
Spinal cord disease (see Chapters 6 and 7)
1. Henson type 2 intervertebral disk disease—Cervical and TL radiographs, myelography, and CT
2. Neoplasia—See type 2 disk disease
3. Degenerative myelopathy—rule out compressive myelopathy
4. Inflammation—CSF

▪ Rule-outs
Seizures (see Chapter 13): Given the dog's age, acquired (secondary) epilepsy is most likely. While there are several causes in this category, neoplasia would be a prime rule-out.
1. Extracranial (metabolic) causes—Complete small animal profile (see Chapter 4)
2. Intracranial causes—Advanced imaging (MR, CT) should be performed (see Chapter 4)

▪ Case Summary
Myelogram and CT—large disk protrusion at C6-7. Compression persists with cervical distraction. Mild compression at C5-6 but disappears with distraction. CT of brain reveals large mass in left frontal lobe displacing frontal bone of sinus. Owner elected cervical decompressive surgery (ventral slot) for the Henson type 2 invertebral disk disease and continued to manage the seizures medically. The paraparesis improved but the seizures became increasingly difficult to control.

CASE STUDY 2-2 ASSESSMENT

■ Assessment (lesion localization and estimation of prognosis)

Dog is presented with clinical signs of a vestibular syndrome (circling, falling, head tilt). The decreased hopping reactions on the right side are consistent with "central vestibular disease." The lesion most likely is located in the right rostral medulla. The sign-time graph is slowly progressive. Neoplastic, degenerative, and inflammation are the primary categories to consider. The musculoskeletal problem is most likely unrelated to the neurologic signs.

■ Diagnostic plan

Rule-outs

1. Neoplasia: MR or CT of the brain is recommended.
2. Inflammation: CSF; MRI of brain
3. Neurodegenerative disease: brain biopsy, MRI of brain

■ Case Summary

- CSF—Normal
- CT—Enhancing mass in right cranial dorsal brainstem and right ventral cerebellum. A meningioma was suspected.
- Pelvic radiographs—severe degenerative joint disease (DJD) of left coxofemoral joint
- Treatment—The owner declined surgery for the mass. No follow up was recorded.
- Final diagnosis—DJS left coxofemerol joint (CFJ); mass right cranial dorsal brainstem

CASE STUDY 2-3 ASSESSMENT

■ Assessment (anatomic diagnosis and estimation of prognosis)

Bilateral, severe brainstem and forebrain disease (medulla, pons, and maybe midbrain). The sign-time graph in this case is acute and progressive over a few hours. Inflammation, toxicity, and vascular infarction are the major categories to consider (see Chapters 12, 13, and 15).

■ Diagnostic plan

Rule-outs

1. Rabies (dumb form)—observation and strict rabies precautions
2. Levamisole toxicity—symptomatic therapy
3. GME—CSF, MRI brain
4. Brainstem infarction—MRI of brain

■ Case Summary

CSF and brain scan would be beneficial. Given dog's condition, possibility of rabies and poor anesthetic risk, dog was managed symptomatically with fluids. Dog made a wonderful recovery. A vascular lesion affecting the rostral brainstem was suspected. Dog did not receive a toxic dose of levamisole. Suspect levamisole killed microfilaria that caused a brainstem vascular occlusion. Rabies was discounted when dog began to improve. The dog was treated as a rabies suspect for several days. He began to walk in 10 days, remained cortically blind and continued to demonstrate forebrain signs. He was nearly normal in 60 days.

CASE STUDY 2-4 ASSESSMENT

■ Assessment (anatomic diagnosis and estimation of prognosis)

The clinical signs are very suggestive of generalized cerebellar disease. The sign-time graph is nonprogressive. One can assume the signs were present at birth.

■ Diagnostic plan

Rule-outs

1. Cerebellar hypoplasia, MRI of brain
2. Cerebellar trauma at birth
3. Cerebellar abiotrophy (unlikely since signs are not progressive)

■ Case Summary

No diagnostic procedures were performed. Given the history and clinical signs, cerebellar hypoplasia is a reasonable clinical diagnosis. There is no treatment and the prognosis is good for this dog. Cerebellar diseases are discussed in Chapter 8.

