

CAUSES OF MORTALITY IN COMMON LOONS

J. Christian Franson
U.S. Fish and Wildlife Service
6006 Schroeder Road, Madison, Wisconsin 53711

David J. Cliplef
Western College of Veterinary Medicine
Saskatoon Saskatchewan, Canada S7N 0W0

ABSTRACT

Summarized are necropsy results from 222 carcasses of Common Loons (*Gavia immer*) submitted to the National Wildlife Health Research Center from 1976 through 1991. The carcasses were from 18 states, and 10 or more birds each were from Minnesota, Florida, Virginia, Michigan, Wisconsin, Maine, and North Carolina. Seventy-three (33%) carcasses were emaciated, and in some of these birds emaciation was thought to be related to exposure to mercury. Over 40% of these emaciated birds were from Florida. Trauma, including blunt trauma of unknown origin, outboard motor propeller wounds, and shooting caused the deaths of 49 (22%) loons, 30 of which were from Minnesota. Diseases, primarily avian botulism type E and aspergillosis, accounted for 39 (18%) mortalities and lead poisoning for 14 (6%), 11 of which had fishing sinkers in their stomachs. Most of the avian botulism type E cases occurred during two outbreaks on Lake Michigan. Seven of the 14 lead-poisoned birds were from Minnesota. Nine (4%) birds died of miscellaneous causes and 9 (4%) of drowning, primarily from entanglement in nets. No diagnosis could be reached for 29 (13%) carcasses. Sample bias precludes interpretation of these data to represent actual proportional causes of mortality in the loon population. However, the sample size is sufficient to clearly identify major causes of mortality.

INTRODUCTION

The Common Loon (*Gavia immer*) is a bird of great mystique and charisma and has been the subject of numerous popular articles and books. Its presence on northern lakes is treasured by many as part of the wilderness experience. Its health and survival has become a focus of effort for many individuals and conservation groups. Part of this effort is based on the recognition that loons may be in jeopardy as a result of contaminants and other human-related mortality factors. For example, mercury exposure from consumption of contaminated prey and lead poisoning, probably resulting from ingestion of fishing sinkers, is a concern that has been identified in Minnesota (Ensor et al., 1992). Pokras and Chafel (1992) found lead poisoning caused by fishing sinkers in 16 of 31 adult loons collected in New England. In an attempt to identify major mortality factors that may be important to loon populations we report here the findings of postmortem examinations of 222 Common Loons, collected during 1976-1991 from several states.

METHODS

From 1976 through 1991, 222 Common Loon carcasses were examined at the National Wildlife Health Research Center, Madison, Wisconsin to determine cause of death (Figure 1). The loons were collected by personnel of the U. S. Fish and Wildlife Service, State Fish and Game Departments, and other agencies. Most birds were found dead, but some that were found sick or injured and were treated in rehabilitation centers but later died, were also examined. Necropsies were conducted by 12 pathologists over the 16-year period covered by this report.

A thorough postmortem examination of each carcass was conducted and tissues were collected for evaluation using standard methods in microbiology, virology, parasitology, toxicology, and histopathology. Testing was guided by lesions observed at necropsy and the history presented with the carcass. The history, necropsy findings, and laboratory results were considered in making conclusions about the cause of death. Cause of death was determined to be the condition responsible for the removal of the bird from the wild as opposed to complications which may have developed secondarily. Differences in proportional mortality between sexes and age groups were tested using contingency tables (Daniel, 1978). Fisher's exact test (Zar, 1984) was used to compare age group difference for specific causes of mortality.

