

# Causes of Mortality in Alaskan Bald Eagles

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The U.S. Fish and Wildlife Service has been documenting the causes of death in Bald Eagles (*Haliaeetus leucocephalus*) from all parts of the United States including Alaska for more than 25 years. The process involves conducting a complete postmortem examination called a necropsy. A necropsy provides a more accurate assessment of the cause of death than would reliance on external appearances or field circumstances alone.

Problems with environmental contaminants and dwindling populations often focus attention on the causes of Bald Eagle deaths in the Lower 48 states. In contrast, understanding the causes of Alaskan Bald Eagle deaths is of particular interest because of the unique population status of eagles in Alaska: They are a stable population, not listed by the Federal Government as threatened or endangered and they're in a relatively contaminant-free environment. The following report summarizes the necropsy findings from 344 Alaskan Bald Eagles.

## **Methods**

From 1975 through February 1989, 344 Alaskan Bald Eagles collected in the wild were submitted to the National Wildlife Health Research Center, Madison, Wisconsin for necropsy (Figure 1). All birds were collected prior to the *Exxon Valdez* oil spill. Necropsy results from 27 eagles collected prior to 1975 were published previously (Coon et al. 1970, Mulhern et al. 1970, Prouty et al. 1977). Any Alaskan Bald Eagle found dead in the wild during the study period was accepted for necropsy; however, it is unlikely that every dead Bald Eagle found was submitted. Bald Eagles found sick or injured and treated for a short period prior to death were also included. Each bird was assigned to one of seven collection locations representing census areas (Figure 2). Carcasses were generally frozen and submitted within weeks or months of collection. Eleven different pathologists performed the examinations over the 15 year period of this study.

The necropsy entailed a methodical and complete examination of all external structures, all internal organs and the selection of appropriate tissue samples for laboratory tests as indicated by examination findings or history (Whiteman and Bickford 1989). Available laboratory tests included bacterial, fungal or viral isolations; parasite collection and identification; histopathology (microscopic examination of tissues); and chemical analysis for selected poisons or environmental contaminants. At the conclusion of all laboratory tests, the results of the necropsy and supportive tests were considered together to reach a knowledgeable conclusion regarding the cause of death. We determined the primary cause of the bird's death to be the initial factor responsible for its demise, not a complication of an original problem. A gunshot wound that resulted in a fatal infection,

for example, would be listed as a gunshot mortality.

Bonferroni simultaneous confidence intervals were used to identify differences in the proportionate causes of death between the sexes or age groups (Kirk 1982). The chi-square test was used to compare body condition indices or the proportionate causes of death at different collection locations (Daniel 1978).

