

# Evaluating and Treating the Nervous System

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The nervous system plays a role in nearly all body processes. Disease syndromes may affect the central nervous system (CNS), which includes the brain and spinal cord, and the peripheral nervous system, which includes cranial nerves, spinal cord nerve roots, spinal nerves, peripheral nerve branches and the neuromuscular junction. Evaluation of the nervous system of birds parallels that of mammals, with modifications specific to avian anatomy, behavior and physiology. An understanding of basic avian neuroanatomy is necessary to evaluate abnormal function of the nervous system.<sup>15,16,62,87</sup>

Suspicion of neurologic dysfunction arises from the history and physical examination. The signalment, presenting chief complaint, time course of clinical signs, and history may suggest the type of disease process or species-specific disorder. A complete neurologic examination is necessary to localize the anatomic distribution, to determine the severity of the disease process, and to assess the prognosis for patient recovery. A minimum database consisting of a complete blood count (CBC), serum or plasma biochemical analysis, and cytologic evaluation of choanal and cloacal swab samples is used to evaluate the contribution of other body systems to the observed clinical signs and physical abnormalities. If neuromuscular disease is suspected, electrophysiological tests are indicated, including electromyography and nerve conduction velocities, and these are becoming more commonly performed in birds. In recent years with the advancement of imaging technology and the improved availability for veterinary patients, scintigraphy, computed tomography and magnetic resonance imaging have been valuable diagnostic commodities in furthering our understanding of avian CNS disease. Only when a disease location and a pathologic process have been identified can appropriate treatment and prognosis be provided.

## Anatomy and Physiology

Complete reviews of avian neuroanatomy are documented in other texts.<sup>15,62,85,87</sup> For a detailed description of the functional organization of the avian spinal cord, the reader is referred to an avian physiology text.<sup>83</sup> The general organization of the avian spinal cord resembles that of all other vertebrates. There are however, some specializations, which birds largely share with the phylogenetically related class of reptiles. Compared to the mammalian cord, the most outstanding deviations are the lack of a *cauda equina* and a *filum terminale* and the occurrence of a *sinus rhomboidalis* or *lumbosacralis*.<sup>83</sup> At the microscopic level there are some significant differences in the organization of cell groups and pathways. Birds differ from most other vertebrates in their locomotion due to bipedal walk and flight. It has to be kept in mind that specializations in the spinal cord may result from adaptations from this peculiar kind of locomotion.

### Control of Eye and Head Movements

A sitting bird is able to survey a large part of its surroundings using both head and eye movements. Several types of eye movements can be distinguished such as saccades (fast flicks elicited by a sudden stimulus) and smooth pursuit (following a moving target).<sup>33</sup> Six muscles are responsible for all movements of the eye. Branches of the oculomotor, trochlear and abducens nerves innervate these extrinsic eye muscles. The two muscles that rotate the eye dorsally are innervated by contralateral centers, with the other muscles innervated by ipsilateral cell groups. These are stimulated by the vestibular centers.<sup>37</sup>

### Control of Jaw and Tongue Movements

The jaw muscles have a visceral origin; therefore, the nerves innervating these muscles and the corresponding motor nuclei are considered visceromotor elements.<sup>33</sup> A complication in parrots is that they possess a so-called kinetic skull. This means that not only the lower jaw but also the upper jaw moves to open the beak.<sup>33</sup> Movement of the beak is achieved by four groups of muscles: two groups of beak openers and two groups of beak closers.

Movements of the tongue are caused by two groups of muscles. The extrinsic muscles are part of the visceral musculature, similar to the jaw muscles, and the intrinsic muscles have a somatic origin innervated by the hypoglossal nerve. Because the jaws and tongue move in close harmony during feeding and drinking, a premotor system is needed to coordinate the activity of the tongue and jaw motor centers.<sup>33</sup>

### Control of Locomotion

The reticular formation in the brain stem also contains

premotor neurons of the motor systems of the spinal cord, thus controlling flying and walking. The final effectors of wing and leg movement are the lower motor neurons of the brachial and lumbar plexuses, respectively. In-depth review of this area is covered in an avian physiology text.<sup>15,33</sup>

### Control of Vocalization and Respiration

Birds use their syrinx, an organ at the transition of the trachea and bronchi, to produce sounds. Vocalization depends upon the control of a few small syringeal muscles and the precise regulation of the stream of (expiratory) air passing through the trachea. This requires a close coordination of the motor centers of the syringeal and respiratory muscles. The caudal part of the dorsal hypoglossal motor nucleus innervates the syringeal muscles, whereas the motor cells of the respiratory muscles are part of the motor system of the spinal cord. However, several cell groups in the cranial and caudal brainstem have a role in the control of respiration. In songbirds, these centers are all under direct telencephalic (cerebral) control.<sup>120</sup> See Chapter 19, Endocrine Considerations.

### Control of Coordination

Birds possess a well-developed cerebellum but, as in mammals, its precise role in the control of motor activity is not well understood. However, there is little doubt that the ease and precision of motor performance depend upon an intact cerebellum. The axons of the Purkinje cells form the output of the cerebellar cortex; the central cerebellar nuclei and some vestibular nuclei are the targets of these fibers.<sup>33</sup> The Purkinje cells have an inhibitory effect on the activity of the central nuclei.

## Neurological Examination and Lesion Localization

A neurological examination is easily integrated into a routine physical examination. The objectives of the neurological examination are to confirm if there is a neurological abnormality and to specifically localize the abnormality within the nervous system. In conjunction with the history, signalment, presenting complaint and the physical examination, the neurological lesion localization is a piece of a jigsaw, essential to creating a list of differential diagnoses for the disease. However, caution must be used, as some manipulations necessary for the neurological examination could exacerbate problems such as spinal cord disease.<sup>80</sup>

### OBSERVATION

Observation of the bird is essential, as it allows evalua-

tion of the bird's mentation (level and content of consciousness), posture, attitude and gait. Changes in mentation are revealed by a history of personality change, change in awareness of surroundings and inappropriate behavioral responses.<sup>26</sup> Consciousness is a function of the brainstem (responsible for arousal) and the cerebral cortex (responsible for content and regulation).<sup>26,40,101,117,119</sup>

## PALPATION

The bird's musculoskeletal system should be palpated for asymmetry, masses, tenderness, contour and tone. A mass effect, tenderness or contour change requires further investigation. The vertebral column should be palpated for deviations and pain, being cautious not to apply too much pressure if there is suspicion of an instability. Unilateral muscle mass loss or atrophy may indicate disuse if it is chronic, or a neurogenic loss if it is acute (within 7 to 10 days).<sup>26</sup>

## CRANIAL NERVES (CNs)

Several differences exist between the neuroanatomy of avian and mammalian species.<sup>15,26</sup> Cranial nerves have specific functions and evaluation of these functions can help to precisely locate a neurological lesion due to their well-documented anatomy. The general functions and specific tests are summarized in [Table 17.1](#).

Simplistically, cranial nerve dysfunction may indicate a CNS lesion (brainstem disease) or a peripheral lesion (affecting the cranial nerves after they have exited the brainstem and course through the skull). Evaluation of the cranial nerves should follow observation and palpation, with particular attention paid to normal functions of eye movement, head movement, blinking, jaw and tongue movement, and general symmetry of the head.

Initially an ophthalmic exam should be performed, which will assist with the evaluation of the optic (CN II), oculomotor (CN III), trochlear (CN IV), abducens (CN VI), and trigeminal (CN V) nerves.<sup>15,16,62</sup> The following tests are essential to the evaluation of cranial nerve function.

### The Menace Response

#### How to Perform

Obscure the vision in one eye and make a slow, threatening hand gesture toward the other eye ([Fig 17.1](#)).

#### How to Interpret

This is a learned response, not a reflex, to a perceived threat, which evaluates CNs II and V (responsible for innervation of the *orbicularis oculi* muscle, which closes the eyelids), as well as the central visual pathways and the cerebellum.<sup>62,87</sup> Birds that are stoic or excited may not show a menace response even though nerve function is

**Table 17.1 | Cranial Nerves: Function and Applicable Tests**

Cranial Nerve	Nerve Function	Applicable Tests
I. Olfactory	Smell	None applicable
II. Optic	Vision	i. Menace response ii. Pupillary light reflex
III. Oculomotor	Extrinsic and intrinsic ocular muscles/ upper eyelid muscle	i. Eyeball position ii. Menace response iii. Pupillary light reflex
IV. Trochlear	Extrinsic ocular muscles	i. Eyeball position
V. Trigeminal	Facial and beak sensation/beak movement	i. Palpebral reflex ii. Jaw palpation
VI. Abducens	Extrinsic ocular muscles and third eyelid muscle	i. Eyeball position
VII. Facial	Muscles of facial expression	None applicable
VIII. Vestibulocochlear	Hearing and balance	i. Startle ii. Oculocephalic reflexes
IX. Glossopharyngeal	Muscles of pharynx, larynx, crop and syrinx	i. Gag reflex
X. Vagus	Muscles of larynx, pharynx, esophagus and crop	i. Gag reflex
XI. Accessory	Superficial neck muscles	None applicable
XII. Hypoglossal	Muscles of tongue, trachea and syrinx	i. Tongue grab/ inspection



**Fig 17.1 |** The menace reaction is performed by making a threatening gesture with fingers or hand toward one eye and watching for a blinking action.

intact. Normal function is demonstrated by a blink. To localize the lesion, other cranial nerve tests would be required.

### The Pupillary Light Reflex

#### How to Perform

Shine a bright light in each eye to evaluate the response of the pupil ([Fig 17.2](#)).

#### How to Interpret

This is a reflex. Light is sensed by CN II; parasympathetic



**Fig 17.2** | The pupillary light response is performed by shining a light source into one eye at a time and monitoring the response of the pupil.

fibers of CN III cause contraction of the iris muscle in normal birds with direct stimulation. There is no consensual response in birds due to the 100% decussation of the optic nerves at the optic chiasm.<sup>26</sup> However, some raptors have an incomplete bony septum between the globes and may respond to light reaching the contralateral retina, mimicking a consensual response.<sup>82</sup> The response may not be as marked as that seen in mammals, as birds have striated muscle in the iris, giving them some voluntary control over pupil size independent of light intensity. This reflex is best evaluated as soon as possible during the examination, as sympathetic tone in restrained birds may override constriction.

## Evaluation of Strabismus

### How to Perform

Observe the bird's head in a normal position for a deviation of one or both globes in the orbit(s).

### How to Interpret

Cranial nerves III, IV and VI aid vision by maintaining the globe in a central position. Deviation of the globe from its central axis indicates dysfunction in one or more of these nerves: ventrolateral — CN III, dorsolateral — CN IV, and medial — CN VI. In contrast to mammals, the third eyelid in birds has striated muscle fibers, innervated by CN VI, that initiate movement of the nictitans across the cornea.<sup>20,59,62,82,87</sup> Prolapse of the third eyelid in birds does not directly indicate loss of sympathetic innervation, as found in Horner's syndrome in mammals.

## The Palpebral Reflex

### How to Perform

Touch the medial canthus of the normal eyelid and watch the response (**Fig 17.3**).



**Fig 17.3** | The palpebral reflex is performed by touching the medial canthus of the palpebral fissures and watching for a blink in response.



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**Fig 17.4** | A cockatiel (*Nymphicus hollandicus*) with eyelid paresis from infraorbital sinus infection demonstrates a cranial nerve V lesion.

### How to Interpret

The normal eyelid should close. Cranial nerve V (trigeminal nerve) is responsible for facial sensation, whereas the motor response to facial sensory stimulation is generally provided by the facial nerve (CN VII). The eyelids are innervated by CN V in birds.<sup>39,62,87</sup> Eyelid sensation is provided by the ophthalmic branch (upper lid) and the maxillary branch (both lids) of CN V. Eyelid closure is provided by the mandibular branch (*orbicularis oculi* muscle) of CN V that, when damaged, may present as eyelid paresis (**Fig 17.4**).

