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Lead Exposure, Diagnosis, and Treatment in California Condors Released in Arizona

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and W. Grainger Hunt*

ABSTRACT.—Lead poisoning was the most frequently diagnosed cause of death among free-ranging California Condors (*Gymnogyps californianus*) released by The Peregrine Fund in Arizona during 1996–2005 and may have caused additional undiagnosed fatalities. We tested condors at least twice per year, and among 437 blood samples analyzed from March 2000 through December 2004 (excluding retests of exposed individuals), 137 showed above-background lead exposure levels of between 15 and 59 $\mu\text{g dL}^{-1}$, and 39 exceeded 60 $\mu\text{g dL}^{-1}$, elsewhere defined as the threshold of clinical affect. Laboratory tests showed that 25 samples among the latter group were above 100 $\mu\text{g dL}^{-1}$, 10 exceeded 200 $\mu\text{g dL}^{-1}$, and 5 were greater than 400 $\mu\text{g dL}^{-1}$. Chelation therapy was administered in 66 cases (28 individuals); all treated individuals survived. Condors showing moderate degrees of exposure were held for retesting to detect trends of blood lead depuration or increase, the latter indicating the need for radiography. Radiographs of seven condors (three alive, four dead) revealed shotgun pellets in their stomachs, and seven more (six alive, one dead) showed ingested lead fragments consistent with those of spent rifle bullets. Surgery or oral doses of psyllium fiber were used to purge lead from the stomachs of surviving individuals. Overall findings indicated that condors in northern Arizona frequently ingest lead and suggest that rifle- and shotgun-killed animals are an important source of toxic exposure for condors.

The endangered California Condor (*Gymnogyps californianus*) is among the most sensitive of all U.S. birds to changes in survival rates. The species defers breeding until six or more years of age and incubates a single egg (Koford 1953). Past data suggest that about one-half of nesting

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attempts succeed, and successful pairs may not renest for 16–18 months after fledging young (Snyder and Snyder 2000). Such low reproductive potential necessitates high individual survival, particularly among the older age categories. Population viability models call for minimum annual adult survival rates in the range of 90–95% (Verner 1978, Meretsky et al. 2000), values that most certainly were not obtained in the wild during the 1970s and 1980s, when the number of individuals counted in surveys declined by about 40 percent (Snyder and Snyder 2000). Known mortality agents at the time included lead poisoning (Janssen et al. 1986, Weimeyer et al. 1988), shooting (Wilbur 1978), powerline collisions (Koford 1953, Brunetti 1965), drowning (Koford 1953), and predator control poisoning (Miller et al. 1965, Borneman 1966, Weimeyer et al. 1988). However, dead condors were usually not recovered, so the relative importance of mortality factors in the condor population could not be accurately determined.

To counter the continuing population decline, the U.S. Fish and Wildlife Service began in 1982 to capture condors for long-term captive propagation. A decision to leave even a few pairs in the wild was thwarted within a six-month period (October 1984–April 1985) when six of the remaining 15 wild condors perished; five of these went unrecovered, and the sixth was found to have died of lead poisoning (Snyder and Snyder 2000). These events prompted the removal of all remaining condors to breeding facilities where success in propagation from the remaining 27 individuals (14 females and 13 males) swelled the population to over 250 birds by 2005, almost half of which have been released to the wilds of California, Arizona, and Baja California, Mexico.

In 1996, The Peregrine Fund began releasing captive-bred condors in northern Arizona (36°N, 112°W) with the goal of establishing a self-sustaining population disjunct from other reintroduced condor populations. The current release site, situated atop Vermillion Cliffs and in view of the Kaibab Plateau to the west, lies approximately 80 km north of the south rim of the Grand Canyon (see Hunt et al. this volume for a description of the northern Arizona environs). Continuing releases brought the number of free-flying birds to about 50 by spring 2005, including three fledged from wild pairs (Woods et al. this volume). Daily monitoring by means of conventional and satellite-based GPS telemetry offered an opportunity to recover condor carcasses and assess proportional impact among the various mortality agents existing outside the immediate areas of release. Lead poisoning was principal among them, accounting for at least six of the 12 condor deaths unrelated to recency of release (Woods et al. this volume).