## Number of Bald Eagles

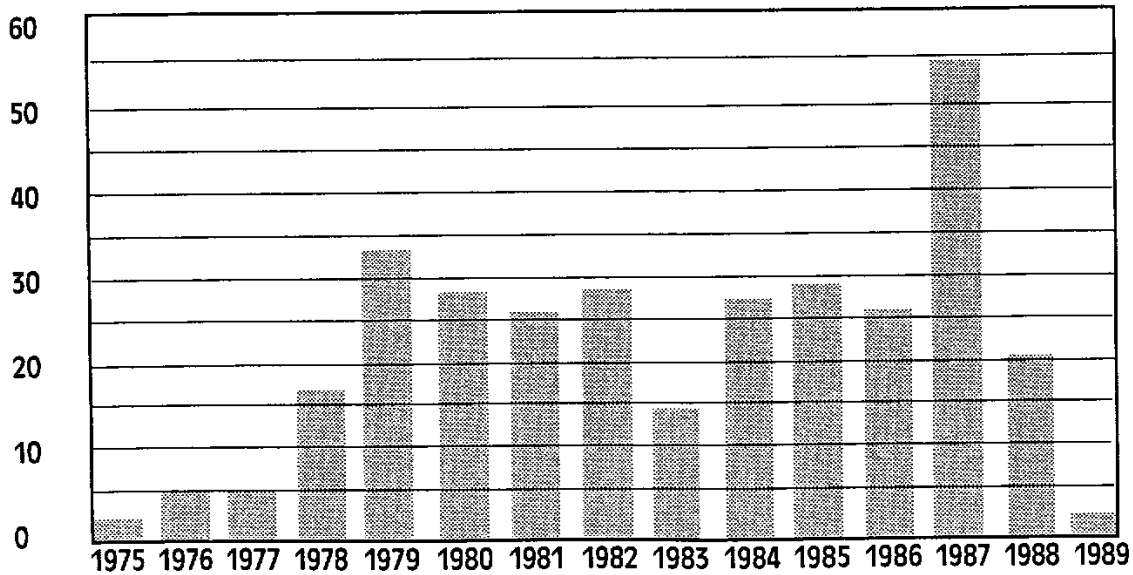


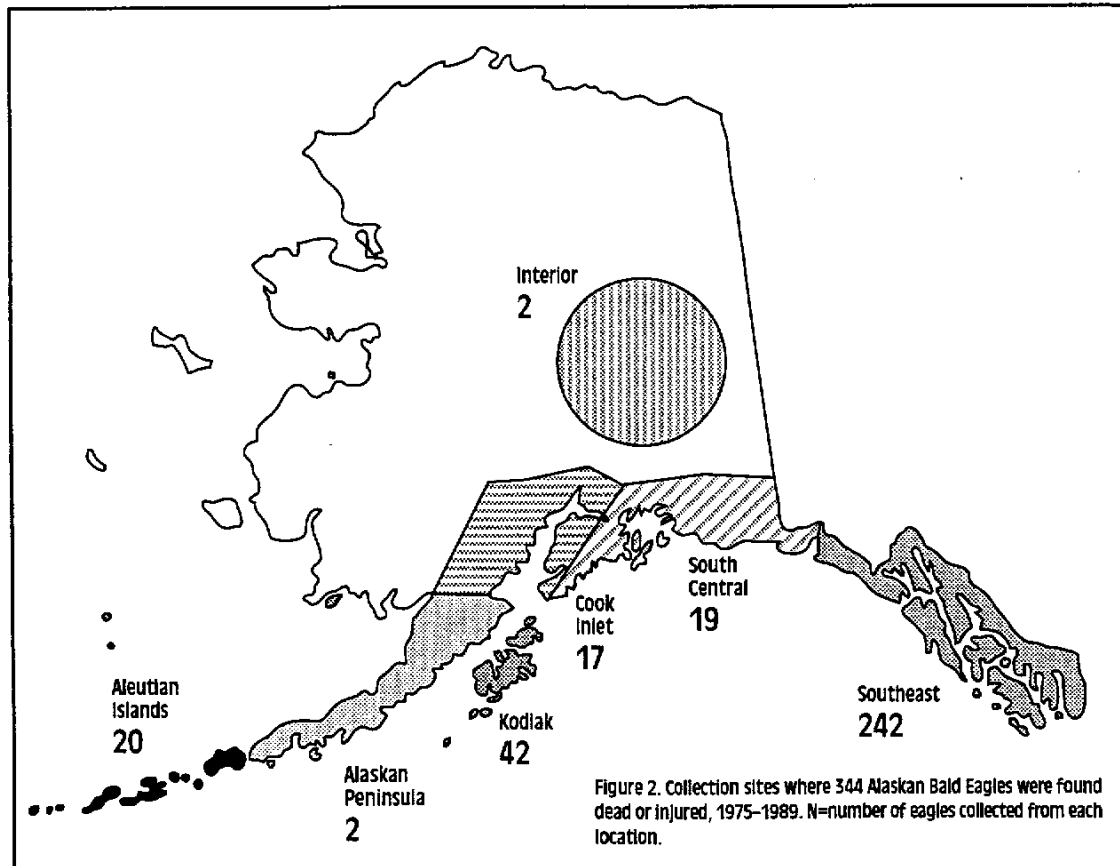
Figure 1. Collection date (year) of 322 Alaskan Bald Eagles, 1975-1989. (Twenty-two eagles with no collection date record are omitted).

