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
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.
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

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

*Parentheses indicate segments that sometimes contribute to a nerve.

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

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# TABLE 2-4

Cranial Nerves

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

Continued
<table>
<thead>
<tr>
<th>Number and Name</th>
<th>Origin or Termination in Brain</th>
<th>Course</th>
<th>Function</th>
<th>Test</th>
<th>Normal Response</th>
<th>Abnormal Response</th>
<th>Occurrence</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>CN VI</strong> abducent</td>
<td>Medulla (rostral and dorsal)</td>
<td>Medulla, lateral to pyramid, Medulla, lateral to retractor bulbi muscles, lateral movement of eye, retraction of globe exits orbital fissure</td>
<td>Lateral rectus and retractor bulbi muscles, lateral movement of eye, retraction of globe exits orbital fissure</td>
<td>Eye position, eye movements</td>
<td>Eye moves laterally and retracts corneal reflex</td>
<td>Medial strabismus, lack of lateral eye movements or retraction of globe</td>
<td>Orbital trauma, orbital mass, brainstem disease</td>
</tr>
<tr>
<td><strong>CN VII</strong> facial</td>
<td>Medulla (rostral and ventrolateral)</td>
<td>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</td>
<td>Muscles of facial expression and taste, rostral two thirds of tongue, and cutaneous sensation of inner surface of pinna</td>
<td>Facial symmetry, palpebral reflex, ear movements Taste: Atropine applied to rostral two thirds of tongue with cotton swabs Sensory: Touch inner surface of pinna</td>
<td>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</td>
<td>Asymmetry of face, ptosis, lip drops, deviation of nasal philtrum, palpebral reflex absent (check CN VI), 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</td>
<td>Idiopathic facial paralysis, polyneuropathies, inner ear infections, brainstem lesions</td>
</tr>
<tr>
<td><strong>CN VIII</strong> vestibulocochlear</td>
<td>Vestibular nuclei medulla; cochlear nuclei medulla; cerebellomedullary angle, medulla</td>
<td>Inner ear, petrosal bone, internal acoustic meatus to medulla (vestibular nuclei)</td>
<td>Vestibular: Posture and gait, eye movements, rotatory and caloric tests Hearing: Startle reaction, electrophysiology (EEG alerting, brainstem-evoked response)</td>
<td>Vestibular: Normal posture and gait, oculocephalic reflex, normal, brief postrotatory nystagmus and caloric-induced nystagmus Hearing: Startled reaction to handclap, evoked response present</td>
<td>Vestibular: Head tilt, head twist, circling, nystagmus, prolonged or absent postrotatory nystagmus, abnormal or absent caloric response Hearing: Poor startle reaction, no evoked response</td>
<td>Otitis media and otitis interna, idiopathic vestibular disease, polyneuropathy, brainstem disease</td>
<td></td>
</tr>
<tr>
<td>Cranial Nerve</td>
<td>Location</td>
<td>Functions</td>
<td></td>
<td></td>
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<tr>
<td>CN IX glossopharyngeal</td>
<td>Medulla (caudal)</td>
<td>Sensory: Solitary tract and nucleus Motor: Parasympathetic, ambiguous nucleus, exit together along lateral surface of medulla, exit through jugular foramen</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>Sensory and motor to pharynx and palate, parasympathetic to zygomatic and parotid salivary glands (in CN V); sensory to carotid body and sinus</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>Gag reflex</td>
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<td></td>
<td></td>
<td>Swallowing</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>Poor gag reflex, dysphagia</td>
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<tr>
<td></td>
<td></td>
<td>Rare, common in rabies; brainstem disease</td>
<td></td>
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<tr>
<td>CN X vagus</td>
<td>Medulla (caudal)</td>
<td>Same as CN IX</td>
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<tr>
<td></td>
<td></td>
<td>Sensory and motor to pharynx and larynx, thoracic and abdominal viscera</td>
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<tr>
<td></td>
<td></td>
<td>Gag reflex, laryngeal reflex, slap test, oculocardiac reflex</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>Swallowing, coughing, bradycardia</td>
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<tr>
<td></td>
<td></td>
<td>Poor gag reflex, dysphagia, inspiratory dyspnea, no abduction of laryngeal folds, regurgitation</td>
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<td></td>
<td></td>
<td>Rare, except in laryngeal paralysis; polyneuropathy</td>
<td></td>
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</tr>
<tr>
<td>CN XI accessory</td>
<td>Medulla (caudal) and cervical spinal cord</td>
<td>Ambiguous nucleus of medulla and cervical gray matter, axons run rostrally from cervical cord to join cranial roots, exit jugular foramen</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>Trapezius and parts of sternocleidomastoid and brachiocephalicus muscles</td>
<td></td>
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<td></td>
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<td>Palpate for atrophy of muscles; EMG</td>
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<td></td>
<td></td>
<td>Normal muscles</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>Atrophied muscles, denervation</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>Rare</td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>CN XII hypoglossal</td>
<td>Medulla (caudal)</td>
<td>Axons exit medulla lateral to pyramid, hypoglossal canal to tongue</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Movements of tongue</td>
<td></td>
<td></td>
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</tr>
<tr>
<td></td>
<td></td>
<td>Protrusion of tongue (wet nose), retraction of tongue</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>Tongue protrudes symmetrically and can lick in both directions, strong withdrawal of tongue</td>
<td></td>
<td></td>
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</tr>
<tr>
<td></td>
<td></td>
<td>Tongue deviates to side of lesion, atrophy, weak withdrawal</td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Brainstem disease, polyneuropathy</td>
<td></td>
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</tr>
</tbody>
</table>

