LEAD POISONING IN WILDFOWL[1]

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Summary

THE symptoms and pathology of lead poisoning are fully described, with special reference being made to diagnostic features which could be used in any quantitative assessment.

The amount of lead shot which constitutes a fatal dose is discussed. It is estimated that 60/80% of adult Mallard with one ingested pellet will succumb, if they are feeding on a diet of wild seeds.

The availability of lead shot pellets to wildfowl on a particular body of water is determined by 1) the shooting intensity and number of shot deposited on the bottom, 2) the nature of the bottom material and 3) the size of the shot pellets involved.

The incidence of ingested pellets can be determined by fluoroscopic examination and examination of viscera material, and will vary with the species and its feeding habits.

Tables showing the incidence of ingested lead shot in four species of dabbling ducks in this country and in comparable species in North America are shown and discussed. There is a marked similarity between Mallard in this country and North America carrying *ingested* lead.

The reproductive capacities of poisoned wildfowl do not seem to be seriously affected.

The variations in mortality between different ages and sexes are attributed primarily to differences in the quality and quantity of food consumed.

Means of reducing or eliminating losses are discussed, including the use of non-toxic shot, encouraging the growth of natural foods most likely to alleviate the poisoning effects, and more care in the choice of shot range. As yet no satisfactory non-toxic shot has been produced.

Introduction

Lead poisoning, caused by the actual ingestion of lead pellets, is a significant mortality factor amongst wildfowl in many parts of the United States, and has caused considerable concern to conservationists for many years. Its occurrence in this country has rarely been recorded in wildfowl (Clapham, 1957 and personal comm.) or in any other birds (Keymer, 1958 and personal comm.), and no quantitative assessments have been made. The purpose of this paper is to show the nature of the disease, its implications, and the various methods which can be used in evaluating the losses due to lead poisoning. Basically it is a review of available literature.

That fatal results are caused by birds of many species—ducks, geese, swans, coots, rails, partridge, and pigeons—eating lead pellets, whether as food or grit, has been recognised since the 1870's (Phillips & Lincoln, 1930). Grinnell (1894, 1901) described the symptoms that appeared following the ingestion of shot by swans, geese and ducks at Currituck Sound on the coast of California, and he also noted three places in Texas where lead poisoning had occurred. Bowles (1908) recorded similar symtoms in a number of Mallard (*Anas p. platyrhynchos*) and McAtee (1908) in the same year gave an account of lead poisoning in Canvasbacks (*Aythya vallisneria*). Wetmore (1915, 1919) not only reported lead poisoning in Whistling Swans (*Cygnus c. columbianus*), Mallard and Pintail (*Anas a. acuta*), but also carried out the first experimental work on lead poisoned ducks, from which he described the premonitory and postmortem symptoms. Since Westmore's pioneering work many instances of plumbism have been observed and recorded (Munro, 1925; Van Tyne, 1929; Howard, 1934; Pirnie, 1935; Shillinger & Cottam, 1937; Jones, 1939; Jones, 1940; Roberts, 1940; Adler, 1942; Mohler, 1945;

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Bellrose, 1947; Ayars, 1947; Yancey, 1953; Wisely & Miers, 1956; Bellrose, 1959).

In some instances the number of deaths directly attributable to lead poisoning reaches spectacular proportions. Bellrose (1959) cites an outbreak in the Claypool Reservoir area near Weiner, Arkansas, where between mid-December, 1953 and mid-February, 1954, an estimated 16,000 ducks, most of them Mallard, succumbed to lead poisoning. This represents a 6.4 per cent mortality among the 250,000 duck present. A further example quoted by Bellrose took place at Dalton Cut-Off in Chariton County, Missouri in 1949, where it was estimated that in a population of 10,200 duck, again mostly Mallard, 1,000 died from the effects of ingesting lead pellets.

Signs and Pathology

The signs of lead poisoning in wildfowl are recognisable before and after death and have been described by a number of people, including Wetmore, 1919; Howard, 1934; Shillinger & Cottam, 1937; Adler, 1944; Jordan & Bellrose, 1951; Coburn, Metzler & Treichler, 1951; Elder, 1954; and Wisley and Miers, 1956. The following account of symptoms and pathological conditions is based mainly on their work with a few observations of my own. The general pathology is similar for wild and captive birds with ingested lead pellets, induced or freely-taken, and for wild-caught birds with an administered lead salt solution. Typically there is a definite pattern with the development of each symptom being followed by an increase in its severity, usually an illness of short duration, ending in death.

