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THE ROLE OF LEAD IN A SYNDROME OF CLENCHED CLAW PARALYSIS AND LEG PARESIS IN SWAMP HARRIERS (*CIRCUS APPROXIMANS*)

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ABSTRACT: We investigated the hypothesis that lead poisoning was the cause of the clinical syndrome of clenched feet paralysis and leg paresis in wild raptors. Swamp Harriers (*Circus approximans*) are one of three extant native raptor species in New Zealand. Harriers with the syndrome were found to have statistically significantly higher blood lead concentrations than those without clenched feet (*t*-test; $t=-4.06$, df=5, $P=0.01$). However, elevated blood lead concentrations were also present in 60% of wild harriers without the clinical syndrome of clenched feet paralysis and leg paresis. There were features of the response to chelation treatment, electroneurodiagnostics, and pathology that were inconsistent with lead poisoning as reported in other birds of prey. We conclude that lead may be a factor in the expression of this clinical syndrome of clenched claw paralysis but that other factors not identified in our study play a role in the expression of the disease.

Key words: Chelation, electroneurodiagnostics, flexor paralysis, plumbism, raptor, toxicity.

INTRODUCTION

A syndrome of flexor paralysis and hindlimb paresis commonly seen in raptors throughout the world is known as clenched claw paralysis (Cooper, 2002). The clinical signs that define this condition include complete or partial peripheral nerve dysfunction in the hind limbs and tightly clenched digits (Cooper, 2002). While many authors suggest the syndrome is known to be caused by lead poisoning (Cooper, 2002; Redig and Arent, 2008), there are no studies in the peer-reviewed literature investigating the role of lead in this syndrome.

Reported neurologic signs of lead poisoning in raptors include seizures, weakness or depression, regurgitation or other gastrointestinal disease, and peripheral neurologic signs such as leg paralysis (Locke and Thomas, 1996; Cooper, 2002; Redig and Arent, 2008). Scavenging and carnivorous birds may ingest lead when feeding on lead-affected animals or animals containing lead shot in their tissues. Acute and chronic poisonings are recognized throughout the world in wild raptors (Locke and Thomas, 1996; Mateo et al., 2001; Krone et al., 2004; Pain et al., 2007). Birds with acute poisoning may present in good condition, but exhibiting neurologic signs such as leg paralysis or seizures, or die without clinical signs (Locke and Thomas, 1996; Redig and Arent, 2008). Chronic poisoning is more insidious and may present as wing droop, reluctance or inability to fly, immune suppression (indicated by secondary infections), poor growth or weight loss, and difficulty hunting, leading to poor condition and death due to starvation or through increased susceptibility to traumatic events such as predation, entanglements, and collisions (Pattee and Pain, 2003; Redig and Arent, 2008).

Swamp Harriers (*Circus approximans*) are one of three extant native raptors in New Zealand. They are ubiquitous throughout both the North and South Islands of New Zealand and areas of Australia and prefer open habitat. Harriers are opportunistic feeders and will scavenge roadkill, particularly the introduced brush-tailed possum (*Trichosurus vulpecula*), and hunt birds and introduced

mammals. Harriers are a common species presented to veterinary clinics throughout New Zealand. The New Zealand Wildlife Health Centre (NZWHC) at the Massey University Veterinary Teaching Hospital has been presented with a number of birds with clenched claws and variable degrees of leg paresis. High blood lead concentrations are typically recorded in these birds; however, chelation therapy has not routinely resulted in resolution of clinical signs.

We investigated the hypothesis that lead was the causative agent of the clinical syndrome of clenched feet paralysis and leg paresis in harriers using a range of antemortem measures including clinical and neurologic examinations, evoked and spontaneous electromyography (EMG), nerve conduction studies, and response to chelation treatment. These findings were compared with a control group of harriers presented for acute musculoskeletal trauma. The results of the antemortem tests were compared to the histopathologic appearance of the nerves and muscles of the pelvic limbs at necropsy.

MATERIALS AND METHODS

Harrier admissions, diagnostics, and treatment

All birds involved in this study (retrospective and prospective) were selected on the basis of having completed a diagnostic workup that included full physical exam, hematology, biochemistry, full body radiography, and blood lead concentration analysis. The case definition for inclusion into clenched claw paralysis was birds that showed partial or complete peripheral neurologic dysfunction in the hind limbs and flexor paralysis of the digits resulting in clenched claws. Birds with neurologic signs consistent with spinal trauma (loss of tail flick reflex, cloacal atony) or radiographic evidence of musculoskeletal trauma to the spine or pelvis or hind limbs were excluded.

