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AVULSION OF THE BRACHIAL PLEXUS IN A GREAT HORNED OWL (*Bubo virginianus*)

MICHAEL P. MOORE, ERIK STAUBER AND NANCY THOMAS

ABSTRACT.—Avulsion of the brachial plexus was documented in a Great Horned Owl (*Bubo virginianus*). A fractured scapula was also present. Cause of these injuries was not known but was thought to be due to trauma. Differentiation of musculoskeletal injury from peripheral nerve damage can be difficult in raptors. Use of electromyography and motor nerve conduction velocity was helpful in demonstrating peripheral nerve involvement. A brachial plexus avulsion was suspected on the bases of clinical signs, presence of electromyographic abnormalities in all muscles supplied by the nerves of the brachial plexus and absence of median-ulnar motor nerve conduction velocities.

An adult male Great Horned Owl (*Bubo virginianus*) was found with a drooping wing in a wilderness area of northern Idaho and presented to the Washington State University Veterinary Clinic for rehabilitation (Fig. 1). Physical examination revealed emaciation (772 g) and drooping of the left wing originating at the scapulohumeral joint. No other physical abnormalities were noted. The following laboratory results were considered normal by our laboratory: WBC = 11 000, heterophils = 78%, lymphocytes = 21%, basophils = 1%, PCV = 36%, total protein (refractometer) = 3.1 g/dL. Four leucocytozoan parasites/100 WBCs were observed; a common finding in wild raptors of this region (E. Stauber, pers. comm.). Whole body radiographs of the owl showed a remodeling fracture of the left scapula near its articulation; bone alignment at the fracture site was considered satisfactory. The wing was immobilized by a bandage to facilitate healing.

The bandage was removed 11 d later with no improvement in the drooped wing. A response to pain could not be elicited and peripheral nerve damage was suspected.

MATERIALS AND METHODS FOR ELECTROMYOGRAPHY AND NERVE CONDUCTION VELOCITY

A commercially available electromyograph[1] equipped with a constant voltage nerve stimulator[2] was used to perform the EMG and motor nerve conduction velocities according to previously described techniques (Steinberg 1979a). A concentric needle electrode[3] was used to record electromyograms of wing and pectoral muscles to serve as a reference and an active electrode. A monopolar ground electrode[4] was placed subcutaneously over radius and ulna The concentric electrode was inserted percutaneously into muscles while observing the response on the oscilloscope of the electromyograph. The concentric needle electrode was withdrawn from the muscle only, and redirected in several planes within the muscle.

Chemical restraint with ketamine hydrochloride,[5] 20 mg/kg given intramuscularly, and xylazine,[6] 4 mg/kg given intramuscularly was necessary before stimulation of the median-ulnar nerve. Monopolar teflon coated needle electrodes[7] were used for median-ulnar nerve stimulation. The cathode was placed approximately 2 cm distal to the anode. The distal 5 mm of the teflon coat was removed from the cathode. The nerve was supramaximally stimulated at a proximal and distal site with a rectangular electrical pulse lasting 0.1 ms. Proximal stimulating electrodes were inserted near the nerve caudal to the biceps brachii muscle. Distal stimulating electrodes were inserted near the nerve caudal to distal insertion of the biceps brachii muscle. Recording electrodes[7] were placed over the flexor carpi ulnaris muscle belly with the reference electrode approximately 1 cm distal to the exploring electrode. The ground electrode was placed between the recording and stimulating electrodes. Placement of stimulating and recording electrodes is depicted in Figure 2.

Median-ulnar motor nerve conduction velocity was calculated by dividing distance between proximal and distal stimulating cathode electrodes by difference in latencies of the evoked compound muscle action potentials (the time between the onset of the stimulation artifact and the onset of the evoked compound muscle action potential). Evoked compound muscle action potentials as the result of stim-

[1] Teca TE4 electromyogram; Teca Corp., White Plains, NY.

[2] NS6 Nerve Stimulator; Teca Corp., White Plains, NY.

[3] CT37 Concentric needle electrode; 37 mm, Teca Corp., White Plains, NY.

[4] RE12 needle electrode; 12 mm, Teca Corp., White Plains, NY.

[5] Ketamine hydrochloride; 100 mg/ml, Bristol Veterinary Products, Syracuse, NY.

[6] Xylazine, Haver, Division of Mobay Corp., Shawnee, KS.

[7] MG37 Monopolar needle electrode; 37 mm, Teca Corp , White Plains, NY.