## Results and Discussion

Certainly every Alaskan Bald Eagle that died during the 15 years of the study is not present in the data set; this fact must be considered in interpreting the results. Public use of an area and public interest can affect the likelihood of a carcass being found and submitted. The mortality factor itself may influence collection probability; for example, an eagle that dies immediately after being struck by a vehicle is more likely to be found than a sick debilitated bird that can hide in remote habitat. However, the data set does identify Bald Eagle mortality factors and documents the minimum number of occurrences.

The causes of mortality by sex and age class for 344 Alaskan Bald Eagles are presented in Table 1. Fifty-six percent of the eagles submitted were males. Sixty-one percent had primarily white head and tail plumage (non-juvenile plumage). There were no significant differences in the causes of death of males versus females ( $P>0.1$ ) or adults versus non-adults ( $P>0.1$ ), so all groups were combined (Figure 3). The causes of death and their proportions were similar to those reviewed by Schmeling and Locke (1982) in reporting the results from examinations of 87 dead eagles early in the study. Statistical comparisons of the causes of death were made among only five collection locations and among the four most frequent diagnoses because only small numbers of birds fell into the other

categories (Table 2). The proportion of electrocution, emaciation and gunshot diagnoses varied significantly ( $P < 0.01$ ) among the collection sites.



The four most frequent diagnoses of trauma, electrocution, emaciation and gunshot accounted for the deaths of 70% of the eagles submitted. Trauma was the single most frequent diagnosis (24.7%). Trauma was diagnosed at similar rates ( $P=0.47$ ) in all five collection sites. Eagles that died from traumatic injuries most often showed the effects of blunt or impact trauma, such as fractured bones or internal hemorrhage. The exact cause of the injuries usually could not be identified by the wounds but the case histories often suggested these birds were hit by vehicles or collided with power lines or power poles. Trauma diagnoses also included birds with injuries that suggested aggressive encounters with other raptors, probably other eagles. Wounds in these cases consisted of relatively shallow punctures that were often associated with extensive muscle tearing or shredding, hemorrhage and sometimes fractures or bone perforations. These injuries were distinctly different from the deeply penetrating tracts of gunshot wounds. Aggression wounds had a somewhat characteristic anatomic distribution that could include the upper shoulders, back of the neck, legs below the knees, head and face and pectoral region.

Table 1. Causes of mortality in 344 Alaskan Bald Eagles, 1975-1989.

Cause of Death	Frequency		Sex*				Age*			
			Male		Female		Adult		Juvenile	
	No.	Percent	No.	Percent	No.	Percent	No.	Percent	No.	Percent
Trauma	85	24.7	44	24.7	36	25.9	57	5	27	20.5
Electrocution	60	17.5	33	18.5	26	18.7	32	15.5	28	21.2
Emaciation	53	15.4	33	18.5	18	13.0	31	15.1	21	15.9
Gunshot	44	12.8	20	11.3	17	12.2	30	14.6	14	10.6
Poisoning	25	7.3	11	6.2	14	10.1	14	6.8	11	8.3
Infectious Disease	13	3.8	5	2.8	6	4.3	5	2.4	7	5.3
Trapping	7	2.0	5	2.8	1	0.7	5	2.4	2	1.5
Other	57	16.5	25	15.2	21	15.0	32	15.5	22	16.7
Total	344	100.0	178	100.0	139	100.0	206	100.0	132	100.0

\*Individuals in which sex/age were not recorded are omitted. There were no significant ( $P>0.1$ ) differences in the causes of death of males versus females or adults versus juveniles.