RESULTS AND DISCUSSION

The 222 carcasses were from 18 states, but Minnesota and Florida accounted for 113 (50%) of the submissions (Table 1). Other states from which 10 or more birds were submitted included Virginia (20), Michigan (19), Wisconsin (17), Maine (15), and North Carolina (13). Age and sex were determined for 208 and 210 carcasses, respectively. The sex ratio was virtually 1:1 (104 males and 106 females); 144 (69%) of the birds were adults. Identified causes of mortality were grouped into six categories: emaciation, trauma, microbial disease, lead poisoning, drowning, and miscellaneous (Table 1). There was no significant difference ($P > 0.2$) in the proportional causes of mortality between males and females. However, there was a difference ($P = 0.056$) in proportional mortality between adults and juveniles with regard to lead poisoning, emaciation, and microbial disease. These differences are identified to indicate possible trends, but more data are needed to clarify the biological significance of these differences. There were no significant differences ($P > 0.15$) between adults and juveniles for the other causes of mortality.

Emaciation

Emaciation was the most commonly diagnosed condition, and was found in 73 (33%) of all loons examined. Emaciation was diagnosed in birds with severe breast muscle atrophy and a complete lack of subcutaneous, coronary, and mesenteric fat and was observed in a significantly ($P = 0.051$) greater percentage of juveniles (40.6%) than adults (26.6%). In seven (10%) of these cases the postmortem condition of the carcass was poor, preventing adequate microbiologic and histopathologic evaluation. In 19 (26%) of the emaciated birds no other lesions were found. The remaining 47 (64%) had lesions that were thought to be incidental findings or that may have been associated with the bird's poor body condition, but were not the primary cause of emaciation. One of the emaciated loons had elevated tissue levels of DDE but not sufficient to have caused death. Emaciation was most frequent in carcasses found in

late winter and spring and lowest in the summer and fall. Thirty-one (42%) of the emaciated carcasses were from Florida (Table 1) and of the states submitting ten or more loons, Florida had the greatest proportionate mortality associated with emaciation (86%) followed by North Carolina (69%), Virginia (30%), Wisconsin (24%), Maine (20%), and Minnesota (18%).

Most of the emaciated loons from Florida were submitted during a die-off of loons in 1983 and during continued elevated mortality of loons along the Gulf Coast in subsequent years. From January to March of 1983, an estimated 2,500 loons died along the coasts of Mississippi and Florida. The most consistent findings were severe emaciation, an incomplete molt, anemia, hemorrhagic enteritis, large numbers of trematodes in the small intestine, and elevated mercury concentrations in tissues of emaciated birds. Mean liver mercury in emaciated birds was 24 parts per million (ppm), wet weight (range 3 to 90 ppm), but was less than 4 ppm in birds in good flesh (Lange and Stroud, unpubl. report). These results are similar to those reported by Ensor et al. (1992) who found that emaciated loons collected in Minnesota had significantly elevated mercury concentrations in livers and feathers. Injured and emaciated loons in that study had a mean liver concentration of 29 ppm mercury, wet weight. It has been suggested that a liver mercury concentration of 20 ppm, wet weight, represents a toxic exposure (Finley et al., 1979; Fimreite and Karstad, 1971). However, mercury levels in tissues are difficult to interpret because of species differences (Eisler, 1987). Liver mercury concentrations associated with toxicity ranged from 17 ppm (wet or dry weight not stated) in red-tailed hawks (Buteo jamaicensis) (Fimreite and Karstad, 1971) to over 700 ppm, dry weight, in grey herons (Ardea cinerea) (Van der Molen et al., 1982), and the mean liver mercury concentration of five apparently healthy loons from Ontario was 43 ppm, wet weight (Fimreite, 1974). A diagnosis of mercury poisoning requires the demonstration of gross and microscopic findings (Fimreite and Karstad, 1971; Heinz and Locke, 1976), combined with analytical results. Some of the emaciated loons reported here showed evidence of nerve degeneration and demyelination but not enough to allow a definitive diagnosis of mercury poisoning. Mercury has also been shown to cause sublethal effects, including decreased hatching success in loons (Fimreite, 1979) and adverse effects on reproduction and behavior in mallards (Heinz, 1979).