The first indication that lead would be a problem for condors in Arizona came in 2000 when at least two died from ingesting shotgun pellets from

an unknown source. Thirteen others showed elevated blood lead levels, and were likely exposed during that same poisoning event (Cade et al. 2004). This episode, followed by a general expansion of condor movement and foraging in the region (Hunt et al. this volume), prompted the development of a regular program of blood lead testing, evaluation, and treatment. Here we report the results of the lead-testing program in Arizona.

METHODS

Lead monitoring.—We began capturing and testing condors for lead exposure during 1999–2001, and have since attempted to test all free-ranging birds at least twice per year. Each condor was identified by a studbook (SB) number assigned at fledging (Mace 2005). We captured condors in a “walk-in” chain-link trap measuring approximately 3.7 m × 3.7 m × 1.6 m in height. Pre-baiting with calf carcasses encouraged condors to enter and exit the trap freely. We observed from a blind and closed the door to the trap by means of a hand-operated cable and pulley system. We then entered the trap, caught each target condor with a hand net, and transported it to a nearby processing area. From one to three people held the condor while a fourth withdrew 1–3 mL of blood from the medial-tarsal vein using a 22-gauge needle and heparinized tubes for sample storage. Using standard techniques for blood collection and lead analysis in the field, we transferred 50 µg of whole blood from each sample to a vial containing 250 µl of 0.35 molar HCl, thence to a sensor strip inserted into a portable blood lead analyzer (LeadCare Blood Lead Testing System, ESA Inc., Chelmsford, Massachusetts) (Fry and Maurer 2003). This instrument determines and displays lead values between 0–65 µg dL⁻¹. We also submitted samples ($n = 163$) for testing to commercial laboratories, some for the purpose of comparison with field-instrument values, but in most cases to accurately determine lead values when they exceeded the field analyzer’s limit of 65 µg dL⁻¹.

Except for occasional aberrations, consistency within samples of blood tested with the field analyzer ($n = 113$) were within the ± 4.6 µg dL⁻¹ standards reported by the manufacturer (Fig. 1A). Laboratory analyses of samples ($n = 56$) were also fairly consistent with duplicate samples sent to the same or different laboratories (Fig. 1B). However, in comparisons of field- vs. laboratory-tested values ($n = 99$), the latter showed higher levels in all but three cases (Fig. 2). For field values of greater than 30 µg dL⁻¹ ($n = 17$ comparisons), the laboratory values averaged 1.8 times higher. By necessity, we made management and treatment decisions primarily in response to the field-tester, but in this report, where both field-tester and laboratory values were available, we list the laboratory values on the assumption of their greater accuracy.