## Evaluation of Jaw/Beak Tone

### How to Perform

Observe the bird for a dropped lower beak and/or an inability to eat with the beak. Assess the strength of the beak safely by manually opening the beak and evaluating the resistance to opening (**Figs 17.5a,b**).

### How to Interpret

The mandibular branch of CN V provides motor func-



**Fig 17.5a** | The beak is assessed for strength and movement.



**Fig 17.5b** | The movement and symmetry of the tongue can be assessed by forcing open the beak or using a speculum.

tion to the jaw.<sup>39,87</sup> A dropped lower beak or the inability to chew indicates damage to CN V. Cranial nerve VII contributes to prehension by partial innervation of the muscles that open the jaw.

### The Oculocephalic Reflex/Interpretation of Nystagmus

#### How to Perform

Move the head from side to side in a horizontal plane and observe the resulting movement of the eyes (**Fig 17.6**).

#### How to Interpret

In normal birds, a physiological nystagmus will be induced, with the fast phase in the direction of head movement. This reflex tests the integrity of CN VIII (vestibulocochlear nerve), which is the sensory arm of this reflex, and CNs III, IV and VI, which are responsible for the motor movement of the eyes. Clinical signs of peripheral vestibular disease are manifest after damage to the inner ear or vestibular branch of CN VIII, which effectively gives unbalanced input to the intact central vestibular system.<sup>26</sup> In the absence of head motion, spontaneous horizontal nystagmus is consistent with CN VIII damage, with the fast component away from the side of the lesion. Unilateral peripheral disease may cause a head tilt with circling toward the side of the lesion.

## POSTURAL REACTIONS

The postural reactions are complex, requiring intact sensory and motor pathways throughout the nervous system, as well as unimpaired processing and integration in the brain.<sup>26</sup> The complexity of the postural reactions allows detection of minor deficits in any key component of the pathway. Postural deficits are seen caudal to or at the level of the lesion.<sup>40</sup> Additional testing must be performed to use the postural deficit to help localize the lesion within the pathway of the deficit.

#### How to Perform

A wing or leg is placed in an abnormal position and a correcting response by the bird is observed. Not all standard mammalian postural reactions can be done due to the modifications of the forelimbs in birds. However, knuckling the toes over while supporting the bird can be done to evaluate how long it takes for the bird to correct. Placing the bird's foot on its dorsal surface against a perch may be a more practical way to test proprioception in the limbs. Alternatively, a piece of paper may be placed under each foot and slowly moved sideways to see if the bird returns its foot to the standing position. Placing reactions of the wings are not evaluated routinely in birds. Placing reactions in the rear limbs may be helpful. Visual placing may be evaluated crudely by offering a perch for the patient to step onto (**Fig 17.7**). Tactile placing may be evaluated in birds such as ratites and raptors that allow hooding. A perch may be touched to the dorsum of the foot to initiate a step-up.

#### How to Interpret

Conscious proprioception is the patient's awareness of limb position and movement without visual information. The sensory branch of proprioception is carried from the skin, muscle and joints of the leg, through the spinal cord and brainstem to the sensory motor cortex where the brain responds by sending messages back to the lower motor neuron for motor function, resulting in a rapid correcting foot placement.<sup>26</sup> Ascending sensory pathways are located in the outermost regions of the spinal cord and are very sensitive to compression.<sup>40,87</sup> With minor spinal cord injury, proprioceptive deficits may be present because of disrupted sensory pathways, while motor function persists because the deeper motor tracts are unaffected. Both visual and tactile placing reactions require an intact motor cortex and intact motor pathways to the involved limb. A cortical lesion may



**Fig 17.6** | By moving the head from side to side, the movements of the eyes can be evaluated for physiological nystagmus.



**Fig 17.7** | Bringing the bird to a perch will assess the ability to place the limbs properly; this is one method of assessing proprioception. The bird also can be visually restricted using hooding.

**Table 17.2** | Clinical Signs Associated with Upper Motor Neuron (UMN) and Lower Motor Neuron (LMN) Diseases

Clinical Function	UMN Disease	LMN Disease
Motor function	Paresis or paralysis	Paresis or paralysis
Muscle tone	Normal to increased	Often reduced
Spinal reflexes	Normal to decreased	Decreased to absent
Muscle mass	Normal to decreased (disuse atrophy)	Dramatically decreased after 5-7 days (neurogenic atrophy)
Conscious proprioception	Often reduced to absent	Often reduced to absent

produce deficits in the contralateral limb, whereas a lower lesion produces deficits in the ipsilateral limb.

## SPINAL REFLEXES

Completion of a reflex requires an intact sensory nerve that provides transmission to the spinal cord and an intact motor nerve that elicits function from the innervated muscle. The reflex arc itself does not involve the brain or the remainder of the spinal cord. Lesions in the motor arm of the reflex arc, termed lower motor neuron (LMN), may cause a decreased or absent reflex (hyporeflexia or areflexia). An exaggerated response (hyperreflexia) results from an interruption in proximal motor pathways that modulate the reflex, termed upper motor neuron (UMN). Lower motor neuron signs indicate damage to one or more components of the reflex arc. Upper motor neuron signs indicate damage anywhere between the reflex arc and the brain (Table 17.2).<sup>39,62,87</sup>

### The Vent Sphincter Reflex

#### How to Perform

Pinch or prick the vent and watch for a wink-like contraction of the external sphincter muscles and a tail bob.



**Fig 17.8** | The pedal withdrawal can be evaluated by applying a pinching stimulus to the digits and watching for flexion and withdrawal of the joints and limb, respectively.

#### How to Interpret

This reflex reveals information regarding the pudendal plexus and caudal segments of the spinal cord. A flaccid, unresponsive vent and overdilated cloaca that is easily expressed indicate LMN damage to the pudendal nerve or its spinal roots. A hypertonic, hyperresponsive vent indicates UMN damage at any point cranial to the pudendal plexus.

### The Pedal Flexor Reflex

#### How to Perform

Apply a pinch stimulus to the skin of each foot and evaluate the response of the ipsilateral and contralateral limb (Fig 17.8).

#### How to Interpret

This is a withdrawal reflex in which stimulation of sensory receptors in the feet elicits contraction of flexor muscle groups in the leg. Presence of a withdrawal



**Fig 17.9** | With the limb allowed free movement, the patella reflex can be performed by applying a stretch stimulus to the patellar tendon.

reflex requires an intact ischiatic nerve (sensory and motor) and an intact spinal segment at the sacral plexus, but does not require transmission along the spinal cord to the brain. Absence of the withdrawal reflex in the leg denotes extensive lower motor neuron damage involving the sacral plexus or the ischiatic nerve. A space-occupying mass within the kidney or pelvic canal, such as a renal tumor or an egg, may impinge upon the lumbosacral plexus and its nerve, causing a hyporeflexic or areflexic withdrawal in the legs.<sup>15</sup>

### The Patellar Reflex

#### How to Perform

A tap stimulus should be applied to the straight patellar tendon and the response of the limb should be evaluated (Fig 17.9). Certain birds, such as ostriches,<sup>26</sup> do not have a patella; in these species, the homologous straight quadriceps tendon is stimulated. Plexor size must be adapted to patient size for improved accuracy. Because of interference from the inguinal web, this reflex may be difficult to elicit in birds.<sup>26</sup>

#### How to Interpret

This is a myotatic (stretch) reflex that effectively stretches the *femorotibialis* muscle.<sup>87</sup> This stretch stimulates the femoral nerve (lumbosacral plexus), which generates muscular contraction to extend the stifle. Upper motor neuron lesions cause hyperreflexia and should be accompanied by weakness.<sup>26</sup> Disease in the lumbosacral plexus or peripheral muscle or nerve (LMN) causes hyporeflexia.

### The Wing Withdrawal Reflex

#### How to Perform

A pinch stimulus to the major digit at the leading edge of the primary flight feathers generates flexion of the wing in the normal bird (Fig 17.10).



**Fig 17.10** | The wing withdrawal can be assessed by stretching each wing out and applying gentle pinching stimulus, watching a complete flexion and withdrawal.

#### How to Interpret

This reflex tests the integrity of the reflex arc to the brachial plexus spinal segment. Absence of the wing withdrawal reflex in a wing indicates damage to the brachial plexus or its nerves. A positive crossed extensor reflex of a wing denotes damage cranial to the brachial plexus within the cervical spinal cord or the brain stem.

## CUTANEOUS SENSATION AND PAIN

Cutaneous sensation testing provides information regarding the location and severity of a spinal cord or plexus lesion.<sup>26</sup> Birds do not have a *cutaneous trunci* muscle, so a panniculus response cannot be used to help localize a spinal cord lesion; however, the feather follicles contain sensory nerve fibers.<sup>31</sup>

Evaluation of nociception (deep pain perception) is reserved for those animals showing evidence of spinal cord disease based on abnormalities in gait, proprioception and spinal reflexes. Nociception requires cerebral perception of painful or injurious stimuli. It is important to remember that a withdrawal reflex is not an indicator of pain perception and may be elicited in an animal whose spinal cord has been transected cranial to the segment responsible for that reflex arc.<sup>26,40</sup> See Chapter 8, Pain Management.

## Ancillary Neurological Tests

### SURVEY RADIOGRAPHY AND MYELOGRAPHY

After a thorough clinical examination, selective or survey radiography may be indicated to evaluate body systems for

their contribution to clinical signs. As it is inexpensive and non-invasive, survey radiography may be one of the best screening tools to uncover a cause for neurologic disease.

Sedation or general anesthesia facilitates exact positioning to yield the best image of the desired area. Both lateral and ventrodorsal projections should be obtained, with the beam centered over the area of interest. The axial skeleton of the bird should be extended, and the spine should be made parallel to the cassette, supporting the bird's body with foam wedges if required. The most helpful spinal views include the following: cervical (lateral and ventrodorsal), thoracic (lateral), thoracolumbar (lateral and ventrodorsal), and lumbar (lateral and ventrodorsal). A fine screen and high-detail films<sup>3</sup> should be used for spinal studies in birds.

Myelography can be performed in birds with normal patient recovery and adds another diagnostic procedure to the armamentarium of avian veterinarians. Myelography is used to identify, characterize and localize spinal cord lesions to the extramedullary or intramedullary compartments. Myelography is indicated if precise localization of a spinal cord lesion is required, such as when surgical intervention is contemplated. A contrast medium is injected into the subarachnoid space and lack of opacification around the spinal cord indicates displacement of that cord segment, preventing contrast material flow and suggesting diffuse cord swelling or a space-occupying mass compressing the cord.

The synsacrum of birds is composed of the fused lumbar and sacral vertebrae and pelvis, and necessitates thoracolumbar rather than lumbar puncture of the subarachnoid space. Another structure unique to birds is the glycogen body, which bisects the spinal cord in the lumbosacral region ventral to the synsacrum and causes a narrowing of the subarachnoid space in this region.<sup>51</sup> It appears that injection of contrast medium into the subarachnoid space in the cerebellomedullary cistern cannot be consistently repeated and trauma to the spinal cord near the brainstem can result in death.<sup>51</sup>

A technique for administering a myelographic contrast agent, iohexol<sup>b</sup>, in the thoracolumbar region has been described.<sup>51</sup> The region for injection is found by placing the thumb and middle finger on the bony prominences of the ileal crests, with the index finger used to palpate to the first indentation cranial to the synsacrum. The quantity of the contrast medium needed to produce a diagnostic myelogram is 0.88 ml/kg, 4 times the 0.22 ml/kg used in mammals in most institutions.<sup>51</sup> The use of a 27-gauge needle for this procedure is currently recommended,<sup>51</sup> and the administration of approximately 0.5 ml/minute has been described as safe.<sup>51</sup> Radiographs should be made within 10 minutes of the contrast

administration. Seizures, the predominant side effect noted in mammalian myelography, have not been observed in birds, even at higher doses than those recommended above. However, this does not rule out the possibility of this occurring with iohexol myelography in birds.<sup>51</sup> There are some avian species in which myelography may not be possible. In duck, goose and swan skeletons, the thoracic and lumbar vertebrae have overlapping plates of bone that would prevent positioning a needle in the subarachnoid space. Potential thoracolumbar puncture sites do exist in psittacines and raptors, which are the most commonly seen patients in private practice and rehabilitation centers.

