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DIAGNOSTIC FINDINGS IN 132 GREAT HORNED OWLS

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ABSTRACT.—We reviewed diagnostic findings for 132 great horned owl (*Bubo virginianus*) carcasses that were submitted to the National Wildlife Health Center from 1975–93. The carcasses were collected in 24 states but most came from Colorado ($N = 21$), Missouri ($N = 12$), Oregon ($N = 12$), Wyoming ($N = 11$), Illinois ($N = 10$), and Wisconsin ($N = 9$). Forty-two birds were emaciated but presumptive causes of emaciation, including old injuries, chronic lesions in various organs, and exposure to dieldrin, were found in only 16. A greater proportion of juveniles (56%) than adults (29%) were emaciated. Twelve owls were shot and 35 died from other traumatic injuries. Poisonings were diagnosed in 11 birds, including five associated with hydrogen sulfide exposure in oil fields and six cases of agricultural pesticide poisonings. Electrocution killed nine birds and infectious diseases were found in six. Miscellaneous conditions, including egg impaction, drowning, and visceral gout were diagnosed in three of the birds and the cause of death was undetermined in 14 owls. While this review identifies major diagnostic findings in great horned owls, sample bias prevents definitive conclusions regarding actual proportional causes of mortality.

KEY WORDS: Bubo virginianus; *disease*; *emaciation*; *great horned owl*; *mortality*; *toxicosis*; *trauma.*

Encuentros diagnósticos en 132 individuos de la especie *Bubo virginianus*

RESUMEN.—Revisamos el diagnóstico para 132 carcasas de *Bubo virginianus* que fueron sometidos al National Wildlife Health Center desde 1975 a 1993. Las carcasas fueron colectadas en 24 estados, pero la mayoría venían de Colorado ($N = 21$), Missouri ($N = 12$), Oregon ($N = 12$), Wyoming ($N = 11$), Illinois ($N = 10$) y Wisconsin ($N = 9$). Cuarenta y dos aves estaban adelgazadas, pero las presuntas causas de adelgazamiento, incluyendo antiguas heridas, lesiones crónicas en varios órganos y exposición al dieldren sólo fueron encontradas en 16. El adelgazamiento ocurrió en mayor proporción en juveniles (56%) que en adultos (29%). Doce búhos fueron cazados y 35 murieron de otras heridas traumáticas. Envenenamientos se diagnosticaron en 11 aves, incluyendo cinco asociadas con exposición a "hydrogen sulfide" en campos petroleros y seis casos de envenenamiento por perticidas de uso agrícola. Nueve aves murieron electrocutadas y en seis se encontró enfermedades infecciosas. Condiciones miscelaneas, incluyendo impacto de huevos, ahogamiento, fueron diagnosticados en tres de las aves y causas de muerte no indentificadas ocurrieron en otros 14 individuos. Mientras esta revisión diagnostica los mayores encuentros en *B. virginianus*, sesgos de la muestra previenen conclusiones definitivas sobre la proporcionalidad de las causas de muerte.

[Traducción de Ivan Lazo]

The great horned owl (*Bubo virginianus*) is widely distributed throughout North America and occupies a greater variety of habitats than any other species of owl (Johnsgard 1988). Great horned owls are adaptable feeders with a highly diverse diet that includes insects, small mammals, and birds (Johnsgard 1988). Although few data are available regarding the longevity of wild great horned owls, one band recovery documents survival for over 20 yr (Klimkiewicz and Futcher 1989). Comparatively little is known about causes of death in this cosmopolitan species. Scattered reports of

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great horned owl mortalities exist in reviews of morbidity and mortality of raptors as a group, but compilations of diagnostic findings specific to great horned owls are lacking. Of the conditions diagnosed in raptors, trauma is among the most frequent and is often associated with human-related causes (Keran 1981, Fix and Barrows 1990, Cooper 1993, Franson et al. 1995). Poisonings are also commonly reported causes of mortality in raptors (Henny et al. 1985, Lumeij et al. 1993, Franson et al. 1995), and compounds previously associated with great horned owl deaths include chlorinated hydrocarbons (Blus et al. 1983a, 1983b, Stone and Okoniewski 1988, Okoniewski and Novesky 1993) and organophosphorus pesticides (Henny et al. 1987). Little is known about the significance of diseases in great horned owl populations, although individual case reports have been published describing several infectious, parasitic, and neoplastic conditions (Halliwell 1971, Keymer 1972, Sileo et al. 1975, Kocan et al. 1977, Clark et al. 1986, Swayne and Weisbrode 1990). We report the results of postmortem examinations of 132 great horned owl carcasses submitted to the National Wildlife Health Center (NWHC), Madison, Wisconsin from 1975–93 to provide additional information on the variety of factors resulting in morbidity and mortality in this species.