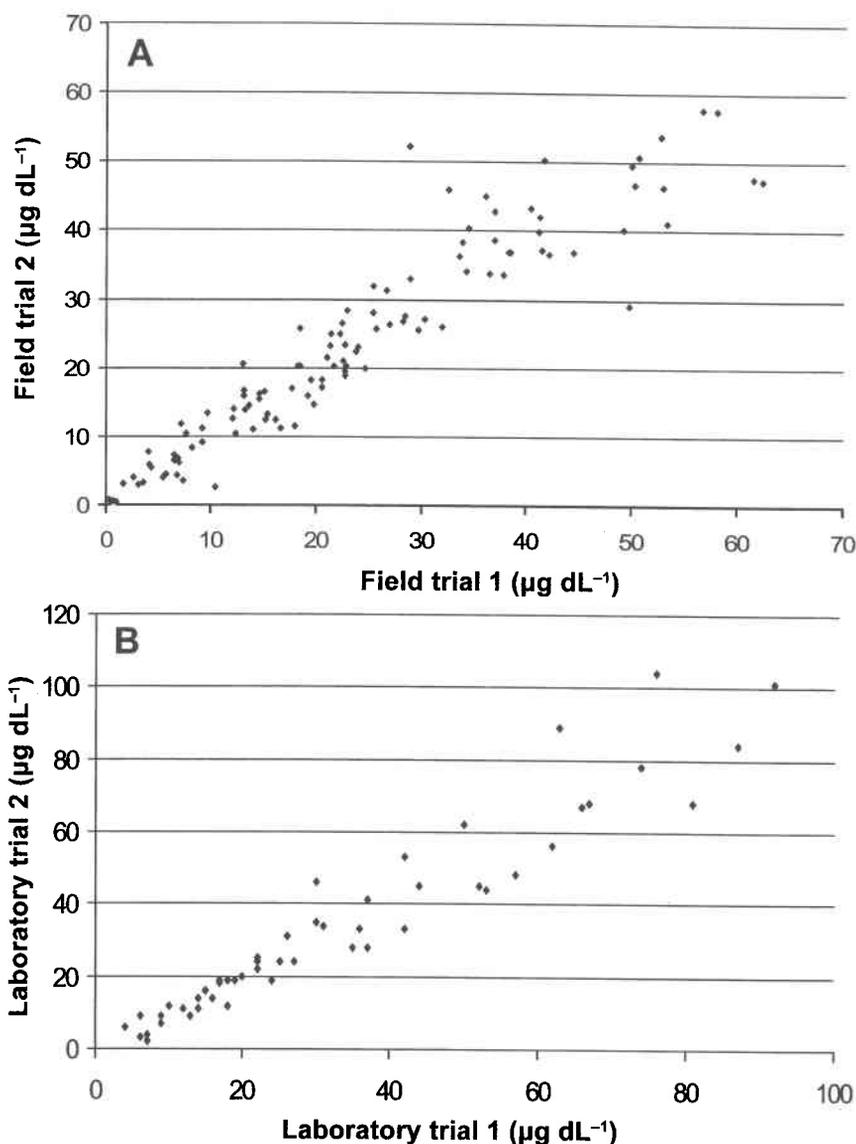


Fig. 1. (A) Comparisons within 113 duplicate sets of condor blood samples from Arizona, 1999–2004, tested with a portable field analyzer. (B) Comparisons within 56 duplicate sets of condor blood samples tested by commercial laboratories. The figure excludes three outliers: (1) $136 \mu\text{g dL}^{-1}$: $189 \mu\text{g dL}^{-1}$, (2) $199 \mu\text{g dL}^{-1}$: $415 \mu\text{g dL}^{-1}$, and (3) $539 \mu\text{g dL}^{-1}$: $570 \mu\text{g dL}^{-1}$.

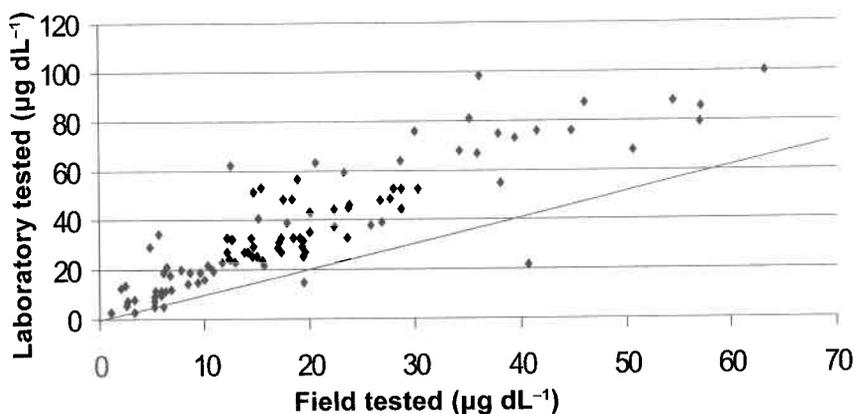


Fig. 2. Comparison of field and laboratory test results of duplicate condor blood samples ($n = 99$), excluding a single outlier (ESA field tester = $35 \mu\text{g dL}^{-1}$, Lab = $212 \mu\text{g dL}^{-1}$). The line depicts the ideal parity of duplicates.

RESULTS

Lead exposure.—We annually tested condors for lead contamination during 1999–2004 (Fig. 3). We analyzed 437 samples during the period, of which 261 (60%) showed “background” lead concentrations of $0\text{--}14 \mu\text{g dL}^{-1}$. Eighty-two samples (18.7%) yielded levels of $15\text{--}29 \mu\text{g dL}^{-1}$ (indicating lead exposure), 55 (12.6%) showed $31\text{--}59 \mu\text{g dL}^{-1}$, and 39 (9%) were over $60 \mu\text{g dL}^{-1}$, the threshold at which the term “clinically affected” has been applied (Fry and Maurer 2003). Laboratory tests showed that 25 of the latter group were above $100 \mu\text{g dL}^{-1}$ (termed “acutely toxic” by Kramer and Redig 1997); 10 of those exceeded $200 \mu\text{g dL}^{-1}$, and 5 showed greater than $400 \mu\text{g dL}^{-1}$. It is important to note that these reported lead levels do not preclude higher degrees of original exposure, as levels are subject to peaking and depuration between lead ingestion and testing (see Fry and Maurer 2003).