## CEREBROSPINAL ANALYSIS

Cerebrospinal fluid (CSF) may confirm pathologic changes, determine the general nature of the pathologic process or reveal a specific cause of disease.<sup>25,64</sup> CSF is most useful in characterizing infectious, neoplastic or inflammatory disorders of the spinal cord or brain. Ideally, this procedure should be performed prior to myelography, as contrast will affect the analysis of the fluid.

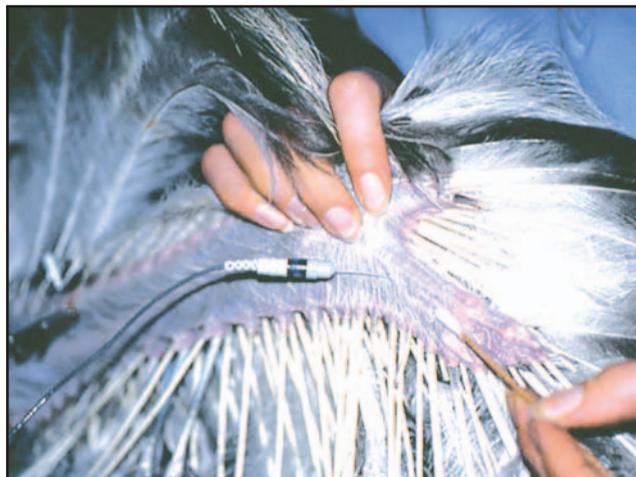
In birds, the cerebellomedullary cistern is very small, and a large venous plexus exists just ventral to it.<sup>62</sup> There is less than a 1 mm space dorsal to the cervical spinal cord in which the tip of the needle could be placed. This can result in blood-contaminated samples, lack of samples and damage to the nervous tissue.<sup>51</sup> The damage, which can be focal and cause acute severe hemorrhage in the subarachnoid space, can result in postanesthetic recovery problems including cardiac arrest and death.<sup>51</sup> Presently, it is recommended to use a 27-gauge needle for this procedure.<sup>30</sup>

### How to Perform

The patient is placed in lateral recumbency with the midline of the neck parallel to the tabletop. The caudodorsal skull region is aseptically prepared. The atlanto-occipital joint is flexed at a 30° angle, and a 27-gauge hypodermic needle is placed at dorsal midline, slightly caudal to the occipital protuberance, and is directed rostrally at a 45° angle to the horizontal axis of the head. The needle is advanced through the skin slowly at 1-mm intervals until a slight change in resistance is felt. Practically, this is not always easily recognized. However, the advantage of using a hypodermic needle with a translucent hub, rather than a spinal needle with a stylette, is that an immediate "flash" of fluid will appear in the hub of the needle once the dura has been penetrated. This limits the risk of advancing the needle into parenchyma. In general, 0.1 to 0.5 ml of fluid may be collected. In small mammals, 1 ml of CSF per 5 kg body weight may be removed safely, although this has not been confirmed in birds.<sup>25</sup>



**Fig 17.11** | A barred owl (*Strix varia*) under general anesthesia for an electromyographic examination.



**Fig 17.12** | A concentric needle is inserted into each muscle for evaluation of the presence of a spontaneous electrical activity.

### How to Interpret

The small sample volume usually obtained precludes extensive biochemical analysis, bacterial or fungal culture, and antibody titer evaluation. Color and clarity of the CSF should be evaluated immediately. Gross visual examination of normal CSF reveals a clear and colorless fluid. Blood contamination may be iatrogenic or due to a pathologic process.<sup>26</sup> Turbidity indicates leukocytosis, high protein content or both. If the sample volume is sufficient, protein content may be evaluated in addition to cytological examination. High protein concentration indicates breakdown of the blood-brain barrier or intrathecal protein synthesis. Preparation of the CSF sample by cyospin centrifugation is recommended because of the small volume and low cellularity of fluid generally recovered.<sup>26</sup> If a cyocentrifuge is unavailable, the cells may be concentrated onto a slide by sedimentation.<sup>25</sup> The cell concentration, population and morphology are evaluated. Relative erythrocyte and leukocyte numbers and protein concentration should be compared with those found in the peripheral blood. Reference values for leukocyte numbers and protein concentration in CSF have not been established in birds. Subjective comparison of CSF values with those of peripheral blood and consideration of parameters used in domestic animals may aid interpretation.

Meningitis caused by infectious agents generally induces a moderate increase in cell numbers and an increase in protein concentration compared with peripheral blood.<sup>3,26</sup> Non-inflammatory conditions caused by degenerative disease or trauma cause a high protein concentration and a minimally increased cell count.<sup>25,26</sup>

## ELECTRODIAGNOSTICS

The use of electrophysiological diagnostic techniques has become a cornerstone in the investigation of neuromuscular disease in animals. Electrodiagnostic evalua-

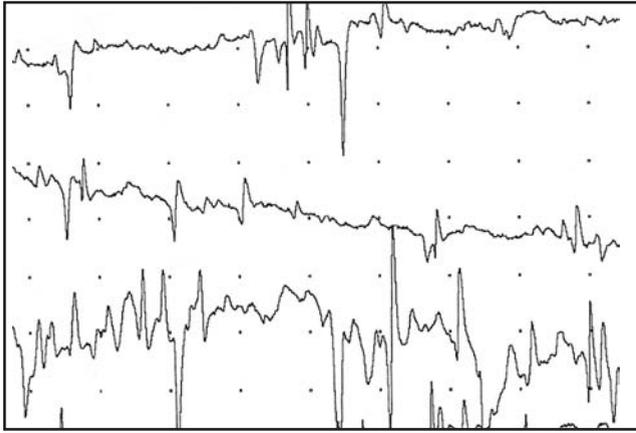
tion of the neuromuscular system is used as an aid in the diagnosis of disease, to assess the progress or regression of the disease, and to assess the degree of recovery.<sup>65,110</sup> Skeletal and smooth muscle as well as the myoneural junction can be examined. Concentric needle electrodes are used to determine the spontaneous or evoked responses. They are usually 27-gauge, subcutaneous or longer, insulated needles with bare tips, which can be inserted into the muscle. A ground electrode must always be used. The patient usually has to be anesthetized for these procedures (Fig 17.11).

### Electromyography

Electromyography (EMG) is the study of the electrical activity of muscle by insertion of a recording electrode into the muscle (Fig 17.12). It examines the integrity of the motor unit, which consists of the lower motor neuron and the muscle fibers that it innervates. Normal resting muscle does not show observable electrical activity once the electrode placement is stabilized and no audible signal is created.<sup>110</sup> Contraindications for electromyography include bleeding tendencies and unusual susceptibility to recurrent systemic infections.<sup>61</sup> Electrical activity demonstrated can be separated into three categories: insertional (associated with electrode movement), spontaneous (associated with resting muscle) and evoked (associated with electrical stimulation of nerves).<sup>61</sup>

#### Insertional

A brief burst of electrical activity accompanies needle insertion into a normally innervated muscle. This collection of monophasic and polyphasic potentials of variable amplitude and duration ends after the needle has stopped its movement. A prolonged discharge of these potentials is sometimes subjectively considered as abnormal, as it represents cellular hyperirritability. Insertional potentials become more noticeable on the



**Fig 17.13** | Fibrillation potentials and positive sharp waves from a denervated muscle 5 days after brachial trauma.

fourth or fifth day after denervation and peak in intensity 8 to 10 days after denervation.<sup>26</sup> These may be decreased or absent in atrophied muscle.<sup>110</sup>

### Spontaneous

Normally, muscles are said to be “electrically silent.” Five to seven days after denervation in the dog, needle electromyography can demonstrate spontaneous, randomly occurring potentials.<sup>26</sup> These are the action potentials of several muscle fibers due to instability in the membrane potential and are termed fibrillation potentials. The time of onset of these potentials in birds has been poorly documented, but may be earlier than seen in the dog. Fibrillation potentials can be monophasic, biphasic and, rarely, triphasic in wave form with a usual amplitude of 20 to 300  $\mu\text{V}$  and a duration of 0.5 to 5 msec (Fig 17.13), both of which decrease with cicatrization.<sup>110</sup> The presence of reproducible discharges in at least two different areas of muscle usually suggests lower motor neuron disease. These include diseases of the ventral horn cells, radiculopathies, plexopathies, myositis, and axonal mono- or polyneuropathies.<sup>61</sup> Positive sharp waves with a sawtooth appearance also are abnormal spontaneous potentials that are detected in motor unit disorders, and it has been suggested that they may precede fibrillation potentials by one or more days.<sup>110</sup> Complex repetitive discharges range from 50  $\mu\text{V}$  to 1 mV in amplitude and up to 50 to 100 msec in duration, representing a group of muscle fibers firing in near synchrony.<sup>61</sup> These discharges typically begin suddenly and maintain a constant firing rate, ceasing abruptly and sounding like a machine gun.<sup>61</sup> These complex repetitive discharges appear as a “train” of identical discharges. These discharges may occur in a variety of myopathic conditions. Needle electromyography has been used in the cat, a species susceptible to organophosphorus-induced delayed neuropathy like the bird, to monitor progression and improvement.<sup>1</sup> This could not be repeated, though, in chickens.<sup>107</sup> Denervation is detectable on needle electromyography in

birds. Fibrillation potentials and occasional complex repetitive discharges were seen in the wing muscles of two red-tailed hawks with traumatic brachial plexus injury. Motor unit action potentials observed in these same birds suggested that intact nerve fibers were present.<sup>108</sup> Fibrillation potentials and positive sharp waves were detected diffusely in limb musculature of a turkey vulture with a peripheral neuropathy associated with lead toxicosis.<sup>92</sup> Spontaneous EMG also has been used to determine prognosis and to direct treatment in seven wild birds with traumatic injuries involving peripheral nerves and musculoskeletal disease.<sup>27</sup> Widespread fibrillation potentials and lack of typical interference patterns suggested denervation in three of these seven birds that had muscle atrophy and wing paralysis or paresis.