One of the first symptoms to appear with experimental birds is a marked lethargy with a lessening resistance to being handled and a quick return to a resting position. This has been interpreted as the beginning of muscle paralysis, though it is probably correlated also with a lowered food intake, where consumption falls to a level below minimum nutritional requirements. At the time of death the body weight may average only 40 per cent of the original weight, with a reduction in, or total absence of any fatty tissues. Bright green droppings (due to excessive bile production) are commonly observed within two days of lead ingestion. Frequent water drinking is usual and a greenish diarrhoea is produced with in some cases a green bile staining of the feathers in the ventral region. By the third and fourth weeks the sternum becomes prominent and there is a characteristic ' roof-shaped ' positioning of the wings as they are held over the back, with an associated dropping of the chest and high carriage of the tail. In some cases the wings of sick birds are extended downwards in a ' wing-drop '—analagous to the characteristic wrist-drop in human lead poisoning.

The most striking post-mortem feature is the extreme emaciation with a loss or reduction of fat deposits in the body cavity. Particularly noticeable is the reduction of the main flight muscles. Flaccid muscle tissue is a general finding. There is usually a marked flabbiness of the heart muscles, exaggerated by the small amount of impoverished blood and often an effusion of the pericardium. There is generally a marked reduction in the size of the liver, which histologically was shown by Coburn, Metzler and Treichler (1951), to be due in part to necrosis. More than the normal amount of bile is present

in the gall bladder and duodenum, and characteristically it is bright green. Regurgitation of bile into the gizzard and proventriculus is common, though it is doubtful if this should be taken as a definite sign of lead poisoning as was done by Anderson (1959). The gall bladder may be enlarged to five times its normal weight. Atrophy of the gizzard muscles is a regular observation. The horny pads of the gizzard may be very stiff, abnormally rough and easily peeled off. Commonly the gizzard is ulcerated and the proventriculus impacted (44 per cent of the penned Mallard used by Jordan & Bellrose, 1951). Anaemia is a constant finding with definite changes in the blood. These changes, particularly affecting the erythrocytes, follow a consistent pattern according to Coburn *et al.* (1951). In their experimental Mallard, dosed with an aqueous solution of lead nitrate, anisocytosis (inequality in size of erythrocytes) was observed early on, followed by poikilocytosis (irregular shape of erythrocytes). In the majority of cases there was a decrease in the numbers of erythrocytes. The normal average number for Mallard is 3.06 millions per cubic millimetre according to Magarth & Higgins (1934), though it will vary with the sex and age of the bird. Though reductions in erythrocyte numbers of up to 40 per cent have been noted by Elder (1954), and it has been suggested that the decrease varies with the dosage and could therefore be used as a measure of toxicity, there does also appear to be a considerable variation between individuals of the same species.

The characteristic basophilic stippling of the erythrocytes first noted by Ehrlich (1885) and correlated with lead poisoning by Behrend (1899), which is so apparent in mammalian plumbism, is not a consistent finding in avian species. Coburn *et al.* (1951) state that they had rarely observed stippling in any avian species. However, Johns (1934) in a careful survey of the blood of wild duck poisoned by lead pellet ingestion, found extensive basophilic stippling. In chronic cases numerous stippled cells begin to appear, coincidental with unmistakeable changes in the nucleus. It was suggested that the direct combination of lead with phosphates on the surface of the erythrocytes and the local liberation of a weak acid, as shown by Aub, Reznikoff & Smith (1924), is sufficiently toxic to produce actual cell death. This has since been disputed by Jandl & Simmond (1957). Whatever the toxic mechanism is, the stippling produced by a basophilic stain is considered to be characteristic of a *dying* cell, seen in the sequence of events in the usual maturation of semi-mature cells in the peripheral circulation. The disagreement between the work of Coburn *et al.* and Johns may possibly be due to differing dosage rates and the difference in acute and chronic cases. More detailed work is needed before stippling of the erythrocytes can be used as a diagnostic character in avian lead poisoning.

It was clearly demonstrated by Jordan & Bellrose (1950) that the toxic effects of ingested shot is due to the lead fraction in the pellet alloy. Abrasion of the pellets in the gizzard results in the circulation of complex lead componds in the blood stream throughout the body. It seems probable that soluble lead salts are formed in the presence of gastric juices (Cantarow & Trumper, 1944). These may form albuminates, peptonates and other more soluble compounds which are readily absorbed and distributed throughout the tissues by the blood stream. Lead compounds may be deposited in varying amounts in the liver, kidneys, bones, nerve and muscle tissues.