CASE STUDY 2-5 ASSESSMENT

▪ Assessment (anatomic diagnosis and estimation of prognosis)

The lesion is located L4-S2 and is more severe on the right side (tends to lateralize). There are LMN signs in right pelvic limb with normal perception of noxious stimuli the limb. This suggests that the lesion is within the ventral gray matter of the spinal cord. The sign-time graph is acute and nonprogressive. Traumatic, and vascular categories of disease should be considered.

▪ Diagnostic plan

Rule-outs

1. Spinal cord infarction: MRI of lumbar spinal cord; rule out other etiologies
2. Intervertebral disk disease: spinal radiographs, myelogram, CT, MRI
3. Trauma: vertebral fracture or subluxation; lumbosacral fracture, subluxation: spinal radiographs, CT, MRI

▪ Case Summary

CaseSpinal radiographs, myelography, and CT of lumbosacral spine are normal. CSF examination is normal. By exclusion, spinal cord infarction was the clinical diagnosis. The dog was given physical therapy and bladder care (assisted urination with gentle manual expression). Within 10 days, dog was more than 50% improved. In 90 days, dog was 90% improved and urinary incontinence resolved. Spinal cord infarction (fibrocartilagenous emboli) is discussed in Chapters 6 and 7.

CASE STUDY 2-6 ASSESSMENT

▪ Assessment (anatomic diagnosis and estimation of prognosis)

The dog has paraplegia with increased reflexes in pelvic limbs and normal thoracic limbs. The lesion is bilateral and located in spinal cord segments T3-L3. Sensory examination further localizes the lesion to the caudal thoracic spinal cord segments. The sign-time graph is acute and nonprogressive. Disease categories to consider are trauma and vascular. Thoracolumbar spinal cord diseases are presented in Chapter 6.

▪ Diagnostic plan

Rule-outs

1. Trauma (vertebral fracture/subluxation): spinal radiographs
2. Intervertebral disk disease (type 1): spinal radiographs, myelography, CT
3. Spinal cord infarction: MRI of spinal cord

▪ Case Summary

After 3 months, the dog is stable and may be slightly improved. Spinal radiographs are normal. Given the history and clinical signs, it is very unlikely that the dog will benefit from more in-depth diagnostic tests. The prognosis for recovery of motor function is poor. The owner was instructed to continue her physical therapy and bladder care.

CASE STUDY 2-7 ASSESSMENT

▪ Assessment (anatomic diagnosis and estimation of prognosis)

The clinical signs are those of a vestibular syndrome (see Chapter 8). The postural reactions are normal, which would support a peripheral vestibular disorder. The signs are bilateral but much worse on the left side. The left sympathetic nerve is affected (often found in otitis media-interna).

▪ Diagnostic plan

The clinical signs are acute and progressive. Categories of disease to consider include inflammation, idiopathic, and toxic.

Rule-outs

1. Bacterial otitis media-interna: otoscopic examination, skull radiographs, and CT of skull / brain
2. Idiopathic feline vestibular disease: exclude other rule-outs

▪ Case Summary

Otoscopic examination revealed inflammation of both tympanic membranes. No exudate was apparent in the middle ear. A clinical diagnosis of bacterial otitis media-interna was made. The cat was placed on amoxicillin and rechecked in 5 days. Cat was clinically improved in 10 days. She had slight head tilt to the left but her gait was remarkably improved.

CASE STUDY 2-8 ASSESSMENT

▪ Assessment (anatomic diagnosis and estimation of prognosis)

The neurologic examination defines a generalized LMN disorder. Given the findings, one suspects a disease affecting motor neurons or motor end plates. The sign-time graph is acute and progressive over several hours. Disease categories to consider are inflammation and toxicity. Generalized LMN disorders are discussed in Chapter 7.