+Percent of eagles per sex or age group.

Table 2. Causes of mortality in 340 Alaskan Bald Eagles from five collection sites, 1975-1989.

Cause of death	Frequency									
	Southeast		Kodiak		Aleutians		South Central		Cook Inlet	
	No.	Percent	No.	Percent	No.	Percent	No.	Percent	No.	Percent+
Trauma+	57	23.5	12	28.5	5	25.0*	7	36.8	2	11.8
Electrocution+	43	17.8	3	7.1	12	60.0*	0	0.0	2	11.8
Emaciation+	48	19.8	2	4.8	1	5.0	1	5.3	0	0.0
Gunshot+	23	9.5	13	30.9	0	0.0	1	5.3	7	41.2
Poisoning	15	6.2	2	4.8	1	5.0	7	36.8	0	0.0
Infectious	11	4.5	2	4.8	0	0.0*	0	0.0	0	0.0
Trapping	6	2.5	1	2.4	0	0	0	0	0	0.09
Other	39	16.2	7	16.7	1	5.0	3	15.8	6	35.2
Total	242	100.0	42	100.0	20	100.0*	19	100.0	17	100.0%

\*Four eagles collected from the Alaska Peninsula and Interior Alaska are omitted because of the small sample size at these sites.

+Collection sites were compared by the chi-square test. Emaciation, electrocution and gunshot varied significantly ( $P<0.01$ ) among the collection sites; trauma did not vary significantly.

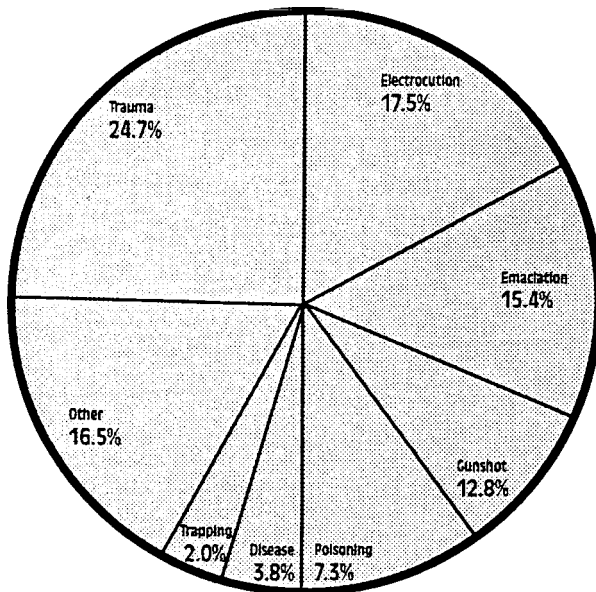


Figure 3. Proportionate causes of mortality in 344 Alaskan Bald Eagles, 1975-1989.

Electrocution was the second most frequent diagnosis (17.5%) among Alaskan eagles. The key to identifying this cause of death was the recognition of burns which could be as subtle as a small perforation in feathers or minute blisters on the feet. Electrocuted eagles were collected at the highest rate (60.0%) in the Aleutian Islands. However, electrocution also caused the death of a substantial number of eagles in Southeast Alaska and occurred in other locations as well.

Raptor electrocution is a manageable problem that can be virtually eliminated by power line or pole modifications and standards for new power line installations (Olendorff et al. 1981). The problem of Bald Eagle electrocution at the military base on Adak Island has been well documented and successfully addressed (see Byrd and Williams 2008).

Emaciation was found in 15.4% of the eagle carcasses examined. This diagnosis was reserved for cases in which there was no apparent physical explanation for the bird's debilitated condition. Birds diagnosed as emaciated had no evidence of crippling injuries that might have caused starvation. No toxicity was found despite the fact that most were tested for a variety of toxic compounds that can lead to emaciation. (Toxicological tests were not available for all emaciated eagles but 87% of the emaciated eagle carcasses were tested for lead; 51% for various organochlorine compounds such as "DDT", dieldrin, endrin and related compounds; and 23% for toxic levels of heavy metals other than lead.)

