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ABSTRACT: A moribund, emaciated California quail (*Callipepla californica*) that was found in an orchard in the state of Washington had an impacted crop and gizzard. Pellets containing the anticoagulant chlorophacinone (Rozol®, RO) were in the crop; the gizzard contents consisted of a pink mass of paraffin that was selectively accumulated from the paraffinized pellets. The plasma prothrombin time of 28 sec was near that determined for control quail. The signs of RO intoxication seen in the moribund wild quail were duplicated in captive quail given ad libitum diets of either RO or another paraffinized chlorophacinone pellet (Mr. Rat Guard II®, MRG). This left little doubt that paraffin impaction of the gizzard was the primary problem. All captive quail fed RO or MRG pellets showed no increases in prothrombin times compared to control values, died in an emaciated condition, and had gizzards impacted with paraffin.

INTRODUCTION

Voles (*Microtus* spp.) are capable of inflicting considerable damage to fruit trees. As a result, a number of chemicals have been used to control vole populations (Byers et al., 1982). Some of the rodenticides such as endrin are exceptionally toxic to non-target wildlife (Blus et al., 1983). As a result there has been a shift to rodenticides such as the anticoagulants chlorophacinone and diphacinone that are considered less hazardous to wildlife. Our recovery of a wild California quail that apparently died as a result of ingesting paraffinized chlorophacinone pellets prompted this investigation. The purposes of this paper are to describe the circumstances related to mortality of the wild quail and to report the response of captive California quail to diets consisting solely of chlorophacinone or diphacinone pellets.

MATERIALS AND METHODS

On 1 December 1981, a moribund, adult female California quail (025) was picked up by an orchardist in Wenatchee, Washington. A blood sample was taken for evaluation of prothrombin time. The bird was killed the same day it was found. Brain, liver, and breast mus-

cle samples were sent to the Patuxent Wildlife Research Center in Laurel, Maryland where they were analyzed for residues of organochlorine pollutants using electron capture gas chromatography (Cromartie et al., 1975; Kaiser et al., 1980).

Groups of pen-reared adult male and female California quail were placed together in cages and given ad libitum diets consisting solely of either paraffinized chlorophacinone pellets (Rozol®, RO, Chempar Chemical Co., Inc., 660 Madison Ave., New York, New York 10021, USA, or Mr. Rat Guard II®, MRG, International 2000, Inc., Oklahoma City, Oklahoma 73111, USA) or diphacinone pellets (Ramik Brown®, RB, Velsicol Chemical Corporation, 341 East Ohio St., Chicago, Illinois 60611, USA). Each treatment group consisted of three or four birds. Mr. Rat Guard II® is similar to RO except for the gray coloration. Diphacinone is also an anticoagulant that is used as a rodenticide in orchards. The apple-flavored high protein RB pellets contain 50 μg/g active ingredient and no paraffin.

