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Retrospective evaluation of feline rodenticide exposure and gastrointestinal decontamination: 146 cases (2000–2010)

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Abstract

Objective – To determine the prevalence of rodenticide exposure in cats, describe the use of gastrointestinal decontamination (GID) after rodenticide exposure, and examine the efficacy of GID following exposure to anticoagulant rodenticides (ACR).
Design – Retrospective study from 2000–2010.
Setting – Emergency service of an urban university teaching hospital.
Animals – One hundred forty-six cats presented for rodenticide exposure.
Main Results – Annually, the number of cats that were presented for rodenticide exposure averaged 13 of 3,336 (0.39%) and totaled 146 cases over 11 years. Cats that had been exposed to rodenticide were significantly more likely to be young ($P < 0.001$), sexually intact ($P < 0.001$), and presented in the fall season ($P = 0.002$). The majority of cats lived indoors (67.6%). The type of rodenticide involved in the exposure was unknown in 50% (71/142) of cases. Of the known types, ACRs were most common (59/142, 41.5%) followed by cholecalciferol (7/142, 4.9%) and bromethalin (5/142, 3.5%). Gastrointestinal decontamination was attempted in 21/36 (58%) cats with exposure to a known ACR. Emesis was attempted in 17/21 (81%) and charcoal administered in 14/21 (67%) cats that underwent GID. This study did not detect an effect of GID efforts on prothrombin time (PT) prolongation 48 hours after exposure to a known ACR.
Conclusions – Cats consume rodenticides. Due to the lack of evidence of altered outcome associated with GID in cats exposed to ACRs, a PT should be evaluated 48 hours after first exposure regardless of whether GID is performed. Treatment should be based on the results of the PT. Gastrointestinal decontamination should be performed at the clinician's discretion based on history, risks, calculated toxic dose, low prevalence of ACR toxicosis in cats, general resistance of cats to ACR toxicosis, and treatment options.

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Keywords: anticoagulant, emesis, poisoning, prothrombin time, toxicology

Abbreviations

ACR	anticoagulant rodenticide
ACRG	anticoagulant rodenticide group
GID	gastrointestinal decontamination
PT	prothrombin time
REG	rodenticide exposure group

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Introduction

Rodenticides are consistently included in the 10 most common poisons reported to be ingested by domestic animals in the United States by the Animal Poison Control Center.[1–4] Historically, anticoagulant rodenticides (ACR) have been the second most common toxin responsible for companion animal death.[5,6] Overall prognosis can be good to excellent but generally depends on client financial limitations, time from ingestion to presentation, severity of clinical signs, and location of hemorrhage.[6,7] The clinical literature regarding cats and rodenticides

is limited to case studies or small case series of clinical toxicosis.[8–10] Published case numbers regarding exposure to or toxicosis from ACR are likely a vast underestimate, since most veterinarians are familiar with the management of these intoxications and because its antidote is readily available.

Given the risk associated with exposure to rodenticides, gastrointestinal decontamination (GID) procedures such as emesis induction and administration of activated charcoal are recommended in veterinary medicine.[6,7] However, the efficacy of GID in multiple toxicoses has come under scrutiny in the human medical community and risk-benefit comparison of GID determines the protocol utilized.[11,12] The American Academy of Clinical Toxicology guidelines state that there is no sufficient evidence to support or exclude ipecac administration after poison ingestion and that activated charcoal as a single dose should not be administered routinely since there is no evidence that administration improves clinical outcome.[13,14] Emesis and administration of charcoal have shown little clinical effect on overall outcome after long acting ACR ingestion in people, likely because such small doses are usually consumed. Gastrointestinal decontamination is therefore only recommended for people in certain cases according to recent consensus guidelines for treatment of ACR ingestion.[15]

The only clinical study in veterinary medicine to examine GID following ACR ingestion in dogs showed that it was safe to treat with oral vitamin K based on prothrombin time (PT) testing 2–3 days following GID.[16] That study did not, however, compare the population of animals that received GID to those that did not, nor did it evaluate the ACR dose ingested to determine in which cases GID might be indicated.