Trauma

Trauma was found in 49 loons and was the second most frequent (22%) cause of mortality. These cases included blunt trauma of unknown origin, outboard motor propeller lacerations, gunshot, wounds from other animals, and entanglement. The state with the most trauma cases (30) was Minnesota (Table 1) and trauma was the leading cause of death in Minnesota, Wisconsin, and Maine. Blunt trauma was diagnosed in 22 (45%) of the trauma cases based on the presence of fractured bones, bruising of various tissues and organs, and hemorrhage. Some of these lesions were likely the result of collisions with boats; propeller lacerations, indicated by deep gashes on the backs of carcasses, occurred in five (10%) trauma cases. Twelve cases (25%) were determined to be the result of gunshot, nine of which occurred in Minnesota. Of six loons from Minnesota exhibiting evidence of animal aggression three were found to have deep puncture wounds thought to have been caused by other loons, two had bite wounds from a small mammal, and one had a pattern of probable talon punctures from an avian predator. The remaining four trauma cases were also from Minnesota and were birds that had become entangled in fishing lines and eventually died of starvation. Most (61%) of the trauma cases were collected in the summer months (Figure 2) which supports the possible correlation with boating and fishing activities.

Microbial Disease

Loons that died of microbial disease were submitted from six states (Table 1). This was the third most frequent (18%) cause of mortality, with 39 cases, and was seen in a significantly ($P = 0.081$) greater percentage of adults (21.7%) than juveniles (10.9%). Fifteen of these cases were caused by avian botulism type E, one was caused by type C botulism, 13 by aspergillosis, and 10 by an assortment of infections. Fourteen of the disease cases were collected in the summer months of June, July, and August, and 16 were collected in November, most of which were associated with avian botulism type E die-offs (Brand et al., 1988).

Avian botulism results from the ingestion of toxin produced by the bacterium Clostridium botulinum, and causes death without producing any visible lesions. This bacterium produces several types of toxins, two of which, C and E, cause epizootics in wild birds. Type C botulism typically occurs during late summer in ducks and other water birds. One loon from Minnesota died in August of type C botulism. Epizootics of type E botulism are more common in late fall in the Great Lakes Basin, particularly in Lake Michigan, and affect primarily loons, gulls, and diving ducks (Brand et al, 1988). Two such epizootics resulted in diagnosis of type E botulism in 15 loons from Michigan and Indiana collected in October and November. The first die-off occurred in 1976, when several hundred loons were picked up along the Michigan and Indiana shores of Lake Michigan (Brand et al., 1983). The second occurred in 1983, when an estimated 592 loons died along the Garden Peninsula of Lake Michigan (Brand et al., 1988). It is probable that loon deaths from type E botulism result from the consumption of fish containing type E toxin.

The 13 aspergillosis cases represented one-third of all disease mortality and were often characterized by emaciation with fungal growth in the lungs and air sacs, from which Aspergillus sp. was cultured. The remaining disease cases included pneumonia (4), enteritis (3), pericarditis (1), septicemia (1), and myositis (1). Organisms cultured included Enterobacter sp., Escherichia coli, and Klebsiella sp.

Lead Poisoning

Lead poisoning was observed in 14 (10%) cases from five states (Table 1) and was diagnosed significantly ($P = 0.069$) more frequently in adults (8.4%) than juveniles (1.6%). Carcasses were typically in poor body condition or emaciated and 11 had lead fishing weights in their stomachs; one additional lead-poisoned bird had fishing line in its stomach. The other two cases were in poor body condition with elevated lead concentrations in the liver, but no lead was found in the stomach. Liver lead concentrations in these 14 cases ranged from 4.7 to 46.1 ppm, wet weight. A liver lead concentration of 4.7 ppm, wet weight, is less than that (8 ppm, wet weight) considered to be consistent with lead poisoning in waterfowl when supported by pathologic findings (Friend, 1985). However, it was the opinion of the pathologist that the loon died as the result of ingesting a lead sinker, found in the stomach, and ensuing toxicity resulting in poor body condition, renal failure, and elevated liver lead. All lead-poisoning cases occurred in birds collected from May through October, with 10 of the 14 cases noted in July, August, and September. This clumping of cases in the summer may be associated with fishing activity and the time required for toxicity to develop after lead ingestion. Lead poisoning affects individual birds and thus mortalities are not as visible as in epizootic diseases, such as avian botulism. Because large numbers of birds are not killed in one location, lead poisoning is less likely to be detected. Its actual significance as a mortality factor may be much higher than is indicated here. Indeed, Ensor et al. (1992) reported a lead-poisoning frequency of 17% in Minnesota, and Pokras and Chafel (1992) reported 50% in New England.

