CHAPTER

# 28

# NEUROLOGY

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Companion and aviary birds frequently develop clinical signs associated with the central or peripheral nervous system. These changes may include depression, blindness, opisthotonos, head tilt, circling, tremors, ataxia, convulsions, paresis and paralysis. Neurologic changes in birds may occur from primary or secondary diseases including genetic abnormalities, neoplasms, metabolic diseases, malnutrition, exposure to toxins, trauma and bacterial, viral, fungal or parasitic infections.

In a retrospective study of avian patients with neurologic disorders, the clinical signs most frequently observed were seizures, ataxia, paresis, paralysis, intention tremors, circling, head tilt, nystagmus, abnormal mentation and visual deficits. General supportive care for patients in this study included antibiotic therapy, fluid support, parenteral vitamin therapy and gavage feeding for nutritional support. The two most common etiologies were lead toxicity and hypocalcemia/vitamin $D_3$ deficiency. Many other neurologic problems resolved with general supportive care and vitamin supplementation.[87]

Diagnostic tests that may be of value in determining the etiology of neural lesions, as well as the prognosis for recovery in some cases, include CBC, biochemistries, specific tests for detecting levels of toxins, radiographs, electroencephalograms, electromyograms, auditory evoke potentials, CT scans and MRI. Some of these diagnostic techniques require specialized equipment, but their efficacy in diagnosing neurologic diseases in birds has been documented. Many of the advanced neurologic diagnostic tests are available at veterinary colleges, and case referral should be considered when one of these techniques is needed to evaluate a patient.

# Neuroanatomy[69]

## Meninges

As in mammals, birds have three meninges: pia mater, arachnoid and dura mater. The subarachnoid space contains cerebrospinal fluid that may be collected from the cisterna magna; however, birds have a large venous sinus located dorsal to the cisterna that is easily damaged during a spinal tapping procedure and, if disrupted, will cause severe hemorrhage.

## Brain

The brain of birds is agyric, with virtually no convolutions (Figure 28.1). The vallecula arises near the rostral end of the median fissure and extends caudad. The sagittal eminence is a prominence medial to the vallecula. The lateral ventricles are displaced dorsally by the large corpus striatum.[83] The olfactory bulbs are pointed at the rostral extent of the brain, and the olfactory center of the brain is rather underdeveloped.[28] The cerebral cortex is also underdeveloped (being two to three cell-layers thick, <1 mm); however, when compared with mammals, the corpus striatum is well developed and is considered the main center for association in birds (Figure 28.2). Consequently, instincts dominate avian behavior, which may account for some of the self-mutilation that occurs in companion birds. Birds have no corpus callosum nor septum pellucidum.

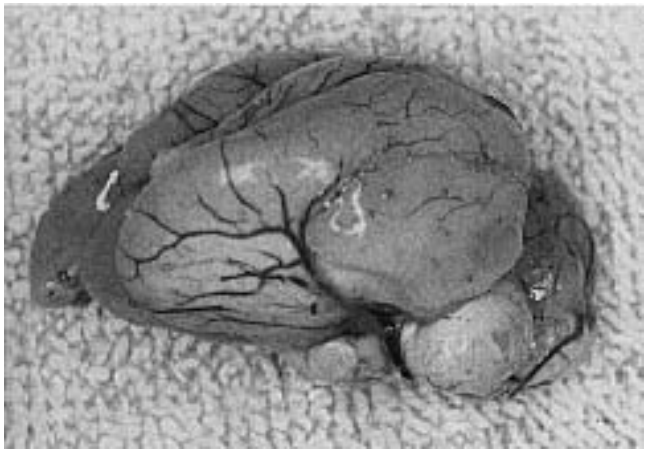


**FIG 28.1** The brain of birds is agyric with virtually no convolutions.

The diencephalon is the location of the pineal body, which lies dorsal and medial between the caudal aspects of the cerebral hemispheres. It is composed of secretory cells, which resemble rudimentary photoreceptors. The nonmyelinated axons are responsive to light. The pathway for stimulation courses from the optic nerve to the cranial cervical ganglion, which has axons to the pineal body. Interestingly, these axons are still responsive to light even if the bird's eyes are removed and the cranial cervical ganglion is removed. The pineal body is involved in reproduction, migration and circadian rhythms. Its secretions exert their effect on the hypothalamus. The hypothalamus is located caudal (or ventral) to the optic chiasm.

### Midbrain

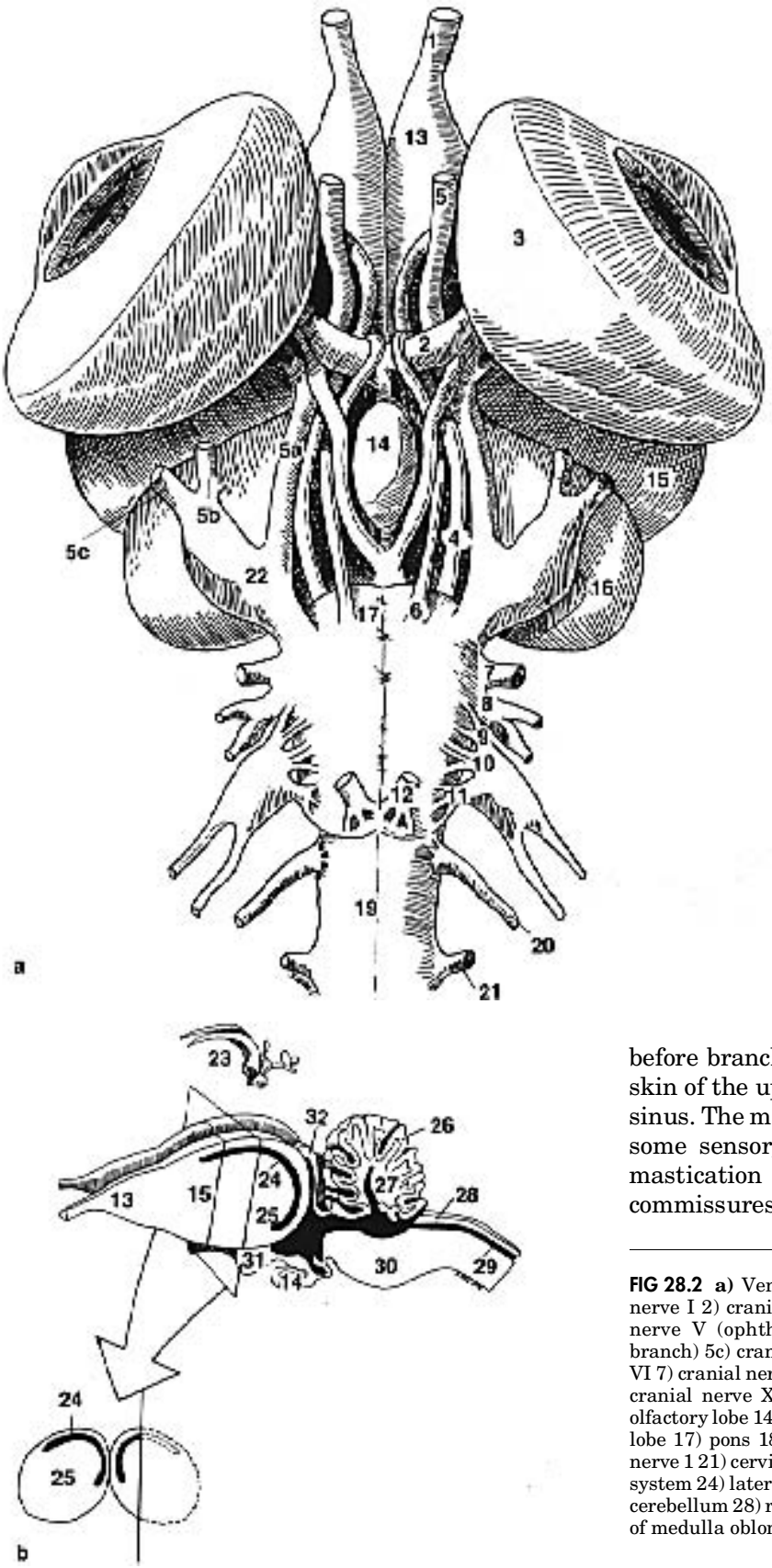
The midbrain is the location of the optic tectum. The avian optic lobes are massive and responsible for the well developed, though monocular, vision of most birds (Figure 28.2). The optic tectum is equivalent to the rostral colliculus of mammals and birds have no caudal colliculus. Cranial nerves III and IV exit from the midbrain. Large motor afferent, efferent and optic fiber systems originate, decussate and terminate in the mesencephalon and myelencephalon. There is complete decussation of tectospinal (motor) and rubrospinal (modulation) tracts in the midbrain, while the tracts of the pons and medulla do not decussate.[1]

### Cerebellum

As in mammals, the cerebellum is the center for coordination of movements and is correspondingly large. It has a single median lobe with transverse sulci and is divided into three main lobes (not four as in mammals) by the fissura prima and fissura secunda, which are deeper transverse sulci. Smaller transverse sulci divide it into ten primary lobules. Each lobule is believed to be responsible for coordination of a specific part of the body. For example, lobules II and III control leg coordination. The lateral flocculus (lobule X) with the paraflocculus (lobule IX) are located at the rostral aspect. The rostral and caudal cerebellar peduncles attach the cerebellum to the medulla. There may also be a cerebellar peduncle.

### Pons

The pons is poorly developed and is present only as a broad band of fibers at the rostral portion of the medulla oblongata. There is no pyramid as found in mammals. Cranial nerves V through XII arise from the medulla.




## Cranial Nerves

The cranial nerves of birds correspond to those found in mammals (Figure 28.3). The olfactory nerve (CN I) is sensory and passes through a single hole in the skull (the olfactory foramen) rather than a cribriform plate. The optic nerve (CN II) is sensory and large in order to accommodate the visual acuity birds require. It is usually more than half the diameter of the spinal cord. The oculomotor nerve (CN III) is motor to the dorsal, ventral and medial rectus muscles and the ventral oblique muscle of the eye. It also supplies the muscles of the eyelids. It has parasympathetic fibers to the gland of the third eyelid, the choroid, the iris and the pecten (triangular pleated membrane extending forward from the optic disc). The trochlear nerve (CN IV) is motor to the dorsal oblique muscle of the eye.

The trigeminal nerve (CN V) contains both sensory and motor fibers. The ophthalmic branch is sensory and consists of two components. The dorsal component innervates the upper eyelid and the skin of the forehead. The ventral component is sensory to the nasal cavity and upper beak. The maxillary branch is sensory and innervates the upper eyelid before branching to supply the lower eyelid, palate, skin of the upper beak, nasal cavity and infraorbital sinus. The mandibular branch is motor but may have some sensory fibers. It innervates the muscles of mastication and the skin and the mucosa at the commissures of the beak.

**FIG 28.2** **a)** Ventral and **b)** lateral view of the brain. 1) cranial nerve I 2) cranial nerve II 3) eye 4) cranial nerve IV 5a) cranial nerve V (ophthalmic branch) 5b) cranial nerve V (maxillary branch) 5c) cranial nerve V (mandibular branch) 6) cranial nerve VI 7) cranial nerve VII 8) cranial nerve VIII 9) cranial nerve IX 10) cranial nerve X 11) cranial nerve XI 12) cranial nerve XII 13) olfactory lobe 14) pituitary gland 15) cerebral hemisphere 16) optic lobe 17) pons 18) medulla oblongata 19) spinal cord 20) cervical nerve 1 21) cervical nerve 2 22) trigeminal ganglion 23) ventricular system 24) lateral ventricle 25) corpus striatum 26) arbor vitae 27) cerebellum 28) roof of medulla oblongata 29) central canal 30) floor of medulla oblongata 31) optic chiasm and 32) pineal body.