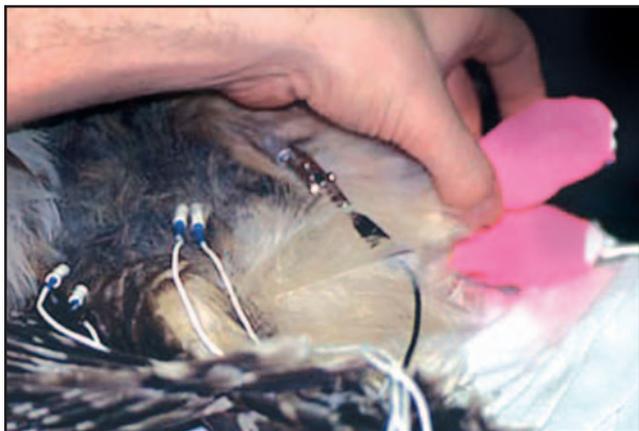
### Evoked

The M wave is an evoked potential resulting from the summation of motor unit potentials. The latency of response represents the time taken for impulse conduction from the point of stimulus to the nerve terminal, including a neuromuscular delay of about 0.5 msec, and the time taken for conduction from the end-plate region to the exploring electrode. The latency can be considered an adequate substitute for the nerve conduction velocity.<sup>26</sup> The amplitude of the evoked potential is determined by the number of muscle fibers activated by nerve stimulation. In a severed motor nerve, conduction stops abruptly after about 5 to 8 days.<sup>26</sup>

### Nerve Conduction Studies

Nerve conduction studies have become a simple and reliable test of peripheral nerve function.<sup>61</sup> The conduction velocity is measured between the two stimulus points on the nerve, thereby eliminating the time for neuromuscular transmission and generation of muscle action potential. It is derived as the ratio between the distance between two stimulation cathodes and the corresponding latency difference.<sup>61</sup> The proximal stimulation site is a point just caudal to the greater trochanter of the femur, and the distal site is just caudal to the distal portion of the tibia (Fig 17.14). At each site, the cathode is placed distal to the anode. The indifferent electrode is placed subcutaneously over the midtarsometatarsus region. The active electrode is placed subcutaneously on the caudal portion of the hypotarsus just distal to the tarsometatarsal joint overlying the digital abductor muscles.<sup>10</sup> Similar electrode configurations are needed for evaluation of the wing nerves (Fig 17.15). The ground electrode is placed subcutaneously between the distal stimulation site and the recording site. This technique has been described in detail for two avian orders.<sup>27</sup>

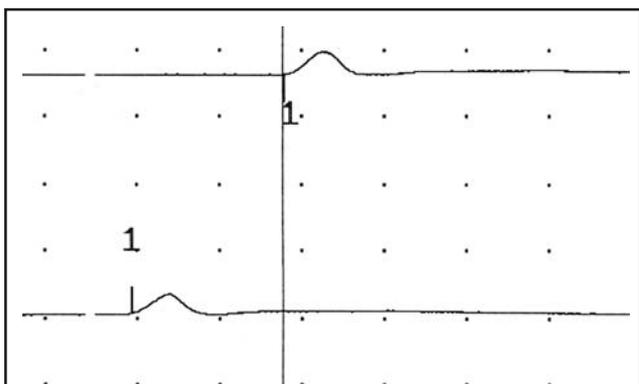
Axonal damage or dysfunction will usually result in loss of amplitude, whereas demyelination leads to prolonga-



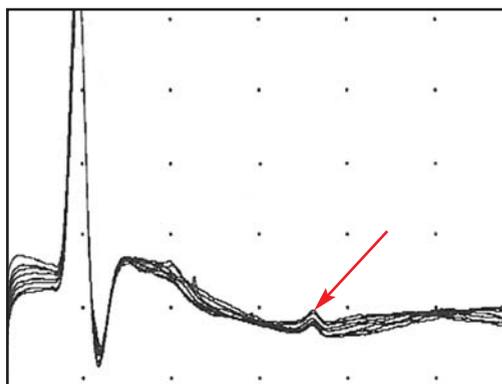
**Fig 17.14** | Needle setup for evaluation of a tibial nerve conduction velocity in a barred owl.



**Fig 17.15** | Needle setup for evaluation of an ulnar nerve conduction velocity in a rhea.



**Fig 17.16** | A reduced nerve conduction velocity is demonstrated after stimulation of the proximal portions (top trace) of the tibial nerve and the distal portions (lower trace). The waveforms (M wave) demonstrated here are typical.



**Fig 17.17** | An F wave (arrow) is demonstrated after the M wave and the stimulation artifact.

tion of conduction time (Fig 17.16).<sup>61</sup> Reference ranges for the motor nerve conduction velocities of the ulnar nerve and the tibial nerve in neurologically normal barred owls and rheas have been established.<sup>27</sup> Based upon these ranges, a turkey vulture with lead toxicosis was shown to have decreased sciatic-tibial nerve conduction velocity compatible with a demyelinating neuropathy.<sup>92</sup> Peripheral nerve histopathology confirmed this finding.

### Effect of Temperature

A linear relation normally exists between motor nerve conduction velocity and temperature of the peripheral nerve segment within physiological limits.<sup>67</sup>

### Effects of Age

Immature animals have slower motor nerve conduction velocity than do adults.<sup>26</sup>

### The F Wave

Measurement of the F wave can help in the assessing of motor conduction along the most proximal segment of the nerve. This is because it results from the backfiring of antidromically activated ventral horn cells. It can supplement the conventional nerve conduction studies, espe-

cially in polyneuropathies where delay of the F wave can markedly exceed the normal range. It occurs after the M response, its initiating electrical stimulation having traveled toward the spinal cord before it returns to activate distal muscles (Fig 17.17). Studies of the F wave can help in the characterization of polyneuropathies and, more specifically, with those that are prominently proximal, such as radiculopathies.<sup>61</sup> Such studies have been attempted in normal birds, but as yet a reference range has not been established for clinical application.<sup>27</sup>

### Repetitive Stimulation

Repeated supramaximal stimulation should create repeatable, identical action potentials in the normal patient. Any evident incremental or decremental response may have physiological significance. Any defect in the neuromuscular transmission will usually produce a maximal drop in amplitude between the first and second responses of a train, followed by further decline up to the fourth or fifth potential.<sup>61</sup> Examples of these disorders would be myasthenia gravis in humans and companion animals and botulism in all species. Repetitive stimulation has been performed in normal birds to

establish reference ranges for clinical application.<sup>27</sup> The techniques were easily performed and demonstrated 22% alteration in potential amplitude.<sup>27</sup>

## MUSCLE BIOPSY

Muscle biopsy is indicated to confirm, define and possibly provide a cause for motor unit disease.<sup>19,88</sup> Biopsy techniques are well established in several mammalian species, but are not known for avian patients. The muscle selected for biopsy should be one that is clinically affected with the disease process. In acute disease, a severely affected muscle is selected. With chronic disease, a muscle demonstrating only moderate changes is the best choice. The muscle should be easily accessible and identifiable so that minimal postoperative discomfort is created. Ideally, the muscle chosen for biopsy would be one for which normal fiber type, distribution and size are known. This is not available for most avian patients. Studies of muscle fiber types have been done in chickens.<sup>16</sup>

## SCINTIGRAPHY

Nuclear imaging or scintigraphy is a non-invasive method for evaluation of soft tissue and osseous structures associated with the nervous system.<sup>112</sup> The technique involves intravenous administration of a small amount of a gamma-emitting radionuclide alone or tagged to some other compound that will allow it to accumulate in certain tissues and exclude it from other tissues. A gamma camera is used to record the amount of radiation emitted from the body and to create images of the distribution of the radionuclide throughout the patient's body. Technetium-99m (<sup>99m</sup>Tc) is used most often, as it has a short half-life (approximately 6 hours) and emits pure gamma radiation. Because the level of radiation is extremely low, its use carries minimal risk for the patient and personnel involved with the procedure. Nuclear scans do not provide detailed anatomic imaging, but do have the potential to provide functional information when used with a digital image processor.

Scintigraphic imaging of the brain has been done in human medicine since 1960.<sup>26</sup> In recent years, cranial scintigraphic imaging has been replaced by computed tomography (CT) and magnetic resonance imaging (MRI), but is still useful where these modalities are considered too expensive or are not available. Bone scanning is particularly useful in identifying spinal abnormalities in birds.<sup>26</sup> Superimposition of osseous structures (ribs, synsacrum) in survey radiographs makes identification of spinal fractures and early vertebral osteomyelitis difficult. In a study of 12 birds with thoracic or pelvic limb paresis, bone scanning identified 100% of the lesions identified on survey radiographs.<sup>75</sup> In addition,

several lesions not identified on survey radiographs, including fractures and early osteomyelitis, were identified by bone scanning. These pathologic lesions were confirmed by the results of necropsy. Basilic vein administration of the radionuclide was found to be superior to medial metatarsal vein administration. This is possibly because the renal portal system provided high uptake of radionuclide by the liver and kidney and obscured visualization of the spine after medial metatarsal vein administration.<sup>75</sup>

## COMPUTED TOMOGRAPHY

Computed tomography imaging requires general anesthesia, uses ionizing radiation and is somewhat costly to perform. The CT scan uses x-rays and computer technology to create cross-sectional images of the patient.<sup>54,68</sup> Regions of the vertebral column and the skull are evaluated for abnormalities in CNS soft tissue or its protective skeleton. Computed tomography provides superior soft tissue imaging with no superimposition of structures compared with conventional radiography.<sup>77,86</sup> With contrast administration, soft tissue structures are better visualized, especially where there is increased blood flow.<sup>86,99</sup>

The greatest value of CT lies in its ability to reveal changes in bony tissues and to provide greater detail of osseous structures than conventional radiography. Slices are most commonly created every 2 to 3 mm in birds.<sup>86</sup> Although these slices may seem very close together, focal lesions may still be missed. Decreasing slice thickness to 2 mm decreases image quality, but may increase sensitivity for small lesions. Images also can be reformatted by the computer to provide images in configurations other than a transverse plane.

Computed tomography has been used to document topographic anatomy of the golden eagle and African grey parrot,<sup>77,86</sup> and it also has been used to specifically demonstrate brain and skull anatomy of the crested breed of the domestic duck (*Anas platyrhynchos*).<sup>12</sup> Computed tomography has been used with some success to locate intracranial lesions in birds, although it was only 80% sensitive.<sup>58,99</sup> A postmortem CT scan was used to identify spinal cord compression in a juvenile penguin (*Aptenodytes patagonicus*) that was euthanized because of an inability to stand despite 6 weeks of medical therapy;<sup>38</sup> radiographs at presentation had failed to identify any spinal abnormalities. Results of necropsy confirmed intervertebral disc rupture and vertebral body displacement.

## MAGNETIC RESONANCE IMAGING

Magnetic resonance imaging (MRI) uses a pulsating external magnetic field and produces radiofrequency signals that are used to generate images.<sup>26,112</sup> Soft tissue con-

trast of the CNS is excellent with MRI, which also provides the spatial orientation of anatomic structures.<sup>26,112</sup> This modality also will help to differentiate central nervous system gray and white matter.

The advantages of MRI compared with CT include the ability to image the cranial brainstem, without artifact, in the caudal fossa of the skull, the increase in soft tissue detail, the creation of images in various planes and the lack of ionizing radiation.<sup>98</sup> However, MRI does not allow a satisfactory depiction of aerated bones in some avian skulls.<sup>12</sup> One main disadvantage of MRI besides the expense, as with CT, is the requirement for general anesthesia, as chemical sedation is not adequate to obtain satisfactory images. It also is necessary to obtain screening radiographs of birds before performing MRI evaluations to rule out the presence of metallic foreign bodies.<sup>98</sup> Avian patients will frequently chew on their cages, etc, and small metal fragments may contaminate commercial feed. Ferrous metal demonstrates susceptibility to the magnetic field and can move within the body.<sup>98</sup>

In a study of normal MRI brain anatomy of pigeons, imaging required approximately 20 minutes, and transverse images were created every 3 mm.<sup>98</sup> A 1.5 Tesla magnet and a human knee surface coil were used. Lesions may be missed because of this distance between slices. This imaging technique also was used in a clinical case in an attempt to identify the cause of seizure activity in a mealy Amazon parrot (*Amazona farinosa*).<sup>98</sup> Without contrast enhancement the lesions were not visualized; however, after the use of contrast material (gadopentate dimeglumine<sup>c</sup> 0.25 mmol/kg IV) perivascular infiltrates were identified, showing disruption of the blood-brain barrier. Because the contrast is eliminated through the kidneys, postcontrast uric acid levels have been compared to precontrast levels to monitor for potential renal toxicity in birds; however, no significant changes have been found.<sup>98</sup> Further, after contrast medium-enhanced MRI studies in birds, recovery has been uneventful and the birds have had normal excreta, appetites and attitudes.<sup>98</sup> Magnetic resonance imaging also has been used to evaluate the cranium of domestic ducks.<sup>13</sup> MRI images were recorded using a relatively large slice thickness of 5 mm, which can lead to difficulty in interpreting scans of small volumes.