Figure 1. Great Horned Owl (*Bubo virginianus*). Note drooping of the left wing.

ulation from proximal and distal sites are shown in Figure 3. A 2 site stimulation method of determining motor nerve conduction velocities is essential to avoid errors due to delayed nerve conduction in terminal nerves, neuromuscular junctions and muscle tissues (Walker et al. 1979; Steinberg 1979a).

RESULTS

After removing the bandage 11 d after admission, needle electromyography (EMG) revealed fibrillation potentials in the left brachial, antebrachial and

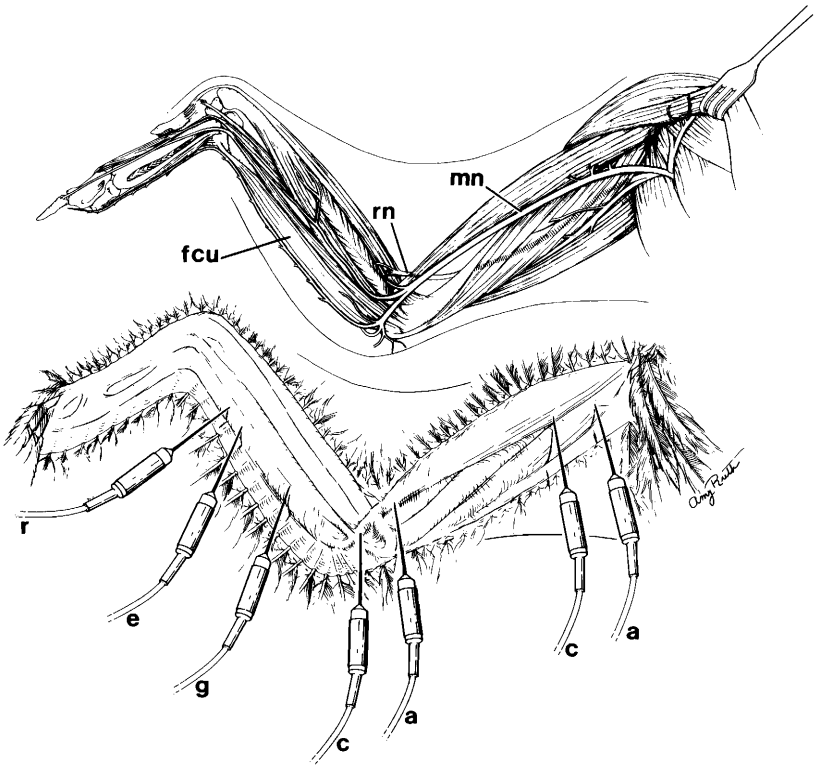

Figure 2. Medial view of the wing depicting placement of electrodes for recording the motor nerve conduction velocity. There was approximately 2 cm between the anode (a) and the cathode (c) of the stimulating electrode and 1 cm between the reference (r) and exploring (e) electrodes. The ground electrode was placed between stimulating and recording electrodes. The top diagram shows distribution of the radial nerve (rn) and the median-ulnar nerve (mn). The flexor carpi ulnaris (fcu) muscle was used to record evoked compound muscle action potential.

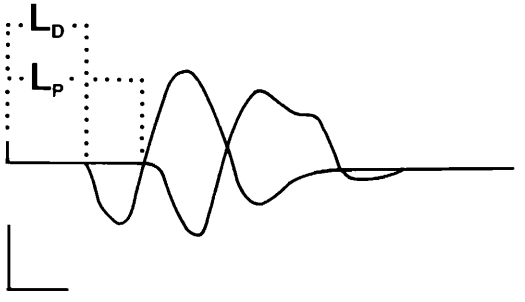

Figure 3. Evoked compound muscle action potentials as the result of proximal and distal median-ulnar nerve stimulations are shown. Motor nerve conduction velocity was calculated by dividing the distance between stimulating cathodes (see Fig. 2) by difference between the distal latency (L_D) and the proximal latency (L_P). The horizontal bar = 1 ms and the vertical bar = 4 mV.

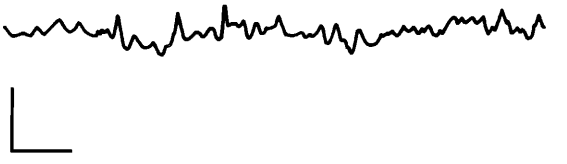

Figure 4. Fibrillation potentials recorded from the left pectoral muscle. The horizontal bar = 5 ms and the vertical bar = 0.1 mV.

Figure 5. Positive sharp waves recorded from the left pectoral muscle. The horizontal bar = 5 ms and the vertical bar = 0.1 mV.