The abducent nerve (CN VI) is motor to the lateral rectus muscles and muscles of the third eyelid. The facial nerve (CN VII) is motor with a small sensory component and parasympathetic fibers. It is not involved in taste as in mammals, but supplies the hyoid and the cutaneous neck muscles. The vestibulocochlear nerve (CN VIII) has separate ganglia for the vestibular and the cochlear nerves. The glossopharyngeal nerve (CN IX) is sensory and motor. Cranial nerves IX, X, XI arise from a row of small roots. Cranial nerves X and XI combine in a common ganglion. The lingual branch of the glossopharyngeal nerve receives sensory input from taste fibers.

The pharyngeal branch has fibers that join with the vagus nerve to innervate the larynx and trachea. The esophageal branch courses along the neck with the jugular vein, supplying the esophagus. The vagus nerve (CN X), CN XI and jugular vein course down the neck in a common sheath to the level of the nodose ganglion. The spinal accessory nerve (CN XI) is enclosed with the vagus and supplies the superficial muscles of the neck. The hypoglossal nerve (CN XII) and CN IX innervate the tracheal muscles. The former has a lingual branch that innervates the tongue muscles and a syringeal branch that courses to the syrinx and tracheal muscles.

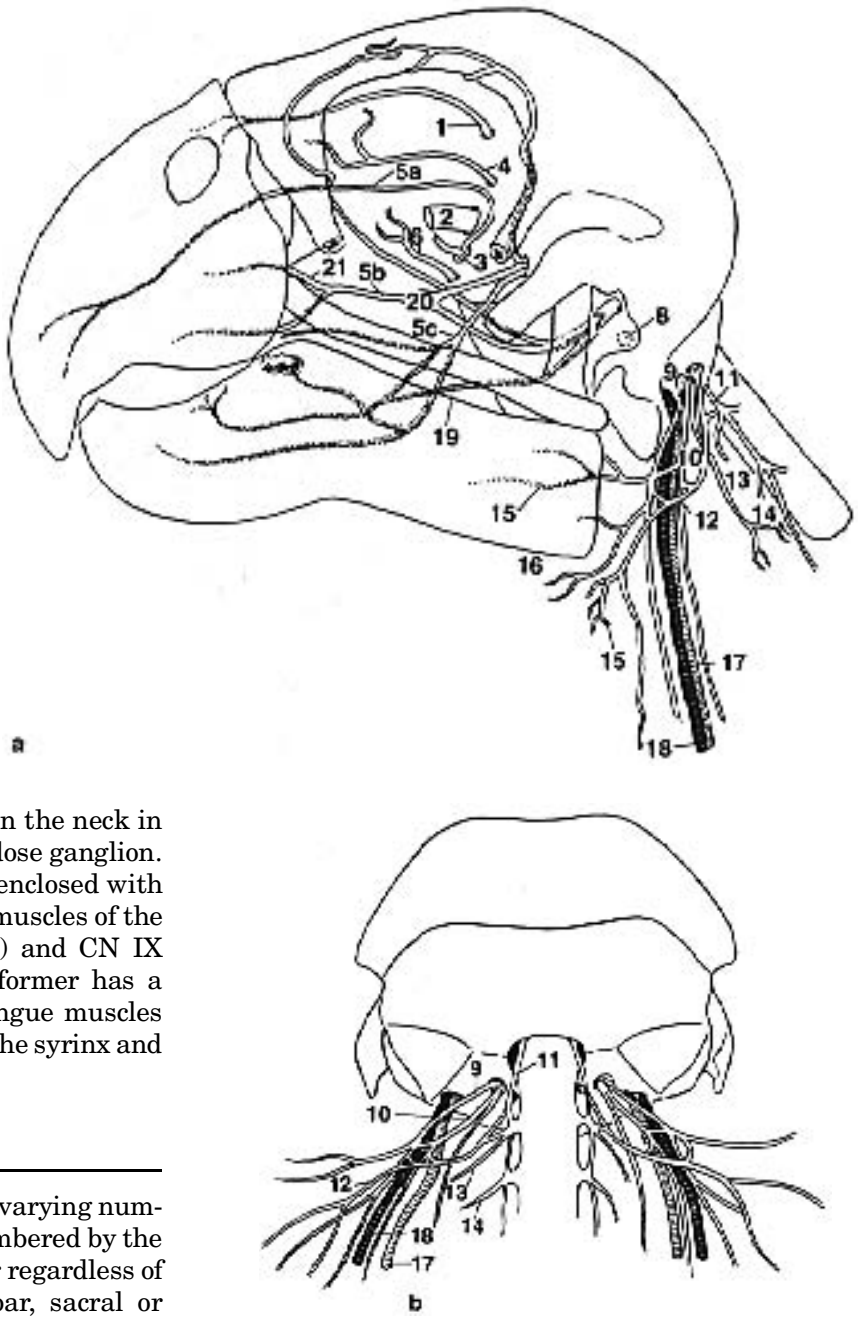



**FIG 28.3** **a)** Lateral and **b)** caudal view of the cranium showing the position of the cranial nerves. 1) cranial nerve I 2) cranial nerve II 3) cranial nerve III 4) cranial nerve IV 5a) cranial nerve V (ophthalmic branch) 5b) cranial nerve V (maxillary branch) 5c) cranial nerve V (mandibular branch) 6) cranial nerve VI 7) cranial nerve VII 8) cranial nerve VIII 9) cranial nerve IX 10) cranial nerve X 11) cranial nerve XI 12) cranial nerve XII 13) cervical nerve 1 14) cervical nerve 2 15) lingual branch 16) pharyngeal branch 17) internal carotid artery 18) jugular vein 19) corda tympani 20) sphenopalatine ganglion and 21) infraorbital nerve.

## Spinal Nerves

Because birds of different species have varying numbers of vertebrae, spinal nerves are numbered by the vertebra caudal to it in numerical order regardless of whether it is cervical, thoracic, lumbar, sacral or coccygeal. In discussions of plexuses, it is customary to refer to the roots making up the plexus by the ordinal number of their cranial to caudal location (Figure 28.4). The lumbar spinal cord contains a dorsal gelatinous structure (glycogen body), which is nestled in a deep cleft between the dorsal columns.[83]

The brachial plexus is formed by ventral branches of three to five spinal nerves. The ram communicans lateralis and medialis connect the second to the third nerve roots and the fourth to the fifth roots (if present). These are sympathetic nerve fibers. The sec-

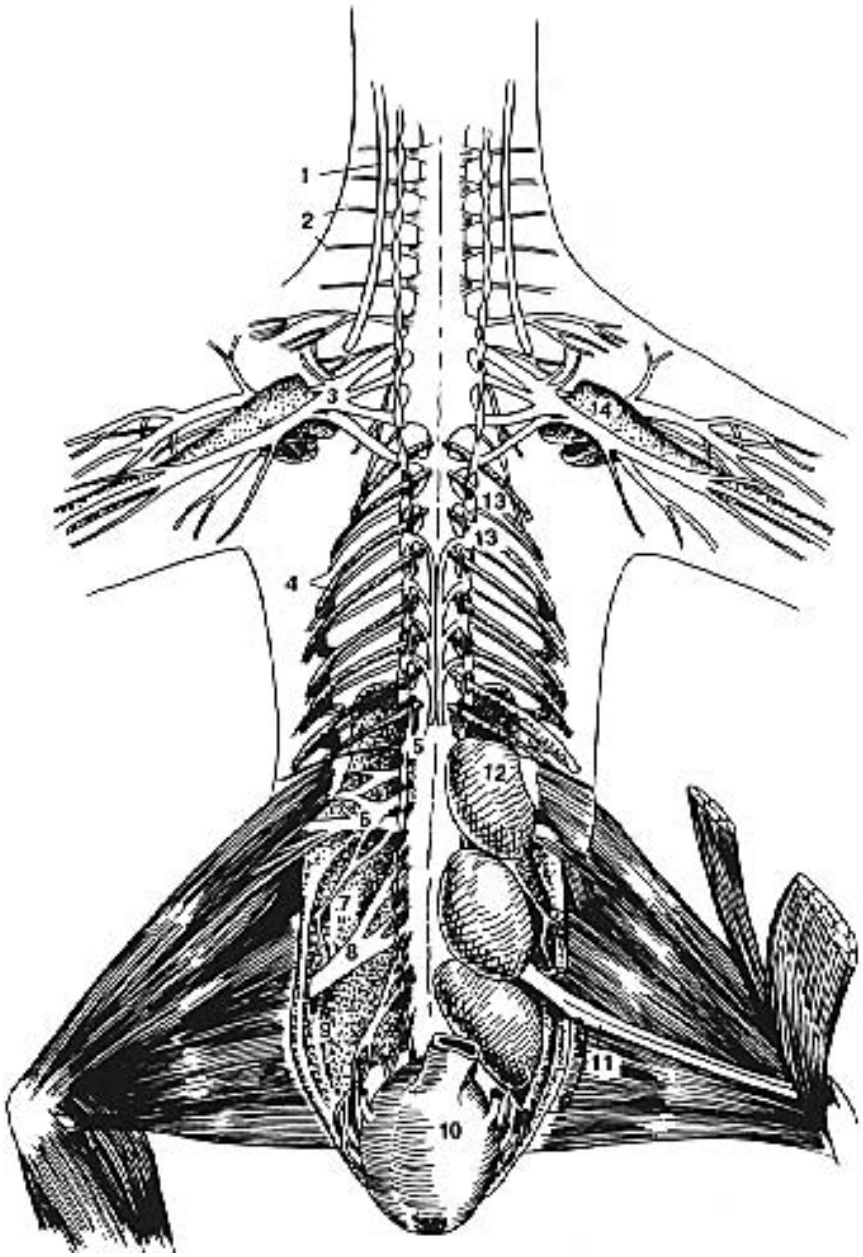



**FIG 28.4** Ventrodorsal view of the spinal nerves and autonomic nervous system. The right kidney has been removed to show their relationship with the lumbar plexuses. 1) vagus 2) cervical spinal nerves 3) brachial plexus 4) thoracic spinal nerves 5) greater splanchnic nerves 6) lumbar plexus 7) obturator nerve 8) sacral plexus 9) pudendal plexus 10) cloaca 11) ischiatic nerve 12) cranial division of kidney 13) autonomic ganglion and 14) humerus.

ond through fourth roots give off rami musculares to innervate the neck muscles. The nerves arising from this plexus innervate the muscles of the wing.

There are three nerve plexuses in the lumbosacral region (lumbar, ischiatic and pudendal). These nerve roots lie embedded in the foveae of the pelvis surrounded by the kidneys (see Anatomy Overlay). All three plexuses are connected to the sympathetic chain. The lumbar plexus is composed of three to four nerve roots (the last two lumbar and first sacral roots). It innervates the cranial thigh muscles and the muscles of the body wall. The obturator nerve, femoral nerve, cranial gluteal nerve and saphenous nerve arise from this plexus.

The ischiatic plexus or sacral plexus is usually made up of six sacral nerve roots, but occasionally four, five or seven roots. The first root arises with the last root of the lumbar plexus. The roots combine to form the ischiatic nerve, which is the largest nerve in body. This nerve should be faintly striated and a loss of striations is suggestive of an inflammatory process. The caudal gluteal nerve also arises from the sacral plexus.[83]

The pudendal plexus is formed by five thin coccygeal nerve roots and supplies the cloaca and tail.

# Neurologic Examination

Two of the primary objectives in performing a neurologic examination are to determine if the neuropathy is focal or diffuse, and to localize focal lesions. The examination should be performed in a consistent, logical manner that starts with a complete history (see Chapter 8). Neuropathies are particularly common secondary to trauma, exposure to toxins and malnutrition. Subtle changes in cranial nerve function and abnormal reflexes are difficult to appreciate and interpret in birds. Assessment of segmental reflexes may be difficult in avian patients, making evaluation of muscle tone, strength and atrophy an essential part of the neurologic examination.