Drowning

Drowning was diagnosed in nine (4%) birds; four each from Virginia and Maryland and one from Wisconsin. Entanglement in nests was suspected in at least five of the drowning cases.

Miscellaneous

Oiling was diagnosed in one loon each from Alaska, Texas, and Virginia. The loon from Alaska was submitted during the Exxon Valdez oil spill, the Texas bird was found on a lake in an oil field, and the Virginia bird was found washed ashore. These carcasses had evidence of heavy oiling of feathers, ingestion of oil with probable toxic damage to organs, and were emaciated or in poor body condition.

Visceral gout occurred in two birds, indicating a primary kidney disease or dietary protein imbalance. One loon died of asphyxiation when a fish became lodged in the trachea, egg yolk peritonitis was diagnosed in one case, and one bird had a greatly enlarged colon. A piece of wire was embedded in the liver of one loon, apparently after penetration through the stomach wall.

Unknown

No cause of mortality could be determined for 29 (13%) loons from nine states. In 16 of these cases there were no findings despite various testing. The remaining 12 had incidental findings of little significance. The cause of death in these birds may have gone unrecognized because of poor postmortem condition or difficulties in culturing some pathogens from specimens that had been frozen for long periods before examination. Type E botulinum toxin is known to decompose rapidly in serum at refrigerated temperatures (Brand et al., 1988), and 15 of the unknown cases were submitted in association with the two type E botulism epizootics mentioned above.

CONCLUSIONS

We have identified several major mortality factors in Common Loons. The reader is cautioned, however, that the causes of mortality reported here cannot be interpreted to be representative of actual proportional causes of death in the loon population because of sample bias. The likelihood of a dead loon being found and submitted forms a major portion of this bias; factors include the remoteness of location, public use of the area where the mortality occurs, presence of interested or concerned persons, and the nature of the mortality factor itself.

ACKNOWLEDGEMENTS

We thank the personnel of various state and federal agencies, particularly D. Helwig and K. Ensor of the Minnesota Pollution Control Agency, for submitting the loons reported here. We also wish to thank L. N. Locke, S. M. Kerr, L. Sileo, R. E. Lange, Jr., R. K. Stroud, S. K. Schmeling, T. J. Roffe, P. A. Gullette, H. A. McAllister, R. Sullivan and C. J. Brand for conducting many of the postmortem examinations that generated these data. M. D. Samuel provided statistical consultation. Staff at the National Wildlife Health Research Center and Patuxent Wildlife Research Center performed numerous laboratory analyses and provided technical support. T. E. Rocke and K. Ensor reviewed the manuscript.