## Intracranial Disease

### CLINICAL SIGNS

Intracranial CNS structures include the forebrain (telencephalon), thalamus (diencephalon), midbrain (mesencephalon), pons (metencephalon), medulla oblongata

(myelencephalon) and the cerebellum. The midbrain, pons and medulla make up the brainstem and are associated with the majority of the cranial nerves. Disease affecting the intracranial CNS may be focal or diffuse. If it is focal, specific deficits may be identified relating to dysfunction of the area affected. The various syndromes and specific signs are described in [Table 17.3](#).

## Seizures

### Definitions and Classifications

A seizure is a sudden, uncontrolled, transient alteration in behavior characterized by a change in motor activity, consciousness, sensation or autonomic function.<sup>28</sup> It is due to a synchronizing abnormal paroxysmal electrical discharge from the brain. A *generalized seizure* is manifested by symmetrical and synchronous clinical signs, which are classically tonic-clonic motions often accompanied by complete loss of consciousness.<sup>28</sup> A *focal*

**Table 17.3 | Intracranial Lesion Localization and Clinical Signs<sup>93</sup>**

Clinical Syndrome/ Lesion Localization	Characteristics of the Location	Specific Clinical Signs
Cerebrum	Damage to cerebrum affects intellectual, learned and sensory activities (vision, hearing, touch, pain)	<ul style="list-style-type: none"> <li>i. Altered mental state</li> <li>ii. Seizures</li> <li>iii. Behavioral change</li> <li>iv. Pleurothotonus and adversion</li> <li>v. Head pressing</li> <li>vi. Central blindness</li> </ul>
Diencephalon	Damage affects autonomic visceral functions and endocrine regulation	<ul style="list-style-type: none"> <li>i. Altered mental state</li> <li>ii. Seizures</li> <li>iii. Behavioral change</li> <li>iv. Endocrine disturbances</li> <li>v. Abnormalities of temperature</li> </ul>
Midbrain	Damage affects control of alert status, as well as CN III and CN IV function	<ul style="list-style-type: none"> <li>i. Altered mental state</li> <li>ii. Opisthotonus</li> <li>iii. Contralateral paresis</li> <li>iv. Ipsilateral CN III dysfunction (mydriasis/ventrolateral strabismus)</li> <li>v. Contralateral CN IV dysfunction (dorsomedial strabismus)</li> </ul>
Pons and Medulla	Damage affects multiple cranial nerves (V-XII) as well as cardiorespiratory centers	<ul style="list-style-type: none"> <li>i. Altered mental state</li> <li>ii. Ipsilateral paresis</li> <li>iii. Irregular respiration</li> <li>iv. Ipsilateral CN V dysfunction (decreased beak strength, decreased palpebral reflex, decreased facial sensation)</li> <li>v. Ipsilateral CN VI dysfunction (third eyelid protrusion and medial strabismus)</li> <li>vi. Ipsilateral CN VII dysfunction (decreased tone in facial musculature, decreased taste, decreased lacrimation)</li> <li>vii. Ipsilateral CN VIII damage (ipsilateral deafness, vestibular dysfunction)</li> <li>viii. Ipsilateral CN IX-XII damage (dysphagia, regurgitation, tongue deviation, inspiratory dyspnea)</li> </ul>
Cerebellum	Damage affects coordinating and reinforcing actions	<ul style="list-style-type: none"> <li>i. Normal mental status and behavior</li> <li>ii. Normal strength</li> <li>iii. Opisthotonus</li> <li>iv. Dysmetric ataxia</li> <li>v. Head (intention) tremors</li> </ul>

*seizure* is manifested initially by focal signs that may secondarily become generalized.

### Treatment

1. Address the underlying cause.
2. Control seizure activity with diazepam (0.5-1.0 mg/kg IV or IO).<sup>28</sup> This can be repeated two to three times if necessary.
3. If diazepam does not stop the activity, administer phenobarbital (1-2 mg/kg IV/IO or IM q 6-12 h to effect).<sup>28</sup>
4. Gaseous anesthesia may be warranted in seizing birds to allow diagnostic sampling and investigation.
5. Convert to oral phenobarbital maintenance therapy within 48 hours (1-10 mg/kg PO q 12 h is standard therapy).<sup>28</sup>
6. Primidone may be an alternative anticonvulsant, although its active compound is phenobarbital. Primidone use (125 mg/day in water source) has been reported in an Amazon parrot.<sup>52</sup>
7. Adjust to lowest dosage required to control seizure activity.

## Head Tilt and Nystagmus

### Definitions and Classifications

A head tilt is due to asymmetrical disease of the vestibular system (Fig 17.18). The vestibular system comprises the peripheral component, which is the vestibular portion of CN VIII arising in the inner ear from the semicircular canals and the central component, which is the nuclei of CN VIII in the medulla.<sup>28</sup> A further central component of this system is the flocculonodular lobe of the cerebellum. Head tilts are usually toward the side of the disease with peripheral vestibular dysfunction, but toward any side with central dysfunction. Nystagmus that is pathological is described as jerky eye movements with two obvious phases; slow and fast. The predominant direction of the eye movements, seen at rest, can be horizontal, rotational or vertical. Nystagmus also can be positional, which means that it is induced by a change in position of the head. With horizontal nystagmus, the fast phase is usually away from the side of the lesion. Vertical and positional nystagmus can indicate that the lesion is central rather than peripheral. All other directions can occur with both peripheral or central lesion localizations.

### Treatment

1. Address the underlying cause and tailor therapy to the specific etiology.
2. Provide supportive care to maintain nutritional and fluid demands.

## Tremors and Involuntary Movements

### Definitions and Classifications

Head and or body tremors may occur with activity and



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**Fig 17.18** | A lovebird (*Agapornis roseicollis*) demonstrates a right-sided head tilt caused by an ipsilateral loss of muscle tone due to peripheral vestibular disease.

appear worse with movement such as eating or drinking. These are called intention tremors and are the cardinal sign of cerebellar disease of any underlying etiology. Generalized body tremors that occur mainly at rest could result from a multifocal or diffuse lesion localization. This may be a disease affecting the meninges, nerve roots, peripheral nerves or muscles. Systemic diseases including electrolyte imbalances, liver and renal disease, and hypoglycemia can cause generalized tremors as can primary neurologic diseases.

### Treatment

1. Address the underlying cause.
2. Attempt treatment with oral anticonvulsant therapy as above.
3. Attempt treatment with anti-inflammatory doses of glucocorticoids.
4. Provide supportive care to maintain nutritional and fluid demands.

## Depression/Stupor/Coma

### Definitions and Classifications

Depression represents a reduced mental state, although one which responds to external stimuli. Depression does not necessarily indicate primary neurologic disease. Stupor is a state of unconsciousness from which the bird can be roused by stimulation with a noxious stimulus (Fig 17.19). Coma is a state of unconsciousness from which the bird cannot be roused by noxious stimuli.

### Treatment

1. Address the underlying cause.
2. Provide supportive care to maintain nutritional and fluid demands.
3. Address ventilatory needs.
4. Address excretion needs and keep clean.



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**Fig 17.19** | A painted bunting (*Passerina ciris*) with profound stupor due to pesticide toxicosis.



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**Fig 17.20** | A gross pathological specimen from an African grey parrot (*Psittacus erithacus*) with cerebral disease due to hydrocephalus.

## DIFFERENTIAL DIAGNOSIS OF INTRACRANIAL DISEASE (Table 17.4)

### Degenerative

#### Lysosomal Storage Disease

Gangliosidosis<sup>18,60</sup>

Neurolipofuscinosis<sup>53</sup>

#### Avian Vacuolar Myelinopathy<sup>71</sup>

This disease more commonly causes spinal and neuromuscular signs, but intracranial signs have been reported.

### Anomalous

Hydrocephalus<sup>100</sup>

Dilated ventricles can be a congenital lesion as well as a postinflammatory consequence (Fig 17.20).

### Metabolic

#### Hepatic Encephalopathy<sup>15,29</sup>

Clinical signs included depression, ataxia and blindness.

#### Hypoglycemia<sup>15</sup>

Seizure activity may occur when the glucose levels drop below 100 mg/dl.

#### Hypocalcemia

Serum calcium levels below 6.0 mg/dl with concentrations as low as 2.4 mg/dl have been reported to cause opisthotonus (Fig 17.21), tonic extension of the limbs and convulsions.<sup>15</sup>

### Neoplasia<sup>100,113</sup>

In a retrospective study of 996 budgerigars (*Melopsittacus undulatus*), 14 (1%) intracranial tumors were detected.<sup>113</sup> Tumors of the ependyma (6), choroid plexus (1), adenohypophysis (6) and one unclassified tumor were found. In this study 87% of the birds were male,

**Table 17.4** | Differential Diagnosis of Intracranial Disease

Mechanism of Disease	Specific Diseases
Degenerative	i. Lysosomal storage disease - Gangliosidosis/Neurolipofuscinosis ii. Avian vacuolar myelinopathy
Anomalous	i. Hydrocephalus
Metabolic	i. Hepatic encephalopathy ii. Hypoglycemia iii. Hypocalcemia
Neoplastic	i. Glial tumors/Ependymoma/Choroid plexus papillomas/Adenomas/Adenocarcinomas/Lymphosarcoma/Lipomas
Nutritional	i. Pyridoxine deficiency ii. Vitamin E deficiency
Inflammatory	i. Viral encephalitis - Proventricular dilatation disease/Avian paramyxovirus type 1 (Newcastle disease)/Polyomavirus/WEE and EEE/West Nile virus/Avian influenza virus/Herpes (duck viral enteritis) ii. Bacterial encephalitis - <i>Salmonella</i> / <i>Pasteurella</i> / <i>Streptococcus</i> / <i>Enterococcus</i> / <i>Chlamydophila</i> / <i>Listeria</i> iii. Vermineous encephalitis - <i>Chandlerella</i> / <i>Baylisascaris</i> (fluke/ <i>Schistosomiasis</i> ) iv. Trichomonas/Protozoal encephalitis - Toxoplasmosis/Leucocytozoonosis/Sarcocystis v. Fungal encephalitis - Mucormycosis vi. Prion - Spongiform encephalopathy
Idiopathic	i. Idiopathic epilepsy
Toxicity	i. Lead ii. Zinc iii. Organophosphates and carbamates
Trauma	i. Head trauma
Vascular	i. Atherosclerosis

indicating a possible sex predisposition, as there were equal numbers of males and females in the study. Typically, neoplasia is a necropsy diagnosis, but it may be found antemortem with a CT or MRI scan. Glial cell tumors, choroid plexus papillomas, adenomas, adenocarcinomas and lymphosarcomas have all been reported as individual primary brain tumors.<sup>15</sup>

### Intracranial Lipomas

Four birds in a flock of 125 purebred crested ducks (*Anas platyrhynchos*) had cerebellar signs of unknown



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**Fig 17.21** | A young cockatiel (*Nymphicus hollandicus*) demonstrates the marked dorsiflexion of the neck seen with opisthotonus.