Chemical analyses of various organs from lead poisoned birds can be used as diagnostic aids, though the rate of deposition is not directly proportional to the dosage level or to the time of poisoning. Coburn *et al.* (1951) found that the most significant increases in lead content were in the liver, where the average value for the poisoned birds was forty times that for the normal controls. Likewise, the lead content of skeletal material from poisoned birds was seven times higher than that found in the controls. Adler (1944), from his work with lead poisoned Canada Geese (*Branta c. canadensis*), has suggested that the liver is the best organ to choose for chemical analysis in aiding diagnosis. By using his approach a more accurate index of lead poisoning may be had. Malysheff (1951), cited by Bellrose (1959), made chemical analyses of the bones and liver of wildfowl taken in the Lower Fraser Valley of British Columbia. He found that 52 per cent of the 79 Mallard he examined had ingested lead at one time or another in their lives, though only about 16 per cent had actually got lead in their gizzards. Recently, Schöberl (1958) has suggested that either a photometric method. using diphenylthiocarbazone, or a polarographic method is most suitable for determining the amount of lead in various tissues.

It seems that the liver is efficient in removing lead from the portal blood but is not so effective in removing it from the systemic circulation. It is possible that lead reaching the liver in the portal system is excreted in the bile and may subsequently undergo reabsorption: this cycle preventing or limiting the amounts of lead that reach the systemic circulation (Cantarow & Trumper, 1944).

The gross pathologic findings are very similar to those produced by starvation, as has been well shown by Jordan (1951, 1953). The suggestion is that death from lead poisoning may be due to, or accelerated by, starvation caused by the paralytic inactivity of the gizzard muscles and a low food intake. Jordan (1951) and Jordan & Bellrose (1951) outlined experiments where they measured the food intake daily of Mallard dosed with one pellet, and fed exactly that amount to a companion control the following day. In nearly all pairs, the weight loss curves, symptoms, gross appearance of viscera and muscles, and mortality were similar, though no impaction of the proventriculus was shown by the deliberately starved birds. Jordan (1953) showed that in intentionally starved Mallard the loss of weight in the liver, kidneys and heart averaged 69.4, 26.8 and 36.7 per cent respectively for males and slightly less for females, with an enlargement of the gall bladder (3 times normal weight) in both sexes.

Fatal Dosage

The amount of lead shot which constitutes a fatal dose varies with the species, the age and sex of the bird, the individual, its general condition, whether it is hand-reared or wild, the feeding habits of the species, and often from author to author. In comparing American work on this subject with available British figures, account must be taken of the differences in shot size. Most of the American work on duck has been done with their No. 6 shot (225 pellets per ounce) or No. 5 (170). Fortunately these shot sizes compare favourably with the sizes usually used in duck shooting in Britain; No. 5 (220) and No. 4 (170). Wetmore (1919) found that six No. 6 shot were

always fatal with hand-reared Mallard. Jordan & Bellrose (1950) found that *one* No. 6 shot was fatal for six out of 10 wild Mallard fed on a diet composed wholly of natural-growing seeds and for seven out of 10 wild Mallards fed on a diet of mixed grains. As a large proportion of the Mallard in England during the shooting season are feeding on a high percentage seed diet (Olney, unpubl. mat.), Jordan & Bellrose's work has obvious importance. They concluded that 60/80 per cent of adult wild Mallard carrying *one* pellet were likely to succumb, if they depended upon diets of wild seeds. It is apparent from their work (1950, 1951) and that of Elder (1954) that the nature of the diet rather than the dose of ingested lead was the more important variable. The effects of lead poisoning are considerably reduced when various leafy, aquatic plants are introduced into a grain or wild seed diet. *Ceratophyllum demersum, Potamogeton pectinatus, Lemna minor* and *Lemna trisulca* were found to be particularly beneficial—probably by acting as buffers and lessening the mutual grinding effect between seed and pellet. In this country, probably only the two duckweed species (*L. minor* and *L. trisulca*) are taken in appreciable quantities by Mallard, Teal (*Anas c. crecca*) or Wigeon (*Anas penelope*) during the winter months.

