A Review of Some Neurologic and Muscular Disorders in Free and Captive Non-domestic Avian Species in Australia

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Introduction

At July 1995 the Taronga Pathology Collection (TPC) has materials from over 7,000 cases on file, of which 2,600 are from non-domestic birds and most of these are of Australian native species. In the TPC are 320 cases with neurologic changes and 110 with muscle changes. About half of these submissions to the TPC came through the Taronga Zoo Wildlife Clinic from free flying birds in the Sydney environment. Others came from various diagnostic laboratories throughout Australia.

This contribution is an adjunct to the excellent review of avian neurologic examination given at the last annual conference of the Australian Association of Avian Veterinarians (Parker, 1994). The pathology of some of the neurologic entities in the TPC has recently been reviewed (Hartley & Reece, 1995).

Clinical Signs

Usually, a very inadequate history was available from neurologic and muscular disorders in submissions from free flying birds. Some birds were found on the ground, apparently unconscious, others were very dejected, others were fluttering around, unable to fly or perch, but bright at the head end and often affected with the clenched claw syndrome. Others showed variable degrees of flaccid paralysis and others convulsions.

On the other hand, in caged birds, signs were usually detected earlier and these included incoordination, dysmetria, tremors, circling, and difficulty in flying or perching, in addition to the signs previously mentioned.

It is important to try and ascertain whether the signs shown are due to central or peripheral nervous system involvement or whether due to muscle involvement. Prognosis is usually bad in the former and usually more favourable in the latter with relevant treatment and hospitalisation and if the birds will feed.

Pathology

For ease of description, entities have been categorised below into those with primarily neurologic lesions and those primarily with muscle lesions. The cause of some conditions is known, whereas in others, it remains to be elucidated.

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1. Neurologic Diseases

a) *Congenital abnormalities*

The only cases on file involved the cerebellum, where there was variable atrophy. This was seen in clutches of double-eyed fig parrots, peach-face love birds and one turquoise parrot. Affected birds at around four weeks of age were ataxic, had whole body tremor and were later unable to fly or perch. Microscopically, lesions ranged from loss of Purkinje cells with or without loss of granule cells and a thin molecular cell layer. These entities are probably inherited.

b) *Trauma and suspected trauma*

Lesions due to this were recognised in 30 cases in the TPC, most of which were seen in free flying birds. In some cases there was gross haemorrhage over the cerebrum and cerebellum or around the spinal cord, sometimes with haemorrhage into the brain or cord parenchyma. Often there was associated subcutaneous haemorrhage and muscle haemorrhage and in a few cases obvious vertebral fractures. In rainbow lorikeets the most common site for trauma was the thoracic spinal cord. Microscopic changes ranged from recent meningeal haemorrhage, often with brain and cord haemorrhagic necrosis to, in more chronic cases, malacic foci with phagocytic response, which in a few cases progressed to cerebral cavitation. Several others showed evidence of Wallerian degeneration of spinal cord white matter.

c) *Spongy vacuolation of the Cerebellum*

This entity has been seen in seven adult birds from six different species and included three species of Psittacines, all of which were caged. Clinical signs which included ataxia, tremors and head tilt had been present for several weeks or months. Microscopically the spongy vacuolation was largely confined to the central cerebellar white matter. The vacuoles were empty but may once have been intramyelinic oedema with loss of axons. The cause of this entity in these birds is unknown, but similar lesions are seen in inherited conditions in association with chronic liver lesions and in several toxicities.

d) *Psittacine focal symmetrical poliomalacia*

Fifteen cases of this unusual entity are on file in the TPC, all of which were Psittacines. It involved eight caged birds, including five superb parrots and six free flying rainbow lorikeets and one free flying scaly-breasted lorikeet from the Sydney area. In the caged birds, often several were affected about the same time with incoordination, difficulty in flying and perching, quickly followed in a few days by quadriplegia. The free flying lorikeets were found on the ground with clenched claws. In all cases there were similar lesions in the spinal cord which were mainly confined to the lumbosacral and cervicothoracic segments. These lesions consisted of variable focal to virtual complete symmetrical non-inflammatory necrosis of the grey matter, particularly involving the areas containing the motor neurones. Consequent to the loss of the latter nerve cell bodies there was extensive Wallerian degeneration of the motor nerves to fore and hind limbs with gradual neurogenic muscle atrophy. In some of the rainbow lorikeets there were similar malacic foci in the optic lobes. The cause of these entities is a mystery and it is not known whether the same cause is responsible in both caged and free flying birds. Similar lesions have been seen in piglets in association with selenium toxicity and with nicotinamide deficiency.

e) *Ataxia in emu chicks*

A syndrome characterised by ataxia, dysmetria, tremors and prostration has been encountered in two to five month old birds at Healesville Sanctuary and on several commercial emu farms in Victoria, Western Australia and elsewhere. Two affected birds at Healesville has moderately severe lesions in the cerebellum of progressive necrosis and loss of Purkinje cells in several folia (no spinal cords were available). Materials from the commercial farms had equivocal loss of Purkinje cells, but had mild to moderate vacuolation in the spinal cord white matter. The latter

appeared to start as segmental intramyelinic oedema which progressed to Wallerian degeneration. There was also active Wallerian degeneration, empty neurilemmal sheaths in some peripheral nerve fascicles. There was also equivocal older bilaterally symmetrical demyelination in the dorsolateral sensory tracts in the spinal cord. Possible causes include genetic, copper deficiency and coccidiostat toxicity.