Neurologic evaluation of neonates is particularly difficult, and making a "side-by-side" comparison with a "normal" clutchmate is the best way to detect subtle changes. Useful tests include evaluation of the feeding response, menace reflex, use of wings to balance, vocalization, perching ability, pain perception and hopping response.[65]

## Mental Status

The patient's mental status and level of consciousness should be evaluated. Any personality changes reported by the owner should be noted. The bird's ability to perform normal activities and its awareness of its surroundings should be assessed. Clients may indicate that the bird acts sleepy, dull, uneasy, nervous, anxious, aggressive or dizzy. It is difficult to differentiate between seizures and syncope based on the history; however, either may suggest an intracranial lesion. Seizures may be characterized by ataxia, disorientation and falling off a perch followed by tonic clonic convulsions.

## Cranial Nerves

An evaluation of the cranial nerves may help localize a focal brain lesion (individual nerves involved) or a generalized encephalopathy (several nerves involved). Olfaction (CN I) is difficult to assess because birds have a poor sense of smell; however, most normal birds will react negatively to noxious odors (eg, alcohol pledget). Birds with CN I dysfunction may exhibit an altered appetite or feeding response.

An ophthalmic examination is essential for detecting neurologic abnormalities (see Chapter 26). Failure to avoid obstacles may indicate vision impairment. Bilateral blindness without ocular lesions may indicate neoplasm, abscess or granuloma formation in the brain.[97] The menace reflex can be used to evaluate CN II and VII; however, depending on the circumstances, the absence of a menace response does not always indicate dysfunction of these cranial nerves. The pupillary light response evaluates CN II and III. Because there is complete decussation of the optic nerves at the chiasm, birds do not have a consensual pupillary light response. [68]

Birds have some degree of voluntary control of pupil size because of skeletal muscles in the iris.[28,68] Excited birds will voluntarily dilate and constrict their pupils. The presence of anisocoria may indicate dysfunction of CN III or a sympathetic neuropathy. Normal eye movements require the coordination of CN III, IV, VI and VIII as well as the cerebellum and brain stem.[87] The presence of nystagmus or strabismus may indicate an abnormality in this system (vestibular). A fundic examination may be performed with the aid of d-tubocurarine. Its action on the avian pupil may be to inhibit the iris constrictor muscles allowing the myoepithelial dilator action to dominate.[68] In some birds, intracameral injection of 0.045-0.09 mg d-tubocurarine chloride produces mydriasis within five minutes without systemic effects.[68]

Cranial nerve V is responsible for facial sensation, movement of the mandible and blinking of the eyelids. Diminished beak strength may indicate an abnormality in CN V. Eye blink involves both CN V and VII. A defect in CN VIII will cause deafness or a head tilt toward the affected side. Cranial nerves IX through XII are involved in normal tongue movement, swallowing and beak strength. Dysfunction is manifested by dysphagia. Deviation of the tongue or atrophy of its muscles is observed with damage to CN XII.

Loss of normal physiologic nystagmus may occur with bilateral CN VIII lesions or with severe brain stem lesions.[87] Altered consciousness is usually an accompanying sign with brain stem lesions. Abnormal, spontaneous nystagmus may result from vestibular lesions. Strabismus may indicate vestibular system dysfunction or a lesion in CN III, IV or VI.[87]

Horner's syndrome may occur with intracranial lesions or with a lesion in the cervical sympathetic tract or the brachial plexus.[87]

## Locating Lesions

Reflexes are evaluated to help determine if a lesion is central (upper motor neuron) or peripheral (lower motor neuron). Wing droop, inability to fly and diminished or absent pain perception may be present with either central or peripheral lesions. Pain perception in the wing requires intact peripheral nerves and the cervical spinal cord. Wing withdrawal is a segmental reflex that is present with intact peripheral nerves, but does not require an intact cervical spinal cord. A spinal cord lesion should cause hyperreflexia; however, hyperreflexia is difficult to distinguish from normoreflexia.

In most situations, it is sufficient to determine if a reflex is present or absent. Weakness in the legs, knuckling over and an inability to grasp perches with the feet may be observed with either upper motor

neuron lesions or lower motor neuron lesions. The patellar reflex is difficult to assess in birds; however, the withdrawal reflex is also a segmental reflex and should be intact with lesions affecting only the spinal cord.[87]

Conscious proprioception requires an intact peripheral and central nervous system. A lesion in either will result in the bird knuckling over. The vent response is a segmental reflex, and the sphincter should be responsive to stimulation if a spinal cord lesion is present and the nerve roots are not affected. A crossed extensor reflex generally indicates a lesion in the spinal cord with a loss of normal central inhibitory pathways.

With cervical spinal cord lesions, dysfunction of the wings, legs and cloaca may be observed while head function and cranial nerves appear normal (Figure 28.5). Weakness in the wings and legs with intact leg and wing withdrawal and vent response would be indicative of a cervical spinal cord lesion. Lesions affecting the thoracolumbar spinal cord will cause leg and cloacal dysfunction without affecting the head, cranial nerves or wings. Cloacal sphincter hypertonia, incontinence and soiling of the vent without signs of head, wing or leg dysfunction are indicative of a lumbosacral spinal cord lesion.

Loss of pain perception indicates a poor prognosis for recovery.[87] It is crucial to differentiate pain perception from withdrawal reflex. Because withdrawal of a stimulated extremity is a segmental reflex and does not require an intact spinal cord for a normal response, movement does not indicate the patient is able to feel the stimulus. Some type of conscious recognition of the stimulus must be identified (eg, vocalization, attempting to bite or escape behavior). This part of the examination is generally reserved for last so that the painful stimulus does not influence the patient's response to other segments of the neurologic examination.

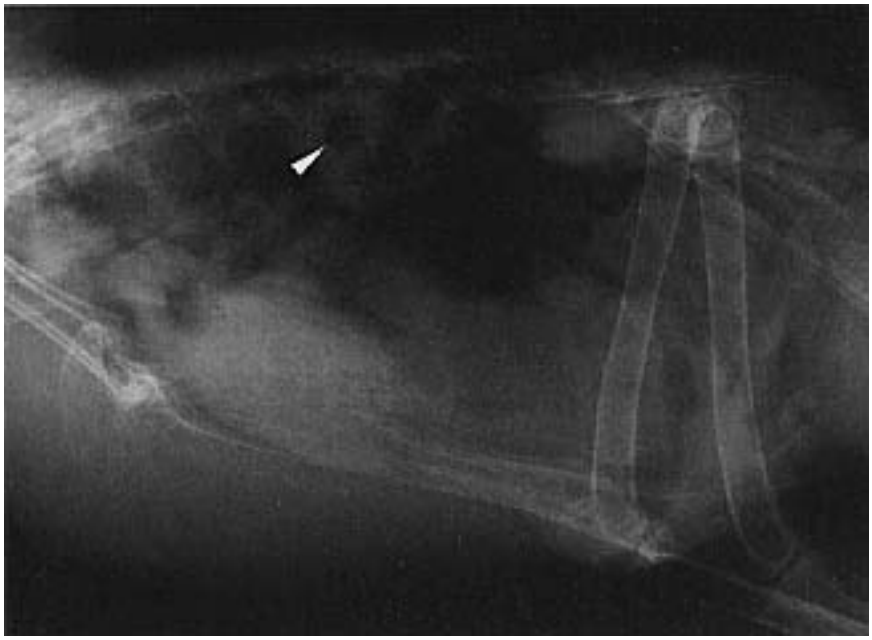


**FIG 28.5** A mature Amazon parrot was presented for emergency evaluation after being bitten by a dog. The bird had several large puncture wounds in the thorax. The bird was recumbent, and a deep pain response could not be elicited from either pelvic limb. Radiographs indicated a puncture wound through the lung (arrow) with an increased soft tissue density (blood) in portions of the lung parenchyma. The bird was placed on broad-spectrum antibiotics and steroids. A deep pain response was noted five days after the initial injury, and the bird slowly improved with a complete return to normal function over a three-month period.

# Diagnostic Techniques

The results of the neurologic examination will suggest which diagnostic tests should be performed. A CBC and serum chemistry profile are indicated if an infectious or metabolic neuropathy is considered. Laparoscopy and organ biopsy may be indicated to further define metabolic neuropathies. Serum for viral diseases or chlamydiosis, and blood levels for heavy metals are indicated in some cases. Radiographs are indicated if spinal trauma or heavy metal intoxication is suspected. TSH stimulation test may be helpful if hypothyroidism is suspected. Electromyograms, nerve conduction velocities, spinal evoked potentials and nerve or muscle biopsies are helpful in evaluating neuropathies.

## Electrodiagnostics

Electrodiagnostic studies are used commonly in mammals for localizing neurologic lesions and aiding in prognostic assessment. When available, electrodiagnostic techniques are valuable in avian patients for distinguishing between a neuropathy and a myopathy, localization of

neurologic lesions and determining the prognosis for return to normal function.

### Electromyogram (EMG)

Diseases of motor neuron cell bodies, ventral nerve roots, nerve plexuses, peripheral nerves, neuromuscular junctions and muscle fibers may alter the electromyogram (EMG), which is a recording of the electrical activity in striated muscle.[27] Normal activity consists of insertion potentials, motor unit potentials and spontaneous waves, which occur infrequently. When the electrode is inserted into the muscle, the intrafascicular nerve branches and muscle fibers are stimulated, creating a brief burst of electrical activity, which ceases immediately after the electrode stops moving. If the electrode is moved, insertional activity will again be recorded. Large positive waves are occasionally observed. If the electrode is inserted coincidentally near a motor endplate, a low continuous level of electrical activity will be recorded with an auditory component sounding like a distant beach surf. Motor unit potentials occur during involuntary muscle contraction or when a motor nerve is stimulated (an M response). The M response has two phases and represents the sum of the electrical activity of all of the muscle fibers in that motor unit.

Fibrillation potentials, positive sharp waves, myopathic potentials and reinnervation potentials are abnormal EMG recordings. Prolonged insertional activity due to muscle hyperexcitability occurs six to ten days following peripheral nerve injury, then gradually decreases. Fibrillation potentials are mono- or biphasic and occur five to seven days following denervation. These spontaneous, repetitive action potentials from muscle fibers, not produced by nerve impulses, occur because of the instability of the cell membrane at the endplate. Fibrillation potentials increase for several weeks after denervation, then decrease as muscle atrophy and fibrosis occur. They stop if reinnervation occurs.

Positive sharp waves are generally associated with denervation, but may be observed with primary myopathies. They are characterized by an initial positive deflection followed by a slower negative potential with a "dive bomber engine" auditory component. They may be single or multiple. Myopathic potentials are generally indicative of a primary myopathy but may be observed with fibrillation potentials following denervation. They are continuous discharges with varying amplitude, duration and frequency with a waxing and waning auditory component sounding like an attacking then retreating dive bomber. As reinnervation occurs, motor unit potentials are initially low amplitude and polyphasic but become larger than normal motor unit potentials and are an indicator of a good prognosis for recovery.

Nerve conduction velocities (NCV) can provide information regarding delayed transmission along a nerve as well as the location of a peripheral nerve lesion. A nerve stimulator is used to generate an M response at two different locations along the course of a peripheral nerve. The distance between the sites is divided by the latency difference in the two M responses to determine the velocity with which the impulse travels along the nerve (m/s). Where there is a peripheral neuropathy such as demyelination, the velocity is slow and the M responses are polyphasic and prolonged. If the nerve has been transected, the stimulation distal to the site will produce an M response, while stimulation of the site proximal to the lesion will not.