Availability

The frequency of occurrence of ingested lead pellets will vary not only with the availability of the pellets, but also with the feeding habits of the different species. Most of the shot fired in the pursuit of wildfowl will in fact fall over water, sink and, depending on the nature of the bottom material, be liable to become ingested by feeding birds. The primary limitation on availability will depend on the shooting intensity and the amount of shot which is deposited on the bottom. It is impossible, and it would certainly be tactless, to estimate accurately the number of cartridges fired for every duck or goose killed. Nevertheless, in certain parts of the country, particularly where most of the shooting is done from behind butts (hides) or over flight ponds, the number of pellets which do not hit a bird, and are deposited in the adjacent mud, must be very high. There will obviously be an increase in susceptibility as the shooting season progresses. The pellet numbers actually available to birds will depend to a large extent on the type of bottom material, and on the size of shot used. This has been shown to be true by Bellrose (1959), using lake beds of different firmness and sinking ceramic pipes in each area with three shot sizes in the top soil of each. By sifting the mud contents a year later, he was able to show that movement of the pellets depended on the degree of firmness of the soil and on the size of shot. The smaller the size of shot, the more likely was it to be dislodged and scattered.

The actual depth to which the bird will dabble in the mud will depend on the species involved and on the food available or preferred. Species of duck differ to some extent in preferred feeding depths, so that the depth of water above the bottom may also determine the availability of pellets. Dabbling ducks usually feed in waters of less than 15 inches in depth, whilst diving ducks feed at depths of many feet, though Pochard (*Aythya ferina*) and Tufted Duck (*Aythya fuligula*) may and often do feed in shallower water. Other species of duck will rarely sift through bottom muds for food,

relying more on leafy aquatic plants (Gadwall, *Anas strepera*), or actually grazing on various grasses and seaweeds (Wigeon, Olney 1957). That the incidence of ingested lead varies considerably with the species and its feeding habits is well shown by American figures (Shillinger & Cottam, 1937; Cottam 1939; Bellrose, 1951; Anderson, 1959). It is probable that the pellets are taken by the birds accidentally or deliberately as or with grit, or accidentally with food material.

Frequency

The incidence of lead shot can be determined either by examining the dead bird (usually as a by-product of a food investigation) or by fluoroscoping live-trapped or dead birds. But the absence of lead shot is not a sure indication that the bird is not suffering or has not suffered from the effects of lead poisoning. Jordan (Bellrose 1959) found in controlled experiments with captive Mallard that 21 per cent of 119 birds dosed with a single No. 6 pellet had *no* pellets in their gizzard at the time of death. Of 1153 Mallard picked up either dead or dying from lead poisoning between 1938 and 1955 in six American states, 132 (11.4 per cent) had no lead pellets in their gizzards (Bellrose, 1959).

The actual time which a pellet has been in the gizzard can often be roughly ascertained by the amount of abrasion and erosion that has taken place over the surface. By using an aspirator (Nord, 1941) to recover pellets from a live duck which has been dosed with shot, it is possible to observe the effects of the digestive processes and grit movements. Signs of erosion are evident within 12 hours. The ridges and craters commonly formed on the pellets when discharged, are smoothed, the surface is pitted and in places a silvery grey cast appears.

Bellrose (1959) provides a comprehensive table showing the incidence of lead shot found in gizzards of various Anatidae in America during the autumn and early winter months of 1938-1953. Parts of this are reproduced opposite (Table 1) in order to show species comparable to those found in this country.

Since 1957 the Wildfowl Trust has been examining viscera and their food contents. Table 2 opposite summarises the numbers and species which were found to contain lead pellets.

Though only three species have so far been found to contain pellets, this is probably due to the smallness of the available sample. Mallard, though they may do considerable feeding in grainfields at certain times of the year, spend much time feeding in marshes and open stretches of water, often heavily shot over. Their habit of deep-puddling into the bottom soil in pursuit of seeds probably brings them into contact with deposited lead shot more frequently than any other dabbling species. The proportion of gizzards from Mallard which contain pellets is remarkably similar for British and American birds. It is significant that of 277 Teal viscera examined in this country *none* contained lead shot, although their diet is similar to that of the Mallard. Field observations suggest that Teal only dabble in the top one or two inches of mud in search of food, and they may therefore be missing the critical depths where lead pellets are lodged. All of the sixteen Mallard found to contain pellets had been feeding inland or in

TABLE 1. Incidence of lead shot in N. American species 1938-53. (after Bellrose, 1959, p.260).

