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Source: Journal of Wildlife Diseases, 29(1) : 177-179

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-29.1.177
Brachial Plexus Injury in Two Red-Tailed Hawks (Buteo jamaicensis)

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ABSTRACT: Two red-tailed hawks (Buteo jamaicensis), found near Deltaville, Virginia (USA), were evaluated because of inability to use a wing. Results of needle electromyographic studies of the affected wing muscles in both hawks were compatible with denervation. On euthanasia, one hawk had extensive axon and myelin loss with multifocal perivascular lymphocytic inflammation of its brachial plexus and radial nerve. Demyelination and axon loss in the dorsal white matter of the spinal cord on the affected side also were found at the origin of the brachial plexus. The other hawk’s wing had not returned to functional status >2 yr after injury.

Key words: Red-tailed hawk, nerve injury, brachial plexus injury, electromyography, denervation, demyelination, Buteo jamaicensis.

Traumatic wing injuries in raptors are a common problem in clinics that treat wildlife. We report brachial plexus injury with radial nerve paralysis in two red-tailed hawks (Buteo jamaicensis). Hawk 1 was maintained for 2 yr after the problem appeared, with marginal clinical improvement. Hawk 2 was euthanized shortly after a presumptive diagnosis of radial nerve paralysis was made.

A mature red-tailed hawk (Hawk 1) was presented to a rehabilitation organization in February 1989. The hawk had been found near Deltaville, Virginia (USA) (37°39’N, 76°20’W) on the side of a road, unable to fly. The left wing dropped noticeably. The hawk was in good condition otherwise and ate well at the rehabilitation center.

Radiographs of the wing were normal, and the bird was returned to the rehabilitator for observation. It was reexamined 2 wk later, with no improvement. The wing at this time seemed slightly atrophied and was noticeably weaker when flexed or extended, compared to the normal wing. A presumptive diagnosis of radial nerve paralysis was made. The rehabilitator was advised to flex and extend the wing regularly to preserve the musculature and to keep the joints flexible.

Hawk 2, also found near Deltaville, Virginia, was presented 5 January 1991, unable to fly, but alert and eating. There were no visible lesions except that the left wing drooped. Based on radiographs of the wing, there was no fracture or luxation. The clinical diagnosis was radial nerve paralysis and the bird was referred to the Virginia-Maryland Regional College of Veterinary Medicine (Virginia Tech, Blacksburg, Virginia) for evaluation of nerve function in the affected wing. Hawk 1 still was in the care of the rehabilitator and also was referred for evaluation. Although a persistent effort had been made to exercise the wing, it still drooped and was not functional. The hawk could not hold the wing high enough to prevent damage unless the flight feathers were trimmed very short.

Neurologic and needle electromyographic (EMG) examinations were performed on each hawk. Needle electromyography was performed according to a previously described technique (Shell et al., 1988) using the Nicolet Compact Four Unit with a two-channel EMG amplifier (Nicolet Biomedical Instruments, Madison, Wisconsin, USA).

Abnormalities of Hawk 1 were restricted to the left wing and included weakness, decreased muscle tone and mass, ankylosis of joints, and lack of voluntary movement. Based on the needle electromyography, there were many fibrillation potentials and occasional bizarre high frequency discharges in the muscles of the left wing, compatible with denervation (Chrisman,
A few motor unit action potentials were observed, which suggested some intact nerve fibers were present. The affected wing of Hawk 2 had very poor muscle tone and would not retract when it was pulled away from the bird's body. There also were many fibrillation potentials and a few motor unit action potentials with the needle EMG. Based on these results, we diagnosed peripheral nerve injury in both birds. Hawk 1 was returned to the rehabilitation organization with a poor prognosis; return of function to the limb after 2 yr was highly unlikely. Hawk 2 was euthanized (Beuthansia-D, 1 ml intravenously, Schering-Plough, Animal Health Corp., Kenilworth, New Jersey, USA) and a complete necropsy performed.

At necropsy, lesions in Hawk 2 were limited to the left antibrachium which had muscle atrophy and some focal hemorrhage. Tissues were fixed in 10% neutral buffered formalin, embedded in paraffin, sectioned at 5 μ, and stained with hematoxylin and eosin for examination by light microscopy. Histologically, there was extensive fragmentation and loss of axons throughout the brachial plexus nerves. Demyelination was nearly complete as assessed with the Luxol fast blue stain for myelin (Pearse, 1960). Multifocal perivascular lymphocytic inflammation was present within the nerves, and dense connective tissue proliferation surrounded the nerve fiber bundles (Fig. 1). The radial nerve also had extensive axon and myelin loss.

Skeletal muscle of the antibrachium had mild myofiber atrophy with no evidence of angular atrophy as seen in neurogenic muscle damage. However, muscle enzyme histochemistry was not done; this precluded proper evaluation of the muscle. Nerve fibers within the muscle had extensive myelin loss. Multifocal mild lymphocytic inflammation was present.

The spinal cord was normal except for demyelination and axon loss in the left dorsal white matter at the origin of the brachial plexus. The final diagnosis was presumed trauma to the brachial plexus with spinal cord and radial nerve degeneration.

Both hawks presumably had traumatic injuries to the brachial plexus region, resulting in radial nerve paralysis. Such injuries have been reported in owls (Bubo virginianus, Otus asio), a gull (Larus argentatus), a crow (Corvus brachyrhynchos), and a red-tailed hawk (Smiley et al., 1988; Moore et al., 1989). In dogs, automobile accidents are the most common cause of brachial plexus injuries; it is hypothesized that the shoulder and scapula are displaced caudally or abducted, putting tension on the dorsal and ventral nerve rootlets or the nerves of the brachial plexus (Griffiths et al., 1974). There was no avulsion of the ventral or dorsal nerve rootlets evident, based on the histopathology of the spinal cord in Hawk 2. However, the demyelination and axon loss in the dorsal white matter were compatible with trauma to at least the dorsal nerve rootlets (Griffiths, 1974). In another report (Smiley et al., 1988) of brachial plexus injury in a red-tailed hawk, trauma to the ventral nerve rootlets was supported by obvious loss of cell bodies in the ventral gray column.

Radial nerve injury in raptors can be difficult to distinguish from muscle or tendon trauma. The natural tendency of hawks...
to resist manipulation complicates diagnosis by making it impossible to discern sensory deficits. A high degree of suspicion for avulsion injuries should be maintained for raptors unable to fly within 2 to 4 wk of injury. Needle electromyography can provide diagnostic (Chrisman, 1975) and prognostic information (Griffiths and Duncan, 1978). In these cases presented here, needle EMG confirmed damage to the muscles supplied by the brachial plexus nerves. Although the needle EMG changes were most compatible with nerve injury, nerve conduction studies would have been helpful to more specifically localize the injury (Moore et al., 1989).

Dogs with radial nerve paralysis sometimes can be managed with physical therapy to allow the muscles to remain healthy until the nerve can regenerate (Griffiths et al., 1974). None of the birds with reported cases of brachial plexus injury have improved. Because hawks are rather poor patients for physical therapy and long-term maintenance can be difficult, a guarded to poor prognosis must be considered if brachial plexus injury is suspected. Referral to a facility capable of electrodiagnostic studies should be considered to confirm the diagnosis. The authors thank Dr. Gouri Krishna for her assistance with the cases.

**LITERATURE CITED**


Received for publication 7 October 1991.