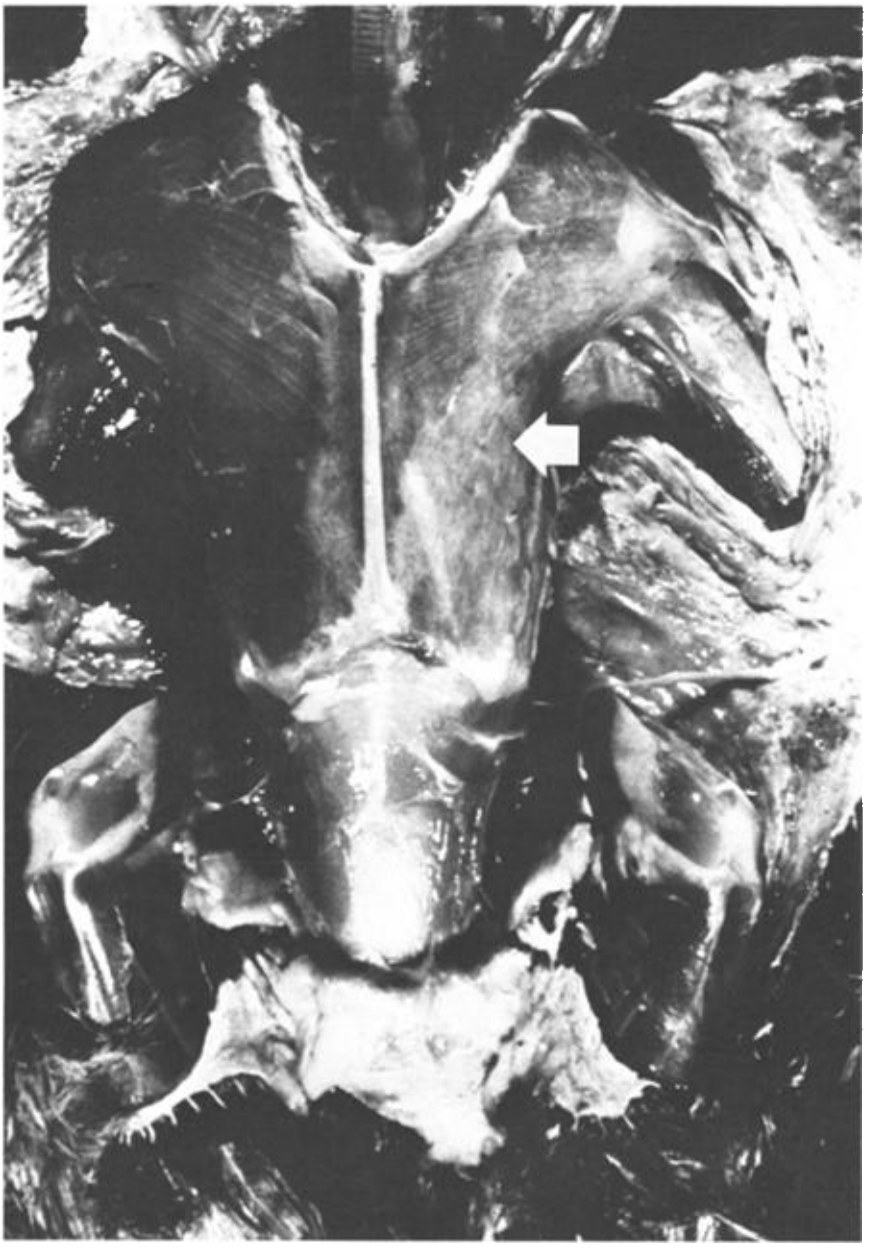

Figure 6. Note the pale streaks in the left pectoral muscle (arrow).

pectoral muscles. Conduction velocity of the median-ulnar nerve was not performed at this time because anesthesia would have been required, and the bird still appeared somewhat emaciated and was considered a poor anesthetic risk. Twenty-two d after admission, pronounced muscle atrophy was detected in all muscles of the left wing. An evoked motor response could not be elicited at a proximal or distal site when the left median-ulnar nerve was stimulated. In addition to fibrillation potentials (Fig. 4),