The birds involved in this study are summarized in Table 1. A retrospective review of harriers admitted to the NZWHC between January 2005 and April 2005 was undertaken. Clinical records were examined for information on clinical presentation, blood lead concentrations, other clinicopathologic values, responses to treatment, and postmortem findings. Three birds with clenched claw paralysis were selected for inclusion in the study, as they had been tested for blood lead concentration with good records of clinical presentation and response to chelation treatment. Prospectively, six of 27 harriers presented to the NZWHC from May 2005 until August 2006 fulfilled the case definition of clenched claw paralysis. Fourteen harriers presented for musculoskeletal trauma (usually motor vehicle collisions) without exposure to lead (i.e., blood lead concentration <0.1 mg/l) were used as controls. Another group of four harriers with elevated blood lead concentrations (i.e., blood lead concentration >0.1 mg/l) but no clinical signs of clenched claw paralysis was identified.

Between 1 and 3 days after admission, birds were anesthetized with isoflurane in oxygen via face mask and blood was collected from the medial metatarsal, ulnar, or jugular veins and placed in 0.4 ml ethylenediaminetetraacetic acid (EDTA) and lithium heparin microcontainers (Becton Dickenson Vacutainer Systems, Preanalytical Solutions, Franklin Lakes, New Jersey, USA) for assessment of hematology, plasma biochemistry, and blood lead concentrations. Whole body ventrodorsal and lateral radiographs were taken. Heparinized blood samples were submitted to a commercial laboratory for hematology and plasma biochemistry analysis for a range of metabolites, or in-house biochemistry was performed on 0.09 ml of heparinized blood using a bench top VetScan (Abaxis Inc., Union City, California, USA) laboratory between 30 min and 1 day after collection. Blood from cases admitted prior to June 2006 ($n=14$) was submitted to a commercial laboratory for lead analysis on 1 ml of blood in EDTA using proprietary methods based on colorimetric measurement of trace metals (Sandell, 1959). Blood collected after June 2006 ($n=11$) was analyzed for lead concentration using a portable lead analyzer (LeadCare®, ESA Inc., Chelmsford, Massachusetts, USA). A 0.05-ml sample of blood was placed in a buffer solution and analyzed for lead content immediately after collection. The analyzer uses anodic stripping voltammetry (Wang, 2000) and has a detection range of 0.0–0.65 mg/l and an analytical reporting range of 0.014–0.65 mg/l. Blood lead concentrations >0.65 mg/l are expressed as "HI". When this upper limit was reached, blood samples were submitted to the commercial laboratory ($n=2$).

Treatment of birds with elevated blood lead concentrations (>0.1 mg/l) consisted of twice-daily chelation with 40–50 mg/kg of CaEDTA

TABLE 1. Summary of sample sizes of New Zealand Swamp Harriers (*Circus approximans*) submitted to various diagnostics used to evaluate a syndrome of clenched claw paralysis.

Treatment group	Sample size	Blood lead concentration on admission	Neurologic examination	Electro-neurodiagnostics	Treatment response	Histopathology
Retrospective	3	3	0	0	3	0
Prospective	6	4	4	4	6	6
Control	14	14	14	0	n/a[a]	1
High lead controls (not clenched)	4	4	2	1	3	2

[a] n/a = not applicable.

(Calcium EDTA Solution 20%, National Veterinary Supplies Ltd., Christchurch, New Zealand) intramuscularly or intravenously (IV) and a balanced electrolyte solution (75 ml/kg IV or per os) for 5 days. The CaEDTA was then discontinued for 2 days and blood lead, packed cell volume, estimated white cell counts, and uric acid were analyzed. Treatment was then reinstituted for 5 days. This cycle of treatment was continued until blood lead concentration was less than 0.1 mg/l or the bird was euthanized. Supportive treatment for other presenting problems such as trauma was also provided.

Electroneurodiagnostics

Birds were anesthetized using inhaled isoflurane in oxygen for the duration of the testing. Evoked and spontaneous electromyograms were carried out on four birds with clenched feet following the protocol of Clippenger et al. (2000) for pelvic limb nerves. Electromyograms were recorded using a signal amplifier (ISO-Dam, World Precision Instruments, Sarasota, Florida, USA) with a gain of 100 and a pass band of 10 Hz. The signal was digitized with a PowerLab A/D converter (AD Instruments, Dunedin, New Zealand) and recorded to a personal computer (Toshiba Satellite) using Chart and Scope software (AD Instruments) for spontaneous and evoked EMG, respectively. Stimulating electrodes were 27-gauge subdermal needle electrodes and recording electrodes were 26-gauge concentric needle electrodes (Medelec Old Woking, Surry, UK). No control birds were available for this section of the study.