Standard EMG equipment may be used to evaluate H-wave and F-wave reflexes. The H-wave reflex evaluates both the afferent and efferent pathways. A peripheral site is stimulated and sensory impulses are carried to the spinal cord, where the alpha motor neurons are activated and discharged, resulting in a compound muscle section potential. The F-wave reflex is evaluated by stimulating the peripheral nerve with a lower intensity than that required to cause an H-wave production, activating the motor neuron to generate an efferent impulse. The H-wave and F-wave reflexes are used in combination to evaluate nerve root avulsion.

Signal-averaging capabilities are required for somatosensory-evoked potentials, spinal-evoked potentials and motor-evoked potentials. Somatosensory-evoked potentials correspond clinically to the presence or absence of pain perception. They may be utilized to determine if failure to react to a painful stimulus is the result of other more painful injuries, stoicism or denervation. This procedure is performed by stimulating a peripheral nerve and recording the response in the cervical spinal cord or cerebral cortex. It is important to recognize that these evoked potentials evaluate sensory, not motor nerve function. A response may persist after permanent loss of motor function.

Spinal-evoked potentials are utilized to determine the location of a spinal cord lesion. Stimuli are applied to peripheral nerves and the responses are recorded by an electrode inserted near the spinal

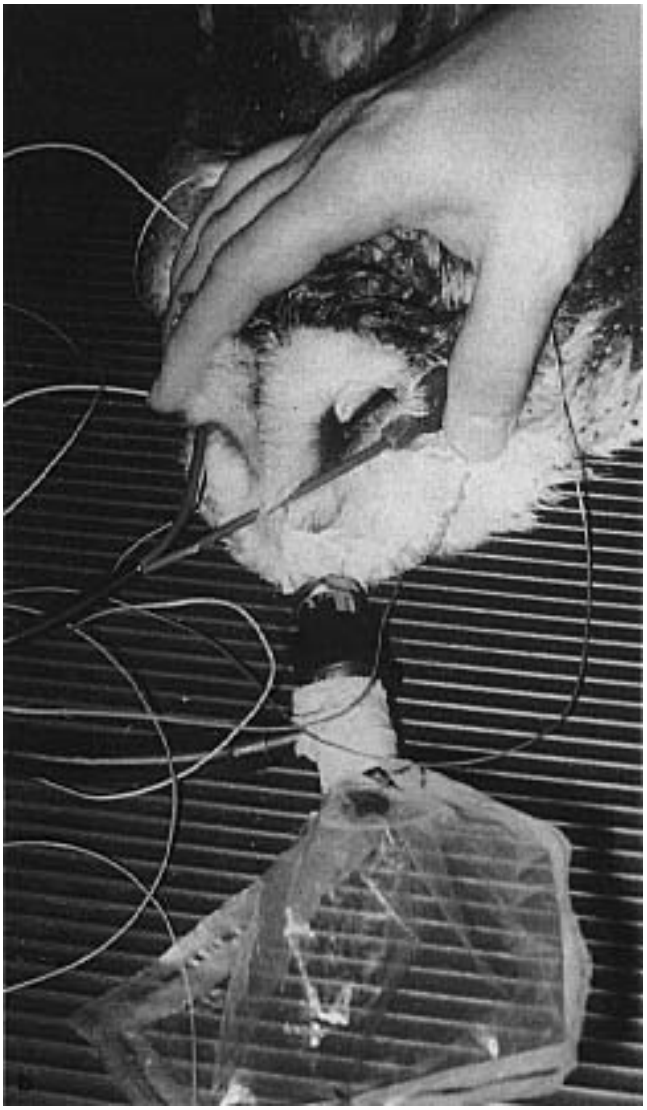


**FIG 28.6** **a)** A Barn Owl was presented with a progressive head tilt and ataxia. Physical examination revealed a discharge of necrotic debris from the right ear canal that contained gram-negative rods. **b)** Auditory-evoked potentials indicated a centralized inflammatory disease (see Color 14).

cord. They can discretely evaluate the sensory pathway of a focal segment of spinal cord such that loss of response cranial to a specific vertebra identifies the location of the lesion. Spinal-evoked potentials also evaluate only sensory function. Motor-evoked potentials are capable of evaluating motor function, but techniques are not well established for animal use. They are currently being evaluated for safety, effects of anesthetics and correlation with injury.

### Electroencephalograms

Electroencephalograms (EEG) are continuous recordings of the electrical activity of the cerebral cortex. They vary with head size, environment, restraint techniques and state of consciousness of the patient. They may be beneficial in monitoring progress in response to brain lesions or injury. Auditory-evoked potentials evaluate the brainstem response to auditory stimuli. They may be used to assess hearing ability and brainstem function (Figure 28.6).

# Neuropathies

## Nutritional

### Hypovitaminosis E and Selenium Deficiency

Deficiencies of vitamin E and selenium have been reported to cause a wide variety of clinical signs and pathologic lesions in birds of all ages. In a survey of central nervous system lesions from animals in a zoological collection, birds had a higher incidence of disease than mammals, and encephalomalacia histologically compatible with hypovitaminosis E was the most common lesion observed.[130]

Vitamin E is a fat-soluble vitamin and depletion of body stores occurs slowly in adult birds, while young birds may develop clinical signs associated with acute deficiency. In young birds, hypovitaminosis E may cause encephalomalacia, exudative diathesis or muscular dystrophy. Encephalomalacia results in ataxia, head tilt, circling and occasionally convulsions and is particularly common in hatchling budgerigars.[51] Exudative diathesis and muscular dystrophy (white muscle disease) occur also with deficiency of vitamin E or selenium. The myositis associated with hypovitaminosis E may cause clinical changes difficult to distinguish from neurologic signs.

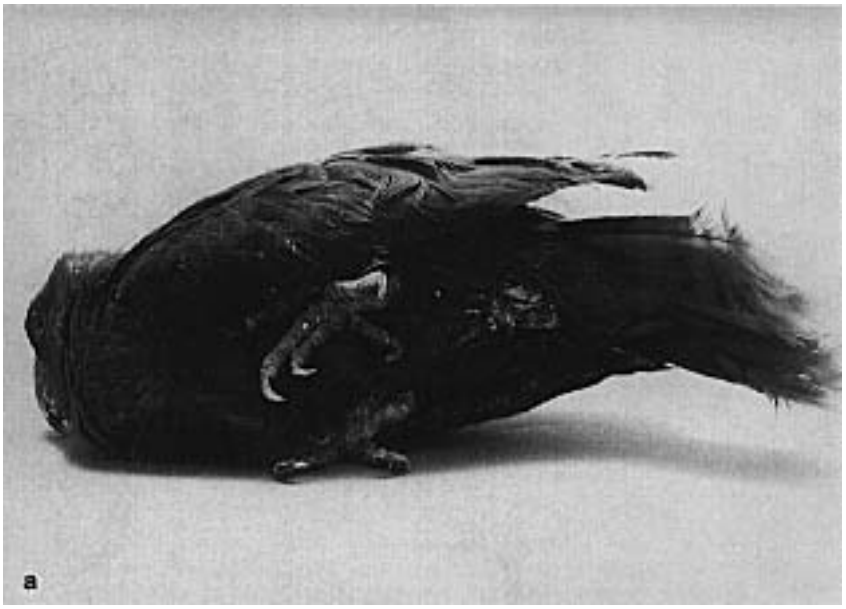




**FIG 28.7** **a)** A two-year-old domestically raised Eclectus Parrot was presented with a one-month history of progressive difficulties in ambulating. At presentation, the bird was recumbent and had stiff, nonmotile thoracic and pelvic limbs, but was bright, alert and responsive. The muscle masses associated with all limbs were atrophied and firm. **b)** EMG findings included fibrillation potentials and positive sharp waves suggestive of denervation. Histopathology indicated severe, progressive, generalized muscle fibrosis of undetermined etiology.

Clinical signs associated with vitamin E and selenium deficiencies include tremors, ataxia, incoordination, abnormal head movements, reluctance to walk and recumbency. [14,63] Postmortem findings suggestive of encephalomalacia include cerebellar edema or hemorrhage (petechia) with flattening of the convolutions.[14] Histologically, there is edema, interruption of vascular integrity with associated capillary hemorrhage and hyaline thrombosis, and cerebellar demyelination with degeneration and necrosis of neurons.[14] Focal or multifocal poliomyelomalacia of the spinal cord may also occur.[40,63] Diagnosis of hypovitaminosis E is frequently presumptive based on clinical signs, history and gross and microscopic pathology. Hypovitaminosis E and selenium deficiency should be considered with pathologic lesions of demyelination and malacia in birds with vague clinical signs or in birds that are found dead in their enclosures with no premonitory signs.

Muscular dystrophy is characterized by light-colored streaks in the muscle fibers (Figure 28.7). Early histologic changes include hyaline degeneration, mitochondrial swelling, loss of striations and central migration of the nucleus.[14] In more chronic cases, muscle fibers are disrupted transversely and macrophages are present engulfing debris from necrotic myocytes.

This deficiency has also been incriminated as the etiology of cockatiel paralysis syndrome. This condition appears to occur most frequently in lutino cockatiels infected with *Giardia* sp. or *Hexamita* sp. Vitamin E- and selenium-responsive neuropathies have been reported in a variety of other species including Blue and Gold Macaws, Severe Macaws, Eclectus Parrots and African Grey Parrots.

Clinical signs include slow or incomplete eye blink due to paresis of the lower eyelid, weak jaw muscles, paresis of the tongue, poor digestion with passage of partially digested food, diminished playful activity, hyperactivity, clumsiness and weak grip, low-pitched and weak vocalization, delayed crop-emptying, spraddle leg, death of young in nest, weak hatchlings,

increased dead-in-shell, increased egg binding that is not responsive to vitamin A and calcium supplementation and decreased fertility. Cockatiels and other psittacine birds showing these clinical signs have responded to vitamin E and selenium supplementation and antiprotozoal therapy. In one study, treatment of giardiasis resulted in an increase in serum vitamin E levels from a deficiency state into the normal range.[49]

A selenium-responsive progressive ascending paralysis in fledgling mocking birds fed a commercial cat food has been reported.[85] Necropsy findings were suggestive of a viral encephalitis; however, clinical signs were similar to vitamin E- and selenium-responsive paralysis in cockatiels. All affected birds responded to parenteral administration of selenium followed by oral vitamin supplementation. Other species of birds being raised at this facility were not affected.

Vitamin E deficiency also occurs in piscivorous birds fed an unsupplemented diet of frozen fish (especially smelt). In birds of the family Ardeidae (herons and bitterns), the deficiency manifests initially as fat necrosis accompanied by steatitis. In pelicans, myodystrophy predominates.[105]

Supplementation with injectable and oral vitamin E is the recommended treatment; however, the patient may or may not respond depending on the severity of damage. Muscular dystrophy may resolve with supplementation, but encephalomalacia rarely responds to therapy. [14]

### Hypovitaminosis $B_1$ (Thiamine)

Clinical signs of hypothiaminosis include anorexia, ataxia, ascending paralysis and opisthotonos.[51] Opisthotonos ("star-gazing") may result from paralysis of the anterior muscles of the neck resulting in pseudohypertonus of the muscles of the dorsal aspect of the neck (see Color 48). Histologically, thiamine deficiency causes a polyneuritis with myelin degeneration of peripheral nerves. Adrenal hypertrophy and edema of the skin are also characteristic of thiamine deficiency. Affected birds generally respond within hours of oral or parenteral administration of vitamin $B_1$. A response to treatment provides a presumptive diagnosis. Administration of thiamine is a useful adjunct to therapy in many nonspecific neurologic disorders.

