the second set was stained with Masson's stain to demonstrate collagen.

After seven days the fibroblast was the predominant cell in the anastomoses performed using monofilament nylon. Other cells present were macrophages and eosinophils. After 21 days there was a thin fibrous tissue capsule around the suture material.

The predominant cell after seven days in the chromic catgut anastomoses was the neutrophil. Other cells were macrophages, giant cells, eosinophils, fibroblasts and lymphocytes. There were new collagen fibres around the suture material. After 21 days the suture reaction was more intense and there was cellular infiltration of the suture material. The suture material showed fragmentation.

Cells observed around polyglactin 910 after seven days were macrophages, giant cells, eosinophils, neutrophils, fibroblasts and lymphocytes. There was cellular infiltration of the suture even after seven days. The cellular reaction was intense at 21 days and macrophages and giant cells were more predominant than in the chromic catgut reaction. Cellular infiltration was greater after 21 days than after 7 days.

Nylon and chromic catgut reaction was similar to that described in literature. The reaction to polyglactin 910 was generally similar to that of chromic catgut, contrary to previous reports. Horton and others (1974) said that the polyglactin 910 reaction was less than that of catgut; Van Winkle and others (1975) reported polyglactin 910 to be more reactive than chromic catgut; Jochen and others (1982) reported polyglactin 910 as producing better or equal tissue reaction as chromic catgut.

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References

CRAIG, P. M., WILLIAMS, J. A., DAVIS, K. W., MAGUON, A. D., LEVY, A. J., BOGDANSKY, S. & JONES, P. R. Jr, (1975) *Surgery Gynaecology and Obstetrics* **141**, 1

GRIER, R. L. (1972) *Iowa State Veterinarian* **34**, 89

HORTON, C. E., ADAMSON, J. E., MLADICK, R. A. & CARRAWAY, J. H. (1974) *American Surgeon* **40**, 729

JOCHEN, R. F., RUTH, D. T. & BURDETTE, B. J. (1982) *Modern Veterinary Practice* **63**, 555

MADSEN, E. T. (1953) *Surgery Gynaecology and Obstetrics* **97**, 73

POSTLETHWAIT, R. W. (1970) *Annals of Surgery* **171**, 892

POSTLETHWAIT, R. W., WILLIGAN, D. A. & ULIN, A. W. (1975) *Annals of Surgery* **181**, 144

VAN WINKLE, W. J. & HASTINGS, J. C. (1972) *Surgery Gynaecology and Obstetrics* **135**, 113

VAN WINKLE, W. Jr, HASTINGS, J. C., BARKER, E., HINES, D. & NICHOLS, W. (1975) *Surgery Gynaecology and Obstetrics* **140**, 7

Lead poisoning in captive birds of prey

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A GREAT deal of attention has been paid to lead poisoning in waterfowl both in the United Kingdom and in the USA, but it is only recently that concern has been expressed over this problem in raptors, especially in the endangered species (Reiser and Temple 1981). Reiser and Temple state that controversy still surrounds the issue of lead toxicity in free-living birds of prey and in an attempt to resolve some of these difficulties, over a period of 30 weeks, 3 mg lead/kg bodyweight was fed daily to nine healthy, but permanently crippled, birds of prey. Data on clinical signs, mortality and blood and tissue lead levels were thus established.

This paper reports the experimental findings in falconers' birds and casualties from wild bird hospitals submitted to the diagnostic laboratory at Lasswade between February 1979 and September 1982.

Ten birds' bodily condition, lesions, bacterial flora and parasite burden were assessed at autopsy as described by Macdonald (1962). The liver and kidney were collected for lead analysis (Thompson and Blanchflower 1971). The carcase was then subjected to pesticide analysis as described by Hamilton and others (1981). The species of the 10 birds examined are listed in Table 1.

Initially the birds were segregated into two groups. The first comprised four birds which were known to have been fed on shot feral pigeons (*Columba livia*) and brown hares (*Lepus europaeus*) and therefore likely to have ingested lead pellets in the food. The second group (controls), comprised six birds fed almost solely on snared rabbits, netted pigeons, beef or one-day-old chicks. As shown in Table 1, three of these controls were subsequently found to be victims of lead poisoning.

In searching for a possible source of lead, it became apparent that trimmings from the carcases of red deer (*Cervus elaphus*) were occasionally offered to the two snowy owls (*Nyctea scandiaca*) to add variety to the diet. Close examination of the trimmings revealed particles of lead arising from disintegration of the rifle bullet on impact with the deers' bones.

The sparrowhawk (*Accipiter nisus*) had been restrained throughout its life on a 3 cm diameter lead pipe which formed a convenient perch. The bird's method of feeding – the prey being held firmly on the perch with the talons and morsels of food detached with the beak – might contribute to contamination of the ingesta with lead. Boredom might also have induced the bird to peck at the perch. Only the last three birds in Table 1 were thought to have died from causes other than lead poisoning and constitute true controls.

The clinical signs varied. Three of the seven poisoned birds showed fits or convulsions and one suffered from imbalance. The head of the lagger falcon (*Falco jugger*) was rotated until the bird was looking backwards, it then fell off its perch and lay rigid on the floor of the cage with its legs fully extended and digits clenched. After about a minute, it regained consciousness, looked around and resumed its perch. During the four succeedings days before death the fits became more frequent and prolonged, lasting up to three minutes. They appeared to be brought on by visual stimuli. No details of the convulsions reported in the two other birds were obtained.