Condors feeding primarily on proffered carcasses (dairy calves) at the release site showed blood lead levels in the range of $0\text{--}12 \mu\text{g dL}^{-1}$. Aside from a shotgun pellet episode in summer 2000 that resulted in the deaths of at least two condors (see Woods et al. this volume), exposure levels did not increase until 2002 when condors began frequenting the Kaibab Plateau during the fall deer seasons (see Hunt et al. 2006). The apparent rise in the overall proportion of exposures during 2002–2004 (Fig. 3) was consistent with this increasing use of the Kaibab Plateau, and the period of highest exposure in each of those three years was during and just after the deer season (Hunt et al. this volume). The difference between the two

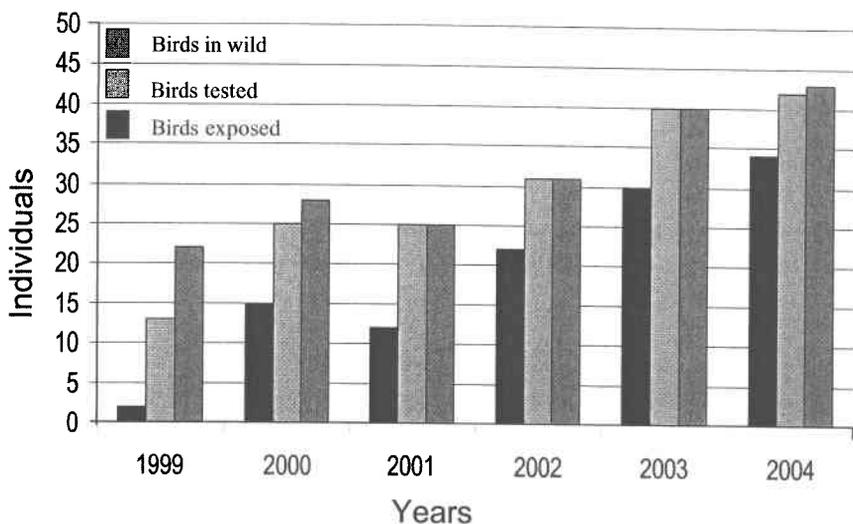


Fig. 3. Trend in lead testing and exposure of California Condors in Arizona during 1999–2004.

three-year periods—1999–2001 and 2002–2004—in the ratio of condors showing background levels ($0\text{--}14\ \mu\text{g dL}^{-1}$) to those indicating exposure was highly significant ($\chi^2 = 15.4$, $df = 1$, $P < 0.001$).

Radiography.—We searched for radio-dense particles in condor radiographs produced by local veterinarians using standard diagnostic radiological equipment usually within 48 hours of blood assays in the field. In 2000, the first year of known exposure, radiographs of eight birds showed five with shotgun pellets in the digestive system; four of these birds were alive and one was dead (Woods et al. this volume). This incident prompted us over the next several years to x-ray all condors showing high lead levels ($>60\ \mu\text{g dL}^{-1}$). However, results showed that of 13 lead-exposed condors radiographed during 2001–2002, only two (one alive, one dead) contained radio-dense fragments; in 2003, three of eight radiographed birds showed fragments. In an effort to reduce unnecessary overall exposure of condors to x-rays, we began radiographing only those retested birds showing increasing blood lead levels or those showing lack of immediate response to chelation therapy. In 2004, a year of many exposures, two condors showed trends of lead increase after capture, and both revealed fragments in radiographs.