Anticoagulant rodenticides are considered a good model for GID efficacy since the PT test can be used to determine the effectiveness of GID after ingestion of a toxic dose prior to development of clinical hemorrhage. This is a safe protocol based on the difference in the half-lives of coagulation factor VII, which prolongs PT results, and factor II, which is the main factor implicated in clinical hemorrhage.[16,17] A recent study demonstrated the efficacy of GID for ACR exposure in dogs through PT time 2–6 days after decontamination,[16] but management of any type of rodenticide exposure in cats with GID has not been reported. Experimental studies in cats after the administration of warfarin, a first generation ACR, have found that the maximum prothrombinopenic response occurred at 24–48 hours,[18] which suggests that the timeline for this model may be similar to that for dogs.

The first aim of this study was to describe the characteristics of cats with suspected rodenticide exposure that were presented to an urban emergency service. The second portion of this study examined the subset of this population that was exposed specifically to ACR to describe the methods and efficacy of GID in a clinical setting.

Materials and Methods

The Matthew J. Ryan Veterinary Hospital of University of Pennsylvania's (VHUP) computerized medical record database was searched to identify cats that were presented to the emergency service between January 01, 2000 and December 31, 2010. The computerized database was searched for the complaint of possible rodenticide ingestion, a final diagnosis of rodenticide exposure, and records that contained the word rodenticide, first generation, second generation, bromethalin, anticoagulant, bromadiolone, warfarin, brodifacoum, difenacoum, coagulopathy, hypercalcemia, seizure, or cholecalciferol.

All of the identified medical files were then reviewed. Cats were included in the rodenticide exposure group (REG) for analysis of prevalence, signalment, and seasonality, and whether or not the owner indicated that there was a known or suspected recent exposure to a rodenticide. Cats were excluded from the study if rodenticide exposure was considered impossible (eg, the cat was a solely indoor cat and no rodenticides were located on the premises), or a definitive diagnosis other than rodenticide exposure was made despite being presented with clinical signs consistent with rodenticide intoxication (ie, coagulopathy, hypercalcemia, or neurologic signs).

Prevalence, signalment, and seasonality of rodenticide exposure

The information collected included signalment, whether the cat was an indoor or outdoor dweller, date and time of presentation, clinical signs, type of rodenticide (if known), and time of possible or known ingestion. Cats were defined as outdoor dwellers if they spent any time outside unsupervised, and cats were classified as indoor dwellers if they remained indoors at all times, were leashed when outside, or were confined to a screened porch. The cats' ages were stratified into 6 age groups based on the AAFP-AAHA Feline Life Stage Guidelines for analysis.[19]

To determine whether the signalment or the proportion of cats varied significantly from the general population of cats that were presented to the emergency service over the same time period, the computerized medical record database was searched for all cats that were presented to the emergency service. For these cats, signalment and date of presentation were used for

analysis. Finally, in the REG the frequency of attempted GID, the type of GID, and the success of emesis induction were recorded.

Gastrointestinal decontamination in the anticoagulant rodenticide group

Only cats with possible exposure to a specifically identified ACR without clinical signs of toxicosis were included in the subgroup to evaluate efficacy of GID. In this anticoagulant rodenticide group (ACRG), the type of rodenticide was based solely on the owner's history without verification with toxicological testing due to the retrospective nature of the study. In the evaluation of GID efficacy, cats with an exposure to an unknown rodenticide or to a rodenticide with a different mechanism were excluded from the ARCG. Cats with exposure to a known ACR were also excluded if a PT result was not available, if they were empirically treated with vitamin K, or if clinical signs of a coagulopathy were present upon presentation.

The additional information recorded from their medical management included method of GID, type and dosage of medications given, and all PT results. The ACRG was then divided into a GID group, which received any form of GID (attempted emesis, administration of activated charcoal, or both) and a non-GID group, which did not receive any form of GID.

The PT results were categorized as either prolonged or normal, based on the standard given with each test result, since several different coagulation analyzers and methods were used over the 11-year period. If the analyzer used had an associated reference interval, the PT was considered prolonged when the result fell outside of this interval. If the analysis results compared the patient result to the pooled control sample, the result was considered prolonged when it was ≥ 125% of the pooled control sample. A modified international normalized ratio was calculated using the patient's PT result divided by the average normal time (or mean of the reference interval) and was then used for reporting prolongation in this publication.[20]

Statistical analysis

Categorical data were compared using the χ^2 test. Statistical significance was set at $P < 0.05$. All statistics were performed with a statistical analysis software package.[a]