### Hypovitaminosis $B_2$ (Riboflavin)

Curled toe paralysis occurs in poultry and is seen in nestling budgerigars with riboflavin deficiency (see Color 48). Other signs include weakness, emaciation in the presence of a good appetite, diarrhea, walking on the hocks with toes curled inward and atrophy of leg muscles. Chicks fed a deficient diet may develop clinical signs as early as 12 days of age.[61] Histologically, a demyelinating peripheral neuritis is observed with edema of the nerves (especially the ischiatic and brachial nerves). There is Schwann cell proliferation and swelling, perivascular leukocytic infiltration and segmental demyelination accompanied by accumulation of osmophilic debris in the cytoplasm of Schwann cells.[61] Gliosis, chromatolysis in the spinal cord and degeneration at the neuromuscular endplate may also be observed. Treatment involves administration of oral or parenteral riboflavin and diet correction; however, many of the changes are irreversible, especially in chronic cases.

### Hypovitaminosis $B_6$ (Pyridoxine)

Neurologic signs associated hypovitaminosis $B_6$ are characteristic, with the bird exhibiting a jerky, nervous walk progressing to running and flapping the wings. The bird then falls with rapid, clonic tonic head and leg movements. These convulsions are severe and may result in death due to exhaustion.[51]

### Hypovitaminosis $B_{12}$ (Cyanocobalamine)

Deficiency in a Nanday Conure was reported to cause subacute, multifocal white matter necrosis.[51]

## Traumatic

### Concussion Lesions

Concussive head trauma is fairly common in free-ranging as well as companion and aviary birds. Head trauma may occur at night when a panicked bird flies into an enclosure wall or if birds fly into windows or mirrors. Injured birds may remain on the bottom of the enclosure and exhibit depression, head tilt, circling or paresis of a wing or leg. Blood may be present in the mouth, ears or anterior or posterior chamber of the eye. Anisocoria and delayed pupillary light response may be present and convulsions may occur if the bird is disturbed.

Fractures of the skull or scleral ossicles may be detected radiographically. In some cases, a bruise might be visualized on the head that is the result of meningeal hemorrhage seen through the cranium. Blood actually leaks into bone and is not subdural (see Color 14).[51] If the vessels in the neck are rup-

tured, the pneumatic spaces in the skull may fill with blood obscuring evidence of trauma. It is very rare for hemorrhage to occur within the brain parenchyma. Unconsciousness with a loss of normal physiologic nystagmus indicates a brainstem lesion with a poor prognosis.[88]

Treatment is supportive and involves maintaining the bird in a dark, quiet, cool area with no disturbances. Dexamethasone appears to be the most important therapeutic agent. The prognosis is guarded-to-poor if the bird is convulsing.

### Compressive Lesions

Pigmented calvarium is characterized by a yellow discoloration (hemosiderin) in the skull, especially the pneumatic spaces of the temporal bones. These pneumatic cavities help regulate CSF pressure. There is no definitive correlation as yet between this condition and neurologic signs. Inflammation of the frontal bone was reported as a cause of opisthotonos and depression with violent convulsions. [51] Jugular stasis occurs secondary to thyroid enlargement and results in increased intracranial pressure. Hydrocephalus and intracranial masses cause compressive injury to the brain or spinal cord. With hydrocephalus, the cortex over the lateral ventricles has a "blister-like" appearance at necropsy.[84] Imaging techniques (CT or MRI) and EEG studies may be helpful in diagnosing these conditions.

### Spinal Abnormalities

Spinal fractures may be the result of injury or metabolic bone disease and may cause compression of the spinal cord. Tumors may affect the spinal cord by direct invasion or compression. MRI, CT scans and scintigraphy are useful imaging modalities for identifying these lesions, which may not be visible with plain radiographs, especially in the acute phase.

The junction of the fixed synsacrum with the more flexible portion of the thoracolumbar spine is a location susceptible to mechanical stress and vertebral subluxation. In broiler chickens, a congenital defect of the vertebral facets of $T_6$ and $T_7$ allows ventral displacement of $T_7$, producing spondylolisthesis and varying degrees of spinal cord compression.[148] In a King Penguin, spondylolisthesis was believed to be the result of trauma because no articular defects were observed.

The intervertebral discs of birds differ from those of mammals. They consist of a fibrocartilaginous central region surrounded by a "C"-shaped synovial cavity that extends around the dorsal and lateral margins of the disc. A fibrocartilaginous, wedge-shaped meniscus protrudes into the joint cavity from the dorsal and lateral margins.[37] This zygapophyseal joint has hyaline cartilage with an intervening synovial cavity. With intervertebral disc rupture, this meniscus is driven into the spinal canal along with the fibrocartilaginous disc material (see Color 14).

In a Black Swan, cervical intervertebral degenerative joint disease and spondylosis caused spinal cord compression and associated neurologic signs. In mammals with spondylosis, ventral and lateral osteophyte formation result in osseous bridging between adjacent vertebrae, while dorsal and dorsolateral osteophyte formation, as occurred in this swan, are rare. Spondylosis is believed to be the result of degenerative changes in the annulus fibrosis and intervertebral disc as a result of continuous heavy stress. In birds, the joints between vertebral bodies are synovial and in this swan, noninflammatory, degenerative changes were associated with the spondylosis. The degeneration, rupture and atrophy of the synovial-lined, fibrous intervertebral meniscus may have been involved in the pathogenesis of this disease.[57]

Dexamethasone and forced rest are the only recommended therapies for birds with spinal lesions. Myelography and spinal surgery have rarely been performed in birds. Naloxone and thyrotropin releasing hormone are beneficial in mammalian patients with spinal cord trauma, but their effects in avian patients are unknown.

## Peripheral Nervous System

### Trauma

Concussive peripheral nerve trauma occurs as a result of long bone fractures or impact trauma. The level of nerve injury (eg, neurapraxia, neurotmesis, axonotmesis or complete transection) determines the prognosis. In many instances, nerve dysfunction is transient; however, as in the case of brachial plexus avulsion, the damage may be permanent.

Brachial plexus avulsion occurs most commonly in traumatized free-ranging birds (Figure 28.8).[34,46] Clinically, there is evidence of denervation of the affected wing including lack of pain perception, paralysis with loss of withdrawal reflex and atrophy of the muscles of the affected wing and the ipsilateral pectoral muscles. Signs of Horner's syndrome may be present including ptosis and a dropped "horn" on the affected side of "horned" owls such as Screech and

**FIG 28.8** **a)** An immature Red-tailed Hawk was found down and unable to fly near a highway. A severe wing droop was present and radiographs indicated a fractured coracoid. The wing was placed in a figure-of-eight bandage and the fracture healed without complication. When the bandage was removed, the bird still had a severe wing droop, no deep pain and muscle atrophy involving most of the palpable muscle masses. **b)** At necropsy, swelling (arrows) and discoloration were present in the brachial plexus suggestive of an avulsion-type injury.

Great Horned Owls. Because of the skeletal muscle in the avian iris, miosis is not a consistent feature of Horner's syndrome. Interruption of the sympathetic pathway from $T_1$ and occasionally $T_2$ segments results in these clinical signs.

A long bone fracture may or may not be associated with peripheral nerve injuries. It may be difficult to determine the level of nerve injury in birds presented with clinical signs associated with a unilateral peripheral neuropathy involving one extremity. Typically, birds with neurapraxia improve clinically within two to four weeks, while those with axonotmesis and neurotmesis (as with avulsion injury) would not.[34] EMG and spinal-evoked potentials can help determine the degree of injury. If these are not available, it is prudent to treat with supportive care for approximately one month, with weekly evaluation for signs of improved neurologic function.

### Abdominal Masses

Compressive peripheral nerve trauma generally occurs secondary to an expanding mass that applies pressure to the nerve (see Figure 25.10). Because the pelvic nerves pass through the renal parenchyma, tumors or infection of the kidneys can damage the nerves (see Color 21). Ovarian tumors, if very large or invasive, have also been reported to damage these nerves. Of 74 budgerigars with abdominal tumors, 64 had paresis of one or both legs.[104] Paresis is usually unilateral in the early stages and is accompanied by abdominal distention.

Renal adenocarcinomas occur more commonly in males than females and the incidence is higher in psittacine than passerine birds (see Color 25). The most common sign is a unilateral leg paresis progressing to paralysis. Affected birds may demonstrate an abdominal lift. The paresis may become bilateral, but systemic signs usually develop before the contralateral limb is affected. The bird may have a palpable abdominal mass with polydypsia and polyuria. Grossly, these tumors are 1 to 2.5 cm in diameter, irregular, ovoid, globular, white or off-white with cysts or necrotic foci. They may metastasize to the liver (see Color 25).

The biologic behavior, clinical signs and gross appearance of embryonal nephromas are similar to renal adenocarcinoma. They usually contain more cysts and necrotic foci and are less frequently associated with paralysis. They occur in younger birds (three to five years old). Ovarian adenocarcinomas and granulosa cell tumors usually cause anorexia, weight loss and diarrhea, but can invade the kidneys and compress the pelvic nerves. If these tumors cause paralysis, the patient is likely to show an abdominal lift.

All of these tumors are associated with a poor prognosis. Excision may be attempted but if neurologic signs are present, the neoplasm is generally not amenable to resection. These tumors cause a peripheral neuropathy with a loss of withdrawal reflex not observed with a spinal cord lesion.

Egg binding or internal trauma associated with oviposition may result in hemorrhage and swelling around the area of the pelvic plexus, causing a transient paresis or paralysis secondary to neurapraxia.

### Circulatory Disturbances

Both peripheral and central neuropathies have been associated with diminished circulation. Atherosclerosis occurs with some degree of frequency in birds. Most commonly, no clinical signs are associated with this condition, and affected birds are simply found dead in their enclosure. In some cases, however, neurologic signs may be observed. Atherosclerosis of the carotid arteries has been described as a cause of ischemia and cerebral hypertension.[22,51,64]

Neurologic signs associated with atherosclerosis include a sudden onset of blindness, ataxia, paresis and seizures. A Blue-fronted Amazon Parrot, which had a 90 to 95% reduction in lumen diameter in one carotid artery and a 60 to 70% reduction in the other due to atherosclerosis, presented with clinical signs of regurgitation once daily, an "aura-like" behavior and holding the right leg in front of the body while going into a semiconscious state. Another Blue-fronted Amazon Parrot with progressive hind limb paresis was found to have severe atherosclerosis at necropsy.[64]

Signs ranging from blindness and ataxia to opisthotonos and seizures have been associated with cerebrovascular accidents and ischemic infarction.[22,46,51,130] MRI, CT scans and EEG may be useful in diagnosing these lesions.

### Primary Neoplasms of the Nervous System

Glioblastoma multiforme, choroid plexus tumors, Schwannomas and astrocytomas, pineal body tumors, undifferentiated sarcomas and hemangiomas have been described in the nervous system of companion birds.[51,116,118,140] Clinical signs vary with the location of the neoplasm. Imaging with MRI or CT may be useful in determining the location of the mass; however, all neural tumors are associated with a grave prognosis. Phenobarbital for control of seizures and dexamethasone to decrease cerebrospinal fluid production (thus, intracranial pressure) may provide symptomatic relief.