Treatment.—Chelation therapy of condors showing high lead levels involved standard intramuscular (pectoral) injections of calcium edatate (or Ca EDTA) twice daily for five days (see Murase et al. 1992). Lethargic birds and those showing signs of dehydration were given oral and/or

subcutaneous fluid (i.e., standard lactated Ringer's solution). Chelation usually resulted in rapid depuration of blood lead levels. For example, condor SB #133 on the first day of testing showed a field-test lead value of $>65 \mu\text{g dL}^{-1}$, and laboratory analysis of the same sample revealed a lead value of $162 \mu\text{g dL}^{-1}$. We began chelation that day. On day three, the level had dropped to $42 \mu\text{g dL}^{-1}$ on the field-tester (lab value = $73 \mu\text{g dL}^{-1}$), and by day five, the field-tester yielded $24 \mu\text{g dL}^{-1}$ (lab value = $39 \mu\text{g dL}^{-1}$). After five days post-treatment, the field-tester showed a lead value of $11 \mu\text{g dL}^{-1}$, and the bird was released. Retesting of this bird three and four months later showed no increase in lead levels.

In some cases, however, a second five-day round of chelation was needed. For example, condor SB #235 showed a field-test lead value of $36 \mu\text{g dL}^{-1}$ on the initial day of testing. We retained this bird to determine whether lead levels were increasing or decreasing. Five days later, the field-tester indicated a blood lead value of $>65 \mu\text{g dL}^{-1}$, and we began chelation. On the fourth and sixth day after treatment began, the lead levels remained at $>65 \mu\text{g dL}^{-1}$. No lead bodies were apparent in a radiograph taken on the eighth day of treatment, but lead levels had by then dropped to $46 \mu\text{g dL}^{-1}$. We stopped treatment, and three days later, lead levels had fallen to $23 \mu\text{g dL}^{-1}$. Differences between these two case histories suggest a difference in the chronology of exposure. Exposure of condor SB #235 was likely more recent than that of condor SB #133 at the time of testing, and lead levels may have been rising as a result of lead bodies remaining in the stomach.

Condors with detectable radio-dense particles were transported to the Phoenix Zoo Hospital for treatment. Shotgun pellets were surgically extracted in two cases. Condors with fragments were treated with fluids, chelation, and oral doses of psyllium fiber to purge lead from the digestive system. For example, 1.5 days after condor SB #235 was observed in the vicinity of a heavily scavenged coyote (*Canis latrans*) carcass, the remains of which were found to contain bullet fragments, the field-tester indicated a lead value of more than $65 \mu\text{g dL}^{-1}$. A laboratory assay of the same blood sample showed a value of $555 \mu\text{g dL}^{-1}$. Radiography revealed fragments in the stomach, and chelation and psyllium purging began within 48 hours of exposure detection. Two days later, laboratory testing showed a level of $489 \mu\text{g dL}^{-1}$. Fecal materials were collected and radiographed to provide an indication of lead fragment passage, and all fragments had passed by the ninth day after their first detection in condor SB #235's stomach. Thirteen and 21 days after exposure detection, under continued treatment, laboratory lead values had declined to 37 and $28 \mu\text{g dL}^{-1}$, respectively.

Although no treated condor died, one poisoning was too far advanced to begin chelation, and the bird died while being transported to the Phoenix Zoo for treatment. In all, 28 of the 50 condors in the Arizona

flock received at least one chelation series during the reporting period, 17 received two chelations (20 injections), 5 were chelated four times, and 2 had six chelations (60 injections each). One of the latter two condors subsequently died of lead poisoning in January 2005, one month after successful treatment of a previous exposure. Eleven of the fourteen condors showing lead-shot (Fig. 4A) or fragments in radiographs (Fig. 4B) were found alive, and three were discovered post-mortem; all of the latter were diagnosed as having died of lead poisoning.

DISCUSSION

Lead toxicity in birds appears to vary broadly among species and even among individuals (Carpenter et al. 2003); for example, Red-tailed Hawks (*Buteo jamaicensis*) and Turkey Vultures (*Cathartes aura*) show greater tolerance than Bald Eagles (*Haliaeetus leucocephalus*) (Reiser and Temple 1981, Carpenter et al. 2003). Clinical signs of lead toxicity, such as depression, lethargy, vomiting, diarrhea, nonregenerative anemia, anorexia, blindness, and seizures, have been observed in waterfowl and raptors with blood concentrations exceeding $100 \mu\text{g dL}^{-1}$ (Locke and Tomas 1996, Kramer and Redig 1997). However, threshold blood lead levels at which such manifestations appear in condors are still poorly known, and may remain undetected until just prior to death (Fry and Maurer 2003). Overt signs of lead poisoning may not be apparent in free-flying condors without close observations, and these are often difficult to make. It is therefore important to obtain a laboratory value as soon as possible when an exposure is detected at the upper limit of a field analyzer (i.e., $>65 \mu\text{g dL}^{-1}$).