Eagle deaths that were attributed to emaciation occurred seasonally (Figure 4). The majority (83%) of the emaciated eagles were found dead from February through July. Emaciation was a consistent diagnosis, found in each of the 11 years from 1978 through 1988. Emaciation from unknown causes was diagnosed at a higher rate (19.8%) in Bald Eagles from Southeast Alaska than in eagles from other locations (0-5.3%).

No single explanation for the cause of emaciation in Alaskan Bald Eagles emerged from the data. A number of problems can be manifested as emaciation. Any individual bird could have had a metabolic or neurological problem that interfered with its normal food-



gathering functions without leaving anatomic clues visible at necropsy. Some birds could have been affected by toxicity from one or more unrecognized compounds. In Southeast Alaska there is potential environmental contamination from organic compounds such as polychlorinated biphenyl compounds (PCBs) associated with pulp mills or with heavy metals such as mercury, which is found in mine tailings or natural deposits. A third possibility is that these birds had an inadequate or inaccessible food supply. The seasonal occurrence of emaciation suggests that inclement weather could be a contributing factor.

This Bald Eagle was obviously this young person's prize and probably was shot to collect the bounty. Photo courtesy of Alaska State Library.

Gunshot mortalities were diagnosed in 12.8% of the eagles submitted. Birds in this category had crippling or fatal injuries characteristic of gunshot wounds. Many of the diagnoses were confirmed by the identification of metal fragments or shot pellets in the wound tissue or by radiograph. The proportion of gunshot mortalities was highest in eagles submitted from Kodiak Island (30.9%) and from Cook Inlet (41.2%). All of the gunshot mortalities from Cook Inlet were collected on the Kenai Peninsula. The gunshot birds from these two locations were collected during every month of the year except July

and August.

The remainder (30%) of the submitted eagles had diagnoses divided among poisoning, infectious diseases, trapping, or miscellaneous categories that were combined as "other." Poisoning caused the deaths of 7.3% of the Bald Eagles examined. The majority of these poisoned birds (16 of 25) died from lead poisoning. Generally the source for Bald Eagle lead poisoning is assumed to be lead shot ingested with the tissues of prey, particularly waterfowl (Pattee and Hennes 1983). The stomachs of lead poisoned eagles are usually empty so there is no specific clue to the source of the lead, but occasionally the birds' stomachs contain lead shot or lead fragments. The 16 lead poisoned Alaskan Bald Eagles all had empty stomachs, but there was some circumstantial evidence suggesting lead shot caused the poisonings. In documenting over 200 Bald Eagle lead poisonings throughout the United States, we found that these cases peak following fall hunting season and occur only from October through June. Alaskan Bald Eagle lead poisonings occurred only from December through June (Figure 5). The majority (13 of 16) of the lead poisoned birds were collected in Southeast Alaska where Bald Eagles prey heavily on waterfowl from October through April (Imler and Kalmbach 1955). Alternatively there has been concern that environmental contamination from lead ore in Southeast Alaska presents a toxic hazard to wildlife. Since there is evidence that bioaccumulation of lead through tissues does not cause raptor lead poisoning (Pattee and Hennes 1983, Custer et al. 1984), it is generally assumed that eagles must ingest the highly concentrated metal itself to become poisoned. What is not clear is how spilled lead ore could become directly accessible to eagles.

Several Bald Eagle poisonings were caused by compounds other than lead. Two eagles died from strychnine poisoning in the Ketchikan area during summer 1979, but we have no further details on the circumstances surrounding this incident. On Kodiak Island in 1987, two Bald Eagles died and two were reported sick with barbiturate poisoning. These birds had been scavenging on the carcass of a horse that had been euthanized with pentobarbital. Five other Bald Eagles died from barbiturate poisoning during one episode on Spike Island in southcentral Alaska. Thirty-eight percent of the 344 eagles examined were screened for organochlorine compounds but none were found to have died from organochlorine poisoning (Kaiser et al. 1980, Reichel et al. 1984, Patuxent Wildlife Research Center, unpubl. data).