METHODS

For this review we selected only intact carcasses, excluding those that were heavily scavenged, in advanced stages of decomposition, or cases in which birds had spent extended periods in rehabilitation. Specimens, submitted by field biologists and others from 1975–93, were stored refrigerated or frozen until examination. Necropsies were conducted by 14 different diagnosticians during the 19-yr period of the study. Each bird was examined by gross inspection to identify, for example, abnormalities in organ systems suggestive of diseases, traumatic injuries indicating gunshot or collisions with objects, burns suggestive of electrocution, gender, stage of maturity (juvenile or adult, based on gonadal development), and to assess the overall body condition. Subsequent laboratory analyses of appropriate tissues were carried out to identify conditions suggested by necropsy observations. Diagnoses of diseases were based on the presence of characteristic lesions at necropsy and histopathological examination of tissues or laboratory isolation of the causative agent. We report the specific causes of death or the most significant findings identified by the diagnosticians, thus omitting incidental conditions unlikely to have caused harm to the birds. Proportional categories of findings, according to gender and age, were compared using the chi-square test for homogeneity (Daniel 1978).

Brain cholinesterase activity, as an indicator of exposure to organophosphorus or carbamate pesticides, was determined for 22 birds using methods described by Ellman et al. (1961) as modified by Dieter and Ludke (1975) and Hill and Fleming (1982). The magnitude of cholinesterase inhibition was calculated by comparison with the mean control value (16 ± 2.5 μmoles/min/g) reported by Hill (1988). Pesticide analyses were done at the Patuxent Wildlife Research Center, Laurel, Maryland. Organophosphorus and carbamate compounds were recovered from stomach contents by column extraction and identified by gas chromatography as described by Belisle and Swineford (1988). Brains were tested for residues of chlorinated hydrocarbons by gas-liquid chromatography (Cromartie et al. 1975). Lead and sulfide residues in tissues were determined according to Boyer (1984) and Feldstein (1960), respectively.

Tissues for histopathology were fixed in buffered 10% formalin, embedded in paraffin, and sectioned for light microscopy; slides were stained with hematoxylin and eosin for routine examination, Ziehl-Neelsen acid-fast for mycobacteria, or Grocott silver for fungi. Bacterial isolation attempts were carried out by inoculating tissues onto 5% sheep red blood agar and eosin-methylene blue plates (DIFCO Laboratories, Detroit, MI U.S.A.), and isolates were characterized and identified with the API-20E system (Analytab Products, Plainview, NY U.S.A.). Cell cultures and embryonating eggs were used for isolation of viruses as described by Docherty and Slota (1988) and Senne (1989).

RESULTS AND DISCUSSION

The 132 specimens were submitted from 24 states, but most came from Colorado ($N = 21$), Missouri ($N = 12$), Oregon ($N = 12$), Wyoming ($N = 11$), Illinois ($N = 10$), and Wisconsin ($N = 9$). Gender was determined for 121 carcasses (61% were female) and stage of maturity was assessed for 116 (84% were adults). Emaciation and trauma were the most frequent diagnostic findings followed by gunshot, toxicoses, electrocution, infectious diseases, and miscellaneous (Table 1). No significant findings were reported for 14 (11%) carcasses. No difference was noted in the distribution of proportional diagnostic findings between males and females, but a slight difference ($\chi^2 = 12.76$, df = 7, $P = 0.08$) was indicated when proportional categories of findings for adults were compared with those for juveniles. Emaciation was the primary source of this difference ($\chi^2 = 4.73$, df = 1, $P = 0.03$), and a higher proportion of juveniles (56%) than adults (29%) were emaciated.

Emaciation. Although emaciation was diagnosed in 42 (32%) carcasses, factors contributing to this condition were identified in only 16. Two had ocular lesions (corneal lacerations and plaques), and one had masses on the eyelids that covered the eyes and may have impaired sight and hence hunting ability. Lesions of the eyes are relatively common in raptors, including great horned owls, and are frequently the result of some type of physical injury (Murphy et al. 1982). Joint dislocations and old fractures in various stages

of healing, including one instance of apparent injury from a leg-hold trap, were found in three carcasses and probably led to decreased mobility and subsequent debilitation through malnutrition. Two emaciated owls had oral lesions, a beak deformity and a proliferative membranous lesion on the tongue that may have hindered consumption of prey.