The appetite of the poisoned birds was not always impaired and forcefeeding sometimes prevented marked loss in bodily condition. No characteristic lesions were observed at autopsy but many of the birds were found to be dehydrated and their kidneys were enlarged and pale. These changes were probably associated with reduced fluid intake shortly before death. Single lead pellets were found in the stomach of the buzzard (*Buteo buteo*) and North American peregrine (*Falco peregrinus anatum*). Histological examination of the kidneys showed acid-fast intranuclear inclusions in the proximal convoluted tubules of only the male snowy owl and lagger falcon. Cultural examinations failed to show the presence of any bacterial or fungal infection. Parasites were rare. The sparrowhawk had a few *Capillaria* species in the crop (probably *C contorta*) and three large nematodes in the duodenum. The peregrine had a heavy louse infestation of *Degeeriella rufa* and *Colpocephalum* species.

Acetylcholinesterase activity in brain tissue was normal and did not suggest organophosphorus poisoning. Alphachloralose was not present.

The sparrowhawk had 2 parts per million (ppm) of mercury and 2 ppm of DDE (a persistent metabolite of the organochlorine pesticide, DDT) in its liver but all the remaining

TABLE 1: Lead poisoning in captive predatory birds

Species	Sex	Weight (g)	Bodily condition	Age	Period in owner's possession	Diet	Duration and nature of symptoms	Lead analysis (ppm dry matter) Liver	Kidney
Buzzard (*Buteo buteo*)	M	787	Good	Not known	3 weeks	Shot feral pigeons	Good appetite, active but unable to fly, sudden death	175	66·7
Sparrowhawk (*Accipiter nisus*)	F	226	Fair	2 years	2 years	Netted feral pigeons	Convulsions of unknown onset, bird confined to lead perch	35·7	31·2
Peregrine falcon (*Falco peregrinus*)	F	921	Fair	9 months	6 weeks	Shot feral pigeons	Inappetance 14 days before death	64·3	34·0
North American peregrine (*Falco peregrinus anatum*	M	450	Poor	13 years	8 months	Shot hares alternate days	Inappetance and convulsions 20 days before death	69·9	221
Lagger falcon (*Falco jugger*)	F	650	Fair	21 months	6 months	Shot hares alternate days	Inappetance and convulsions five days before death	56·9	193
Snowy owl (*Nyctea scandiaca*)	M	1116	Poor	8 years	4 years	Beef, snared rabbits, venison	Inappetance and lethargy three days before death	44.7	67·6
Snowy owl	F	—	Poor	10 years	9 years	Beef, snared rabbits, venison	Good appetite, imbalance and lethargy seven days before death	204	146
Buzzard	M	900	Good	Not known	4 years	Beef	Sudden death after laparoscopy	2·3	2·4
Peregrine	F	868	Fair	Not known	7 months	Day-old chicks	Sudden death after anaesthesia	5·3	2·7
Kestrel (*Falco tinnunculus*)	F	270	Good	Not known	2 months	Day-old chicks	Lethargy for two days before death from reproductive disorder	2·0	2·2

birds had less than 1 ppm of these substances. Such residues occur widely in animals and were lower than have been measured in many free-living predatory birds which have died from causes other than poisoning.

The lead levels in the controls in the present findings are identical to those reported by Reiser and Temple (1981). However, those authors found only about 10 ppm of lead in the liver and kidney of their chronically poisoned birds compared with 35 to 200 ppm found in the present study. The precise amount of lead ingested by the casualties in this study was unknown. Three of the nine deliberately poisoned birds studied by Reiser and Temple (1981) died three to four weeks after the onset of clinical signs whereas the illness in the birds in this study usually ran a much shorter course and the neurological disturbances were also more severe.

Under the conditions described a daily lead intake greater than 3 mg seems likely since a single lead pellet of No 6 shot weighs 100 mg. Radiological examination of a single illegally shot golden eagle (*Aquila chrysaetos*), which was submitted as part of the programme monitoring the effects on wildlife of toxic chemicals used in agriculture, revealed 27 lead pellets embedded mainly in the musculature. It is probable that much of the lead shot is regurgitated by falcons along with the undigested feathers, fur, teeth and bones but lead pellets were found in the folds of the gastric mucosa in two birds.

Locke and others (1967) have shown that the presence of intranuclear inclusions is not a consistent diagnostic criterion for lead poisoning. The present findings support this conclusion in that such inclusions were found in only two of the seven birds proven by chemical analysis to have high levels of lead in their tissues. Reiser and Temple (1981) review the synergism of pathogenic organisms and toxic chemicals but no evidence of this phenomenon was found. Since medieval times, fits in falcons have puzzled their owners (Cooper 1978). One possible cause might be lead poisoning.

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References

COOPER, J. E. (1978) Veterinary Aspects of Captive Birds of Prey. Saul, Gloucestershire Standfast Press p112

HAMILTON, G. A., RUTHVEN, A. D., FINDLAY, E., HUNTER, K. & LINDSAY, D. A. (1981) *Biological Conservation* **21**, 315

LOCKE, L. N., BAGLEY, G. E. & YOUNG, L. T. (1967) *Bulletin of the Wildlife Disease Association* **3**, 176

MACDONALD, J. W. (1962) *Bird Study* **9**, 147

REISER, M. H. & TEMPLE, S. A. (1981) Recent Advances in the Study of Raptor Diseases. Eds J. E. Cooper and A. G. Greenwood. Keighley, Chiron Publications p21

THOMPSON, R. H. & BLANCHFLOWER, W. J. (1971) *Laboratory Practice* **20**, 859