Results

Prevalence, signalment, and seasonality of rodenticide exposure

The original computerized database search yielded 196 cases. Of these 196 cases, 54 were excluded; 9 cases were related to recalled pet foods that were originally (and erroneously) believed to be adulterated with a rodenticide, and 45 cases were presented with clinical signs of coagulopathy or seizures. Of cases that were presented with clinical signs, only 4 had a clear final diagnosis of known or highly suspected rodenticide toxicosis based on history, clinical signs, imaging, or biochemical or toxicological testing. Three of these 4 cases had a final diagnosis of ACR toxicosis based on history of known exposure or confirmation via rodenticide screening test (positive for bromadiolone). The other case's final diagnosis was unconfirmed but suspected to be ACR toxicosis. One of these cases suffered respiratory arrest and died in hospital before aggressive treatment or further diagnostics could be performed.

The remaining 142 cases that were presented without clinical signs were included in the REG. To examine the prevalence of rodenticide exposure and signalment of the REG, the emergency service's general feline population was used for comparison. On average, approximately 13 cats were presented to the emergency service for rodenticide exposure annually between 2000 and 2010. Based on the electronic medical records between 2000 and 2010, the annual number of cats that were presented to the emergency service averaged 3,336. Approximately 0.39% (13/3,336) of cat cases that were presented to this urban emergency service were thus related to rodenticide exposure.

No breed was overrepresented in the REG when compared to the emergency service population. Kittens ($P < 0.001$) and junior-aged cats (all cats < 2 years old; $P < 0.001$) were overrepresented (111/142, 78.2%) in comparison to the entire emergency service population (12,125/36,529, 33.2%). Within the REG, there was no significant difference between the male (70/142, 49.3%) and female population (72/142, 50.7%), but when compared to the general emergency service population, intact animals were overrepresented in the REG ($P < 0.001$).

The majority of the REG were indoor dwellers (67.6 %), whereas only 14.7% of cats were outdoor dwellers. No environment was specified for 17.6% of cats. These categories were not able to be compared to that of the general emergency service population. A recent change in environment was noted in the medical record of approximately 20% of the REG cats (10.3% of cats having recently moved into a new residence with their current owners and 13% of the cats having been acquired within the last month). Cats in the REG were significantly more likely to be presented in the fall season ($P = 0.002$) and significantly less likely to be presented in the spring ($P = 0.023$).

A summary of the class of rodenticide (ie, unknown, anticoagulant, cholecalciferol, bromethalin) implicated for the 142 cases in the REG is provided in Table 1.

Table 1: Type of rodenticide implicated in 142 cats presented for rodenticide exposure without clinical signs

Type of rodenticide	Number of cats	Percent of total population	Percent by generation
Not specified	71	50	
Cholecalciferol	7	4.9	
Bromethalin	5	3.5	
Anticoagulant	59	41.5	
First generation	2	1.4	First generation
Wafarin	2	1.4	100
Second generation	57	40.1	Second generation
Brodifacoum	30	21.1	53
Bromadiolone	24	16.9	42
Difethialone	2	1.4	4
Diphacinone	1	0.7	2

Although the 142 cats were believed to have been exposed to some type of rodenticide, the specific toxicant was recorded in only half (71/142) of the cases. It is unknown whether this was due to recording error, but it was frequently noted that the owners lacked the knowledge of the specific product used. Anticoagulant rodenticides were the most common type of known rodenticide followed by cholecalciferol and bromethalin. Of the ACRs, second generation were far more common than first generation ACRs.

The reported time of presentation after first possible exposure was highly variable and often imprecise, ranging from <1 hour to >48 hours. Most frequently, the owners reported a range of time over which ingestion could have occurred; commonly the owners found scattered or displaced pellets upon waking or returning from being out of the house. Almost one-third of cats were presented within approximately 6 hours of first possible exposure. Of the remaining cats, the time of exposure was unknown.