Spontaneous EMGs were recorded from the cranial and caudal muscle groups proximal to the stifle, and those distal to the stifle but proximal to the tarso-metatarsal joint, in both legs of four affected birds. Two birds were examined twice during treatment.

Evoked EMG following stimulation of a motor nerve was used to evaluate nerve conduction in the right and left tibial nerves in three affected birds and in the ulnar nerve of one bird. Recordings were made in the same four locations as those for spontaneous EMG. Stimulating electrodes were placed in the area of the greater trochanter 10 to 15 mm apart. A grounding electrode was placed between the distal stimulating electrode and the recording electrode. Evoked EMGs were recorded using signal averaging of 16 trials and a 10V stimulus with a 0.5 ms duration using a Grass S48 nerve stimulator (Grass Technologies, West Warwick, Rhode Island, USA).

Pathology

Standard necropsy was carried out on six lead-affected birds with clenched feet and one control bird (blood lead concentration <0.1 mg/l at presentation). Samples of tissues were preserved in 10% neutral buffered formalin. Blocks of preserved tissues were routinely embedded in paraffin and sections were cut to three microliters and stained with hematoxylin and eosin. Routine histopathology was carried out on liver, kidney, spleen, lung, heart, brain, and gastrointestinal tract. Representative transverse and longitudinal sections of the cranial and caudal femoral and tibial muscle groups were examined with light microscopy. The ischiatic nerve was dissected from the hip to the hock joint and examined along its length in longitudinal and transverse sections. Representative samples of nerve and muscle sections were stained with luxol fast blue and counter-stained with silver.

Statistical analysis

Statistical analysis was performed using SigmaStat© for Windows, version 3.5 (SPSS Inc., Chicago, Illinois, USA). Normally distributed data were compared using a *t*-test. For the purposes of statistical analysis, blood lead levels below the analytical reporting range of the colorimetric method were reported as

0.1 mg/l and those below or above the analytical reporting range of the LeadCare analyser were reported as 0.014 mg/l and 0.65 mg/l, respectively. Means are reported plus or minus standard deviation.

RESULTS

Clinical presentation

For nine birds (30% of harriers admitted), the major presenting sign was paralysis of the feet in a clenched position. The flexor tendons were contracted and the toes were knuckled under, with most of the birds presenting with pressure sores and inflammation of the digits. In some birds, the toes could be straightened manually; however, the birds showed no voluntary movement in the digits and the feet would clench soon after straightening. Most birds showed evidence of pain perception assessed by response to skin pin-prick. Seven (78%) of the birds with clenched feet also had hock paresis and were unable to stand in a normal upright position. Three (33%) of the nine birds were in poor body condition and one had a history of seizures.

Four birds with elevated blood lead (mean = 1.29 ± 1.02 mg/l; range = 0.65–2.8 mg/l), but without signs of clenched claw paralysis, were also examined. One of these birds had hock paresis without clenching of the feet. Two of the birds were ataxic and one was presented with a history of seizures.

On full body radiographs, none of the birds showed evidence of metallic material within the gastrointestinal tract.

Blood lead concentrations

The mean blood lead concentration of all 25 harriers was 0.806 ± 1.155 mg/l with a range of 0.014–3.7 mg/l. The mean blood lead concentrations of harriers presenting with (n = 6; mean = 2.35 ± 1.179 mg/l; range 0.5–3.7 mg/l) and without (n = 19; mean = 0.318 ± 0.589; range 0.014–2.5 mg/l) clenched claw paralysis were significantly different (P = 0.01).

Neurologic examination

Standardized neurologic examinations were performed on four of nine harriers presenting with clenched claw paralysis and two of the birds presenting with elevated blood lead concentrations without clenched feet. Birds with clenched feet all had knuckling of digits and no grasping reflex. Three of the four had detectable muscle atrophy of the legs. Birds from both groups displayed abnormalities in leg strength and withdrawal as well as balance loss. Birds from the control group showed no neurologic abnormalities (Table 2).

Electroneurodiagnostics

Spontaneous EMGs of the proximal muscle groups had normal electrical activity. Examination of the distal muscle groups showed evidence consistent with lower motor neurone disease in all four birds examined. Abnormal signs included denervation potentials and reduced or absent insertion potentials.