Jan Hooimeijer

**Fig 17.22** | A pigeon (*Columba livia*) with paramyxovirus infection of the central nervous system demonstrates profound torticollis of the neck.

etiology.<sup>14</sup> Gross necropsy of the euthanized ducks revealed yellow intracranial masses in the brain of each. Histologically, these masses were intracranial lipomas consisting of fatty tissue separated into lobules by connective tissue. These lipomas were similar to those recorded in other animals and humans.<sup>14</sup>

## Nutritional

### Pyridoxine (Vitamin B<sub>6</sub>) Deficiency<sup>15</sup>

#### Vitamin E Deficiency

#### (Nutritional Encephalomalacia)<sup>6</sup>

The recommended dietary level of vitamin E for domestic poultry is 10 to 25 IU/kg feed dry matter (DM), but this is not known for many other birds. Clinical signs are commonly seen in young chicks (2-6 weeks old) and include ataxia, head retraction and “cycling” with legs.<sup>6</sup> This has become known as crazy chick disease.<sup>66</sup> Gross and histological lesions of encephalomalacia are primarily found in the cerebellum, with evidence of ischemic necrosis of the cerebellar cortex and white matter, capillary thrombi, hemorrhages and malacia.

## Inflammatory

### Viral Encephalitis

#### Avian Paramyxovirus Type 1 (Newcastle Disease)

causes a non-suppurative encephalomyelitis and has been reported to cause head shaking, head tremors, head tilt, circling and torticollis (**Fig 17.22**).<sup>8,69</sup> Infection is usually reported in juvenile wild birds, racing pigeons and domestic poultry.<sup>57,69</sup> The virus will commonly cause motor dysfunction and so is discussed below.

**Avian Polyomavirus (APV)**, also known as budgerigar fledgling disease, is a member of the family Papovaviridae.<sup>72</sup> Polyomavirus infection in psittacine birds may be subclinical or may present as a fatal, acute, multisystemic disease.<sup>72</sup> Although observed more frequently in budgerigars (*Melopsittacus undulatus*), APV infection may result in death of both juvenile and adult non-budgerigar

psittacine species.<sup>72</sup> Viral inclusions have been found in brain tissue, however, reports of virus-induced neurologic disease are rare.<sup>72</sup> Tremors of the head, neck and limbs, incoordination and ataxia have been described in nestling budgerigars with APV infection. Approximately 10% of susceptible neonates exhibited neurologic signs.<sup>72</sup> In addition, an adult Moluccan cockatoo (*Cacatua moluccensis*) with APV infection exhibited inability to perch properly and had a slight torticollis that progressed to a semicomatose state.<sup>72</sup> Neurologic deterioration was observed over a 2-day period following a 1-week history of illness. Body tremors and unsteadiness on the perch were signs documented in a Ducorps’s cockatoo (*Cacatua ducorps*) with concurrent APV and psittacine beak and feather disease virus (PBFDV).<sup>72</sup> PBFDV (circovirus) infection is often associated with clinical evidence of acquired immunodeficiency, leading to a variety of secondary or opportunistic infections.<sup>72</sup>

**Proventricular Dilation Disease (PDD)** is a progressive, variably contagious and often fatal disease of psittacine birds, which is presumed to be caused by an unidentified neurotropic virus.<sup>17</sup> The disease was first recognized in macaws in the late 1970s in the USA and is now known to have a worldwide distribution affecting more than 50 species of psittacine birds.<sup>17</sup> Neurologic clinical signs include ataxia, tremors and seizures. Definitive diagnosis in live and dead birds is based on demonstration of characteristic lymphoplasmacytic infiltrates within autonomic nerves and ganglia at various levels of the digestive tract.<sup>17,48</sup>

**Equine Encephalitis** - Viral infections have been reported in commercial, domestic, and free-living flocks of passerines and Columbiformes for several decades.<sup>96</sup> Most infections are attributed to eastern equine encephalitis (EEE), but several outbreaks of western equine encephalitis (WEE) have been documented.<sup>96</sup> Many types of birds are natural hosts of EEE and WEE viruses.<sup>96</sup> The diseases are caused by an alpha virus in

the arbovirus group/family, and are transmitted through the bites of mosquitoes.

Specific signs of WEE and EEE have been vague. In 1991, the first confirmed outbreak of EEE was reported in ratites, specifically emus (*Dromiceius novaehollandiae*) in southeastern Louisiana.<sup>96</sup> This outbreak coincided with unseasonably heavy rainfall, an abundance of arthropod vectors and close proximity to reservoir host species.<sup>96</sup> Affected emus exhibit variable signs, becoming very drowsy and depressed, reluctant to stand up, trembling, often remaining sitting, and developing an S-shaped cervical curvature. Aroused sick birds are ataxic with leg weakness.<sup>96</sup> Both infections can progress to cause paralysis and death.<sup>114</sup>

Diagnosis is confirmed by isolation of the virus from the brains of birds that die. Serum antibody titers for EEE, WEE and Venezuelan equine encephalitis exposure can be evaluated by hemagglutination inhibition.<sup>96</sup> Recently, an immunohistochemistry technique for confirmatory diagnosis of EEE infection in birds has been developed.<sup>121</sup> Treatment has been supportive, consisting of oral electrolytes and broad-spectrum antibiotics.<sup>96</sup> Protection of commercial emu flocks may be attempted by a routine vaccination schedule for both EEE and WEE. Vaccine efficacy recently has been proven in emus, and it currently is the only protective action that exists, other than mosquito control.<sup>96,114</sup>

**West Nile Virus (WNV)** emerged in the northeastern USA in the summer of 1999 causing death to thousands of wild and zoo birds.<sup>41,106</sup> Previously this infection has been reported around the Mediterranean basin.<sup>81</sup> It is a mosquito-borne flavivirus that is endemic in Africa and Asia, occurring sporadically in temperate regions of Europe.<sup>106</sup> It has been shown to cause a diffuse encephalitis and has been recovered from the brains of birds in winter, long after the mosquito vectors ceased to be active, suggesting a prey-to-predator mode of transmission.<sup>41</sup> Live and inactivated vaccines for protection against WNV recently have been evaluated in young domestic geese with initial promising results.<sup>78</sup>

**Avian Influenza Virus** has been reported to cause 75 to 100% mortality in birds within 10 days of infection.<sup>91</sup> Depression and neurologic dysfunction were common clinical manifestations of the disease.<sup>91</sup> These signs include attenuated motor functions, such as paresis to paralysis, vestibular dysfunction as indicated by torticollis and nystagmus, and general behavior aberrations.<sup>91</sup>

**Herpes (Duck Viral Enteritis)** causes photophobia, ataxia, seizures and penile prolapse.<sup>15</sup>

**Bunyavirus**<sup>109</sup> rarely causes clinical disease after trans-

mission of the virus from mosquitoes.

### Bacterial Encephalitis

*Listeria monocytogenes* causes intracranial infections of birds resulting in opisthotonus, ataxia, and torticollis.<sup>15</sup>

*Chlamydophila psittaci* will occasionally cause neurologic signs in birds following acute respiratory or gastrointestinal disease.<sup>15</sup>

Abscesses, granulomas, encephalitis and meningitis has been reported to be due to *Salmonella typhimurium*,<sup>53</sup> *Pasteurella multocida* (fowl cholera),<sup>50</sup> Lancefield group D *Streptococcus* sp.,<sup>24,47</sup> and *Enterococcus* sp. (neonatal multifocal encephalomalacia with sepsis).<sup>24,44</sup>

### Verminous Encephalitis

*Candlerella quiscali* is a filariid nematode of grackles that has been reported to cause cerebrospinal nematodiasis in emus.<sup>15</sup> Clinical signs in the young include torticollis, ataxia, recumbency and death.

*Baylisascaris procyonis* - The common raccoon ascarid is known to cause life-threatening visceral, cerebral and ocular larva migrans in birds in North America.<sup>23,32</sup> Infected asymptomatic raccoons can harbor a large number of worms and shed large numbers of infective eggs into the environment.<sup>23</sup> Affected birds can develop ataxia and dysmetria that progresses to inability to stand, with severe torticollis and a head tilt.<sup>23</sup>

*Trichomonas gallinae* causes disease in the domestic pigeon and birds that feed on pigeons such as eagles, falcons and hawks; it has been responsible for avian enteric trichomoniasis.<sup>89</sup> Caseous masses in the roof of the mouth may extend to involve the brain.

**Schistosomiasis** causing granulomatous encephalitis has been reported in swans.<sup>15</sup>

### Protozoal Encephalitis

*Toxoplasma gondii*, a cyst-forming coccidium, is not frequently identified as pathogenic in captive birds. Asymptomatic infection with this parasite is reportedly common.<sup>74</sup> Outbreaks of toxoplasmosis have been reported worldwide in chickens, and in passerine and psittacine birds and birds exhibited in zoos.<sup>74,122</sup> Naturally occurring infections have been diagnosed in chickens, ducks and many wild birds.<sup>122</sup> Because toxoplasmosis is a zoonotic disease, the source of the infection should be determined, if possible, to prevent infection of humans. Household cats should be tested. Common clinical signs of *T. gondii* infection in avian species include anorexia, blindness, head tilt, circling and ataxia.<sup>74,122</sup> Canaries (*Serinus canaria*) may be susceptible to a form of toxoplasmosis of the central nervous system and eyes that differs from the acute form seen in other species.<sup>74</sup>

Partridges (*Perdix perdix*) have been reported to be more susceptible to toxoplasmosis than other gallinaceous birds.<sup>104</sup>

Serological antibody titers have been reported (latex agglutination test) for infected birds, but have not been helpful. A single titer can rarely specifically diagnose the disease when the birds have been previously exposed.<sup>42</sup> Treatment of suspected cases and all in-contact birds with trimethoprim 0.08 g/ml H<sub>2</sub>O and sulfadiazine 0.04 g/ml in water for 3 weeks has been advised, but eradication from a captive flock may be difficult.<sup>122</sup>

### *Trichomonas gallinae*<sup>89</sup>

#### Leucocytozoonosis<sup>95</sup>

*Sarcocystis* spp. have been reported to cause toxoplasma-like infections of birds with a non-suppurative meningoencephalitis.<sup>49</sup> Infection has been reported in over 60 species of birds, with Old World psittacines apparently more susceptible (see Fig 17.25).<sup>15</sup>

This disease has been reported in 53 capercaillies (*Tetrao urogallus*) examined at necropsy in Sweden and one capercaillie from Finland.<sup>34,49</sup> Protozoa were mainly confined to the brain, but systemic disease is common.<sup>34</sup> Little is known about sarcocystosis of birds in general. In North America, *Sarcocystis falcatula* is the most pathogenic and well-defined species among birds, with schizonts persisting in the tissues for up to 5.5 months.<sup>15,49</sup> The predominant lesion in fatal *S. falcatula* infection is pneumonia, however, *Sarcocystis*-associated encephalitis has been described in a golden eagle (*Aquila chrysaetos*) and in a straw-necked ibis (*Carphibis spinicollis*) in the USA.<sup>35,49</sup>

#### Fungal Encephalitis

**Mucormycosis** is a rare infection in birds.<sup>84</sup> The order Mucorales includes a number of saprophytic fungi that have been mentioned as possible etiologic agents of meningoencephalitis in birds.<sup>84</sup>

#### Spongiform Encephalopathy

Three cases of a spongiform encephalopathy of unknown etiology have been reported in ostriches (*Struthio camelus*) from two zoos in northwestern Germany.<sup>63</sup> The birds were euthanized after showing progressive ataxia and uncoordinated feeding behavior. The lesions identified by light microscopy were similar to those of prion-induced transmissible encephalopathies in mammals and had gray matter vacuolation. However, a toxic or nutritional etiology could not be ruled out.<sup>63</sup>

### Idiopathic

#### Epilepsy

Intermittent seizures with no other abnormality can indicate idiopathic disease, especially if there is a long his-

tory of seizures.<sup>100</sup> In chickens, epilepsy has been found to have genetic basis.<sup>100</sup> Idiopathic epilepsy also may be acquired as a result of a cerebral insult and residual brain damage.<sup>100</sup>