f) *Suspect viral polioencephalomyelitis of free flying rainbow lorikeets*
This is the most commonly encountered neurologic entity seen in the Sydney area and, in some seasons, is responsible for many deaths. What is probably the same entity has been seen recently in free flying rainbow lorikeets at the Currumbin Sanctuary. Nothing comparable has been seen in any other local avian species. It was seen at any time of the year in subadult and adult birds. They were found unable to fly and had the clenched claw syndrome, but will eat and may survive for weeks or longer. Microscopically, the lesions were mainly seen in the spinal cord and usually were most severe in the lumbar region. They consisted of moderate to severe small mononuclear inflammation affecting particularly the ventral horn grey matter, in association with necrosis and loss of motor neurones and sometimes also spongy vacuolation. As a result of these lesions, there was an associated Wallerian degeneration of the motor nerves and gradually neurogenic atrophy of supplied muscles. Sometimes similar inflammatory lesions were also seen in the central cerebellar white matter, posterior brain stem and peripheral nerves.

g) *Miscellaneous inflammatory lesions in the Central Nervous System*
Isolated cases of bacterial and fungal encephalitis and/or myelitis have been recognised, also three cases of Toxoplasma encephalitis. Two cases of presumed Haematozoan schizogony and associated vasculitis are on file and one of these, a little penguin had small malacic lesions.

h) *Neurologic signs with no observable lesions*
Botulism: This entity may occur in summer months in outbreak form, particularly in water birds. The history and signs of flaccid paralysis in the absence of any neurologic lesions, is strongly indicative of this intoxication.

Convulsions in tawny frogmouths: This entity has occurred in small numbers, most winters, but in 1994 there were heavy losses, at least along the eastern parts of New South Wales. Birds were often found on the ground, disorientated, with increasing convulsions and death. The cause is thought to be due to organochlorine toxicity.

2. **Muscular Diseases**

These may be secondary to lesions in the central or peripheral nervous systems leading to neurogenic atrophy or may be primary to a variety of causes.

a) *Trauma*
This may result from cat attack or from flying into objects. In the former there was often puncture wounds to the skin and muscles with extensive haemorrhages. Some of these birds with treatment survived, unless secondary infection occurred. In birds that flew into objects, in addition to muscle damage there was often spinal cord damage and occasionally fractured vertebrae. Recent lesions consisted of exclusive intramuscular haemorrhage with early myonecrosis often with oedema. Older lesions were seen as myonecrosis with macrophage response and some haemorrhage.

b) *Suspect Haemoproteus myopathy of pied currawongs*
Around 30 cases of this entity are in the TPC and all have occurred in the Sydney basin. It was seen mainly in subadult birds in their first summer and affected birds were usually found fluttering on the ground, unable to fly or perch and some had clenched claws. Affected birds on

hospitalisation and if they will feed may make a complete clinical recovery in three weeks or so and fly away. The lesions were readily seen and palpated in the live bird under the skin in the breast muscles. These consisted of hundreds of slightly raised white foci, up to 1 cm long and 0.5 cm wide, often with a central grey streak. They were located throughout the body in skeletal musculature usually with lesser numbers in cardiac and gizzard musculature. Microscopically the lesions consisted of a row of up to 30 protozoan megaloschizonts in varying stages of maturity, each up to 250 microns in diameter. These were surrounded in early cases by a broad zone of myonecrosis and in those birds that survived longer, a macrophage response. At maturity the schizonts collapsed liberating the contained thousands of zoites - which then invaded erythrocytes to form the sexual cycle. The latter stages were first seen as ring forms, which with increasing length of survival of the bird, developed into sausage-shaped pigmented gamonts, characteristic of a *Haemoproteus* spp. Lesions in gizzard and cardiac muscle were similar, but the surrounding reaction was much less severe.

c) *Suspect Haematozoan myopathy of rainbow and scaly-breasted lorikeets*
This entity has recently been recognised in one of each of these birds, and suspected in others, at the Currumbin Sanctuary near Brisbane. Almost identical lesions to those seen in pied currawongs were seen in skeletal and cardiac musculature in association with protozoan megaloschizonts in varying stages of maturity. As no bloods have been available from affected birds, it is not known what presumed Haematozoan may be involved.

d) *Nutritional myopathy of free living lorikeets*
This entity was reported at the last AAV meeting (Wilson, 1994) from south-eastern Queensland in free flying young rainbow and scaly-breasted lorikeets. There were gross focal lesions in skeletal muscle and myocardium of myonecrosis and inflammatory response, suspected of being associated with a nutritional deficiency, possibly Selenium-vitamin E related. Some affected birds recovered in two to three weeks. The relationship between this entity and the one just described above remains to be further investigated.

e) *Miscellaneous myopathies*
Other myopathies contained in the TPC include several cases of multifocal necrotising myosotis, due to Salmonella typhimurium infection. Also two cases of massive mycotic necrotising myosotis of the breast muscle which probably spread from lung involvement. Additional lesions were granulomas associated with viable and dead spargana in tawny frogmouths and pseudotrichinella granulomas in a masked owl from Tasmania. There were also several cases each of recent or older standing focal or diffuse myonecrosis, the cause of which was not apparent in the available sections.

REFERENCES

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