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. 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.

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). 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. 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. 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.
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. Menta
tion 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.

Localizing 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

**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**

<table>
<thead>
<tr>
<th>Left</th>
<th>Reactions</th>
<th>Right</th>
</tr>
</thead>
<tbody>
<tr>
<td>NE</td>
<td>PL</td>
<td>NE</td>
</tr>
<tr>
<td>NE</td>
<td>TL</td>
<td>NE</td>
</tr>
</tbody>
</table>

**Spinal reflexes**

<table>
<thead>
<tr>
<th>Left</th>
<th>Reflex, Spinal Segment</th>
<th>Right</th>
</tr>
</thead>
<tbody>
<tr>
<td>+3</td>
<td>L4-6</td>
<td>+2 – +3</td>
</tr>
<tr>
<td>+2</td>
<td>Extensor carpi radialis C7-T1</td>
<td>+2</td>
</tr>
<tr>
<td>+2</td>
<td>Triceps C7-T1</td>
<td>+2</td>
</tr>
<tr>
<td>+2</td>
<td>Flexion–PL L5-S1</td>
<td>+2</td>
</tr>
<tr>
<td>+2</td>
<td>Flexion–TL C6-T1</td>
<td>+2</td>
</tr>
<tr>
<td>Absent</td>
<td>Crossed extensor</td>
<td>Absent</td>
</tr>
<tr>
<td>+2</td>
<td>Perineal S1-2</td>
<td>+2</td>
</tr>
</tbody>
</table>

**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**

**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 anti-inflammatory 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
*PulSATx:* Pain and crepitus in right coxofemoral joint

**Postural reactions**

<table>
<thead>
<tr>
<th>Left</th>
<th>Reactions</th>
<th>Right</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Proprioceptive positioning</td>
<td></td>
</tr>
<tr>
<td>+2</td>
<td>PL</td>
<td>+2</td>
</tr>
<tr>
<td>+2</td>
<td>TL</td>
<td>+2</td>
</tr>
<tr>
<td>+2</td>
<td>Wheelbarrowing, hopping</td>
<td>+2</td>
</tr>
<tr>
<td>+2</td>
<td>PL</td>
<td>+1</td>
</tr>
<tr>
<td>+2</td>
<td>TL</td>
<td>+1 +2</td>
</tr>
<tr>
<td>NE</td>
<td>Extensor postural thrust</td>
<td>NE</td>
</tr>
<tr>
<td>+2</td>
<td>Hemistand-hemiwalk</td>
<td>+1 +2</td>
</tr>
<tr>
<td>+2</td>
<td>Tonic neck</td>
<td>+2</td>
</tr>
<tr>
<td>NE</td>
<td>Placing, tactile</td>
<td>NE</td>
</tr>
</tbody>
</table>

