Chapter (non-refereed)

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LEAD POISONING IN MUTE SWANS - AN EAST ANGLIAN SURVEY

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ABSTRACT

From October 1981 to September 1982, 320 dead mute swans (*Cygnus olor*) were examined and total lead levels were determined in liver, kidney, and bone. Chemical analyses revealed that 70% (224) of these swans had died of lead poisoning. In the majority of cases, death was due to an acute exposure. Thirty-six dead birds and 8 birds which were given a veterinary examination before they died had tissue levels of lead which did not conform to the accepted levels that would confirm fatal lead poisoning. These birds were included in the total of lead poisoned birds after taking 3 further points into account: (i) veterinary diagnosis; (ii) post-mortem evidence, including the presence of lead fishing weights in the gizzard; and (iii) the effect of a fall in body temperature usually experienced by lead poisoned birds prior to death which was made worse by the very low environmental temperatures prevailing at the time these 44 birds were examined.

INTRODUCTION

The poisoning of wildfowl by the ingestion of spent gun-shot pellets is well documented throughout Europe and North America (Belrose 1959; Del Bono 1970; Clausen *et al.* 1975; Beer & Stanley 1964; French 1982). In Britain, a joint study carried out in 1973 by ITE and the Veterinary Investigation Service in Nottingham was able to identify for the first time that ingested anglers' weights were responsible for the deaths of mute swans on the River Trent in Nottingham (Simpson *et al.* 1979). In America, several common loons (*Garia immer*) died after ingesting lead anglers' weights (Locke *et al.* 1982). Swans and other waterfowl are assumed to take in the lead weights along with, or in mistake for, grit which they need to aid digestion. Following the joint ITE-VI Centre study, the NCC report on lead poisoning in swans was published (NCC 1981). This report identified lead poisoning from anglers' weights as being the biggest single cause of mute swan deaths in Great Britain. It also identified several 'hot spots' throughout Britain where 70-90% of reported swan deaths were due to lead poisoning. In some areas, previously substantial swan populations have declined markedly, eg River Thames (Birkhead 1982), and in others they have disappeared completely (eg River Avon near Stratford).

This report outlines some results of a project investigating the effects of lead on swans in East Anglia. A major objective of this study is to determine the proportion of swans dying through the ingestion of lost or discarded fishing weights.

METHODS

Between October 1981 and September 1982, a study was done on the rivers Welland, Nene, Ouse, and Cam, a total river length of approximately 300 miles. Also included were gravel pits and reservoirs within the same area. During this time, 320 mute swan (*Cygnus olor*) carcases were examined.

Chemical analyses of liver, kidney, and bone were done to help establish the cause of death. The contents of the gizzard and proventriculus were examined and pieces of lead shot identified by eye. In the majority of cases where lead shot was found, it was possible to distinguish visually between fishing weights and spent gun-shot. Where there was doubt, the material was tested chemically to differentiate between the 2 sources. Only 4 mute swans were shown to have carried gun-shot in their gizzards. These 4 birds were eliminated from the study, even though 2 contained anglers' weights as well as the spent gun-shot.

RESULTS AND DISCUSSION

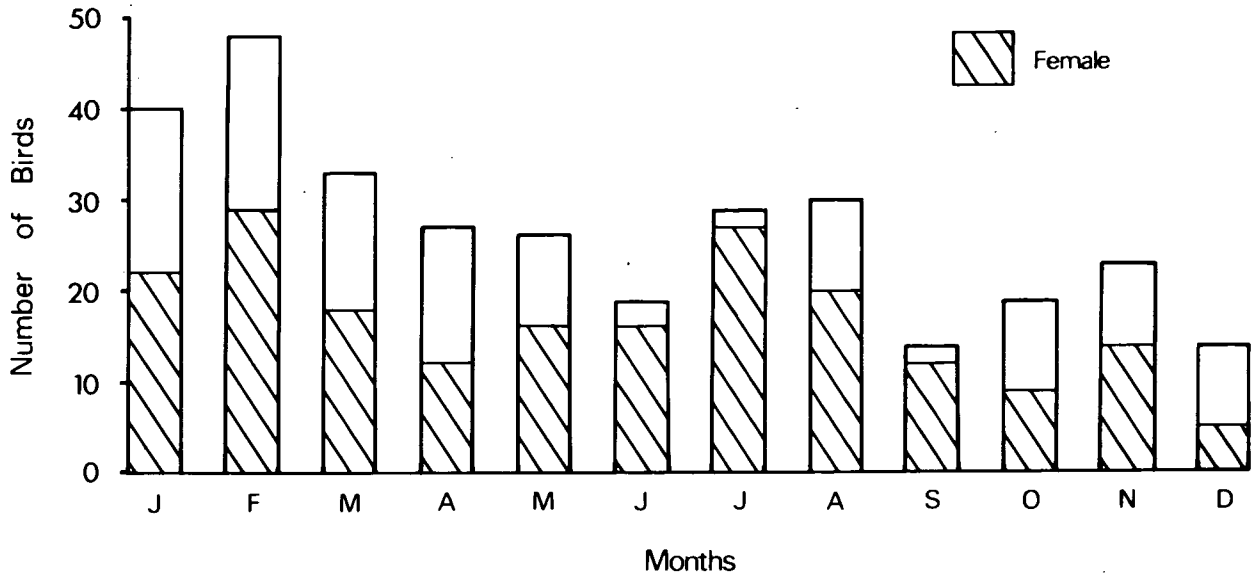
The majority of the swans were corpses with no known history, but in 8 cases clinical observations were available following X-ray and veterinary examination. The symptoms in these 8 cases were anorexia, weakness, inability to move correctly and impaction of the oesophagus. These birds were very emaciated. These are all signs of lead poisoning; in addition, a marked and sustained fall in body temperature, observed up to 72 hours prior to death, was noted in the 8 live birds. A similar drop in body temperature has been demonstrated with pigeons dosed with lead fishing weights in this laboratory. The body weights of all 320 birds in the study are given in Table 1. These findings are consistent with our original observations (Simpson *et al.* 1979).