Six carcasses had lesions of other organ systems thought responsible for emaciation, including one carcass each with intestinal nematode impaction (*Porrocaecum* sp.), abdominal adhesions secondary to a healing puncture wound, granulomatous hepatitis of undetermined etiology, and necrotizing verminous pneumonia. One carcass had a swollen foot and bacterial cultures of the foot and liver yielded heavy growth of *Serratia* sp., indicating a possible septicemia. Another bird had a laceration of the skin over the back of the neck, surrounded by an accumulation of tissue debris, fly ova, and maggots. This condition was thought to be antemortem and contributory to emaciation.

Elevated dieldrin residues were found in the brains of two emaciated owls. An adult female found dead in Minnesota in 1981 had 2.8 ppm wet weight dieldrin in its brain. Another adult female, found moribund in late 1981 in Illinois, had a brain dieldrin concentration of 4.4 ppm wet weight. Brain residues of 4–5 ppm wet weight dieldrin are considered to be the lower threshold of toxicity (Stickel et al. 1969). Although the use of most organochlorine pesticides is now banned in the United States, avian mortality continues from exposure to historically treated areas (Okoniewski and Novesky 1993), and these compounds should be considered in cases where emaciated birds are found dead.

For the remaining 26 emaciated owl carcasses, no etiology was revealed by necropsy or laboratory evaluations of tissues. Although heavy parasitism by lice (*Strigiphilus* sp.), gastrointestinal parasites (*Porrocaecum* sp. and unidentified capillarids), and renal coccidia occurred in four of these carcasses, it was concluded that these infections were secondary to debilitation, not the cause of it. Liver lead concentrations were determined for 16 of these birds and in all cases were less than 2 ppm wet weight, which is considered to be within normal limits of exposure for many species of birds (Franson 1996). Brains of two carcasses were analyzed for organochlorine pesticides, with negative results. In eight emaciated owls, no testing beyond gross inspection at necropsy was conducted, precluding any further diagnosis. These 26 emaciated birds were found during all months of the year, although slightly more were found from April to September ($N = 15$) than October to March ($N = 11$). This may simply reflect an increased number of observers in the field during the summer months. Of the 24 birds in this group for which stage of maturity was determined, eight were juveniles and 16 were adults. Overall, a greater proportion of juveniles (56%) than adults (29%) were emaciated. Indeed, most (eight of 10) of the emaciated juveniles were in this category of unidentified etiology, suggesting that these birds may have been too naive to obtain adequate food. These results agree with other reports, including Cooper (1993) who found a similar frequency of starvation in barn owls (*Tyto alba*), and Kenward et al. (1993) who reported that juvenile northern goshawks (*Accipiter gentilis*) were more frequently diagnosed with starvation than adults.

Table 1. Categories of diagnostic findings in 132 great horned owls.

DIAGNOSTIC FINDING	*N*	%
Emaciation	42	32
Trauma	35	26
Undetermined	14	11
Gunshot	12	9
Toxicoses	11	8
Electrocution	9	7
Diseases	6	4
Miscellaneous[a]	3	2

[a] Visceral gout, egg bound, drowning.

Trauma. Trauma, excluding gunshot, was the second most frequent (26%) finding. Types of trauma suggested by information provided by the submitter or lesions observed at necropsy included collision with a moving vehicle or stationary object, being struck by a blunt object, and non-gunshot puncture wounds. Extensive skin and feather damage were noted in eight owls and skeletal fractures in 17. Hemorrhage, most frequently of the head, body cavity, and air sacs, was present in 21 of the 35 trauma cases. Internal organs including liver, spleen, duodenum, and stomach were ruptured in six carcasses. Concurrent abnormalities, including lesions of owl herpesvirus, renal gout, septicemia, and pododermatitis, were noted in four of the birds that died from trauma. These findings did not alter the cause of death as trauma, but may have rendered the owls more susceptible to traumatic injuries.

Gunshot. In 12 (9%) of the carcasses examined, gunshot wounds were diagnosed based on the presence of shot in association with fractures, recent hemorrhage, and other trauma or when tracts indicating the

path of a bullet or pellet could be identified. Seven were shot with a rifle, three with a shotgun, and two with an undetermined type of weapon. The frequency of cases with physical injury as the primary diagnosis (trauma plus gunshot) was 36%, somewhat less than the 43% reported in barn owls from England (Cooper 1993).

Toxicoses. Toxicoses were identified in 11 (8%) great horned owls. Hydrogen sulfide poisoning was diagnosed in five owls collected in North Dakota oil fields in 1982. The owls were found near flare or vent pipes, perhaps used as perches, designed to burn off or vent natural gases released during crude oil production, storage, and pipeline operations (Bicknell 1984). Hydrogen sulfide (1.5–4.0 ppm wet weight) was found in pulmonary fluid of each carcass. Hydrogen sulfide acts as a direct irritant, producing a chemical pneumonitis, and combines with and inhibits metabolic enzymes (Robinson et al. 1990, Short and Edwards 1989). Inhibition of the central nervous system respiratory drive produces apnea, the major cause of death (Warenycia et al. 1989).