Evoked EMGs were performed on the legs of three affected birds and on the ulnar nerve of one bird. Neuromuscular conduction was present in the proximal ischiatic nerve of two affected and one control bird. There was no conduction in the distal nerves of the two birds with clenched claws, while conduction was present in the ulnar nerve of affected birds and in all nerves examined in the control bird. These nerve conduction studies are consistent with an absence of neurologic function in the peripheral nerves distal to the stifle in birds with clenched claws.

Treatment response

Nine birds with clenched feet underwent treatment. Hospitalization time ranged from 4–185 days with a mean of 46 days ±56.6 and a median of 21 days. Five birds showed a reduction in blood lead concentration with chelation treatment; the remaining four were euthanized

TABLE 2. Selected neurologic exam results for six New Zealand Swamp harriers (*Circus approximans*) presenting with elevated blood lead concentrations. All abnormalities detected were bilateral[a].

Bird	Posture and gait	Mental status	Cranial nerves	Balance	Proprioception	Cloacal function	Leg strength and tone	Leg muscle	Leg superficial pain	Leg deep pain	Leg withdraw	Leg grasping	Knuckling of digits
Clenched claws													
1	Ab	N	N	N	Ab	N	Ab	Ab	N	N	N	Ab	Ab
2	Ab	N	N	Ab	Ab	N	N	Ab	N	N	Ab	Ab	Ab
3	N	N	N	N	N	N	N	Ab	Ab	N	N	Ab	Ab
4	Ab	Ab	N	Ab	N	N	Ab	N	N	N	Ab	Ab	Ab
Unclenched													
1	Ab	Ab	N	Ab	Ab	N	Ab	N	Ab	N	Ab	Ab	N
2	Ab	N	N	Ab	N	N	Ab	N	N	N	Ab	N	N

[a] Ab = abnormal; N = no abnormalities detected.

before an effect of chelation on blood lead concentration could be demonstrated. A clinical response was observed in one bird that regained foot function over 27 days while blood lead concentration decreased from 3.7 mg/l to <0.1 mg/l. The bird was sent to a rehabilitation facility; however, its feet reclenched almost immediately and it was readmitted. The bird was euthanized following incomplete resolution after a further 5 mo of treatment.

Three birds with elevated blood lead concentrations without clenched feet underwent treatment. Hospitalization time ranged from 30–35 days with a mean of 33 days ±2.65. All three birds showed a reduction in blood lead concentration and a resolution of clinical signs of lead toxicity in response to chelation treatment. Two of the three birds were sent to rehabilitation for eventual release and one remained in permanent captivity as a retired advocacy bird. A fourth bird was euthanized on presentation due to severe trauma.

Histopathologic lesions

Histopathologic sections of the leg muscles from six birds with clenched claw paralysis were examined. High numbers of myofibrils showed pathologic changes including centralizing nuclei and swollen hyper-eosinophilic myocytes consistent with myonecrosis, fatty infiltration and replacement of muscle fibers with adipocytes, and regenerating muscle fibers and variation in myocyte size. These pathologic changes were found in iliotibialis cranialis, femorotibialis, flexor cruris medialis, iliofibularis, tibialis cranialis, and gastrocnemius muscle sections examined from all six birds. Sections from four birds also showed small, multifocal inflammatory foci within the muscles consisting primarily of mixed lymphocytes and macrophages; however, granulocytes were also observed in some areas. One bird had evidence of fibrosis. One harrier with clenched claws had more severe changes within its femorotibialis muscle, with inflammation throughout the transverse

section of muscle and segmental necrosis of muscle fibers observed in the longitudinal section. The flexor hallucis longus muscle was examined in one affected bird and revealed a moderate numbers of cells with centralizing nuclei, necrosis, and fatty infiltration with variation in cell size and occasional regenerating muscle cells. In the control bird, there were also occasional central nuclei, necrotic cells, and regenerating muscle fibers observed in the muscle sections but at a much lower incidence within the muscle bundles. Sarcocysts were present in almost all sections examined from both affected and control birds but were not associated with any histopathologic changes identifiable with light microscopy. No acid-fast intranuclear inclusions of the renal tubular cells were observed in six harriers with elevated admission blood lead or in a control harrier. No abnormalities were identified in peripheral nerves of the legs in any birds examined.