The general procedures described by Quick (1957) were followed in determination of the one-stage prothrombin time using blood plasma. Blood samples were taken from the brachial or jugular vein; samples were cooled to about 5 C and processed within several hr after collection. Freeze-dried thromboplastin (Difco Chick Embryo Extract EE100®, Difco Laboratories, P.O. Box 1058-A, Detroit, Michigan 48232, USA) was placed in a 10-ml solution containing 0.025 M $CaCl_2$ in 0.85% NaCl and incubated in a water bath for 45 min at 39 C; the thromboplastin mixture was refrigerated and used within 7 days of preparation. The throm-
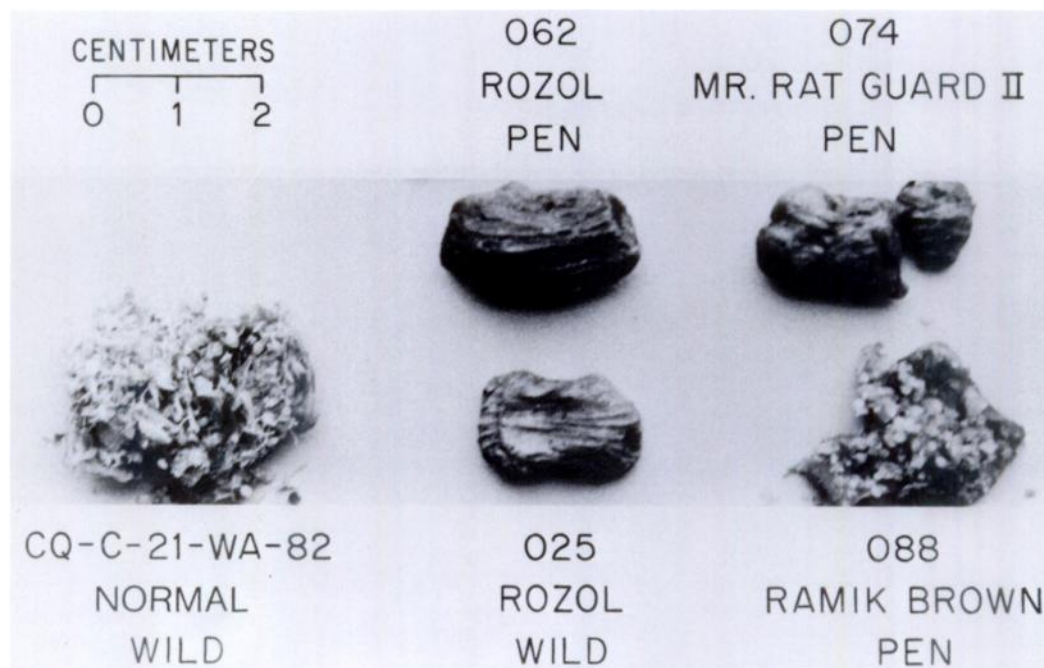
FIGURE 1.   Gizzard contents of California quail. Paraffin impaction in a wild quail (025) and in two pen-reared birds (062 and 074) fed chlorophacinone pellets. Contents of a bird (088) fed Ramik Brown® and a normal wild quail (CQ-C-21-WA-82) (gizzard linings attached) are included for comparison.

boplastin mixture (0.2 ml) and oxalated blood plasma (0.1 ml) were transferred to separate chambers on a fibrometer (Fibrosystem®, BBL, Division of Becton, Dickinson and Company, Cockeysville, Maryland 21030, USA) where they were warmed to reaction temperature (37 C). The plasma was then pipetted into the thromboplastin mixture and the timer activated. The fibrometer automatically stopped when the first fibrin strand was formed; the prothrombin time was displayed on a digital readout. Prothrombin time was determined for one or more control birds each day that samples were run.

For quail on toxic diets, one-way analysis of variance was used to compare pre- and post-test mean body weights and to compare mean prothrombin times with the control value. Considering both body weight and prothrombin time, means were separated by a multiple range test (Kramer, 1956). A paired t-test was also used in comparing pre- and post-test body weights of individual birds on each toxic diet. The level of statistical significance for all tests is $P \leq 0.05$.

RESULTS

Necropsy of California quail 025 revealed that it was extremely emaciated (hatchet-breasted). All lipid reserves including coronary fat had apparently been mobilized. The impacted crop was full and contained about equal parts of seeds and a pink material subsequently identified as RO pellets that are broadcast in orchards in late fall to control voles. The cylindrical pellets (5 mm diameter and irregular lengths up to about 20 mm) contain inert ingredients (primarily grain and paraffin) and 50 μg/g of the anticoagulant chlorophacinone. The gizzard was impacted (Fig. 1), and the contents consisted of a pink compact mass that contained little grit.

The prothrombin time of 28 sec for quail 025 was slightly above the mean prothrombin time of 21.2 sec (range 19 to 23 sec) determined for 19 control California quail in this study. Residues of organochlorines in tissues (Table 1) were all well below levels associated with mortality or sublethal effects.