Of the 142 cats in the REG, 60 cats underwent attempted emesis induction; however, in 3 of the cases, when an emetic was given, it was not recorded whether emesis was achieved. In the remaining 57 cases, emesis was successfully induced in 19 (33%). The emetics used for GID included oral hydrogen peroxide dosed at 3–40 mL/cat doses repeated up to 3 times, 5–10 minutes apart; IV apomorphine[b] dosed at 0.015 mg/kg; IV xylazine[c] dosed at 0.2–0.44 mg/kg; or a combination of these medications. The emetic agent was not specified in 9 cases. Hydrogen peroxide induced vomiting in only 2/14 cases (14%), apomorphine in 0/3 (0%), and xylazine in 14/34 (41%). For 3 cases with successful emesis, the induction agent was not listed in the medical record or was achieved after multiple induction agents were given. Although the sample size was insufficient to document a statistically significant difference in emetic efficacy ($P = 0.098$), a much larger portion of cats vomited when adminstered xylazine than with any other emetic agent. Activated charcoal administration was attempted in 52 cats.

Table 2: Method of gastrointestinal decontamination in 36 cats and development of prolonged prothrombin times following exposure to anticoagulant rodenticides

Form of decontamination	Number of cats	Prolonged prothrombin time 24–48 hours after exposure
Emesis only	7	1
Activated charcoal only	4	0
Emesis and activated charcoal	10	2
None	15	3

Gastrointestinal decontamination in the ACRG

Thirty-six cases met the criteria for the ACRG. Twenty-one of the 36 ACRG cats received some form of GID (an emetic [$n = 7$], activated charcoal [4], or an emetic followed by activated charcoal [10]) and 15 received no GID. In each group, 3 cats developed a prolonged PT. No difference between the GID group and non-GID group could be detected ($P = 0.22$; Table 2). Table 3 summarizes the active ingredient, time from first possible ingestion to measurement of PT, and calculated modified international normalized ration for the 6 cats that developed a prolonged PT. None of these cats had clinical signs requiring treatment other than vitamin K administration.

Although no follow-up other than the review of the medical records was conducted, there were no recorded clinical signs of coagulopathy in the medical records between initial presentation and PT results in the ACRG.

Table 3: Description of gastrointestinal decontamination and active ingredient for cats that went on to develop a prolonged PT

Cat	Emetic given	Activated charcoal given	Ingestion to PT time	PT INR	Rodenticide active ingredient
1	No	No	Unknown	1.55	Brodifacoum
2	No	No	Unknown	1.28	Brodifacoum
3	Yes	Yes	Approximately 60 hours	1.49	Brodifacoum
4	Yes	Yes	Approximately 45 hours	1.29	Bromadiolone
5	Yes	No	Unknown	1.26	Bromadiolone
6	No	No	Approximately 24 hours	**	Bromadiolone

**The PT range interval was not indicated in the medical records but was stated to be mildly prolonged. INR, modified international normalized ratio (patient's PT/mean reference value); PT, prothrombin time.

Discussion

Anticoagulant rodenticide ingestion is a common intoxication in dogs[1,2,4–7] and although it is believed to occur significantly less frequently in cats,[6,8] no study has addressed the prevalence of rodenticide exposure in cats. In this study, approximately 0.39% of feline cases presented to an emergency service had a complaint associated with rodenticide. In comparison, a study in an urban emergency service examining canine cases reported 105 of 7,788 dog cases per year (1.35%) presented with complaints related to rodenticide.[16] Therefore, among owners seeking emergency veterinary services in an urban environment, dogs appear to be presented 3.5 times more frequently than cats for possible rodenticide ingestion.

No breed was overrepresented in the REG. Similar to the Kohn study,[8] cats in the REG were more likely to be young cats. Intact animals were overrepresented, which is likely biased by the age of the cats since 31% (44/142) of cats were <6 months old.

Rodenticide exposure group cases were more likely to be presented in the fall season, which is consistent with other reports of increased rodenticide use as rodents move indoors when the precipitation increases and temperature decreases.[6] This information is not always reported and is likely dependent on the weather and climate.[6]

Cats in this study were more likely to be indoor than outdoor dwellers. This finding is in direct contrast to Kohn's study[8]; however, these studies differ because the current study examines suspected exposure to ACRs rather than presentation for hemorrhage. Indoor dwelling cats are more likely to be observed ingesting or playing with rodenticide; whereas ingestion may not be observed in cats that dwell outdoors such that cats would not be presented until clinical signs occurred. Additionally, in the United States and in contrast to many European countries, indoor cats are very common.

Approximately one-fifth of the owners reported having recently acquired the cat or had recently moved the cat to a new environment. Therefore, when a client acquires a new cat or takes the cat to a new environment, they should be reminded to check for rodenticides.