Poisonings by phorate, fenthion, and an unidentified organophosphorus compound were diagnosed in three owls. One owl was found dead in South Dakota in 1982 in association with over 275 other dead birds, primarily ducks. Brain cholinesterase activity in this bird was inhibited by 83% and phorate (Thimet®) residues, 200 ppm wet weight, were recovered from stomach contents that consisted of feathers and grain. Phorate poisoning was also diagnosed in several of the ducks, and the owl apparently fed on those carcasses. Another great horned owl found dead in 1993 in Missouri had a 98% inhibition of brain cholinesterase activity and its stomach contents, consisting primarily of avian remains, contained 14.7 ppm wet weight fenthion. Brain cholinesterase activity in a third great horned owl, found in Utah in 1991, was inhibited by 88% without reversal after incubation of the sample. These findings are consistent with exposure to an organophosphorus pesticide (Hill and Fleming 1982, Smith et al. 1995) but, because the avian remains found in the stomach were not analyzed, the specific compound was not identified. Secondary poisoning by organophosphorus compounds, including fenthion, has been previously reported in several species of raptors (Henny et al. 1985, 1987, Bruggers et al. 1989, Hunt et al. 1991).

The brain cholinesterase activity of an owl found dead in Delaware in 1989 was within the normal range, but carbofuran (4.6 ppm wet weight) was recovered from the feathers and flesh found in its stomach. The carcass was partially decomposed and the lack of cholinesterase inhibition was probably the result of postmortem reactivation of the enzyme (Hill and Fleming 1982), one of the factors that contribute to a lack of correlation between cholinesterase inhibition and carbamate exposure (Greig-Smith 1991). Two birds died of poisoning by chlorinated hydrocarbons (endrin and chlordane) and those cases are described elsewhere (Blus et al. 1983a, 1983b).

Electrocution. Nine (7%) great horned owls died of electrocution, including three from Colorado, two from Oregon, two from Illinois, and one each from Arkansas and Wisconsin. The frequency of electrocution in this group of owls is considerably lower than the rates of 12% and 25% reported for bald eagles (*Haliaeetus leucocephalus*) and golden eagles (*Aquila chrysaetos*), respectively (Franson et al. 1995).

Infectious Diseases. Infectious diseases were diagnosed in six (4%) of the great horned owls. Two owls found dead in Nebraska in 1992 had gross and microscopic lesions consistent with owl herpesvirus infection (Green and Shillinger 1936, Sileo et al. 1975). Nodular lesions characteristic of avian tuberculosis were found in the liver and spleen of an owl collected in Nevada in 1984 and one found in Nebraska in 1987. Acid-fast bacteria, consistent with tuberculosis, were seen microscopically in impression smears of tissues and *Mycobacterium avium* was isolated from the liver of both birds. Bacterial pneumonia was diagnosed histologically in an owl found in 1980 in Oregon, but bacterial cultures of lung were negative. In 1980 an owl from Wisconsin was found to have numerous small abscesses in its enlarged liver and spleen and bacterial culture of spleen yielded heavy growth of *Staphylococcus aureus.*

Miscellaneous. Unusual diagnoses were reached in three of the 132 cases reviewed. One adult female had extensive bruising and tissue damage surrounding a fully formed egg in the distal oviduct, and apparently was egg bound. Drowning was diagnosed upon discovering water in the anterior thoracic air sacs of another owl. Severe visceral gout characterized by urate deposits within the kidneys and throughout the intestinal tract was found in a third owl.

Cause of death was not determined in 14 (11%) of the great horned owl carcasses examined. All of the carcasses included in this category were found to have adequate stores of body fat and no evidence of trauma. In five of the birds, no testing beyond gross inspection at necropsy was conducted. Liver lead concentrations

were determined in five cases and brain cholinesterase activities were evaluated in two of the 14 birds, but those results were within normal limits. Isolation of infectious agents was attempted in six of the 14 cases, but proved unsuccessful.

The significance of these results and their impact on great horned owl populations are difficult to evaluate because of the retrospective nature of the study and the nonrandom carcass collection techniques. However, this report does serve to identify major diagnostic findings in great horned owls. Increasing educational efforts may help mitigate causes of mortality related to human activities. More judicious use and monitoring of pesticides should help prevent poisonings and modifications to structures such as power lines and utility poles may reduce electrocution and trauma mortalities. Emaciation of undetermined etiology is a significant finding and should be further evaluated. A study designed to examine emaciation in great horned owls with regard to age, food availability, and the temporal and geographic distribution of emaciated birds may help to identify potential causes of emaciation.

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