LITERATURE CITED

- Brand, C. J., R. M. Duncan, S. P. Garrow, D. Olson, and L. E. Schumann. 1983. Waterbird mortality from botulism type E in Lake Michigan: an update. *Wilson Bull.* 95:269-275.
- Brand, C. J., S. M. Schmitt, R. M. Duncan, and T. M. Cooley. 1988. An outbreak of type E botulism among Common Loons (*Gavia immer*) in Michigan's Upper Peninsula. *J. Wildl. Dis.* 24:471-476.
- Daniel, W. W. 1978. Applied nonparametric statistics. Houghton Mifflin Co., Boston. 503 pp.
- Eisler, R. 1987. Mercury hazards to fish, wildlife, and invertebrates: a synoptic review. U.S. Fish Wildl. Serv. Biol. Rep. 85(1.10). 90 pp.
- Ensor, K. L., D. D. Helwig, and L. C. Wemmer. 1992. Mercury and lead in Minnesota Common Loons (*Gavia immer*). Minnesota Pollution Control Agency, Minneapolis. 32 pp.
- Fimreite, N. 1979. Accumulation and effects of mercury on birds. Pages 601-627 in J.O. Nriagu (ed.). *The biogeochemistry of mercury in the environment.* Elsevier/North-Holland Biomedical Press, New York. 696 pp.
- Fimreite, N. 1974. Mercury contamination of aquatic birds in northwestern Ontario. *J. Wildl. Manage.* 38:120-131.
- Fimreite, N. and L. Karstad. 1971. Effects of dietary methyl mercury on red-tailed hawks. *J. Wildl. Manage.* 35:293-306.
- Finley, M. T., W. H. Stickel, and R. E. Christensen. 1979. Mercury residues in tissues of dead and surviving birds fed methylmercury. *Bull. Environ. Contam. Toxicol.* 21:105-110.
- Friend, M. 1985. Interpretation of criteria commonly used to determine lead poisoning problem areas. U.S. Fish Wildl. Serv. Fish Wildl. Leaflet. 2.
- Heinz, G. H. 1979. Methylmercury: Reproductive and behavioral effects on three generations of mallard ducks. *J. Wildl. Manage.* 43:394-401.
- Heinz, G. H. and L. N. Locke. 1976. Brain lesions in mallard ducklings from parents fed methylmercury. *Avian Dis.* 20:9-17.
- Pokras, M. A. and R. Chafel. 1992. Lead toxicosis from ingested fishing sinkers in adult Common Loons (*Gavia immer*) in New England. *J. Zoo Wildl. Med.* 23:92-97.
- Van der Molen, E. J., A. A. Blok, and G. J. De Graaf. 1982. Winter starvation and mercury intoxication in grey herons (*Ardea cinerea*) in the Netherlands. *Ardea* 70:173-184.
- Zar, J. H. 1984. Biostatistical analysis. Prentice-Hall, Inc., Englewood Cliffs. 718 pp.

Table 1. State of origin and causes of mortality for 222 Common Loons (*Gavia immer*), 1976-1991.

State	Emaciation	Trauma	Microbial Disease	Lead Poisoning	Drowning	Misc.	Unknown	Total
Minnesota	14	30	15	7	0	4	7	77
Florida	31	1	0	0	0	1	3	36
Virginia	6	4	3	0	4	2	1	20
Michigan	0	0	10	0	0	0	9	19
Wisconsin	4	6	2	2	1	0	2	17
Maine	3	5	4	3	0	0	0	15
North Carolina	9	1	0	0	0	0	3	13
Indiana	0	0	5	0	0	0	2	7
Maryland	0	0	0	0	4	0	0	4
Texas	2	1	0	0	0	1	1	5
Washington	2	0	0	0	0	0	0	2
Wyoming	0	0	0	0	0	0	1	1
Alaska	0	0	0	0	0	1	0	1
Delaware	1	0	0	0	0	0	0	1

Table 1 (Continued)

Idaho	0	1	0	0	0	0	0	0	0	1
Vermont	0	0	0	1	0	0	0	0	0	1
West Virginia	1	0	0	0	0	0	0	0	0	1
New Hampshire	0	0	0	1	0	0	0	0	0	1
Totals	73	49	39	14	9	9	9	29	222	