**Spinal reflexes**

<table>
<thead>
<tr>
<th>Left</th>
<th>Reflex, Spinal Segment</th>
<th>Right</th>
</tr>
</thead>
<tbody>
<tr>
<td>+2</td>
<td>Quadriceps L4-6</td>
<td>+2− 3</td>
</tr>
<tr>
<td>+2</td>
<td>Extensor carpi radialis C7-T1</td>
<td>+2</td>
</tr>
<tr>
<td>+2</td>
<td>Triceps C7-T1</td>
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</tr>
<tr>
<td>+2</td>
<td>Flexion-PL LS-S1</td>
<td>+2</td>
</tr>
<tr>
<td>+2</td>
<td>Flexion-TL C6-T1</td>
<td>+2</td>
</tr>
<tr>
<td>+2</td>
<td>Absent</td>
<td>+2</td>
</tr>
<tr>
<td>+2</td>
<td>Crossed extensor Perineal Absent</td>
<td>+2</td>
</tr>
</tbody>
</table>

**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**

**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 levafoxione 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
*PulSATx:* Negative

Continued
**Postural reactions**

<table>
<thead>
<tr>
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<th>Left</th>
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<tbody>
<tr>
<td>Reactions</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Proprioceptive</td>
<td>0 PL</td>
<td>0</td>
</tr>
<tr>
<td>position</td>
<td>0 TL</td>
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</tr>
<tr>
<td>Wheelbarrowing</td>
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<td></td>
</tr>
<tr>
<td>Hopping</td>
<td>0 PL</td>
<td>0</td>
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<tr>
<td>0 TL</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Extensor postural</td>
<td>0 thrust</td>
<td>0</td>
</tr>
<tr>
<td>thrust</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>NE</td>
<td>Tonic neck</td>
<td>NE</td>
</tr>
<tr>
<td>Placing, tactile</td>
<td>NE PL</td>
<td>NE PL</td>
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<tr>
<td></td>
<td>NE TL</td>
<td>NE TL</td>
</tr>
<tr>
<td>NE</td>
<td>Placing, visual</td>
<td>NE Placing, visual</td>
</tr>
<tr>
<td></td>
<td>NE PL</td>
<td>NE Placing, tactile</td>
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<tr>
<td></td>
<td>NE TL</td>
<td>NE Placing, tactile</td>
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</table>

**Spinal reflexes**

<table>
<thead>
<tr>
<th></th>
<th>Left</th>
<th>Right</th>
</tr>
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<tbody>
<tr>
<td>Reflex, Spinal Segment Quadriceps</td>
<td>+2–+3</td>
<td>+2–+3</td>
</tr>
<tr>
<td>L4-6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Extensor carpi radialis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C7-T1</td>
<td>NE</td>
<td>NE</td>
</tr>
<tr>
<td>Triceps</td>
<td></td>
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<tr>
<td>C7-T1</td>
<td>NE</td>
<td>NE</td>
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<tr>
<td>Flexion-PL</td>
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</table>

*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.

---

**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**

**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.
- **Palpation:** normal

**Postural reactions**

<table>
<thead>
<tr>
<th>Left</th>
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<tbody>
<tr>
<td>+2 PL</td>
<td>0</td>
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<tr>
<td>+2 TL</td>
<td>+2</td>
</tr>
<tr>
<td>PL</td>
<td>0</td>
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<tr>
<td>TL</td>
<td>+2</td>
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<tr>
<td>NE</td>
<td>NE</td>
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<td>NE</td>
<td>NE</td>
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<tr>
<td>NE</td>
<td>NE</td>
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</table>