### Toxicity

#### Lead<sup>100</sup>/Zinc<sup>73</sup>

Caged birds that eat their cage wire can become intoxicated if the wire is high in zinc content. A new wire fence syndrome has been reported in emus.<sup>11</sup> The birds gnaw away at their cage made of zinc material and eventually ingest so much that toxicity occurs. Some galvanized coatings contain as much as 99.9% zinc, but galvanized wire also may contain lead. The white rust associated with galvanized wire also is toxic.<sup>11</sup> Waterfowl may be exposed to elevated zinc concentrations through ingestion of contaminated vegetation and sediments due to the recently approved zinc-coated iron shot used for waterfowl hunting in the USA.<sup>73</sup>

Seizures can be a common clinical sign of zinc toxicity, in conjunction with paresis, polyuria/polydipsia, weight loss, anemia and gastrointestinal abnormalities. Sudden death has been reported in orange-bellied parrots (*Neophema chrysogaster*) due to zinc toxicoses, however, it was thought to be a result of the birds colliding with walls or structures in the aviary, causing head trauma.<sup>55</sup> Serum concentrations of zinc can be used to confirm the diagnosis and should be compared to normal birds as controls. Syringes with rubber stoppers should be avoided as this may be a source of zinc contamination.<sup>11</sup> In general, zinc levels greater than 2 ppm in emu are considered diagnostic for toxicity. Tissue levels can be evaluated postmortem in suspicious cases.<sup>55</sup> Published reference values of zinc levels in tissues of normal birds are as follows: macaws, 75 µg/g (tissue unspecified);<sup>55</sup> cockatiels (*Nymphicus hollandicus*), 59 µg/g in the kidneys, 72 µg/g in the liver and 94 µg/g in the pancreas;<sup>55</sup> goldeneye ducks (*Bucephala islandica*), 35.9 µg/g in the liver;<sup>55</sup> and peach-faced lovebirds (*Agapornis roseicollis*), 21 and 33 µg/g in the liver.<sup>55</sup> Treatment is discussed in Chapter 31, Implications of Toxins in Clinical Disorders.

#### Organophosphates, Carbamates and Pesticides

There are over 80 different registered organophosphates and carbamates in the USA, with both classes being acetylcholinesterase inhibitors that bind to and subsequently inactivate acetylcholinesterase, causing an accumulation of acetylcholine at the postsynaptic receptors.<sup>15</sup> Birds are 10 to 20 times more susceptible to these inhibitors than mammals. Acute intoxications can cause seizure activity. Exposure to carbamate insecticides can be detected using brain cholinesterase reactivation techniques.<sup>5,56,105</sup>



Johnathon Cracknell

**Fig 17.23** | A rose-crowned fruit dove (*Ptilinopus regina*) is examined after head trauma, demonstrating superficial skin lesions.

## Trauma

### Head Trauma (Fig 17.23)<sup>15,53</sup>

## Vascular

### Atherosclerosis

Atherosclerosis occurs with some degree of frequency in birds.<sup>15</sup> Often this can cause acute death, but can cause sudden onset blindness, ataxia, paresis and seizures.<sup>15</sup> Clinical signs may be from the cerebrovascular accidents, and MRI or CT may be useful in their diagnosis.<sup>15</sup> See Chapter 12, Evaluating and Treating the Cardiovascular System.

# Spinal and Neuromuscular Disease

## CLINICAL SIGNS (Table 17.5)

### Monoparesis/Monoplegia/Hemiparesis/Hemiplegia

#### Definitions and Classifications

Paresis indicates the presence of reduced motor function in the limbs. The suffix “-plegia” indicates the complete loss of motor function. The prefix mono- indicates the involvement of just one limb (Fig 17.24) and the prefix hemi- indicates the involvement of both limbs on one side of the body with normality on the contralateral side. Monoparesis sometimes may be expressed as a “clenched” claw (Fig 17.25).

#### Treatment

1. Specific treatment of the underlying etiology.
2. Skin care for recumbent birds or those that are dragging their limbs.
3. Physiotherapy.
4. Supportive care if unable to eat and drink.

## Tetraparesis/Paraparesis/Paralysis

### Definitions and Classifications

The prefix tetra- indicates the involvement of all four limbs. Tetraplegia is very rare due to the negative effect such a lesion would have on the respiratory capabilities of the patient. The prefix para- indicates the involvement of just the pelvic limbs, and paralysis or the equivalent paraplegia indicates complete loss of motor movement in the pelvic limbs (Fig 17.26).

#### Treatment

1. Specific treatment of the underlying etiology.
2. Skin care for recumbent birds or those that are dragging their limbs.
3. Physiotherapy.
4. Supportive care if unable to eat and drink.

## Ataxia

### Definitions and Classifications

Ataxia indicates an uncoordinated gait and does not necessitate an impairment of motor function, but there may be a concurrent paresis. Ataxia is often due to spinal disease, but may be due to either vestibular or cerebellar disease. There will be no motor dysfunction in the case of pure cerebellar ataxia.

**Table 17.5** | Spinal Lesion Localization and Clinical Signs<sup>93</sup>

Clinical Syndrome/ Lesion Localization	Characteristics of the Location	Specific Clinical Signs
Cranial cervical spine	Damage to cranial cervical spinal cord reflects damage to peripheral white matter pathways	i. No cranial nerve deficits ii. UMN <sup>a</sup> wing, leg and vent signs iii. Possible neck pain iv. Very rare loss of pain perception
Cervico- thoracic spine	Damage to the cervical intumescence produces clinical signs that reflect damage to the grey matter and brachial plexus <sup>b</sup>	i. No head signs ii. LMN <sup>c</sup> wing signs iii. UMN leg and vent signs unless brachial plexus lesion, only (ii)
Thoraco- lumbar spine	Damage to spine caudal to cervical intumescence <sup>d</sup> and cranial to lumbar intumescences reflects white matter lesions	i. No head or wing signs ii. UMN leg and vent signs iii. Possible kyphosis, spinal ± abdominal pain <sup>d</sup> iv. Variable loss of pain perception in limbs
Lumbosacral syndrome	A lesion in the lumbar intumescence <sup>e</sup> reflects damage to central gray matter and lumbosacral plexi <sup>f</sup>	i. No head or wing signs ii. LMN leg signs iii. LMN vent signs unless peripheral limb nerves only affected iv. Variable loss of sensation in legs and vent

a UMN = upper motor neuron. See Table 17.2.

b Brachial plexus includes the pectoral, medianoulnar, bicipital, ventral propatagial, axillary and radial peripheral nerves.

c LMN = lower motor neuron. See Table 17.2.

d A space-occupying mass within the kidney or pelvic canal may place pressure on lumbosacral plexus and its ischiatic nerve, mimicking lumbosacral syndrome.

e Lumbar intumescence encompasses lumbar, sacral and pudendal plexi.  
f Lumbosacral plexi includes the femoral, obturator, ischiatic, pudendal, pelvic and coccygeal peripheral nerves.



Greg J. Harrison

**Fig 17.24** | A palm warbler (*Dendroica palmarum*) with pesticide toxicosis demonstrating incoordination.



Greg J. Harrison

**Fig 17.25** | An African grey parrot (*Psittacus erithacus*) with suspected *Sarcocystis* infection of the central nervous system exhibits torticollis and bilateral clenched claws.



Greg J. Harrison

**Fig 17.26** | A cockatiel (*Nymphicus hollandicus*) with a spinal lesion of unknown etiology demonstrates a posture often seen with paraparesis.

**Treatment**

1. Specific treatment of the underlying etiology.
2. Skin care for recumbent birds or those that are dragging their limbs.
3. Physiotherapy.
4. Supportive care if unable to eat and drink.

**DIFFERENTIAL DIAGNOSIS OF SPINAL AND NEUROMUSCULAR DISEASE** (Table 17.6)

**Degenerative**

**Psittacine Focal Symmetrical Poliomyelomalacia**

This lesion has been documented in 15 adult birds in Australia, including five superb parrots, two budgerigars and one lovebird (*Agapornis* sp), all of which were from aviaries.<sup>53</sup> In caged birds, there was a history of one or more birds suddenly becoming uncoordinated, having difficulty perching, followed by posterior paralysis. Some birds could still fly. Others became tetraplegic within 2 or 3 days.<sup>53</sup>

**Table 17.6** | Differential Diagnosis of Spinal and Neuromuscular Disease

Mechanism of Disease	Specific Diseases
Degenerative	i. Psittacine focal symmetrical Poliomyelomalacia ii. Lysosomal storage disease - Gangliosidosis iii. Avian vacuolar myelinopathy iv. Lafora
Neoplastic	i. Abdominal neoplasia - ovarian pathology/renal masses
Nutritional	i. Vitamin E deficiency ii. Thiamine (vitamin B <sub>1</sub> ) deficiency iii. Riboflavin (vitamin B <sub>2</sub> ) deficiency iv. Pyridoxine (vitamin B <sub>6</sub> ) deficiency
Inflammatory	i. Viral - Proventricular dilatation disease/Marek's disease /Avian paramyxovirus type 1 (Newcastle disease)/Polioencephalomyelitis of rainbow lorikeets/Borna/Eastern and western equine encephalitis/West Nile virus/Picornavirus (duck viral hepatitis) ii. Fungal - <i>Aspergillus</i> spp.
Toxicity	i. Lead ii. Zinc iii. Tick Paralysis iv. Organophosphates, carbamates and insecticides v. Plant toxins vi. Botulism
Trauma	i. Peripheral nerve trauma ii. Spinal trauma
Vascular	i. Fibrocartilaginous embolic myelopathy

**Lysosomal Storage Disease**

**Gangliosidoses** are most commonly caused by deficient activity in one of the two enzymes necessary for ganglioside catabolism.<sup>18</sup> Specifically, an inherited defect in  $\beta$ -galactosidase is associated with GM<sub>1</sub> gangliosidosis and a similar defect affecting the activity of  $\beta$ -hexosaminidases is associated with GM<sub>2</sub> gangliosidosis.<sup>18</sup> The enzymatic defects lead to an accumulation of GM<sub>1</sub> or GM<sub>2</sub> respectively, within the neurons of the central and peripheral nervous system. Two related juvenile emus have been reported with severe, progressive, eventually fatal intraneuronal gangliosidosis.<sup>18</sup> Analysis of affected brain tissue demonstrated significant elevations in total ganglio-

sides with low levels of lymphocyte  $\beta$ -galactosidase.<sup>18</sup>

### Avian Vacuolar Myelinopathy (AVM)

AVM has been diagnosed in wild birds in the southeastern USA. It was first documented in bald eagles (*Haliaeetus leucocephalus*) in 1994 and also has been found in American coots (*Fulica americana*).<sup>115</sup> Bald eagles are frequently found dead, but have been noted to have difficulty flying and can crash into or overfly perches. Affected coots appear to fly or are wobbly in flight; they are uncoordinated on land and may swim in circles or on their backs.<sup>71</sup> On neurologic examination, the majority of coots have been documented with ataxia, decreased withdrawal reflexes, proprioceptive deficits and decreased vent responses.<sup>71</sup> Other signs seen in less than half of examined coots include beak and tongue weakness, head tremors, absent pupillary light responses, anisocoria, apparent blindness and nystagmus.<sup>71</sup>

Birds that die have no gross lesions of the nervous system, and some coots have been documented to recover from this disorder with supportive care.<sup>71</sup> Histologically, the disease is characterized by diffuse, spongy degeneration throughout the white matter of the CNS with the optic tectum most severely affected.<sup>71</sup> The etiology remains unknown.<sup>115</sup>

### Lafora's Disease

Lafora's disease is a presumed inherited defect of carbohydrate metabolism first documented in humans.<sup>103</sup> This disturbance of the carbohydrate metabolism causes the formation of typical Lafora-bodies, which consist of polysaccharide complexes. Similar changes have recently been described in two cockatiels (*Nymphicus hollandicus*).<sup>15</sup>

### Neoplasia

Primary spinal neoplasia is rare in birds. Compressive peripheral nerve trauma generally occurs secondary to an expanding mass that applies pressure to the nerve, because the pelvic nerves pass through the renal parenchyma.<sup>15</sup>

### Nutritional

#### Vitamin E/Selenium Deficiency

Clinical signs associated with vitamin E and selenium deficiencies include tremors, ataxia, incoordination, reluctance to walk and recumbency.<sup>15</sup> This deficiency has been incriminated as the etiology of cockatiel paralysis syndrome. This condition appears to occur most frequently in lutino cockatiels, but similar syndromes have been reported in a variety of other species including blue and gold macaws, eclectus parrots and African grey parrots.<sup>15</sup> 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.