Pituitary adenoma or pituitary chromophobe adenoma occurs in young (four years old), predominantly male budgerigars with a two to three percent prevalence. These tumors arise from chromophobe cells and may or may not be functional, secreting tumors. The avian pituitary gland secretes LH, FSH, TSH, ACTH, growth hormone, melanocyte stimulating hormone (MSH) and prolactin (in Columbiformes). These tumors are reported to cause a classic clinical syndrome described as somnolence with occasional convulsions, uncoordinated wing-flapping and clonic leg twitches, followed by unconsciousness.[51] Clinical signs are usually the result of compression of the brain and cranial nerves. Incoordination, tremors and inability to perch have also been reported.[91] Polydipsia and polyuria, cere color change, feather abnormalities and obesity may occur with functional tumors. Exophthalmos, visual deficits, lack of pupillary light response and mydriasis may be present also.[97,140]

A tentative diagnosis may be confirmed with contrast-enhanced CT scanning of the skull. Because clinical signs are primarily related to compression of the brain and not to adrenal hypersecretion, therapy with o,p'-DDD is not generally indicated.[140] Radiation therapy is effective in treating human and canine pituitary tumors and may be efficacious in birds. A report suggests that this tumor is transmissible; however, this theory has not been confirmed.[54]

## Metabolic Neuropathies

### Hepatic Encephalopathy

Birds with severe liver disease may demonstrate signs of hepatic encephalopathy. Hepatic lipidosis, mycotoxicosis, hemochromatosis and vaccine-induced hepatopathy have been reported to cause clinical signs of depression, ataxia, diminished conscious proprioception and seizures (see Color 20).[98,110,135,146] With hepatic encephalopathy, these signs usually occur shortly after eating when the blood levels of neurotoxins absorbed from the gastrointestinal tract (and not properly processed by the liver) are high. Various compounds are believed to contribute to the clinical signs associated with hepatic encephalopathy including ammonia, glutamine, glutamate, alpha ketoglutarate, aromatic amino acids, methionine, mercaptans and short chain fatty acids.[18] In addition, abnormal neurotransmitters, alterations in the blood-brain barrier, alterations in neuroreceptors, al-

tered cerebral sensitivity and hypoglycemia contribute to the development of clinical signs associated with hepatic encephalopathy.

Postprandial blood ammonia concentrations may be elevated and can be determined using a conspecific control bird.[135] In a Toco Toucan with hepatic encephalopathy, the three-hour postprandial blood ammonia level was 350 μg/dl, while the conspecific control was only 86 μg/dl. Ammonia tolerance tests may be beneficial in diagnosing some cases; however, a parallel test should be performed on an asymptomatic bird of the same species. Serum bile acids assays are a more reliable and safer test of hepatic function.

Treatment should be directed toward the underlying cause of hepatic failure. Lactulose syrup and a low-protein, high-carbohydrate, high-quality protein diet with a vitamin supplement may provide symptomatic relief while the underlying hepatopathy is corrected.[135] Neomycin sulfate may decrease the formation of ammonia by reducing the quantity of gram-negative bacteria in the colon.

### Hypocalcemia

A syndrome characterized by opisthotonos, tonic extension of the limbs and convulsions has been described in young (two- to five-year-old) African Grey Parrots.[43,53,95,101,122,123] Initially, an affected bird may only seem uncoordinated and fall from its perch. The frequency gradually increases, seeming to be precipitated by some external stimulation. Eventually, seizure activity is pronounced and may become constant or prolonged. Serum calcium levels are below 6.0 mg/dl with concentrations as low as 2.4 mg/dl reported. It has been shown that parathyroidectomized birds begin to have seizures when the serum calcium concentration falls below 5.0 mg/dl.[127] Birds demonstrating only intermittent incoordination may still have normal serum calcium levels. This condition has also been observed in Amazon parrots and conures.[122]

At necropsy, the parathyroid glands of affected African Grey Parrots are grossly enlarged, presumably in response to the low serum calcium concentrations (see Color 14). Parathyroid hyperplasia and degeneration are prominent histologic findings. Vacuolation of the cells of the adrenal glands is a consistent feature and may be an indication of stress. There is no skeletal demineralization present in this syndrome.[122] In most birds with signs of nutritional secondary hyperparathyroidism, serum alkaline phosphatase activity is increased; however, this change has not been found to occur with the hypocalcemia syndrome in African Grey Parrots.[53]

The etiology and pathogenesis of this condition remains speculative. Affected birds are usually wild-caught and are maintained on a diet deficient in calcium and vitamin $D_3$ (usually a whole-seed diet). It appears that these birds are not able to mobilize body calcium stores (as occurs in cows with "milk fever"). Vitamin A deficiency may also play a role, as hypovitaminosis A has been shown to inhibit osteoclast activity.[123] It has been postulated that a virus that affects parathyroid function is the cause of this problem; however, attempts to demonstrate viral particles using electron microscopy have failed. Another hypothesis suggests that there is increased renal excretion of calcium.[95,122]

Diazepam may be used as an anticonvulsant, but birds generally rapidly respond to the parenteral administration of calcium gluconate. Seizure activity would be expected to cease well before serum calcium levels return to normal.[53] Corticosteroids should not be used in these patients because they increase urinary excretion and decrease intestinal absorption of calcium.

Once the serum calcium concentration has returned to within normal limits, the patient should be placed on a proper diet with calcium and vitamin supplementation. Foods such as dairy products should be encouraged, while those high in fat such as seeds should be eliminated. Serum calcium concentration should be evaluated periodically (every two to four months) to determine if alterations in therapy are indicated. The prognosis for full recovery appears to depend on the severity of damage to the parathyroid glands. If the condition is detected before complete

### CLINICAL APPLICATIONS

- The corpus striatum is well developed and is considered the main center for association in birds; consequently, instincts dominate avian behavior, which may account for some of the self-mutilation that occurs in companion birds.
- Birds are ten to twenty times more susceptible than mammals to acetylcholine inhibitors found in organophosphate and carbamate pesticides. Young birds and males are also more susceptible.
- Because withdrawal of an extremity following stimulation is a segmental reflex that does not require an intact cervical spinal cord for normal response, movement does not indicate the patient is able to feel the stimulus.

degeneration of the glands has occurred, the prognosis is favorable.

Because it appears that these birds cannot mobilize body stores of calcium, long-term prevention of recurring problems requires that birds receive adequate levels of calcium in proper balance with phosphorus, as well as sufficient levels of vitamins A, $D_3$ and E.

### Hypoglycemia

Hypoglycemia may occur as a result of starvation or malnutrition, hepatopathy, endocrinopathies and septicemia. Blood glucose less than 150 mg/dl (or half the species' normal value) may be an indication of hypoglycemia.[146] Seizure activity usually occurs once the blood glucose level falls below 100 mg/dl. Therapy should consist of 1.0 ml/kg IV of a 50% dextrose solution for acute relief of clinical signs, while the underlying cause of the problem is being determined and corrected. Dextrose solutions (>2.5%) should be administered intravenously with caution because they are hypertonic and may cause tissue damage if perivascular leaking occurs. Dextrose will compromise a patient's acid-base balance and should not be used in dehydrated birds.

## Seizures and Idiopathic Epilepsy

Seizures in birds can have numerous etiologies and various clinical presentations (Table 28.1). A typical seizure may consist of a short period of disorientation with ataxia followed by falling to the enclosure floor as a result of the loss of the ability to grip the perch (Figure 28.9). The bird may remain rigid or have major motor activity for a few seconds or a few minutes. Voiding may or may not occur. The postictal phase is variable.

**TABLE 28.1 Common Causes of Seizures in Birds**

- Nutritional (Calcium, phosphorus and vitamin $D_3$ imbalances, vitamin E and selenium deficiencies, thiamine deficiency, hypovitaminosis $B_6$)
- Metabolic (Heat stress, hypocalcemia, hypoglycemia, hepatic encephalopathy)
- Toxic (Heavy metals, insecticides)
- Infectious (Bacterial, fungal, viral or parasitic)
- Traumatic
- Neoplastic
- Hypocalcemic (African Grey Parrots)
- Hypoglycemic (Raptors)

### Idiopathic Epilepsy

Idiopathic epilepsy is used as a diagnosis when other causes of seizures have been ruled out. A syndrome of idiopathic epilepsy has been described in Red-lored Amazon Parrots that has been suggested to have a genetic basis.[124] Seizures of undetermined cause occur with some degree of frequency in Greater Indian Hill Mynahs as well. Mild to severe seizure activity may occur in these birds with signs ranging from "periodic trance-like states" and "stiffening up" to grand mal-type seizures.

Diazepam can be used to temporarily interrupt seizure activity.[125,126,146] Long-term phenobarbital at a dose of 4.5-6.0 mg/kg PO BID titrated to effect, appears to be beneficial in reducing the frequency and severity of seizures in these birds.[124] Blood phenobarbital concentration should be determined approximately one month after institution of therapy to evaluate the dosage. Owners should be advised that the therapy does not "cure" the condition, and they should keep a calendar recording seizure activity and severity.

The use of electroencephalograms in diagnosing epilepsy is difficult in any species of animal. The knowledge of normal electroencephalogram patterns for avian species is limited as is the availability of the equipment and trained personnel to make and evaluate the EEG. Even in species where EEG patterns indicative of epilepsy are established, it is recognized that between seizures, the EEG may be normal.[146]

## Lafora Body Neuropathy

Lafora body neuropathy has been reported as a cause of fine, continuous myoclonus in one cockatiel and weakness, anorexia and dyspnea in another cockatiel.[1,9,11] This disease is characterized by the formation of glycoprotein-containing cytoplasmic inclusion bodies within neurons. This accumulation of glycoproteins is believed to be the result of a defect in intracellular metabolism. In humans, the condition is known as familial progressive myoclonic epilepsy. It has also been diagnosed as a cause of spontaneous convulsions or epilepsy in beagles, miniature poodles and basset hounds. The inclusions may be found in other organs including the liver, heart, skeletal muscle and sweat glands. In the affected cockatiel, Lafora-like particles were identified diffusely throughout the liver.[9,11]

**FIG 28.9** A mature Umbrella Cockatoo that was maintained in a dark room on a wild-bird-seed diet was presented for an acute onset of convulsions. The bird was severely depressed and was unable to stand. Note the poor general condition of the feathers. The bird did not respond to supportive therapy. Necropsy findings included microhepatia, hypertrophy of the parathyroid glands and enlarged adrenal glands.

## Xanthomatosis

The etiology of xanthomatosis is unknown but it seems to develop as a response to deep inflammation (see Chapter 25). The clinical manifestation is thick yellow skin usually in the area of the sternum and ventral abdomen. Xanthomatosis may affect the brain where it appears in association with blood vessels.[51]

## Toxic Neuropathies

### Heavy Metals

Lead and zinc poisoning are the most common causes of toxicity in birds (see Chapter 37).[92] In addition to common sources of lead contamination, chronic exposure to automobile exhaust has been shown to contribute to the cumulative lead concentrations in body tissues.[88]

Clinical signs associated with lead intoxication are dependent on the amount ingested and the chronicity of the intoxication. Lead adversely affects all body systems by inhibiting enzyme activity and protein formation.[100] Nervous, digestive and hematopoietic systems are most affected. Neurologic changes suggestive of plumbism include lethargy, depression, weakness, ataxia, paresis, paralysis, loss of voice, head tilt, blindness, circling and seizures.

Lead intoxication causes a demyelination of the vagus nerve and a block of presynaptic transmission by competitive inhibition of calcium. Demyelination produces the clinical signs associated with peripheral neuropathy. Lead encephalopathy is the result of diffuse perivascular edema, increases in cerebrospinal fluid and necrosis of nerve cells.[100]

Histologically, neurologic lesions associated with plumbism include neuronal degeneration with shrunken, angular, basophilic neurons of the cerebral cortex and edema of Virchow-Robin spaces and leptomeninges.[59,106] Vacuolation of the neuropil of the cerebral cortex, optic lobes and medulla may be present. Gross and microscopic neurologic lesions may be absent, even in birds with neurologic signs.[32]

### Botulism (Limber Neck)

Botulism in birds is usually the result of ingestion of the exotoxin of *Clostridium botulinum* type C. Occasionally *C. botulinum* type A and type E are involved. The organism is an anaerobic, spore-forming bacillus. Spores are present in the soil of many wetlands and are numerous in marsh areas with a history of the disease. The spores are environmentally stable, can survive in the soil for years and are resistant to heat and chemical disinfectants.