All seven captive California quail that

TABLE 1.   Organochlorine residues in tissues of a wild California quail (025).

Tissue	μg/g (fresh wet wt.)[a]						
	DDE	Dieldrin	HE	OXY	TNCH	Endrin	PCB's
Brain	10.0	0.11	0.03	ND[b]	0.06	0.07	0.48
Breast muscle	2.1	ND	ND	ND	ND	ND	ND
Liver	12.0	0.19	0.04	0.03	ND	0.93	ND

[a] DDE = dichlorodiphenyldichloroethylene, HE = heptachlor epoxide, OXY = oxychlordane, TNCH = trans-nonachlor, PCB's = polychlorinated biphenyls resembling Aroclor 1260.
[b] ND = no residue detected.

were fed chlorophacinone pellets consumed approximately 0.4 to 0.7 g pellets/bird/day and were severely emaciated when they died after 7 to 12 days on the toxic diet (Table 2). These birds lost a significant amount (27 to 60%) of their pretreatment body weights (paired t-test). The gizzards of all birds were impacted (Fig. 1); coloration of the contents was pink in those fed RO and gray in those fed MRG. The gizzard impaction was related to the selective accumulation of paraffin from the pellets into a compacted mass. The gizzard contents of four birds on chlorophacinone diets melted when heated with a lighted match. Gizzards of these birds contained an abnormally low quantity of grit. The body condition of the captive California quail fed chlorophacinone pellets was similar to that noted in quail 025 except that their crops contained little or no food and were not impacted. Prothrombin times of four quail fed chlorophacinone pellets ranged from 21 to 25 sec (99 to 118% of the mean control value)

after 7 to 11 days even though most of these determinations were made when the birds were near death (Table 3).

The quail given the RB diet ingested approximately 8.3 g of pellets/bird/day and generally maintained their body weight. Birds were killed after 14 to 36 days on the toxic diets; post-treatment weights (Table 2) ranged from −11 to +9% of pretreatment values, and were not significantly different (paired t-test). Crops and gizzards of all birds on the RB diet appeared normal (Fig. 1).

Prothrombin times of four quail fed RB ranged from 17 to 28 sec (80 to 132% of our control values). There were no significant changes in prothrombin time through 36 days on the RB diet, and prothrombin values were not significantly different from those of birds fed RO (Table 3).

Quail 025 was the only wild California quail of 59 examined in the Wenatchee area in 1981 to 1983 that had chlorophacinone pellets in its crop. We found RB pellets in crops of two wild quail found

TABLE 2.   Body weight relations of pen-reared California quail given no-choice diets of anticoagulant pellets.

Treatment[a]	n	Sex	Status[b]	Days on diet	Mean body wt. (g)[c]		% Weight change
					Pre	Post	
RO	3	2M, 1F	D	8–12	175 AB	106 C	−27 to −48
MRG	4	2M, 2F	D	7–10	195 A	85 C	−53 to −60
RB	4	2M, 2F	K	14-36	173 AB	167 B	−11 to +9

[a] RO = Rozol®, MRG = Mr. Rat Guard II®, RB = Ramik Brown®.
[b] D = died on dosage, K = killed.
[c] A significant difference is indicated for those means not sharing a common letter.

TABLE 3.   Plasma prothrombin time of pen-reared California quail given ad libitum diets of chlorophacinone and diphacinone pellets relative to control values.

Treat-ment[a]	Days on diet	n	% of control value	
			Mean[b]	Range
RO	7	3	109	99–118
RB	7	4	109	95–132
RB	14	4	103	80–110
RB	23	3	109	103–111
RB	36	2	98	92–105

[a] RO = Rozol®, RB = Ramik Brown®. Prothrombin time (108% of control value) for a quail on the Mr. Rat Guard II® diet for 11 days not included in the statistical analysis.
[b] Means not significantly different.

dead in 1980–1981 and in crops of two ring-necked pheasants (*Phasianus colchicus*) obtained from hunters in 1979.