Infectious diseases caused the deaths of 3.8% of the Alaskan Bald Eagles examined. The single most common disease identified was avian pox, found in six eagles collected in Southeast Alaska and on Kodiak Island. Pox infection in Alaskan eagles was reported previously (Schmeling and Docherty 1982). Avian pox is a viral disease which can cause massive eruption of proliferative skin nodules, particularly in non-feathered skin. The nodules can impair vision and hearing and lead to severe debilitation or secondary bacterial infections. As in many other viral diseases, pox is more common in young immunologically naive birds, so this disease poses a particularly serious threat to translocation projects. The early lesion of avian pox may be only an unapparent small blister. If unrecognized, the disease may spread rapidly by contact among all birds in a hacking tower. If infected birds are released, pox may spread to other wild free-flying

eagles in the area. Even if the infected birds are not released, the disease may be spread to wild birds via mechanical transmission by insects (Karstad 1971).

### Number of Emaciated Bald Eagles

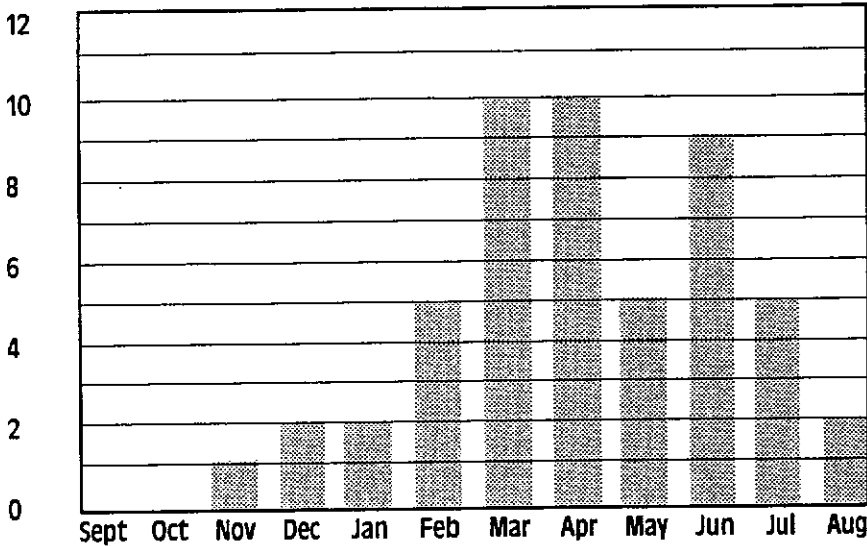


Figure 4. Collection date (month) of 51 emaciated Alaskan Bald Eagles (two emaciated eagles with no collection date record are omitted).