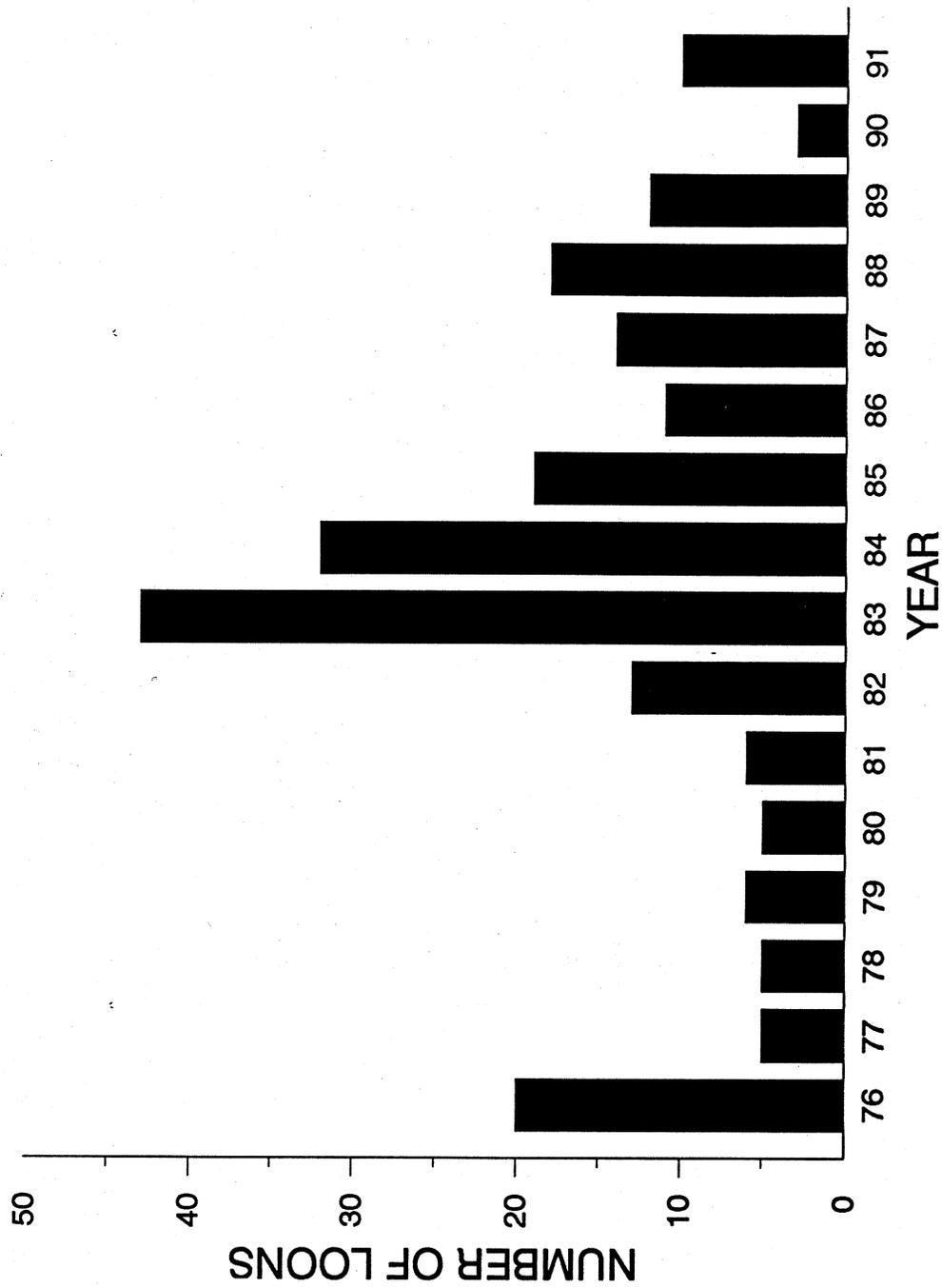


Figure 1. Year of collection for 222 common loons (*Gavia immer*).

Figure 1. Year of collection for 222 common loons (*Gavia immer*).

