

Introduction

The number of publications describing the avian nervous system and its associated disorders although limited, is increasing. There is enhanced awareness that neurological signs can be present due to primary neurological disorders, or as sequelae of systemic conditions. This review aims to provide the practitioner with a brief outline of avian neuroanatomy and a practical approach to the neurological examination of the bird, thus providing a starting point for the diagnosis, prognosis and treatment of neurological disease in avian practice.

Neuroanatomy

The avian nervous system can be divided into the central nervous system which comprises the brain and spinal cord and the peripheral nervous system which includes the cranial and spinal nerves, their ganglia and plexi and the peripheral portions of the autonomic nervous system.

The Central Nervous System (CNS)

The avian brain is lissencephalic (lacks convolutions), fills the skull completely and is easily damaged, especially in cockatiels and budgerigars due to the thinness of the roof of the skull (Shivaprasad 1993). The brain narrows rostrad to the small underdeveloped olfactory lobes (Cooper & Clay 1979), but widens caudally to the cerebral hemispheres. The cerebral cortex is underdeveloped, being two to three cells thick in the budgerigar. The characteristic features of the avian brain are the large corpora striata of the forebrain, the large optic lobes of the midbrain and the large median cerebellum of the hindbrain.

In the forebrain (telencephalon) the lateral ventricles are displaced dorsally by large corpora striata that are the main centres for senso-motor correlation in birds and are well developed in comparison to mammals. Consequently, instincts dominate avian behaviour (Portmann & Stingelin 1961).

In the midbrain (mesencephalon) the optic lobes are displaced ventro-laterally by the large corpora striata and median cerebellum, they are massive and responsible for the well developed optico-sensory system of birds. The midbrain also contains the oculomotor centre and connections necessary for transmitting impulses to muscles involved in locomotion and flight. The torus semicircularis is the region involved in the production of bird song (Brown 1969, 1971; Newman 1972; Potash 1970). The interbrain (diencephalon) is small in birds and consists of the pineal (epiphysis) dorsally, the thalamus centrally and the hypothalamus and pituitary (hypophysis) ventrally. The pineal is involved in reproduction, migration and circadian rhythms and is composed of secretory cells which resemble rudimentary photoreceptors. Its secretions exert their effects on the hypothalamus. The pituitary gland is relatively large in the budgerigar and is located ventrally between the large optic chiasma and the medulla. It consists of a large adenohypophysis and a small neurohypophysis. The intermediate lobe is absent.

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In the hindbrain (metencephalon) the cerebellum lacks the lateral lobes of the mammalian brain and functions to regulate movements to maintain spatial orientation. The medulla contains the reticular formation and several reflex centres necessary for maintaining life, such as cardiovascular and respiratory centres. Cranial nerves V through to XII (excepting most of the fibres of XI) originate on the ventral surface of the medulla. The pons is poorly developed and there is no pyramid as in mammals.

The Spinal Cord

The avian spinal cord is an extension of the medulla, extending throughout the entire length of the spinal canal as no cauda equina or pronounced filum terminale is evident. The cervical, thoracic and lumbosacral spinal cord regions correspond to those areas of the vertebral canal. The avian caudal vertebral canal contains the coccygeal region of the spinal cord. The cervical and lumbosacral areas contain many neurones in their grey matter and hence are enlarged. These neurones give rise to axons that form the brachial and lumbosacral plexi that supply the limbs. Histologically, the avian cord resembles that of mammals with the sensory fibres passing into the dorsal horn and the motor fibres arising in the ventral horn (Ariens-Kappers *et al* 1936; Pearson 1972). The glycogen body is a formation of astrocytes that stores glycogen. It is located in a cleft in the dorsal midline of the lumbosacral cord (Lowenstine 1987). Its function is unknown. It is large in the developing embryo and persists in varying degrees in adults in some species.

The Peripheral Nervous System (PNS)

Birds, like mammals, possess twelve pairs of cranial nerves that can be divided anatomically and functionally into three groups (Bubien-Waluszewska 1979).

Group one consists of cranial nerves that are entirely sensory such as olfactory I, optic II and vestibulocochlear VIII.

Group two consists of the cranial nerves that are entirely motor such as oculomotor III, trochlear IV, abducent VI and hypoglossal XII.

Group 3 consists of cranial nerves that are mixed such as trigeminal V, facial VII, glossopharyngeal IX, vagus X and part of the accessory XI.