TABLE 1 Mute swan body weights (kg)

	Lead poisoned (lead shot present)	Lead poisoned (lead shot absent)	Cause of death other than lead poisoning
Number	174	50	96
Range	(5.1-9.8)	(4.2-7.9)	(8.1-13.2)
Mean	7.1	6.0	9.8

At post-mortem, sexes were determined and 70% (224) of the birds examined were female. The sexes and monthly receipts of swan bodies are shown in Figure 1.

Figure 1 The total number of birds received each month



Lead shot was present in the alimentary tract of 174 swans and in all cases death was due to lead poisoning. Fishing hooks and attached line were found in 6 birds and in only one case could these have contributed to death. None of these 6 birds died of lead poisoning.

Chemical analyses of kidney, liver, and bone showed that 70% (224) of the birds examined had died of lead poisoning. Of the 224, 180 birds in the survey had levels of lead in the tissues in excess of the values that would confirm fatal lead poisoning in mammals. These values are 125 mg/kg dry wt lead in the kidney and 50 mg/kg dry wt lead in the liver (Clarke & Clarke 1975). Forty-four birds with lower levels of lead in their tissues were also included in the lead poisoned group, because the post-mortem and veterinary evidence obtained were classically those of lead poisoned birds. The levels of lead in these 44 swans were (expressed as mg/kg dry wt): 13-32 (mean 28) in liver, 38-64 (mean 53) in kidney, and 40-86 (mean 63) in bone. However, at post-mortem these birds exhibited the accepted signs of lead poisoning, ie impaction of the oesophagus, proventriculus and gizzard, wasting of the muscle, a high level of iron in the liver, an enlarged gall bladder and the presence of lead fishing weights in the gizzard. In addition, 8 of these 44 swans were the live birds that received a veterinary examination prior to death, and whose classical signs of lead intoxication have already been mentioned.

One explanation as to why these swans died with relatively low levels of lead in their tissues could be that they died in the winter of 1981-82 when temperatures of $-17^{0}C$ were recorded in the East Anglian region. These cold conditions could have exacerbated the hypothermia that may be associated with lead poisoning in birds, making it impossible for the birds to recover or causing them to succumb more easily.

The deposition of lead in the intoxicated and dead birds can provide evidence of the type of exposure that has taken place, namely a high bone and low soft tissue level indicates a chronic exposure and a low bone, high soft tissue level indicates an acute form of exposure (Clarke & Clarke 1967). In all but 20 birds, kidney lead levels were higher than bone levels, indicating that death was due, in a majority of cases, to acute exposure.

The high number of swans reported dead and confirmed as lead poisoned in this study supports previous findings from other areas (NCC 1981; Birkhead 1982).

The number of female and young non-breeding swans dying of lead poisoning is difficult to explain. Lead retention in rats is known to be associated with age (Shields *et al*. 1978). Young rats fed lead acetate retained more lead at a given dietary intake than mature rats. This work may go someway to explain our observations. Breeding females need calcium for egg production and this can be furnished in several ways by mobilising stored calcium in the skeleton - up to 40% of the stored calcium can be used in this way (Sturkie 1954) - or the need can be met from an increased absorption from the alimentary canal (Barltrop & Khoo 1976). Because the metabolism of lead follows closely that of calcium, a high demand for calcium yields a high availability of lead to the soft tissues. Female ducks in breeding condition are known to accumulate more lead in their tissues than do males (Finley *et al*. 1976; Finley & Dieter 1978).

Finally, we could find no influence of the close fishing season on the availability of lead to swans. We received more lead poisoned birds during the close season than in the equivalent previous or following period of time. Swans can take up to 3 weeks to die from lead intoxication after ingesting fishing weights, so this fact must be taken into account when interpreting the close season data.

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