▪ Diagnostic plan

Rule-outs

1. Botulism is the number 1 rule-out given the combination of autonomic and LMN involvement
2. Tick paralysis—usually no autonomic involvement. No engorged female ticks found on the dog.
3. Polyradiculoneuritis—usually no autonomic involvement

Diagnostic procedures

1. Assess dog for megaesophagus and aspiration pneumonia—thoracic radiographs, CBC
2. Assess dog for intestinal ileus and detrusor atony—abdominal radiographs
3. Assess dog for systemic inflammation—CBC
4. Rule out any metabolic consequences of vomiting—biochemical profile, UA
5. Botulism—consider mouse inoculation or immunologic testing of feces for botulinum toxin.
6. EMG

   The CBC, biochemical profile, and urinalysis were normal. Thoracic radiographs: dilation of entire intrathoracic esophagus with a mixture of air and fluid. Early alveolar pulmonary opacity involving the ventral aspects of the right cranial, right middle, and left cranial lung lobes. Radiographic diagnosis: generalized megaesophagus and aspiration pneumonia involving the right cranial, right middle, and left cranial lung lobes.

▪ Case Summary

The differential diagnosis included botulism, tick paralysis, and polyradiculoneuritis. Organophosphate intoxication was also considered but excluded early in the case evaluation.

Diagnosis: Given the history of multiple dog involvement, exposure to carrion 4 days before development of clinical signs and the presence of autonomic nervous system involvement, botulism was the most likely diagnosis. While several seed ticks were present on the dog, no engorged female ticks were identified and the dog did not improve when ticks were removed. Polyradiculoneuritis does not cause autonomic signs and was deemed less likely.

Treatment: Aspiration pneumonia: IV antibiotics (ampicillin, enrofloxacin), terbutaline SC, thoracic coupage and nasal O_2

Regurgitation: metoclopramide SC q8h

Physical therapy: Passive manipulation of limbs several times a day. Sterile indwelling urinary catheter. Manual evacuation of feces and enemas.

The dog began to improve in 6 days. Over several months, she gradually recovered normal motor function.

CASE STUDY 2-9 ASSESSMENT

▪ Assessment (anatomic diagnosis and estimation of prognosis)

The lesion is localized to the right lower sciatic nerve involving the tibial and peroneal nerves. The sign-time graph is acute and nonprogressive. Categories of disease to consider are trauma and vascular. Peripheral nerve disorders are discussed in Chapter 5.

▪ Diagnostic plan

Rule-outs

1. Sciatic nerve trauma: pelvis and right pelvic limb radiographs
2. Ischemic myoneural necrosis (vascular occlusion): check for other evidence of thrombosis (cardiac radiographs, echocardiography, thoracic radiographs)

▪ Case Summary

Radiographs of the pelvis and right pelvic limb were normal. Evaluation of the heart and lungs were normal. Owner agreed to surgical exploration of the sciatic nerve in the caudal thigh region. A small, dark mass was found around the sciatic nerve just above the bifurcation into the peroneal and tibial nerves. The mass was an organized hematoma. Following surgery, the cat improved about 50% over the next 6 months.

CASE STUDY 2-10 *ASSESSMENT*

▪ Assessment (anatomic diagnosis and estimation of prognosis)
The dog has a right forebrain lesion (compare the gait and with the postural reactions). We know that trauma is the etiology in this case. The dog most likely has a contusion affecting the right cerebral cortex. The prognosis is good. The dog should be observed for seizures in the future. Head trauma is discussed in Chapter 12.

▪ Diagnostic plan
At this point, the dog is recovering following treatment for head trauma. No further diagnostics are indicated.

CASE STUDY 2-11 *ASSESSMENT*

▪ Assessment (anatomic diagnosis and estimation of prognosis)
Cranial nerves I (right), V (right and left), and VII (right) are affected. The sign-time graph is chronic and progressive. Categories of disease to consider include neoplasia, degeneration, and inflammation/infection. Prognosis is guarded.

▪ Diagnostic plan

Rule-outs
1. Meningioma (and other tumors) affecting base of brain—MRI of brain
2. Fungal infection—MRI of brain, CSF, serology
3. Abscess—MRI of brain, CSF
4. Neurodegenerative disease—MRI of brain

▪ Case Summary
Owner elected euthanasia. Necropsy revealed a large expanding meningioma extending from the olfactory tracts on the right side to the medulla on both sides. Cranial nerves were involved after their exit from the brainstem. This explains the normal gait and posture but multiple cranial nerve involvement. Cranial nerve disorders are discussed in Chapters 9 and 11.

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