Each vertebral segment has a pair of spinal nerves which exit from the intervertebral foramen. The spinal nerves are numbered by the vertebra caudal to them, in numerical order. Since birds of different species have different numbers of vertebrae, the number of cervical spinal nerves varies among species being 14 in the pigeon (Huber 1936) and 12 in the budgerigar (Evans 1969). The fusion of the last few thoracic vertebrae with the lumbar, sacral and first few caudal vertebrae to form a rigid synsacrum, results in a loss of epaxial musculature and diminution of the dorsal branches of the spinal nerves. The ventral branches remain large and coast above or through the kidneys, supplying the body wall, pelvic limbs and tail. Three nerve plexi are distinguishable topographically: the crural, ischiatic and pudendal. The autonomic nervous system is similar to that of mammals.

The Neurological Examination

When performing a neurological examination on a bird, the main aim is to determine if the neuropathy is focal or diffuse. If focal, an attempt is then made to localise the lesion to the head, cervical, thoracolumbar or lumbosacral spinal cord. If the examination fails to localise the lesion, a metabolic or generalised neuromuscular lesion should be considered. Lesions causing neurological deficits can

be localised to peripheral nerves, their corresponding spinal cord segments or sites rostral in the central nervous system. If the examination implies dysfunction at more than one level, the lesion is assumed to be located at the higher location or to be multifocal.

The examination should be performed methodically and logically. The order of the neurological examination depends on the clinical condition and cooperation of the patient. The degree of cooperation of the patient should be considered when interpreting responses to neurological tests. Awareness of potential injury sustainable from an uncooperative patient should be exercised at all times. Practicing the skills required to perform a neurological examination on normal birds will facilitate the interpretation of the examination on abnormal birds. Diagnostic tests that may be of value to support neurological diagnoses and prognoses are haematology and biochemistry, toxin analysis and radiography. If necessary, cases requiring advanced neurological diagnostic tests such as electroencephalography, electromyography, computer tomography and magnetic resonance imaging may be considered for referral.

A. **History**

A detailed history is crucial to a thorough neurological examination. Neuropathies are particularly common secondary to malnutrition, trauma or toxicity. Ascertain the bird's age as hereditary and inflammatory conditions are more likely in the young bird while degenerative and neoplastic conditions more likely in the older bird. Chronological development of the clinical signs can be very informative for example, posterior paresis followed by forelimb involvement suggest ascending myelitis. Conditions associated with acute onset include toxicity, trauma, vascular accident or fulminating inflammation while degenerative, neoplastic or low grade inflammation are more likely chronic. Degenerative changes for example may be indicative of a progressive condition, a post traumatic event of a static condition and an intervertebral disc displacement of an intermittent condition.

Information on lineage may suggest a hereditary disorder. Details on the bird's mental state and demeanour may be useful. Questions should be asked relating to its normal "personality", recent aggressiveness, disorientation or inappetence for example.

Seizures may be difficult to differentiate from syncope. Generally, seizures are characterised by ataxia, disorientation and falling off the perch, remaining rigid or having some form of motor activity for varying lengths of time. Both seizures and syncope may indicate an intracranial lesion.

B. **Distant Examination**

Observation of the bird's mental status, flight, posture, gait and behaviour is a non invasive means of acquiring information. See Table 1.

C. **Physical Examination**

Evaluate by palpation, the tone of pectoral and hind limb musculature. The presence of muscle atrophy is suggestive of reduced innervation to that musculature. Palpate the skeletal system for evidence of crepitus or deviations.

D. **Cranial Nerves**

Tests exist to evaluate normality and abnormality. Often other neurological lesions/conditions

should be considered as well as cranial nerve dysfunction. See Table 2.

E. Spinal Reflexes

Spinal reflexes, although difficult to assess objectively in birds, can help determine if a lesion is centrally or peripherally located. In most situations determining if a reflex is present or absent is sufficient and symmetry of response should receive particular attention. Spinal reflexes require reflex arcs only to be intact, no other parts of the central nervous system are required. See Table 3.

F. Reactions

These require reflex pathways, ascending and descending fibre tracts in the spinal cord and higher centres. Once again look closely for subtle asymmetrical deficits. It is important that the wings are tested when they are both free (if possible) and also if one, then the other is restrained. Comparison of spinal reflexes and reactions helps to localise lesions. If a reflex exists, but its corresponding reaction does not, a lesion exists within the central nervous system rostral to the segment involved in the reflex arc. See Table 4.

Alternative Approach to Localise Lesions

Lyman (1986) grouped neurological findings according to the areas they represent.