**Spinal reflexes**

<table>
<thead>
<tr>
<th>Left Reflex, Spinal Segment</th>
<th>Right</th>
</tr>
</thead>
<tbody>
<tr>
<td>Quadriceps</td>
<td></td>
</tr>
<tr>
<td>+2 L4-6</td>
<td>0</td>
</tr>
<tr>
<td>Extensor carpi radialis</td>
<td>+2</td>
</tr>
<tr>
<td>C7-T1</td>
<td>+2</td>
</tr>
<tr>
<td>Triceps</td>
<td>+2</td>
</tr>
<tr>
<td>C7-T1</td>
<td>+2</td>
</tr>
<tr>
<td>Flexion-PL</td>
<td>0</td>
</tr>
<tr>
<td>L5-S1</td>
<td>+2</td>
</tr>
<tr>
<td>Flexion-TL</td>
<td>+2</td>
</tr>
<tr>
<td>C6-T1</td>
<td>0</td>
</tr>
<tr>
<td>Crossed extensor</td>
<td>0</td>
</tr>
<tr>
<td>Perineal</td>
<td>+1</td>
</tr>
<tr>
<td>S1-2</td>
<td>0</td>
</tr>
</tbody>
</table>

**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**

**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

<table>
<thead>
<tr>
<th>Left Reactions</th>
<th>Right Reactions</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 Proprioceptive positioning</td>
<td>0 Proprioceptive positioning</td>
</tr>
<tr>
<td>+2 PL</td>
<td>+2 PL</td>
</tr>
<tr>
<td>+2 TL</td>
<td>+2 TL</td>
</tr>
<tr>
<td>Wheelbarrowing Hopping</td>
<td>Wheelbarrowing Hopping</td>
</tr>
<tr>
<td>0</td>
<td>NE</td>
</tr>
<tr>
<td>+2</td>
<td>+2</td>
</tr>
<tr>
<td>NE Extensor postural thrust</td>
<td>NE Extensor postural thrust</td>
</tr>
<tr>
<td>NE Hemistand-hemiwalk</td>
<td>NE Hemistand-hemiwalk</td>
</tr>
<tr>
<td>NE Tonic neck Placing, tactile</td>
<td>NE Tonic neck Placing, tactile</td>
</tr>
<tr>
<td>+2 NE +2 NE</td>
<td>+2 NE +2 NE</td>
</tr>
<tr>
<td>Placing, tactile</td>
<td>Placing, tactile</td>
</tr>
<tr>
<td>NE PL</td>
<td>NE PL</td>
</tr>
<tr>
<td>NE TL</td>
<td>NE TL</td>
</tr>
<tr>
<td>Placing, visual</td>
<td>Placing, visual</td>
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<tr>
<td>NE</td>
<td>NE</td>
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<tr>
<td>NE</td>
<td>NE</td>
</tr>
</tbody>
</table>

### Spinal reflexes

<table>
<thead>
<tr>
<th>Left Reflex, Spinal Segment</th>
<th>Right Reflex, Spinal Segment</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 Quadriceps</td>
<td>0 Quadriceps</td>
</tr>
<tr>
<td>+2 L4-6</td>
<td>+2 L4-6</td>
</tr>
<tr>
<td>+2 Extensor carpi radialis</td>
<td>+2 Extensor carpi radialis</td>
</tr>
<tr>
<td>+2 C7-T1</td>
<td>+2 C7-T1</td>
</tr>
<tr>
<td>+2 Triceps</td>
<td>+2 Triceps</td>
</tr>
<tr>
<td>+2 C7-T1</td>
<td>+2 C7-T1</td>
</tr>
<tr>
<td>Flexion-PL</td>
<td>Flexion-PL</td>
</tr>
</tbody>
</table>

### Cranial nerves: normal

**Sensory evaluation**

1. Hyperesthesia: none
2. Superficial pain perception: poor caudal to T13; the cutaneous trunci reflex is absent behind caudal to T13 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**

### 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

<table>
<thead>
<tr>
<th>Left Reactions</th>
<th>Right Reactions</th>
</tr>
</thead>
<tbody>
<tr>
<td>+2 Proprioceptive positioning</td>
<td>+2 Proprioceptive positioning</td>
</tr>
<tr>
<td>+2 PL</td>
<td>+2 PL</td>
</tr>
<tr>
<td>+2 TL</td>
<td>+2 TL</td>
</tr>
</tbody>
</table>

### 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.

CASE STUDY 2-8  CHLOE

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

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.