### Number of Lead Poisoned Bald Eagles

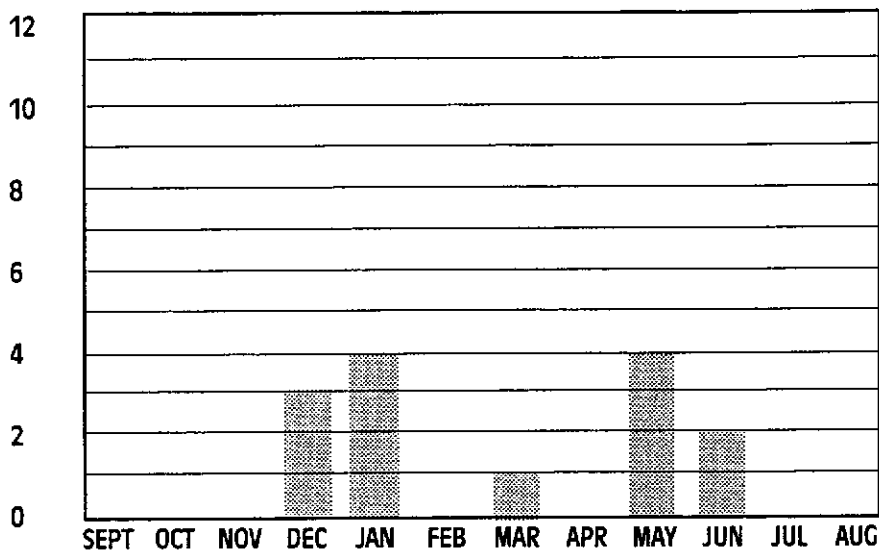


Figure 5. Collection date (month) of 14 lead poisoned Alaskan Bald Eagles. Two lead poisoned eagles with no collection date record are omitted.

Additional infectious diseases diagnosed in the Alaskan Bald Eagles examined included fungal and bacterial infections. Three eagles died from aspergillosis, a respiratory infection named after the causative agents, fungi of the genus *Aspergillus*. This disease is most common in eagles held in close confinement; however, only one of these three birds had any history of captivity for rehabilitation. Three eagles died from bacterial infections that were either generalized or confined to a joint. The bacterium identified in two of these birds was *Staphylococcus aureus*, a bacterium that may gain entry through skin wounds and tends to localize in the joints of young birds. One eagle had both a bacterial



joint infection and avian pox.

Accidental trapping in leghold traps caused the deaths of 2.0% of the Bald Eagles submitted. Trapped eagles had constrictive or lacerating trap injuries to toes or legs. Some birds had additional physical complications from capture such as abrasions, bruising, evidence of drowning or emaciation.

The last diagnostic category, a miscellaneous category called "other," included 16.5% of the Alaskan Bald Eagles submitted. Approximately one-third of these carcasses were too decomposed to allow a meaningful examination. In another one-third no cause of death could be identified; and, in fact, the common causes of death were ruled out in these birds. The remaining one-third of eagles in the "other" category had complicated diagnoses in which the causes of their conditions could be one of several factors or several factors combined. Included in this category were eagles that died with visceral gout, the final condition produced by any of a variety of toxic, infectious or even nutritional factors that cause kidney damage.

The proportionate causes of death for Alaskan Bald Eagles were compared with similar data for 1919 other Bald Eagles collected in the Lower 48 states (Figures 3 and 6). Both data sets were similar. In both cases trauma was the most frequent diagnosis and trauma along with electrocution and gunshot comprised three of the four most frequent mortality factors. There were, however, two notable differences in the data sets. Emaciation was diagnosed in Alaskan Bald Eagles at almost three times the rate for eagles in the Lower 48 states. To examine this difference a little further, we also compared the adequacy of fat and muscle deposits described at necropsy in Alaskan Bald Eagles versus that in eagles from the Lower 48 states. We confined the comparison to the mortality factors that were not expected to be debilitating. Although 72% of the Alaskan Bald Eagles dying from trauma, electrocution and gunshot injuries were in normal body condition, a significantly ( $P < 0.002$ ) greater proportion (82%) of the eagles dying from the same factors in the Lower 48 states were in normal body condition. This finding suggests that the general state of nutrition of Alaskan Bald Eagles may be relatively poor in comparison to eagles in the Lower 48.

In the second notable difference, poisoning was diagnosed at more than twice the rate in Bald Eagles in the Lower 48 states than it was in eagles from Alaska. Approximately one-half of the poisonings in the Lower 48 states were caused by lead and the majority of the remainder were due to pesticides. The historic problems with organochlorine compounds such as DDT were represented in the earlier period of the data set, however, recent poisonings in the Lower 48 states were often due to the organophosphorus or carbonated pesticides, the pesticides that replaced "DDT." Although the compounds used in later years break down relatively rapidly in the environment, birds including Bald Eagles are exquisitely sensitive to many of them. These new pesticides are the documented cause of more than 100 Bald Eagle deaths, usually subsequent to the eagles' feeding on carrion or bait in secondary poisoning episodes. Fortunately it appears that Alaska has been spared this problem to date, due to the sparsity of agricultural pesticide use.