Species	No. of gizzards examined	1 pellet No.	1 pellet %	Over 1 pellet No.	Over 1 pellet %	Total No.	Total %
Mallard *Anas p. platyrhynchos*	17,066	757	4.44	402	2.35	1,159	6.79
Gadwall *Anas strepera*	1,141	14	1.23	7	0.61	21	1.84
Baldpate *Anas americana*	1,577	42	2.66	8	0.51	50	3.17
Pintail *Anas a. acuta*	4,530	241	5.32	161	3.55	402	8.87
Green-winged Teal *Anas crecca carolinensis* ..	2,272	23	1.01	8	0.35	31	1.36
Shoveler *Anas clypeata*	1,439	19	1.32	4	0.28	23	1.60
Redhead *Aythya americana*	597	56	9.38	25	4.19	81	13.57
Lesser Scaup *Aythya affinis*	886	67	7.56	49	5.53	116	13.09
Canada Goose *Branta c. canadensis*	511	4	0.78	0	0	4	0.78

TABLE 2. Incidence of lead shot in British species 1957-59.

Species	No. of gizzards examined	1 pellet No.	1 pellet %	over 1 pellet No.	over 1 pellet %	Total No.	Total %
Mallard *Anas p. platyrhynchos* ..	244	14	6	2	1	16	7
Wigeon *Anas penelope*	288	4	1	0	0	4	1
Teal *Anas crecca*	277	0	0	0	0	0	0
Shoveler .. *Anas clypeata*	14	1	7	0	0	1	7

a slightly brackish habitat, and none of the birds shot whilst *feeding* on saltmarsh species contained any pellets.

A large sample of diving ducks is needed before any figures comparable to American species are available. Possibly in certain areas diving ducks are particularly vulnerable to lead ingestion—at least the American figures indicate that is so.

A small number of birds sent in for post-mortem examination have been found to be suffering from lead poisoning. The most startling case was a first-year drake Mallard, hand-reared though not pinioned, which had 41 pellets in the gizzard, and not surprisingly showed the typical lead poisoning syndrome symptoms. In this case, although the pit where the ducks were kept had not been shot over, it is thought that some old cartridges may have been thrown into the water.

Fertility and Fecundity

Apart from direct losses, some concern has been engendered by evidence in other animals that their reproductive capacities are impaired by lead poisoning (Cole & Bachhuber, 1914—rabbit and fowl; Aub *et al.* 1926—man). Shillinger & Cottam (1937), Cheatum & Benson (1945) and Elder (1954) have voiced concern over possible sterility as an after effect of lead poisoning in wildfowl. However, Cheatum & Benson (1945) concluded that no significant loss of fertility resulted from the ingestion of lead shot in male Mallard. In a series of experiments by Elder (1954) to test the effects of lead poisoning on fertility and fecundity in Mallards, he administered 18 No. 6 shot to his experimental birds. Although he managed to show that fecundity was reduced, while fertility, embryonic success and hatchability were not, his results are somewhat nullified by his use of such a large dosage level. Rarely will 18 pellets be ingested by a duck, and seldom will significant numbers recover from the resultant severe poisoning.

Movement and Mortality Rates

Field experiments conducted by Bellrose (1959) in the years 1949-51 showed that birds dosed with lead shot had 1) a greater vulnerability to being shot, 2) a lower ability to migrate and 3) higher over-all mortality rates in the first year after being ringed and released. Wild Mallard that were dosed with one No. 6 shot each and then released were 1.5 times as vulnerable to shooting as were undosed controls; those dosed with two No. 6 pellets were 1.9 times as vulnerable and those dosed with four No. 6 pellets were 2.1 times as vulnerable. In the dosed Mallard the effects of the ingested shot did not appear to affect their behaviour until 5 days, when the proportion of ring returns became higher than for undosed birds. The period of affliction appeared to persist for about 15 days or slightly longer until the ringing returns reverted to more similar figures for both dosed and undosed birds. It is suggested that either the duck is shot or dies of lead poisoning in the second or third week following ingestion, or they begin to recover by the early part of the fourth week. The weakness and fatigue symptoms so apparent as part of the lead poisoning syndrome, are likely to reduce their movements. That it has a pronounced effect on their local movements has been shown experimentally by Jordan & Bellrose (1951) and Bellrose (1951, 1959). The more ingested lead pellets there are per bird, the greater is the reduction in the movements of those birds.

The variations in mortality rates with different sexes and ages were attributed primarily to differences in the quality and quantity of food consumed. It is known that the food intake of juvenile Mallard exceeds that of the adults, and it has been shown experimentally (Jordan & Bellrose, 1951) that the juvenile Mallard mortality rate from lead poisoning is less than the adult rate. The female mortality was higher than comparable male mortality in all months apart from the spring months when the female is known to eat more than the drake. At all other seasons the female is thought to eat less than the male (Jordan & Bellrose, 1951). Lower air temperatures which are known to increase food consumption may well have an effect on the mortality numbers.