DISCUSSION

We investigated the hypothesis that lead is a causal factor in a syndrome of clenched claw paralysis and leg paresis in Swamp Harriers in New Zealand. There was an association between high blood lead concentrations and clenched claw paralysis in the harriers. However, if lead was the sole causal factor in the clenched claw syndrome, we would have expected to see a strong response to prolonged chelation therapy (Dumonceaux and Harrison, 1994), electroneurodiagnostics consistent with a peripheral neuropathy (Bleecker et al., 2005), and pathology consistent with demyelination of the peripheral nerves (Hunter and Wobeser, 1980; Verity, 1997; Dart et al., 2004). In the birds examined, we found a marginal response to chelation therapy, electrodiagnostics consistent with total absence of nerve conduction distal to the stifle, and an absence of demonstrable peripheral neuropathology.

Further confounding the results is that, although the mean blood lead concentrations of control birds were significantly lower than birds with the clenched claw syndrome, there was an overlapping range of lead concentrations in the two groups, and not all birds with elevated blood lead concentrations exhibited clenched claw paralysis. It is possible that the birds with clenched claw paralysis had chronic toxicity, with a greater exposure to lead over time, or that there are other factors contributing to the clenched feet syndrome. The absence of metallic material within the gastrointestinal tract of birds in our study contrasts with results by Samour and Naldo (2002), who found 35% of captive falcons treated, including 78 Saker (*Falco cherrug*), 12 Peregrine (*Falco peregrinus*), and 6 Lanner (*Falco biarmicus*) Falcons, had radiographic evidence of lead pellets or fragments in the gastrointestinal tract. The source of the lead in the harriers in New Zealand is either from ingestion of prey with high tissue levels of lead, such as waterfowl, or represents chronic lead toxicity where the original source of the lead has already transited the gastrointestinal tract. Lead shot was banned for use by waterfowl hunters in New Zealand as recently as 2002, but is still used in terrestrial areas, those defined as greater than 200 m away from waterways.

Treatment of the harriers with prolonged chelation was successful in producing a decline in the blood lead concentration over time, as detected by serial blood lead measurements. The chelation did not result in full resolution of the clinical syndrome although, in most birds, an improvement in neurologic signs was seen. This chelation protocol is generally successful in the treatment of uncomplicated lead toxicity in other species of birds (Dumonceaux and Harrison, 1994), domestic mammals (Knight and Kumar, 2003), and humans (Needleman, 2004; Gilbert and Weiss, 2006). Our failure to completely resolve the clinical syndrome seen in the harriers with

prolonged chelation is strongly suggestive that factors other than lead contributed to the clenched claw paralysis.

In humans, low-level chronic lead exposure causes impaired learning ability, neuromuscular defects, and altered heme metabolism (Silbergeld and Goldberg, 1980). Alterations in the function of these systems may occur when lead is present at levels less than that known to cause overt clinical signs. Some authors suggest that blood lead concentrations <0.1 mg/l may have a significant effect (Needleman, 2004; Gilbert and Weiss, 2006). Electroneurodiagnostic measures of chronic blood lead exposure in humans are associated with progressive impairment of large and small myelinated sensory nerve fibers (Bleecker et al., 2005). In our study, electroneurodiagnostic testing of the legs suggested an absence of neurologic function in peripheral nerves distal to the stifle.

Spontaneous and evoked electromyography (SEMG and EEMG) enables assessment of the nerve and muscle function in live patients. The SEMG examines the health of the muscle cells and the integrity of the muscle unit including muscle and nerves, while an EEMG examines the ability of a compound action potential to be propagated by a peripheral nerve in response to electrical stimulation (Clippenger et al., 2000). Reductions in the insertion potential activity can be suggestive of reduced muscle responsiveness, such as that seen with atrophied muscle (Sims, 1996). While this was observed in some birds, the lack of normal recordings for the length of insertion potentials in this species makes it difficult to draw definitive conclusions. The SEMG indicated that there was possible abnormal electrical activity in the distal muscle groups. Denervation or fibrillation potentials, observed as biphasic or triphasic potentials, are caused by the firing of single muscle fibers in denervated muscle and may be caused by neurogenic or myopathic disorders (Sims, 1996). The frequency of denervation potentials decreases over time as the muscle further atrophies (Sims, 1996).

Evoked EMG involves repeated stimulation of a nerve and motor units to achieve a recording of a compound muscle action potential (CMAP). Neuromuscular disease results in reduced amplitude CMAP and demyelination results in a prolonged latency CMAP although, more commonly, both pathologies tend to have a mixed effect (Rubens et al., 2001). In our study, abnormalities in conduction were observed as a complete loss of the CMAP when recording was attempted between the stifle and the foot. This was also observed when the nerves were dissected out and conduction was evaluated.