Botulism is uncommon in companion birds, but occurs with some degree of frequency in waterfowl. Decaying organic matter provides adequate substrate for development of the clostridial spores. The toxin can persist for months under alkaline conditions (pH 9). Intoxication occurs following ingestion of contaminated food such as necrotic tissue (plant or animal) or dipterous maggots and other invertebrates. Maggots concentrate the toxin without being affected. Birds eat the toxin-laden maggots and disseminate the disease. It is generally the larval stages of blowflies and fleshflies (Calliphoridae and Sarcophagidae) that are found in high concentrations on decomposing carcasses and carry the greatest concentrations of toxin.[39] In an outbreak in pheasants,

20,000 maggots were collected from one carcass, each containing $4x10^4$ mouse MLD of type C toxin/g of maggots. It has been estimated that one ounce of type C toxin could kill the entire population of the United States.[95]

The toxin interferes with the release of acetylcholine at motor endplates causing signs of peripheral neuropathy. All peripheral nerves, including cranial nerves, are affected. The classic clinical sign is a limber neck resulting from paralysis of the cervical musculature. Most birds exhibit hindlimb paresis first, which is characterized by sitting on their sternum with legs extended behind their body.[12] Paralysis of the wings followed by loss of control of the neck and head are observed in the terminal stages. Green diarrhea with pasting of the vent are also common with botulism.[12,39,48] Inconsistent clinical findings with botulism are chemosis, swelling of the eyelids and nictitans, ocular discharge and hypersalivation.[12,48]

Gross and histologic examination generally fail to reveal any lesions. In some cases, maggots may be observed within the proventriculus, and some birds have a pericardial effusion.[12,48] The toxin can be detected in stomach contents, serum or plasma. The mouse protection test is used to provide an antemortem diagnosis in most cases. Mice are challenged with the serum of an affected bird and control mice are left untreated while others receive antitoxin. A positive diagnosis is made if only unprotected mice die. This test is accurate approximately 75% of the time.[48,62] In an outbreak where toxin was not detected in serum, the toxin was recovered from the spleen and liver.[39]

Therapy is primarily supportive. Cathartics, laxatives and drenches are used to flush unabsorbed toxins through the gastrointestinal tract. Tube-feeding provides nutritional support for birds that are unable to eat and drink. Antitoxin may be administered intraperitoneally, but it is not commercially available and its benefits are equivocal.[12,48,72]

Efforts should be made to prevent exposure of birds to maggots and other sources of botulism toxin. Ponds and lakes should not be used for disposal of carcasses and organic debris. They should be dredged periodically, and a fountain or other means of aeration should be provided.

### Pesticides

The United States Environmental Protection Agency has registered 36 different organophosphate and 46 carbamate compounds such as carbaryl (Sevin dust), dursban (chloropyrifos), diazanon, malathion, dichlorvos and methyl carbamate.[113] Both of these compound classes are acetylcholinesterase inhibitors that bind to and subsequently inactivate acetylcholinesterase causing an accumulation of acetylcholine at the postsynaptic receptors. Organophosphate bonds are considered irreversible, while carbamate bonds are slowly reversible (spontaneous decay in several days). Acetylcholine is the neurotransmitter found at autonomic ganglia (both sympathetic and parasympathetic); postganglionic parasympathetic nerves affecting smooth muscle, cardiac muscle and exocrine glands; and at neuromuscular junctions of the somatic (skeletal muscle) nervous system.[72,119]

Birds are 10 to 20 times more susceptible to these acetylcholine inhibitors than mammals. Young birds and males are also more susceptible.[94] The development of clinical signs is dependent on the concentration of the pesticide, the route and proximity of exposure, the amount of ventilation, the species of bird and its physical condition.[72] Exposure in companion birds is generally accidental or the result of inappropriate use of insecticide products. The most common route of exposure is inhalation.

Two types of neuropathy and corresponding clinical signs have been described related to toxicosis with acetylcholinesterase inhibitors.[72] Acutely, clinical signs are related to excessive stimulation of acetylcholine receptors. Signs include anorexia, crop stasis, ptyalism, diarrhea, weakness, ataxia, wing twitching and muscle tremors, opisthotonos, seizures, bradycardia and prolapse of the nictitans.[72,94,113,119] Bradycardia and dyspnea with crackles and wheezes may occur as the toxicosis progresses. Respiratory failure is usually the cause of death and results from increased mucus secretion, bronchoconstriction and paralysis of respiratory musculature.

The second type of neuropathy is an organophosphate ester-induced neuropathy, which is not associated with an inhibition of acetylcholine.[73] The onset of clinical signs is delayed (7 to 21 days after exposure) and is the result of a symmetric distal primary axonal degeneration of the central and peripheral nervous systems, with secondary myelin degeneration. Clinical signs include weakness, ataxia, decreased proprioception and paralysis.

Diagnosis of acetylcholinesterase inhibition is usually based on clinical signs and a history of exposure

to these compounds. Cholinesterase assay may be performed on blood, plasma, serum or brain tissue. A decrease in acetylcholinesterase of 50% from normal is considered diagnostic.[73] Normal avian plasma cholinesterase levels are reported to be greater than 2000 IU/l. A new cholinesterase test requires only 0.01 ml of serum and results may be available in five minutes.[113]

Intoxication is best treated with atropine, pralidoxime chloride (2 PAM) and supportive care (see Chapter 37).[119]

The exact mechanism of action of organochloride insecticides, such as DDT, is unknown, but clinical signs are usually neuromuscular, resulting from either stimulation or depression of the central nervous system.[11] There is no known antidote for organochloride intoxication. Birds with seizures or other signs of CNS stimulation should be tranquilized or lightly anesthetized with a long-acting agent such as phenobarbital. In cases of CNS depression, stimulants may be beneficial. Cathartics, activated charcoal and general supportive care should be provided as necessary (see Chapter 37).

#### Therapeutic Agents

Dimetridazole was commonly used to treat trichomoniasis, giardiasis and histomoniasis. Toxicity results in birds with increased water consumption (increased intake of drug). Convulsions, wing flapping and opisthotonos have been reported in budgerigars, goslings, pigeons and ducks. Histologically, large clear spaces are present around blood vessels, neurons and glial cells. Neurons have a pyknotic nucleus and eosinophilic cytoplasm. Dimetridazole is no longer commercially available in the United States.

Metronidazole toxicity has been reported in dogs and cats, but not in companion birds. Clinical signs relate primarily to the vestibular system and include ataxia, incoordination, proprioceptive deficits, nystagmus, depression, paresis, tremors and seizures. Treatment involves limiting further absorption of the drug (eg, absorptives, emetics and cathartics), fluid therapy to increase renal excretion, control of seizure activity and supportive care.

#### Other Neurotoxins

Many plants are toxic to the nervous and digestive systems. Generally, birds are more resistant than mammals to plant-derived toxins (see Chapter 37).

Citreoviridin and tremorgens are two types of mycotoxins that primarily affect the neural system. Fusariotoxins and ochratoxins also produce nervous disorders. A syndrome characterized by cervical paresis in free-ranging Sandhill Cranes has been associated with mycotoxicosis.[107]

Domoic acid poisoning was diagnosed as the cause of death in Brown Pelicans and Brandt's Cormorants exhibiting neurologic signs. Twenty-seven of 39 affected birds died within 24 hours.[153] Domoic acid is a neurotoxin produced by marine diatoms. It is an excitatory toxin that binds to both pre- and postsynaptic kainate receptors in the brain, resulting in continuous depolarization of neurons until cell death occurs. It was theorized that this epornitic was caused by the ingestion of contaminated anchovies, which had fed on the diatoms.

## Infectious Neuropathies

### Fungal

The nervous system may be a secondary site for aspergillosis lesions that may cause ataxia, opisthotonos and paralysis. In infected birds, yellowish, mycotic nodules may be grossly visible within the brain or spinal canal. Fungal granulomas may compress or invade peripheral nerves and cause unilateral or bilateral paresis or paralysis (see Color 21).[47] Spinal aspergillosis has been diagnosed in several penguins at the San Francisco Zoological Gardens. Fungal elements are usually detected histologically (see Chapter 35).

*Dactylaria gallopava* has been reported in Grey-winged Trumpeter Swans and gallinaceous birds. The organism is present in wood chips and bark litter. Infections characterized by encephalomyelitis occur following the inhalation of fungal spores. *Cladosporius* (*Exophiala*) and *Mucomyces* have also been reported to cause meningoencephalitis.[31]

### Parasitic

#### Toxoplasma

*Toxoplasma gondii* infections are reported primarily in Galliformes and Passeriformes with lesions involving the brain and skeletal muscles. Cats are the only host known to excrete infectious oocysts.[36] Considering avian species, it would seem that raptors are

most likely to become infected because they prey on the same types of animals as cats. Although toxoplasmosis has been reported in raptors, they appear to be more resistant to infection than other birds.[36,78]

Clinical signs include anorexia, pallor, diarrhea, blindness, conjunctivitis, head tilt, circling and ataxia. Infection may occur from ingestion of coprophagic arthropods or food and water supplies contaminated with feces from infected cats. Encapsulated cysts (round or oval) are found in the brain histologically. A definitive diagnosis is made using a latex agglutination serum test or immunohistochemical staining of affected tissues. There is variation among avian species with respect to the antibody response generated against *Toxoplasma* sp. Chickens do not develop antibodies, while raptors, pigeons, canaries and finches have been shown to develop positive agglutination antibody titers.[36,70,144] In experimentally infected raptors, cysts were found in the brain and muscles (skeletal and cardiac) even when no clinical abnormalities were noted.

In canaries and finches, toxoplasmosis has been shown to cause loss of myelinated axons in the optic nerve resulting in blindness and conjunctivitis.[144] Tachyzoites could be demonstrated in the detached and intact retinae, lenses and exudate from the vitreous humor. Focal, disseminated, chronic inflammation with accumulations of tachyzoites characterized the histologic findings in the brains (see Chapter 36).

### Sarcocystis

*Sarcocystis* spp. infect a broad range of hosts in several orders of birds. Infection in birds is reported to be less common than in mammals; however, infection has been reported in over 60 species of birds, with Old World psittacines apparently more susceptible (see Chapter 36). Unlike many other species of *Sarcocystis, S. falcatula* schizonts can persist in avian tissues for up to 5.5 months.[36] It is believed that cockroaches and flies may be transport hosts for the parasite.[26,52] Raptorial species may become infected by ingestion of prey containing the encysted organism.[1,36]

Histologically, the organisms are elongated, spindle- or banana-shaped, and grouped into packets within a spherical- to spindle-shaped cyst. Granulomatous inflammation consisting of macrophages and lymphocytes forming nodular and diffuse aggregates is associated with these cysts. With electron microscopy, characteristic polar rings and micronemes may be discernable.[58]

### Schistosomiasis

Granulomatous encephalitis caused by the blood fluke *Dendritobilharzia* sp. has been reported in swans.[77,147] Neurologic signs included head tilt, circling, weakness and extension of the head and neck. Granulomas containing macrophages, giant cells, lymphocytes and occasional heterophils and fibroblasts were identified within the cerebrum and cerebellum of affected birds. Ova of the parasite were identified within these lesions. Adult flukes were identified within the blood vessels in one bird. Adult schistosomes usually live within veins; however, those of the genus *Dendritobilharzia* live within arteries. Adults have little host-specificity.