Reduction of losses

In an effort to eliminate the losses due to lead poisoning among wildfowl various non-toxic shot alloys have been advocated and tested. Jordan and Bellrose (1950) reviewed previous work and tested a number of possible alloys. Several metals regarded as being non-toxic were considered as substitutes for lead. The availability, physical and chemcial properties, price, and ballistic performance were factors which had to be taken into consideration. Green & Dowdell (1936) suggested that a lead-magnesium alloy shot would not cause lead poisoning. They suggested that the magnesium would be hydrolysed by water which would produce irregular cracks across the surface and the final disintegration of the pellet. Unfortunately their findings were not substantiated by Jordan & Bellrose's careful experiments. Three other lead-alloy shots tested, lead-phosphorus, lead-calcium and a copper-alloy coated lead shot, were not less toxic than the usual lead-alloy shot used. Attempts to coat lead shot with a nylon plastic in order to lessen the abrasion effects were unsuccessful (Bellrose 1959). However an iron-alloy was found to be non-toxic when dosed to penned wild Mallard, but it has the disadvantage of not being so effective a shot as a lead shot when fired at maximum ranges, because of its lower density (Bellrose 1953). Ignoring their availability and price, there are many metals which could probably make good or even better shot pellets than the lead-alloy now used. The physical properties of gold would make it a good example—though its use as a shot would probably be confined to a favoured few.

As the effects of lead poisoning can be greatly minimised by the form of food consumed, one of the means of alleviating losses would be to encourage the growth of suitable leafy, aquatic plants. Though often freely given, corn is the least beneficial in reducing the poisoning effects.

It would probably be impertinent to suggest that less pellets would be available for ingestion if wildfowlers were more careful in their selection of shot ranges. Nevertheless, more careful shooting would undoubtedly reduce the chances of lead pellets being obtainable by feeding birds.

There are no records in this country comparable to the spectacular local outbreaks of lead poisoning recorded in the United States. It is probably more in the dispersed, day to day mortality that our losses occur. Such mortality can occur constantly, and generally will pass unnoticed, particularly if there is cover available for the sick birds to hide until they die or are taken by predators.

Bellrose (1959) has estimated that approximately one-fourth of the wild Mallards in North America in any year ingest lead shot, and that in the Mississippi Flyway approximately 4 per cent. of the Mallards die as the direct result of lead poisoning, with an additional 1 per cent afflicted with lead poisoning being shot. For all wildfowl species in North America the annual loss due to lead poisoning is estimated to be between two and three per cent. of the population. These figures can only suggest what may be the extent of the problem in this country, and until we have more information no accurate assessment can be made. At the present time losses due to lead poisoning are probably small, but their possibility should certainly be taken into account in the dynamics of any wildfowl population.

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Appendix A

An adult female Pochard (*Aythya ferina*) found dead in a duck trap at Abberton Reservoir, Essex, on 20th February, 1960 proved on examination to contain *84* ingested lead pellets. Not surprisingly the bird exhibited signs of actue lead poisoning.

It is difficult to theorise as to the reason why such a large number were taken. Possibly the total is an accumulation of many ingestions—though the amount of erosion as shown in the photograph on p. 188, suggests that if this was so, it was not over a very long period.

Appendix B

An interesting paper by M. N. Rosen and R. A. Bankowski (A diagnostic technic and treatment for lead poisoning in swans, *Calif. Fish & Game* 46, 3:81-90, 1960) was unfortunately received too late to be included in the above review. Their work at Tule Lake National Refuge in Northern California on Whistling Swans (*Cygnus c. columbianus*) showed a range of 1.0 to 3.3 per cent. mortality due to lead poisoning. Of particular interest is their use of calcium versenate (calcium disodium ethylene diamine tetra-acetate) as a diagnostic aid and possible cure. An intravenous inoculation of this compound will cause a temporary alleviation of the lead poisoning effects by detoxification within the blood and tissues. The detoxification is due to the calcium versenate forming a soluble complex with lead which is rapidly excreted. It is however only a temporary remission, for any lead shot retained in the gizzard is unaffected and as more lead was absorbed the bird would suffer a relapse. Continued administration of calcium versenate effectively cured some swans, though those that had too many lead shot, or had the additional complication of impaction did not survive.

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