DISCUSSION

The duplication of signs of RO intoxication of California quail 025 in captive birds leaves little doubt that the moribund condition of the wild California quail was caused by impaction of the gizzard through selective accumulation of paraffin from the pellets. Digestion of food seemed impossible in those birds with impacted gizzards.

According to Fitzek (1978), the toxicity of all anticoagulants is due to depression of prothrombin level of blood that leads to uncontrollable bleeding. One-stage prothrombin time provides a means of detecting changes in prothrombin level (Quick, 1957). Even large changes in prothrombin time in animals ingesting anticoagulants are difficult to interpret. Several golden eagles (*Aquila chrysaetos*) fed diphacinone-contaminated meat for 5 to 10 days experienced increases in prothrombin time from a control value of 23 sec up to 900 sec (Savarie et al., 1979). All eagles survived, but several experienced weakness and hemorrhaging wounds. This indicates that chlorophacinone and diphacinone had little effect on the vascular system of California quail and that problems

from chlorophacinone pellets were related to paraffin impaction

This is apparently the first record of birds dying from impaction of their gizzards with paraffin from anticoagulant pellets. The observations that some wild birds are ingesting these toxicants and that one of these birds suffered almost certain lethal effects indicate that there is some hazard to individuals. Adverse effects of these anticoagulant pellets on wildlife populations are probably minimal, although the evidence is inconclusive.

ACKNOWLEDGMENTS

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BOOK REVIEW . . .

The Ticks of California (Acari: Ixodida), Deane P. Furman and Edmond C. Loomis. Bull. Calif. Insect Survey 25: i–viii, 1–239, University of California Press, Berkeley, California 94720, USA. $25.00 US.

A total of 49 described species (seven genera) of ticks are recorded from California. One species [*Argas* (*A.*) *cooleyi*] consists of populations typical of this species plus a population of a species in the process of being described as new. Two other species have been introduced repeatedly into California. In this state, ticks harm wildlife, livestock, and man by causing irritation, anemia, toxemia, allergic sensitization, and paralysis. They transmit five serologically distinct strains of spotted-fever-group rickettsiae and the agents causing Q fever (*Coxiella burneti*), bovine anaplasmosis (*Anaplasma marginale*), Colorado tick fever (CTF) (an apparently new CTF-related virus), Powassan encephalitis (POW virus), tularemia (*Francisella tularensis*), tickborne relapsing fevers (*Borrelia hermsi, B. parkeri*), Lyme disease (*B. burgdorferi*), canine jaundice and possibly human babesiosis (*Babesia microti*), an uncharacterized malaria-like disease of rodents and lagomorphs, and epizootic bovine abortion (agent and wildlife associations undetermined). To aid in tick identification, the first section of the monograph briefly reviews the six species most commonly encountered in California, lists taxonomic characters and their definitions, and keys to tick families, genera, and species by adult and immature stages. The obviously expert review of each species includes taxonomy, diagnosis, bionomics, and medical importance. Each species review is supported by a map of distribution in California and by one or more plates of illustrations (a total of 356 figures). The first four of the 75 plates are line drawings with explanations of structural details. The remaining 71 plates consist of scanning electron microscope illustrations of definitive characters. There are eight pages of references.

The illustrations are excellent and outstandingly useful. The smoothly flowing text contains a wealth of easily managed detail. The entire monograph is marked by a high degree of accuracy, meticulous attention to detail, clarity, and intimate knowledge of the variety of subjects treated. It is a model for workers elsewhere and a boon to entomologists, parasitologists, zoologists, epidemiologists, and specialists in human and veterinary medicine.

A good deal of effort is being made by several California specialists, independently and in collaboration with others elsewhere, to answer fascinating questions regarding several tickborne agents and their epidemiology in this state. Much information is presented for a number of species of ticks in California, but other poorly known species, as well as some California tick-associated infections, cry for more intensive field and laboratory investigations.

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