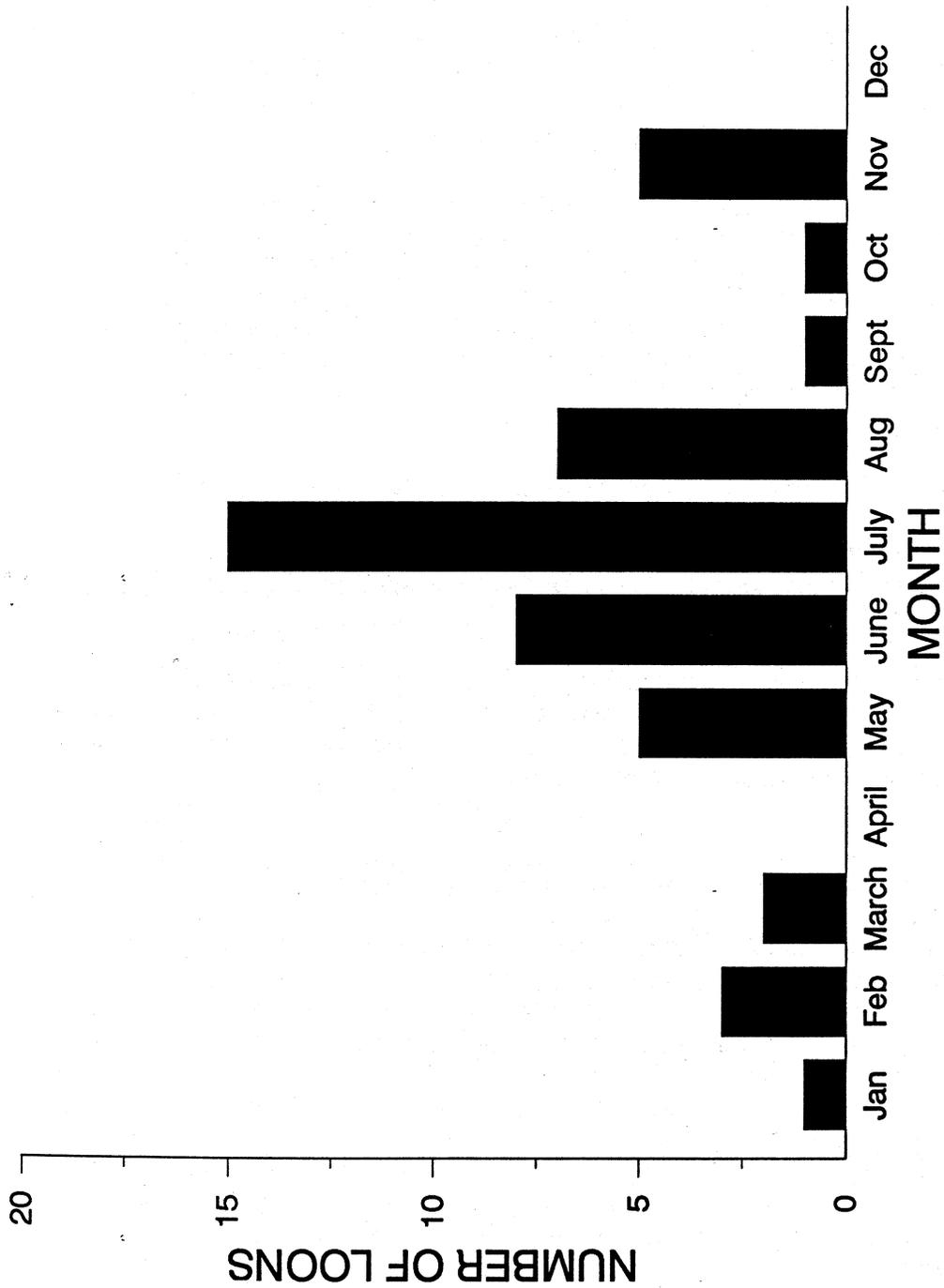


Figure 2. Month of collection for 48 trauma cases in common loons (*Gavia immer*), 1976-1991.