Histopathologic examination of the nerves and muscles of the legs of six affected birds failed to establish an etiology for the condition. Surprisingly, there was a complete lack of observable histologic change in peripheral nerves examined by light microscopy. This was contrary to the clinical signs, the lack of detectable conduction in some nerves, and the elevated blood lead concentrations. Lead is known to cause segmental demyelination and axonal degeneration in many species (Hunter and Wobeser, 1980; Verity, 1997; Dart et al., 2004). Demyelination is thought to be caused by toxicity to Schwann cells (Verity, 1997), which may progress to axonal degeneration. Demyelination results in generalized weakness and symptoms such as foot drop and hand drop in humans (Anderson et al., 1996) and leg and wing paresis or paralysis in birds. In cases of chronic lead exposure, different stages of demyelination and remyelination may be seen histopathologically (Feldman, 1977). In lead-poisoned Mallards (*Anas platyrhynchos*), Hunter and Wobeser (1980) found swelling and fragmentation of myelin nerve sheaths occurring in the vagus, brachial, and sciatic nerves. In some cases, clinical disease in peripheral nerves may precede disease in other tissues (Hunter and Wobeser, 1980).

While no evidence of lead-induced neuropathology was observed in the sections examined, moderate to severe muscle pathology was observed in all affected birds. This was particularly evident in the muscles distal to the stifle, although one bird was found to have severe changes in its femorotibialis muscle. None of the muscular histologic changes identified are specific to any etiology; however, muscle fiber degeneration and disintegration with no inflammatory response has been observed in Mallards dosed with lead (Clemens et al., 1975). It is likely that prolonged recumbency may have contributed to the muscle changes observed. Prolonged chelation therapy and reduced blood lead concentrations in these harriers may have reduced the ability to detect acute microscopic changes in the nerves; however, chronic changes such as demyelination and remyelination would have been expected.

The hind-limbs of raptors have specialized anatomic features that form a mechanical locking mechanism (Einoder and Richardson, 2006), composed of the automatic flexor mechanism that occurs in most birds and a tendon-locking mechanism of the digits that is well developed in raptors (Einoder and Richardson, 2006; Fowler et al., 2009). The automatic flexor mechanism is activated by flexion of the leg joints, causing tension of the flexor tendons, which results in passive flexion of the digits independent of muscular contraction (Einoder and Richardson, 2006). The tendon-locking mechanism of the digits consists of interlocking tubercles on the ventral surface of the flexor tendons and plications on the opposing tendon sheaths. The tendon-locking mechanism is most important in securing the grip of raptors on prey and is activated when resistance is encountered on flexion of the feet (Einoder and Richardson, 2006). The characteristic clinical signs of clenched claw paralysis in birds are, therefore, produced by the automatic flexor mechanism in the absence of extensor muscle activity in the limb. It is, therefore, likely that the syndrome of clenched claw paralysis reported in raptors has multiple etiologies and can include any condition that produces neural or muscular dysfunction in the limbs.

We suggest that, while underlying spinal trauma or infectious disease are possible causal factors in the syndrome of clenched claw paralysis, these causes are unlikely in our study population given the results of radiographs, neurologic examination, and postmortem examination of dead birds. No histologic evidence of spinal trauma or Wallerian degeneration in peripheral nerves was observed. We suggest that the presence of sarcocysts in the muscle of these birds is an incidental finding and is not associated with the clinical syndrome of clenched claw paralysis. The pathogenic *Sarcocystis* disease described by Olias et al. (2009, 2010) in pigeons caused encephalitis and severe lymphohistiocytic and eosinophilic myositis with degeneration and rhabdomyolysis. The sarcocystis found in our study was present in both affected and control birds and was not associated with inflammation.

In summary, birds with clenched claw paralysis had clinical signs and electroneurodiagnostic findings most consistent with a peripheral neuropathy or myopathy. All birds had elevated blood lead concentrations and mild anemia that was responsive to chelation therapy; however, many failed to respond clinically to chelation therapy, and one bird that showed an apparent response to treatment demonstrated a rapid clinical relapse. With no evidence of neuropathology, and with EMG testing indicating lack of function in nerves distal to the stifle, the clinical syndrome is most suggestive of a functional distal neuropathy and may be due to a variety of causes.

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