### *Baylisascaris* sp.

*Baylisascaris procyonis* is the ascarid of raccoons and is a zoonotic organism that can cause fatal meningoencephalitis in humans. Over 40 species of mammals and birds have been shown to develop clinical cerebrospinal nematodiasis following infection.[4,5,68,99] Free-ranging birds are infected by ingesting raccoon feces, while companion birds may become infected by ingestion of food contaminated with parasite eggs. The eggs may remain viable and infective in the environment for years.

Clinical signs are nonspecific and include depression, ataxia and torticollis. The onset may be acute or chronic, possibly related to the number of larvae involved. Prolonged migration of a single larva within the brain could produce chronic, progressive signs. Treatment with ivermectin has been completely ineffective.[5]

Gross lesions are generally absent. Edema of the brain and spinal cord, encephalitis, encephalomalacia, eosinophilia around sections of larvae, degenerative foci with heterophils in the neuropil, perivascular cuffing and glial cell proliferation are observed histologically.[4,5,38,67,99] In some cases, a cross section of the parasite may not be observed as the larvae migrate even after host death.[99]

### Filaria

*Chandlerella quiscali* is a filariid nematode of grackles that has been reported to cause cerebrospinal nematodiasis in emus.[7] Gnats are the vectors for natural infection. The gnat is ingested and the larvae migrate into the brain or spinal cord and then into the lateral ventricles of the cerebrum where they mature and produce microfilaria. Affected emu chicks demonstrated torticollis, ataxia, recumbency

and death. Circulating microfilaria were not detected (see Chapter 36).

## Viral Neuropathies

The following viral diseases of birds have neurologic clinical or histologic abnormalities as part of their pathophysiology; most produce lesions in other systems as well. Only their effects on the nervous system will be discussed here. For a more complete discussion of these viruses, see Chapter 32.

### Paramyxovirus

Paramyxoviruses (PMV) 1, 2, 3 and 5 have been isolated from companion birds, and groups 1 and 3 have been associated with lesions of the central nervous system.[75] Pigeon paramyxovirus causes a nonsuppurative encephalitis similar to that described with Newcastle disease virus.[129]

Clinical signs associated with PMV-1 in companion birds are variable depending on the virulence of the strain and the species of bird affected. In some cases, the only signs may be acute death and high mortality. Other signs are associated with abnormalities of the respiratory, digestive and nervous systems.[23,24,30] Neurologic signs including depression, hyperexcitability, ataxia, incoordination, torticollis, head tremor, opisthotonos, muscle tremors and unilateral or bilateral wing or leg paresis or paralysis occur more commonly in older birds and with chronic infections.[24,30] Some birds clench their feet while others lose control of the tongue and their ability to grip with the beak.[23]

All reflexes are depressed but neurologic signs are exacerbated by excitement. Seizures and running movements are often observed just prior to death. Neurologic signs generally persist in birds that survive the acute infection.[142]

At necropsy, there may be petechiae on the surface of the cerebrum and cerebellum.[23] Histologically, central nervous system lesions are commonly observed in the cerebellum, brainstem, midbrain and spinal cord. Neuronal degeneration, gliosis, endothelial cell hypertrophy and lymphocytic perivascular cuffing characterize the lesions.[30,114]

Lymphoplasmacytic meningoencephalitis outbreaks have been described in *Pionus* species and *Neophema* species from a quarantine station. These deaths were caused by an unclassified hemagglutinating virus that morphologically resembles paramyxovirus.[82,84] The disease produced high morbidity and moderate mortality. Muscle tremors, circling, ataxia, torticollis, weakness, depression, paresis and paralysis were the major clinical signs.

A yellowish periventricular discoloration was the only abnormal gross brain lesion noted. Histologically, there was lymphoplasmacytic perivascular cuffing adjacent to the ventricles, within the choroid plexus and around the central canal of the spinal cord, sometimes accompanied by edema of the neuropil. Some ependymal cells and few subependymal glial cells had equivocal eosinophilic Cowdry type A inclusions. On electron microscopy, these contained virions compatible with paramyxovirus.

### Neuropathic Gastric Dilatation

There is strong evidence that neuropathic gastric dilatation is caused by a virus (possibly a paramyxovirus). Anorexia, regurgitation, changes in fecal consistency, weight loss, pectoral muscle atrophy and depression are presenting clinical signs.[55,64,90,139,151] The clinical signs of this disease primarily relate to the gastrointestinal system, with central and peripheral neurologic signs occurring only occasionally.[64,150] However, the main histologic lesions involve the nervous system.

Creatine kinase is an enzyme released from damaged nerves and muscles. It has been reported that some birds with neuropathic gastric dilatation have elevated serum CK levels; however, an elevation of serum CK activity is not specific for this disease and may occur with septicemia, neuropathies and myopathies.[60]

Microscopic lesions generally involve the brainstem, the ventriculus and the proventriculus. Multifocal lymphocytic encephalitis with lymphocytic perivascular cuffing, gliosis and neuronophagia characterize the brainstem lesions.[55] Asymmetric lymphocytic poliomyelitis, lymphocytic perivascular cuffing and gliosis are present in the spinal cord. Multifocal lymphocytic leiomyositis with smooth muscle degeneration and fibrosis are common in the ventriculus and proventriculus.

There is a general loss or depletion of myenteric ganglion cells (myenteric ganglioneuritis). The ganglia of gastric and duodenal myenteric plexuses demonstrate round cell accumulations (lymphocytes, plasma cells, macrophages). Intranuclear and intracytoplasmic eosinophilic inclusion bodies have been described within the perikaryon of the celiac ganglion and myenteric plexus.[90] Virus particles associ-

ated with the inclusions have been morphologically similar to paramyxovirus particles.

### Avian (Picornavirus) Encephalomyelitis

This picornavirus has been associated with gastrointestinal and neurologic signs in Galliformes, Anseriformes and Columbiformes.[8,51,89,132,133] Only one serotype is recognized but strains vary in neurotropism. Clinical signs include depression, ataxia, paresis or paralysis and severe, but fine head and neck tremors. Neurologic signs occur only in birds less than 28 days of age.[51] No characteristic gross lesions are noted at necropsy. Neuronal degeneration with lymphocytic perivascular cuffing and gliosis in the brain and spinal cord characterize the disease histologically.[89]

### Polyomavirus (Budgerigar Fledgling Disease)

Although not the primary lesions, tremors of the head, neck and limbs, incoordination and ataxia have been associated with polyomavirus in infected birds. Histologically, large, slightly basophilic intranuclear inclusion bodies can be identified in the cerebellar ganglionic layers.[88,91,131]

### Reovirus

Reovirus is commonly reported in imported birds and primarily affects African Greys, cockatoos and other Old World Psittaciformes. Clinical signs include uveitis, depression, emaciation, anorexia, incoordination, ataxia, paresis and diarrhea. Histologically, multifocal necrosis of the liver is present. This is not a neurotropic virus, and the paresis or paralysis is the result of vascular thrombosis of the extremities.

### Togaviridae

Viral encephalitis caused by a number of togaviruses has been reported in numerous species of birds. Clinical signs include depression, ruffled feathers, decreased appetite, dyspnea, profuse hemorrhagic diarrhea (in emus), ataxia, muscle tremors, weakness, unilateral or bilateral paresis or paralysis, torticollis and death. At necropsy, the cerebral hemispheres may be softened. Histologically, lesions include nonsuppurative encephalitis, patchy neural necrosis, cerebral vasculitis, leukocytic perivascular infiltrates, microgliosis, meningitis, neuronal degeneration and myocardial necrosis.[121] These changes have a rostral distribution contrary to most avian viral encephalitides. Vaccines are available and appear to be beneficial in outbreaks.[15,51,56,21,121,138]

### Marek's Disease

This lymphoproliferative disease is caused by a herpesvirus. Peripheral nerve dysfunction occurs secondary to lymphoid infiltrates. Often birds display a spraddle-leg paralysis with one leg extended forward and the other back. Grossly, the ischiatic nerves appear enlarged with a loss of striations and a gray discoloration. In a study of neoplasms of budgerigars, many birds with abdominal tumors also had evidence of infection with avian leukosis virus; however, similar findings were not confirmed in another study.[43,104]

### Encephalomyelitis in Lorikeets

Encephalomyelitis was described in free-ranging Australian lorikeets.[102] Affected birds demonstrated a progressive bilateral paralysis with clenched feet. Perivascular macrophage infiltrates, vascular endothelial cell proliferation, gliosis, neuronal necrosis and neuronophagia were observed in the brainstem and spinal cord of affected birds. Edema of the ischiatic nerve was also a feature. A viral or protozoal etiology has been suggested.

### Duck Viral Enteritis

Duck viral enteritis is primarily a concern where feral populations of ducks mingle with captive birds. Clinical signs include photophobia, ataxia, seizures, penile prolapse, lethargy, hemorrhagic diarrhea and serosanguinous nasal discharge. The most notable finding at necropsy is hemorrhagic bands on the small intestine (see Color 14).

### Duck Viral Hepatitis

Duck viral hepatitis is caused by a picornavirus. Clinical signs include lethargy, seizures, opisthotonos and death. Ducklings suffer the highest mortality, and Muscovy Ducks appear to be resistant to infection with this virus. At necropsy, the liver, spleen and kidneys are enlarged with petechial hemorrhages. It is recommended to vaccinate breeders before the onset of laying.

## Bacterial Neuropathies

### Listeriosis

*Listeria monocytogenes* is a small, gram-positive, nonsporulating, motile rod that is frequently confused with a hemolytic *Streptococcus* sp. The organism is ubiquitous and can survive for years in the environment. Intracranial infections cause opisthotonos, ataxia and torticollis (Figure 28.10).[29] Brain lesions consist of microabscesses with heterophil and giant cell infiltrates, diffuse gliosis, meningeal hyperemia, degeneration of Purkinje cells and perivascular cuffing (see Chapter 33).

**FIG 28.10** An adult Amazon parrot was presented with a history of progressive depression, weight loss and ataxia. When undisturbed, the bird would exhibit repeated periods of opisthotonos with shifting phase nystagmus. Abnormal clinicopathologic findings included WBC=26,000 (toxic heterophils), PCV=26, total protein=7.5 and CPK=1200. The bird did not respond to supportive care. Histopathology indicated a severe perivascular cuffing with neuronal necrosis of undetermined etiology.

### Chlamydiosis

Occasionally, *Chlamydia psittaci* will cause neurologic signs in birds that survive the acute respiratory or gastrointestinal phase of the disease. Signs include seizures, torticollis, tremors and opisthotonos (see Chapter 34).[88]

### Granulomas

Avian tuberculosis can cause neurologic signs if the granulomas occur intracranially or adjacent to peripheral nerves.[51,108] Osteomyelitis caused by mycobacterium may produce a lameness that could be misinterpreted as a neuropathy. Abscesses, granulomas, encephalitis, myelitis and meningitis may be caused by any bacterial organism. *Salmonella, Streptococcus, Staphylococcus, Pasteurella multocida, Mycoplasma* and *Clostridium* spp. have been isolated.[19,46,51,73] Clinical signs depend on the location and extent of the lesions.

### Otitis

Otitis media and interna in companion birds may cause neurologic signs. Otitis interna produces a head tilt and circling toward the affected side (see Figure 28.6). If the infection progresses, other cranial nerves and the midbrain may become affected.

## Congenital Abnormalities

The incidence of developmental abnormalities of the avian nervous system is not well established. Studies in poultry suggest that meningocoele and other related anomalies result from early malformation and forking of the neural tube during embryonic development.[79] In turkeys, hydrocephalus, shortened beaks and absence of the terminal digits have been linked to an autosomal recessive semilethal gene.[103] In laboratory animals, virus infections, certain chemicals and malnutrition (hypovitaminosis A, hypervitaminosis A, hypovitaminosis $B_{12}$ and $B_6$ and