In our study, laboratory results almost invariably exceeded those reported by the field-tester. However, the economics, portability, and speed of assay of the field instrument made it essential for classifying exposure levels for management decisions, for example, whether or not to hold a condor for further testing or for the return of laboratory results. Accordingly, we used the field tester's indicated value of about $60 \mu\text{g dL}^{-1}$ as the treatment threshold for condors, whereas laboratory comparisons suggested that, on average, the true value was nearly double (180%) that concentration, or about $108 \mu\text{g dL}^{-1}$ (Fig. 2).

Unfortunately, the lag in timing between field and laboratory testing, coupled with the logistical challenge of transporting condors for radiography, can hinder the process of evaluation and decision-making regarding treatment. Accurate assessment is further confounded by the question of when the condor was exposed versus when it was tested. Lead half-life in avian blood is estimated at 7–20 days, whereas lead in other tissues and bone may persist for many months (Reiser and Temple 1981, Eisler 1988, Fry and Maurer 2003). A high value may indicate recent exposure, but it may also reflect a

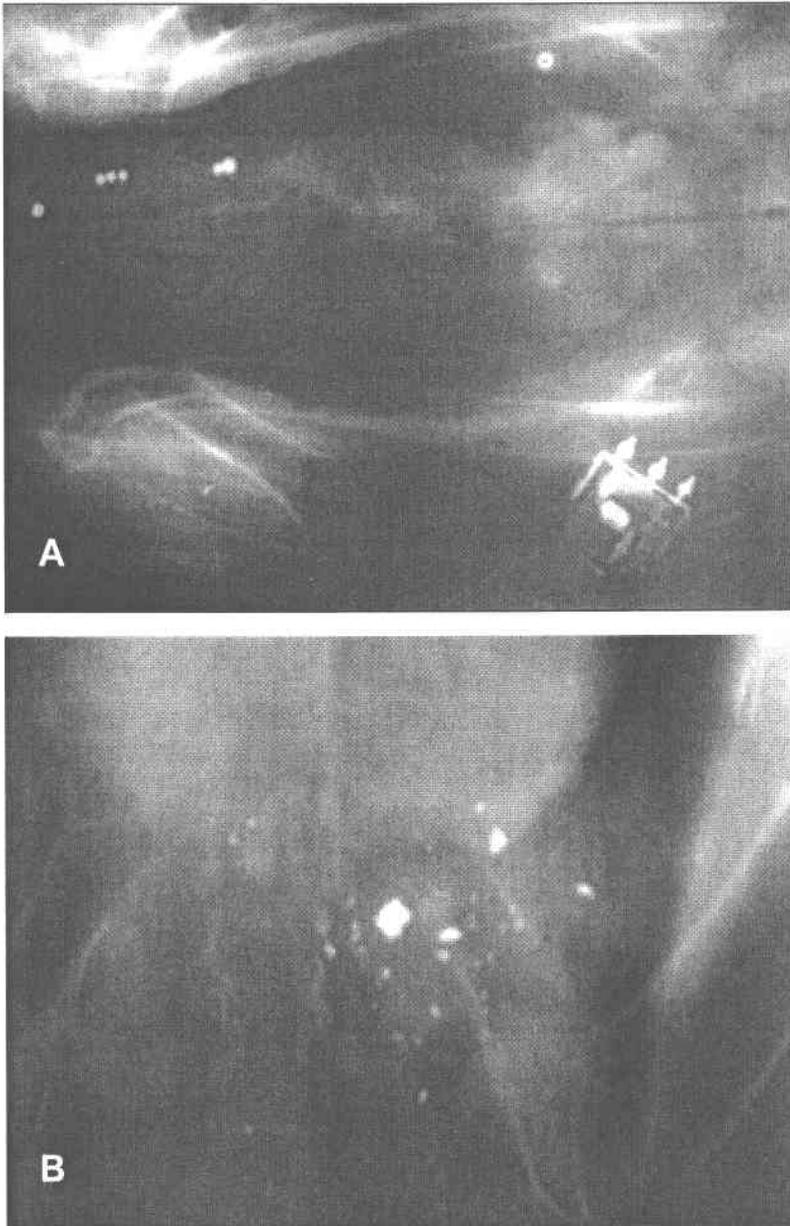


Fig. 4. (A) Radiograph of the digestive tract of condor SB #165 containing lead shotgun pellets of two sizes. Lead poisoning was the diagnosed cause of death (VHF transmitter visible). (B) Radiograph of condor SB #243's stomach containing lead bullet fragments; its blood lead level four days later showed $691 \mu\text{g dL}^{-1}$.

point along a trend of depuration from an even higher level, or the continued presence within the stomach of lead bodies that may cause levels to rise after testing. It is thus important to consider that a measurement of moderate blood lead concentration at the time a free-ranging condor is captured for sampling may not reflect the degree of exposure. Thus, deciding whether to begin chelation is based on (1) an in-the-field detection of a high lead level ($\sim 60 \mu\text{g dL}^{-1}$), (2) a clear trend of increase toward a higher level over several days, or (3) the continuance of a moderately high level over time (Fig. 5). Whereas the