Head signs are loss of cerebral, cerebellar, brain stem or cranial nerve functions. Reece *et al* (1986) describe cerebellar defects in parrots. Dooling *et al* (1987) describe effect of deafness on the contact call of the budgerigar. Jacobsen *et al* (1984) describe a cockatiel suffering Sarcocystis encephalitis that was unable to shell its seeds. Myers *et al* (1983) describe a cockatiel with cerebrospinal nematodiasis and torticollis, opisthotonus, ataxia and trembling.

Wing signs are asymmetrical wing carriage without fracture, atrophy of the proximal musculature, decreased wing strength or inability to fly. Hayes *et al* (1992) described drooping wings in wild turkeys with reticuloendotheliosis. Shell *et al* (1993) used electromyographic studies to evaluate brachial plexus injury in red-tailed hawks that showed noticeable wing drooping.

Leg signs are weak gait, absence or reduction of pain perception, inability to grasp, proximal muscle atrophy, knuckling of a digit or contraction of a limb. Blue-McLendon *et al* (1992) describe the cerebrospinal nematodiasis causing abnormal gait in emus. Clubb (1992) explains that aging macaws demonstrate muscle wasting and degenerative neurological disease. McOrist and Perry (1986) describe leg paralysis in free living rainbow lorikeets. Greenacre *et al* (1992) describe leg paresis in a palm cockatoo caused by aspergillosis.

Vent signs are absent or weakened vent reflex, vent sphincter hypertonia, incontinence or soiling of the vent. Sammartino (1933) extirpated the entire lumbar cord of pigeons and reported total loss of cloacal sphincter tone.

Cervical spinal cord lesions should be suspected when wing, leg and vent signs are observed, and head function and cranial nerves appear normal. Wing, leg and vent spinal reflexes are normal. Thoracolumbar spinal cord lesions will cause vent and leg signs, without affecting the head, cranial nerves or wings, while lumbosacral spinal cord lesions are indicated by vent signs with lack of head, wing or leg signs.

Table 1: Distant Examination

		Clinical Signs	Site: Lesion/Condition
Mental Status	Aggressiveness		Brain: Psychomotor like epilepsy, space occupying mass in forebrain
	Depressed	↓ response to stimuli	Brain stem, forebrain
	Stuporous	Partial consciousness	Brain: Acute encephalitis, forebrain trauma or neoplasia
Flight	Uncoordinated flight	non- rhythmic fanning of wings	Cerebellum, vestibular system, <i>corpora striata</i>
	↓ Obstacle avoidance	Collision with objects	Eyes, CN II, visual centre in brain: neoplasm, abscess, granuloma
	Poor take off/landing	Uncoordinated launch/ landing	Cerebellum, vestibular system, visual system, spinal cord, peripheral nerve
	Paresis	Flight not sustainable	Spinal cord, peripheral nerve. Consider systemic disorder
Posture	Head tilt	Rotation/deviation of head	CN VIII
	Tremor	Continuous twitching	Cerebellum, vestibular system
	Falling	Loss of balance	Vestibular system
Gait	Ataxia	Muscular incoordination	Cerebellum, vestibular system
	Dysmetria	Inability to control or limit movement	Cerebellum
	Circling	Persistent walking in circles	Unilateral vestibular system, <i>corpora striata</i>
	↓ obstacle avoidance	Collisions with objects	Eyes, CN II, visual centre
	↓ righting reflex	↓ correction of posture after displacement	Vestibular system, corpora striata, visual or proprioceptive systems, cerebellum, spinal cord, peripheral nerve
Behaviour	↓ prehension	Abnormal tongue movement, reduced beak strength	CN V, IX- XII
	↓ perching ability	↓ ability to grasp perch and support weight	Peripheral receptors, corpora striata, vestibular or visual system, cerebellum, spinal cord, peripheral nerve