Documentation of the causes of Alaskan Bald Eagle "sea" deaths over the past 15 years has provided an extensive database for future reference and has identified several problems pertinent to the management of this species. Losses of eagles due to electrocution have been greatly reduced in the Aleutian Islands by developing raptor-safe power line and power pole configurations. Similar management techniques could be successful in other areas. Because avian pox poses a threat to the success of translocation projects, particular caution must be applied to screening or quarantining eagles donated to such programs. Cases with law enforcement implications such as gunshot or trapping mortalities are probably underrepresented in this study since the carcasses of birds killed by these means may be purposely hidden. The numbers in this data set could indicate just the tip of the iceberg.

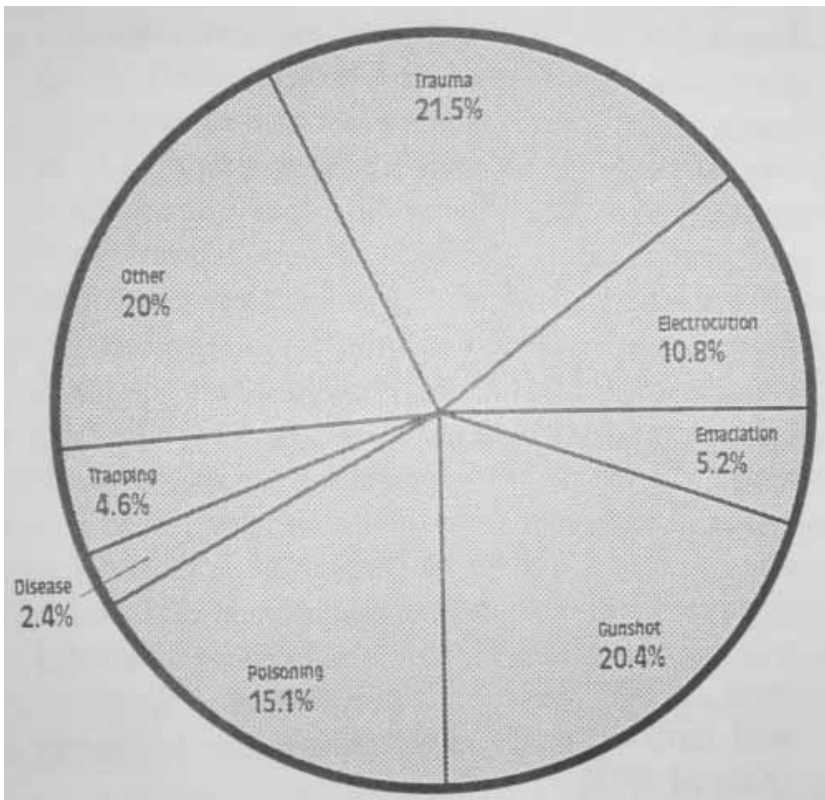


Figure 6. Proportionate causes of mortality in Bald Eagles collected in the contiguous United States, 1975-1988. N=1919.

These data also raise a number of interesting questions. What is the source of the lead that poisons Alaskan Bald Eagles? There are similarities to lead poisoning patterns in the remainder of the United States, but, alternatively, unique environmental sources may exist in Alaska. Why does emaciation appear to be particularly common in Bald Eagles of Southeast Alaska? As some other papers in this volume suggest, nutrition, toxins or other factors may be affecting the body condition or reducing the survival of Alaskan Bald Eagles. These and other questions will be answered only by future investigations.

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