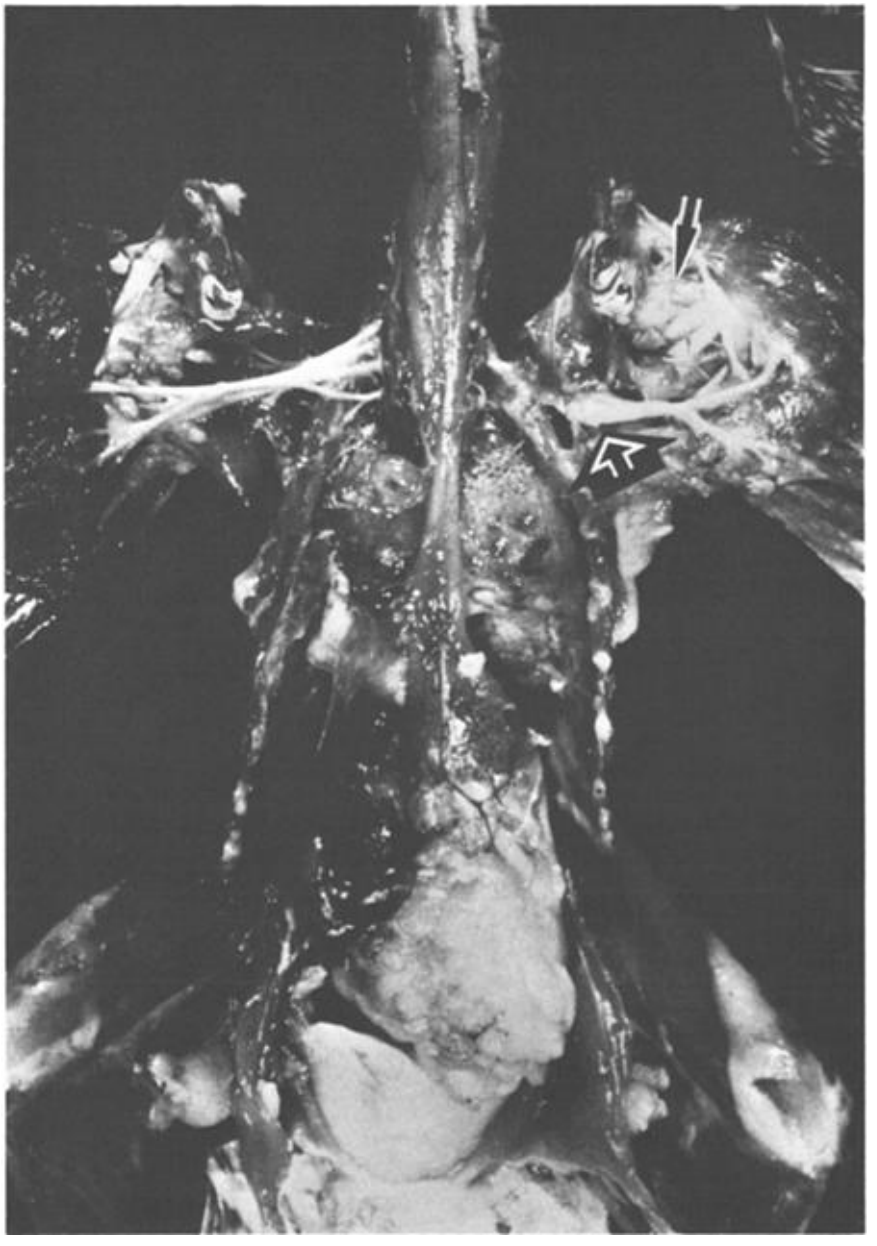

Figure 7. Note the avulsion of the nerve roots from the spinal cord (large arrow). The 1.0 cm granuloma due to *Aspergillus* sp. has been removed in order to more clearly demonstrate the avulsion. There is a 1.0 cm granuloma of *Aspergillus* sp. situated in the facial planes of the shoulder (small arrow) that was continuous with the granuloma that was removed.

positive sharp waves (Fig. 5), were now present. Conduction was present (50 m/s) in the contralateral median-ulnar nerve. Brachial plexus avulsion was suspected because of the EMG abnormalities in every muscle supplied by the brachial plexus and the absence of the evoked motor response from the median-ulnar nerve. The bird was euthanatized because of the negative prognosis for rehabilitation.

At necropsy the bird was judged to be in good nutritional condition and weighed 1100 g. Left pectoral muscles and muscles of the left wing were thin, brown and diffusely pale streaked (Fig. 6). Roots of the left brachial plexus were separated from the cervical spinal cord and displaced 1.5 cm lateral to their origin forming a 0.75 cm spherical enlargement (Fig. 7). A 1.0 cm encapsulated mass of gray-yellow caseous inspissated material was seen between avulsed segments of nerve roots and was continuous with a 1.0 cm friable, white powdery plaque positioned in the facial planes of the shoulder. Fracture of the left scapula was bridged by a bony callus and held rigidly with the caudal fragment ventral to normal position. Several small masses similar to that seen in the left shoulder region were seen in thoracic and abdominal airsacs and were identified as granulomas of *Aspergillus* sp. Histologic examination of tissues showed neurilemmal cell proliferation, demyelination and axon degeneration leading to muscle cell atrophy and degeneration.