Assessment (anatomic diagnosis and estimation of prognosis)

Diagnostic plan

Rule-outs
1. 
2. 
3. 
4. 

Postural reactions

Spinal reflexes

Cranial nerves

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.
**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**

**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**

<table>
<thead>
<tr>
<th>Left</th>
<th>Reactions</th>
<th>Right</th>
</tr>
</thead>
<tbody>
<tr>
<td>+2</td>
<td>Proprioceptive positioning</td>
<td>0</td>
</tr>
<tr>
<td>+2</td>
<td>PL</td>
<td>+2</td>
</tr>
<tr>
<td>+2</td>
<td>TL</td>
<td>+2</td>
</tr>
<tr>
<td>+2</td>
<td>Wheelbarrowing</td>
<td>0</td>
</tr>
<tr>
<td>+2</td>
<td>Hopping</td>
<td>+2</td>
</tr>
</tbody>
</table>

**Spinal reflexes**

<table>
<thead>
<tr>
<th>Left</th>
<th>Reflex, Spinal Segment Quadriceps</th>
<th>Right</th>
</tr>
</thead>
<tbody>
<tr>
<td>+2</td>
<td>L4-6</td>
<td>+2</td>
</tr>
</tbody>
</table>

**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

**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**

<table>
<thead>
<tr>
<th>Left</th>
<th>Reactions</th>
<th>Right</th>
</tr>
</thead>
<tbody>
<tr>
<td>+2</td>
<td>PL</td>
<td>+1</td>
</tr>
<tr>
<td>+2</td>
<td>TL</td>
<td>+1</td>
</tr>
<tr>
<td>+2</td>
<td>Wheelbarrowing</td>
<td>+1</td>
</tr>
<tr>
<td></td>
<td>Hopping</td>
<td></td>
</tr>
<tr>
<td>+2</td>
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<tr>
<td>+2</td>
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<td>+1</td>
</tr>
<tr>
<td>+2</td>
<td>Extensor postural thrust</td>
<td>+1</td>
</tr>
<tr>
<td>+2</td>
<td>Hemistand-hemiwalk</td>
<td>+1</td>
</tr>
<tr>
<td>NE</td>
<td>Tonic neck</td>
<td>NE</td>
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<tr>
<td></td>
<td>Placing, tactile</td>
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<td>PL</td>
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<tr>
<td>NE</td>
<td>TL</td>
<td>NE</td>
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**Spinal reflexes**

<table>
<thead>
<tr>
<th>Left Reflex, Spinal Segment</th>
<th>Right Quadriceps</th>
</tr>
</thead>
<tbody>
<tr>
<td>+2</td>
<td>L4-6</td>
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<td>Extensor carpi radialis</td>
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<tr>
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<td>C7-T1</td>
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<tr>
<td>+2</td>
<td>Triceps</td>
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<tr>
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<td>C7-T1</td>
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<td>+2</td>
<td>Flexion-PL</td>
</tr>
<tr>
<td>+2</td>
<td>L5-S1</td>
</tr>
<tr>
<td>+2</td>
<td>Flexion-TL</td>
</tr>
<tr>
<td>+2</td>
<td>C6-T1</td>
</tr>
<tr>
<td>Absent</td>
<td>Crossed extensor</td>
</tr>
<tr>
<td>Absent</td>
<td>Perineal</td>
</tr>
<tr>
<td>+2</td>
<td>S1-2</td>
</tr>
</tbody>
</table>

**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

**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
CHAPTER 2  Localization of Lesions in the Nervous System

CASE STUDY 2-1

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 intervertebral 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 (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 meningocele 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 coxofemoral joint (CFJ); mass right cranial dorsal brainstem

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**CASE STUDY 2-3**

**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 (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

- **Case Summary**
  Case spinal 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 O2

  Regurgitation: metoclopramide SC q8h


  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.
CHAPTER 2 Localization of Lesions in the Nervous System

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.

- **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.

REFERENCES