Table 2: Cranial Nerves

Test that indicates normality	Description
Menace: CN II & VII	Eye-blink, pulling away of head or aggressive action of beak can be provoked by bringing the hand towards each eye.
Pupillary light: CN II & III	The avian pupil's movements are poor in response to light, but rapid in response to accommodation or voluntary control (Gelatt, 1991). No consensual pupillary light response as there is complete decussation of the optic nerves at the chiasm (Kern <i>et al</i> , 1984).
Pupil size: CN III	Pupils should be symmetrical. If not, also consider intra-ocular inflammation, ocular structural lesions or sympathetic neuropathy
Corneal: CN V & VII	A symmetrical eye blink normally occurs when the cornea is gently touched with a moist cotton swab
Smell: CN I	Reaction to noxious odours, appetite/ feeding response
Facial sensation, Palpebral response: CN V & VII	Symmetrical eye-blink should be elicited by touching each side of face, lateral canthus
Doll's eye CN VIII (bilateral damage)	Symmetrical deviations of the eyes when the head is moved in different positions, always returning to centre Also consider brain stem (especially pons and midbrain) lesion.
Fundus CN II	The pupil must be dilated in order to examine the fundus. The fundus is more developed in diurnal species (Gelatt, 1991). Parasympatholytic drugs have no appreciable affect on the iris musculature in birds. Short acting combinations of ketamine hydrochloride and xylazine has been used successfully in raptors for mydriasis (Greenwood and Barnett, 1981). Topical application of d-Tubocurarine produces variable effects (Kern <i>et al</i> , 1984). Aqueous d-tubocurarine is not effective (Bellhorn, 1973).
Nystagmus CN VIII	Periodic, rhythmic, involuntary movement of both eyeballs in unison. Slow component in one direction and quick return. May be vertical, horizontal or rotary. In horizontal nystagmus the fast component opposes the side of the lesion. Also consider cerebellar lesion or increased intracranial pressure.
Strabismus: CN III, IV or VI (Lyman, 1986)	Involuntary deviation of the eye. Also consider vestibular lesion.
Dysphagia: CN IX - XII	Abnormal tongue movement. A deviated tongue opposes the side of the lesion
Horner's syndrome	Enophthalmos, upper eyelid ptosis, slight elevation of the lower lid, pupil constriction and narrowing of the palpebral fissure. Consider an intracranial lesion or one affecting the cervical sympathetic tract or brachial plexus (Kern <i>et al</i> , 1984)
Reduced Beak strength: CN V, IX to XII	Reduced beak strength when biting or eating
Torticollis: CN XI	Cervical muscle contraction producing neck torsion

Table 3: Spinal Reflexes

Body Balancing	With wings held in to body, suspend the bird vertically, head down, then quickly rotate up to horizontal position and observe fanning of the tail feathers. Dip the bird forward back to ventral position and observe tail flick up. This reflex is present in pigeons with transected spinal cords and functions to maintain balance (Bolton, 1976).
Wing Withdrawal	Lightly touch a wing and observe it being pulled away. A segmental reflex, if present indicates that the reflex arc and associated spinal cord segment in the cervicothoracic cord is intact. Damage may still exist higher in the central nervous system.
Leg Withdrawal	Lightly touch a leg and observe it being pulled away. A segmental reflex, if present indicates that the reflex arc and associated segment in the thoracolumbar spinal cord is intact (Lyman, 1986).
Vent Reflex	Touch the vent mucosa with a fine object and observe it close tightly. A segmental reflex, if present indicates that the reflex arc and associated lumbosacral spinal cord segment is intact.

Table 4: Reactions

Proprioception: wings	Note the resting wing carriage. Pull the wing out of its resting position and note the time taken for its return. Only normally innervated wings will correct displacement.
Unilateral/ Bilateral wing fanning	Unilateral and bilateral wing fanning should be assessed whilst the bird is moved up, down and from side to side (feet and lower back restrained). Forceful, rhythmic, fanning out of the wings should be evident and in the bilateral test wing movements should be simultaneous. Unilateral wing fanning will occur only in the normal innervated wing, while in the bilateral test, a lesion causing loss of afferent stimulus on one side will be compensated for by afferent stimulus from the other side and normal wing movements will occur (Trendelenberg, 1910).
Proprioception: legs	Observe bird at rest. Knuckling of the foot is quickly corrected in normal birds.
Placing	With wings held into body and legs free, approach a horizontal surface such as a desk top and as soon as any part of the foot touches the surface, the feet should swiftly position themselves accurately on the surface, to support the bird's weight.
Pain perception: wings and legs	The differentiation between pain perception and withdrawal reflex is critical. Movement does not indicate that the patient is able to feel the stimulus. Some type of conscious recognition of the stimulus is required eg. vocalisation, attempts to escape or bite. This part of the examination is best kept till last, so as not to influence the patient's behaviour to other segments of the neurological examination. Loss of pain perception is a poor prognostic sign. Deficits in a particular wing may only be obviously detected if its counterpart is restrained (Ten Cate, 1936; 1937).

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