DISCUSSION

Cause of the fractured scapula and avulsion of the brachial plexus could not be determined. The reason for infection with *Aspergillus* sp. at the brachial plexus was not apparent. Perhaps infection was related to a pre-existing condition in adjacent airsacs which later spread to the fracture at the time of the suspected trauma, or infection could have been associated with the fracture and extended into the thorax and around nerve roots of the brachial plexus. There was no histological evidence that the *Aspergillus* sp. granuloma was invading peripheral nervous tissues. Therefore, brachial plexus avulsion was most likely the result of trauma. Also, *Aspergillus* sp. infection is common in raptors, and therefore could have been coincidental with fracture and avulsion (Redig 1980). Other cases of brachial plexus avulsion in raptors diagnosed subsequently have been related to collision with automobiles.

Nerve injury in raptors can be difficult to differentiate from musculoskeletal causes of limb dysfunction. Electrodiagnostic evaluations have been useful in pathoanatomical localization of disease within the motor unit (Steinberg 1979b). Needle electromyography detects electrical activity of muscle by inserting needles into muscles, amplifying the response, observing the response on an oscilloscope and/or permanently recording the response for further analysis (Kimura 1983). Sound characteristics are also recorded. Abnormalities of resting muscle are detected by both visual inspection of recordings and by sound characteristics. Muscle diseases, neuromuscular junction, peripheral nerve, spinal motor nerve roots or the ventral motor horn cells may be detected by the use of needle EMG (Chrisman 1982). When EMG is combined with nerve conduction studies, the lesion can be localized to the peripheral nerve, the neuromuscular junction or the muscle itself. Wallerian degeneration of nerves will occur with avulsion or injury of the nerve resulting in an inability to conduct an impulse (DeLahunta 1983)

Electrodiagnostic evaluations were helpful in this case for diagnostic and prognostic purposes. Musculoskeletal injury to the scapula and antebrachium may result in brachial plexus and nerve root injury (Griffiths 1974; Griffiths et al. 1974). In this case neurologic dysfunction resulted from total avulsion of nerve roots of the brachial plexus rather than a lesion within the peripheral nerve. Information regarding nerve conduction techniques in raptors has not been previously described. The depiction of median-ulnar motor nerve conduction velocity should provide valuable information for those interested in clinical electrodiagnostic assessment of raptors.

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LITERATURE CITED

CHRISMAN, C. L. (ED.). 1982. Special ancillary investigations. Pages 67–89. *In* Problems in small animal neurology. Lea and Febiger, Philadelphia, PA.

DELAHUNTA, A. (ED.). 1983. Lower motor neuron–general somatic efferent system. Pages 65–66. *In* Veterinary neuroanatomy and clinical neurology. W. B. Saunders Company, Philadelphia, PA.

GRIFFITHS, I. R. 1974. Avulsion of the brachial plexus, Part 1. Neuropathology of the spinal cord and peripheral nerves. *J. Sm. Anim. Practice* 15:165–176.

———, I. D. DUNCAN AND D. D. LAWSON. 1974. Avulsion of the brachial plexus, Part 2. Clinical aspects. *J Sm. Anim. Practice* 15:177–182.

KIMURA, J. (ED.). 1983. Techniques and normal findings. Pages 235–257. *In* Electrodiagnosis in diseases of nerve and muscle: principles and practice. F. A. Davis, Philadelphia, PA.

REDIG, P. T. 1980. Aspergillosis in raptors. Pages 117–129. *In* J. E. Cooper, and A. G. Greenwood, EDS Proceedings of the International Symposium on Diseases of Birds of Prey: Recent advances in the study of raptor diseases, London.

STEINBERG, S. H. 1979a. A review of electromyographic and motor nerve conduction velocity techniques. *J. Am. Anim. Hosp. Assoc.* 15:613–619.

———. 1979b. The use of electrodiagnostic techniques in evaluating traumatic brachial plexus root injuries. *J. Am. Anim. Hosp. Assoc.* 15:621–626.

WALKER, T. L., R. W. REDDING AND K. G. BRAUND. 1979. Motor nerve conduction velocity and latency in the dog. *Am. J. Vet. Res.* 40:1422–1439.

Department of Veterinary Clinical Medicine and Surgery and Department of Microbiology and Pathology, College of Veterinary Medicine, Washington State University, Pullman, Washington 99164. Current address of third author: 5913 Lincoln Road, Oregon, Wisconsin 53575.

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