interval between lead ingestion and testing will usually remain unknown, as will the form and severity of exposure, retaining a condor and monitoring the trend of blood lead concentration over several days may shed light on the question of continued mobilization of lead into the bloodstream that may suggest the presence of lead in the condor's stomach (Fig. 4). This procedure minimizes the necessity of routine radiography and its potential for damaging DNA, particularly germ line DNA.

In conclusion, The Peregrine Fund has settled on a management program based upon the periodic testing of blood lead concentrations at a minimum of twice per year and concentrating effort at times of expected contamination based on exposure histories and seasonal events, particularly the fall deer hunting seasons when condors encounter lead in the form of spent bullet fragments (Hunt et al. 2006, this volume). Anomalous episodes, like those of shotgun pellet ingestion, are more difficult to anticipate, although close monitoring of condor movements and behavior have occasionally allowed us to identify exposed birds. By examining data on

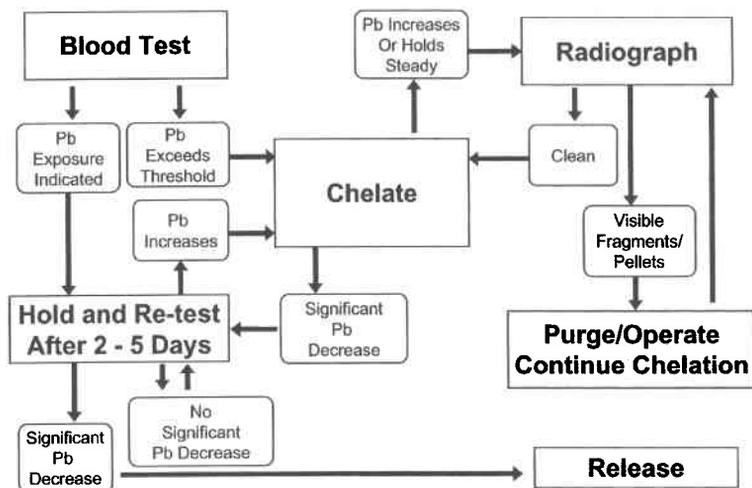


Fig. 5. Flow diagram representing The Peregrine Fund's protocol for evaluating and treating condors exposed to lead in Arizona.

the movements of condors associated with the affected individual(s) in the weeks prior to presumed exposure, we are able to identify and target those additional birds in need of testing (see Hunt et al. this volume). Among the many unknowns is whether or not the current blood lead thresholds (field-test value of $\sim 60 \mu\text{g dL}^{-1}$) are the appropriate levels at which treatment should commence. There are also the uncertain effects of multiple exposures within a short time span or the long term effects of more widely-spaced, multiple, subclinical exposures. As of September 2005, every condor in Arizona that is two years old or older has been exposed to lead, and eight of ten condors nine years old or older have shown lead levels exceeding $100 \mu\text{g dL}^{-1}$. Whether such frequent and long-term exposure to lead will affect future reproductive capacity and survival is as yet unknown.

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