#### Thiamine (Vitamin B<sub>1</sub>) Deficiency

Clinical signs of thiamine deficiency include ataxia, ascending paralysis and opisthotonus.<sup>15</sup> A response to treatment provides a presumptive diagnosis, as affected birds generally respond within hours of oral or parenteral administration of vitamin B<sub>1</sub>.<sup>15</sup>

#### Riboflavin (Vitamin B<sub>2</sub>) Deficiency

Birds with this deficiency have weakness, atrophy of the leg muscles and are seen to walk on their hocks with their toes curled inward, although this does not always happen because death may occur first.<sup>15,118</sup> The condition has occasionally occurred in chickens, but since riboflavin has been added to poultry feed it has become quite rare.<sup>118</sup> Leg and wing paralysis has been reported in racing pigeons with this disorder.<sup>118</sup> The deficiency causes a demyelinating peripheral neuritis. Treatment involves administration of oral or parenteral riboflavin and diet correction.

#### Pyridoxine (Vitamin B<sub>6</sub>) Deficiency

Deficiency of this vitamin causes characteristic jerky, nervous walking, progressing to running and flapping the wings.<sup>15</sup>

### Inflammatory

#### Viral Disease

**Proventricular Dilation Disease (PDD)** has been reported to cause peripheral (sciatic, brachial and vagal) neuritis in psittacine birds.<sup>17</sup>

**Marek's Disease Virus (MDV)** in chickens is caused by an oncogenic herpesvirus and is characterized by lymphomas and paralysis.<sup>43</sup> Paralysis associated with MDV infection has usually been attributed to peripheral nerve lesions because gross enlargement of nerves with lymphoid infiltrates is a common feature of MD, and CNS damage has not been found consistently in paralyzed birds.<sup>43</sup> The onset of both lymphomas and paralysis usually occurs 4 to 12 weeks after infection with MDV.<sup>43</sup> A paralytic syndrome involving the brain also is induced by MDV and is now designated transient paralysis (TP).<sup>43</sup> Clinically, TP is characterized by the development of a flaccid paralysis 8 to 12 days after infection with an oncogenic MDV. This initially affects neck muscles and later tends to become generalized.<sup>43</sup> Most birds with TP recover completely within 24 to 48 hours, although a few birds that become severely affected may die within the same period of time.<sup>43</sup> A more acute and fatal form of TP has recently been described in young chickens.<sup>123</sup>

Once known as fowl paralysis and range paralysis, it was considered that gross enlargements of the peripheral nerves were a pathognomic lesion.<sup>123</sup> However, gross enlargement of peripheral nerves also can be induced by reticuloendotheliosis virus (REV).<sup>7</sup> Both REV and

subgroup J avian leucosis virus can induce lymphoid infiltrations in peripheral nerves, indicating that additional criteria may be needed for confirmation of etiology.<sup>7</sup>

**Avian Paramyxovirus Type 1 (Newcastle Disease)** is a variable disease with different susceptibilities between avian orders.<sup>8</sup> The disease is produced following infection and varies with virus pathotype; other factors include the species, age, immune status, general health of the bird and environmental conditions.<sup>8</sup> Clinical neurologic findings in affected birds include ataxia, torticollis, opisthotonus, head shaking and tremors, head tilt, leg paralysis progressing to lateral recumbency and blindness.<sup>8</sup> Some birds may survive for up to 18 months after developing CNS signs, but the CNS signs do not regress before death or euthanasia.<sup>8</sup> Definitive diagnosis requires virus isolation, demonstration of the viral antigen, or rising specific antibody titers.<sup>8</sup> A titer of 1:8 is usually considered evidence of exposure to this virus, and in unvaccinated birds when coupled with clinical signs is considered strong diagnostic evidence of infection.<sup>8</sup>

**Suspected Viral Polioencephalomyelitis of Rainbow Lorikeets (*Trichoglossus haematodus*)** has been documented in 35 rainbow lorikeets in Australia.<sup>53</sup> It affects adults at any time of the year and usually causes an inability to fly or perch. The birds are commonly alert and will eat, but are characteristically affected with clenched claws.<sup>53</sup> The cerebellum was affected in about half of the birds, and the lesions were largely restricted to the central cerebellar white matter and involved the roof nuclei.

**Borna Disease Virus** has been documented to cause a paretic condition in young ostriches in Israel.<sup>4</sup> Birds between 14 and 42 days old were affected. In a small number of cases, clinical signs of incoordination were seen 1 to 3 days before paresis supervened. At this stage of the disease, the birds could stand and move with difficulty if given external support.<sup>4</sup> Mortality can be remarkably high. About 1% of the ostriches seemed to recover if they were given intensive supportive care, however, they relapsed several months later.<sup>4</sup>

**Eastern and Western Equine Encephalitis** infections can cause weakness and paralysis of the legs.<sup>96</sup>

**Picornavirus (Duck Viral Hepatitis)** has been associated with neurologic signs in Galliformes, Anseriformes and Columbiformes of less than 28 days of age.<sup>15</sup> Clinical signs documented include depression, ataxia, paresis or paralysis, and severe but fine head and neck tremors.

**West Nile Virus** has been documented to cause paresis and paralysis in many avian species.<sup>78</sup>

## Fungal Disease<sup>21,22,46,102</sup>

### Toxicity

#### Lead

Lead toxicity, one of the most commonly recognized poisonings of companion and free-ranging birds, is a common cause of neurologic abnormalities and can be fatal.<sup>92</sup> Peripheral neuropathies caused by lead intoxication have been reported, but it is more common to see central nervous system abnormalities.<sup>92</sup> Various sources of lead have been documented, but the source of lead in any specific poisoning often remains difficult to determine. A classic source of lead exposure for birds is ingesting lead shot from the bottom of lakes in heavily hunted areas.<sup>92</sup> The combination of the grinding action of the ventriculus and its low pH (2.0-3.5) acts to solubilize ingested shot.<sup>92</sup> Raptors may eat carcasses containing lead shot and become intoxicated.<sup>92</sup> Inhalation of fumes from leaded gasoline is another possible route of exposure.<sup>92</sup> Lead poisoning as a result of lead shot embedded in soft tissues is rare because of the avascular fibrous tissue that develops around these foreign bodies.<sup>92</sup>

Clinically, lead toxicosis may be an acute or chronic problem, with clinical signs dependent on the amount and surface area of lead ingested.<sup>92</sup> Clinical signs in birds include behavioral changes, lethargy, anorexia, vomiting, diarrhea, ataxia, limb paresis or paralysis, seizures, anemia and emaciation.<sup>92</sup> Death may occur within 48 hours after the first appearance of clinical signs.<sup>92</sup>

Whole, unclotted blood is the sample of choice for determining lead concentrations because 90% of circulating lead is contained within RBCs.<sup>92</sup> Concentrations of >20 µg/dl lead in whole blood are suggestive of lead intoxication in psittacine birds, and concentrations >40 to 60 µg/dl are diagnostic of lead toxicosis.<sup>92</sup>

#### Zinc<sup>73</sup>

See Intracranial Disease above.

#### Tick Paralysis

Tick paralysis is a disease caused by neurotoxins associated with 60 species of hard and soft ticks in 10 genera.<sup>76</sup> Generally, the disease is considered a motor polyneuropathy characterized by a progressive, ascending, flaccid motor paralysis. Clinical signs include ataxia, paresis, paralysis, areflexia, hypotonus and respiratory failure.<sup>76</sup> Death is common if the tick is not removed.<sup>76</sup> The toxin is associated with the female tick's salivary glands and is probably secreted during feeding.<sup>76</sup> *Ixodes brunneus* has been associated with tick paralysis in birds.<sup>76</sup> All three stages of the tick feed exclusively on birds, particularly passerines and group-feeding species; at least 64 species of birds are known to be hosts for this tick.<sup>76</sup> Adult females are found on adult birds primarily in the colder

months of the year, with larvae and nymphs occasionally present at the same time, causing clinical signs during winter months.<sup>76</sup>

A definitive diagnosis of tick paralysis can be based only on dramatic clinical improvement and recovery of the host, usually within 24 to 72 hours following removal of the tick.

### Organophosphates and Carbamates

Organophosphorus-induced delayed neurotoxicity (OPIDN) is a neurologic condition characterized clinically by the delayed onset of a progressively developing hindlimb ataxia and paralysis.<sup>90,116</sup> Two categories of OPIDN — Type I and Type II — have been identified,<sup>116</sup> and differ from each other in the length of the delay period prior to onset of symptoms, the type of resultant clinical signs and the extent of central nervous system involvement. Type I OPIDN has a longer delay period (typically 10-21 days) and affects only the spinal cord and brainstem, whereas type II OPIDN has a shorter delay period (4-7 days) and results in additional degeneration in the midbrain and forebrain. Widespread neuropathology follows both oral and injectable forms of the compounds.<sup>116</sup>

### Plant Toxins

*Nerium oleander*<sup>2</sup> (See Chapter 31, Implications of Toxic Substances in Clinical Disorders.

### Botulism (Limberneck)

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.<sup>15</sup> This is an uncommon problem in companion birds but frequent in waterfowl.<sup>15,45,70</sup> The classic sign is limberneck resulting

from paralysis of the cervical muscles.<sup>15</sup> Most birds exhibit hindlimb paresis first, which progresses to paralysis of the wings, followed by loss of control of the neck and head in the terminal stages.<sup>15</sup> Species' susceptibility and clinical manifestations vary, as has been evidenced in rehabilitation facility outbreaks.

## Trauma

### Spine Trauma/Peripheral Nerve Trauma

Trauma is fairly common in free-ranging as well as companion and aviary birds. The consequences, diagnosis and management of these traumas have been well discussed in other texts.<sup>15</sup>

## Vascular

### Fibrocartilaginous Embolization

Emboli may involve vessels of the spinal cord, leptomeninges or both. Fibrocartilaginous embolism (FCE) and ischemic myelopathy have been reported in several 15-week-old tom turkeys with peracute onsets of paresis and ataxia.<sup>111</sup> Recovery was noted in some affected birds suspected to have this disease, but cartilaginous emboli were found in the spinal cord vasculature accompanying myelomalacia in three turkeys that did not recover.<sup>111</sup> The articular cartilage of the vertebral body endplates are suggested to be the source of the emboli, as there were articular cartilage defects found in the affected birds. There is no known treatment in any species and diagnosis is by exclusion. Recovery is possible in some cases, but supportive care would be necessary.

## Products Mentioned in the Text

- a. Lanex fine screens/TML film, Kodak, Rochester, NY
- b. Omnipaque 240, Winthrop Pharmaceuticals, New York, NY
- c. Magnevist, Berlix Laboratories, Wayne, NJ

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