The most common category of rodenticides reported in the REG was ACR. In the United States, frequency of ACR exposure and the prevelance of the specific mechanism of rodenticide may change in upcoming years as under the Federal Insecticide, Fungicide, and Rodenticide Act. Specific products like the common second generation ACRs cannot be sold to the general populous. Exposure to other ACRs will likely decrease with the use of bait stations.[4]

The second goal of this study was to evaluate the medical management of cats that were presented for ACR exposure. The reported LD_{50} for warfarin in cats and dogs is similar, and is 5–50 mg/kg and 5–58 mg/kg, respectively. Cats, however, are significantly more resistant (greater than 10–25 fold) than dogs to some second generation ACRs such as difenacoum, difethialone, and bromadiolone.[6,21] Despite a possible increased resistance to some ACR active ingredients, the current study reinforces previous case reports that cats can ingest sufficient a quantity of ACR to experience toxicosis.[8] The current feline population included a cat that died after having been witnessed eating an ACR approximately 2 weeks before presentation and had been reportedly bleeding from the hind end for days before presentation, and another cat that presented anemic and coagulopathic and tested positive for bromadiolone. There was no indication in the medical record that either patient had received GID after witnessed ingestion or antidote.

Recommended GID methods for toxin exposure include the use of emetics, adsorbents, and possibly a cathartic to limit the absorption of the toxin within a few hours of ingestion.[6] After ACR exposure, decontamination can be followed by approximately 4 weeks of empirical vitamin K1 therapy with the duration dependent on type of ACR, chronicity of exposure, and individual factors that may alter toxin clearance.[6,7] The treatment should be followed by a PT measurement

2–3 days following discontinuation of treatment to ensure no coagulopathy develops.[16] However, owner compliance for oral medications can be difficult to achieve, particularly in cats, which can make this treatment plan unsuitable; therefore, checking a PT 36–48 hours following exposure may be preferable.

Routine GID has recently been called into question in the human medical field and multiple toxicological societies have concluded that no method of GID should be performed routinely on the poisoned human patient.[11,22,23] Although the veterinary field still depends heavily on GID as a mainstay treatment after ingestion of toxic substances, the benefit and risks associated with GID for each patient and each toxin need to be assessed before GID is undertaken.[12]

When determining the benefit of GID, in contrast to other types of rodenticides, ACR intoxication is treatable, particularly if the ingestion is known before toxicosis develops.[6,8] When considering GID, information provided by the client such as the number of exposures, time since exposure, and dose ingested should be considered. In this study, only 20 of 142 cats (14%) were actually witnessed ingesting rodenticide. The most common history involved finding scattered rodenticide pellets. It is uncommon for a client to observe a cat eating rodenticide and even less common for a client to be able to estimate the dose consumed; therefore, actual exposure and likelihood of toxic dose ingestion are usually unknown. Experimentally, it has also been documented that cats are more resistant to specific ACRs than other domestic animals.[6,21] However, this study also shows that cats are capable of ingesting toxic doses. In the 11 years of this study, 2 cats were identified with rodenticide intoxication presenting with anemia and coagulopathy, while in Kohn's study 7 cats were presented over a 6-year period.[8]

The outcome benefit of GID in cats after potential ACR ingestion is also questionable based on this study's inability to demonstrate a significant difference in the incidence of prolonged PT between the GID and non-GID ACR groups. A 4–6-hour period after ingestion is commonly used in veterinary medicines as the window for GID,[6,16] especially when the exact time of ingestion is not known.[24] In this study, only 33% of the REG cases were presented within 6 hours of first possible exposure. In the remaining 66% of cases, the possible exposure may or may not have occurred as many as 6 hours prior to presentation, which may render GID less effective for the majority of cats that present for rodenticide exposure. Alternately, our findings may be explained by the fact that cats rarely ingest toxic doses of ACR based on less dietary indiscretion and/or higher LD_{50} as discussed above.

In people, the value of administration of activated charcoal >1–2 hours after ingestion is also debated, especially in low risk toxicoses.[22] Human studies have shown a significant reduction in efficacy of activated charcoal with increasing time from ingestion and clinical studies have found no difference in outcome with administration of activated charcoal in many situations, including long acting ACR.[15] In healthy cats, gastric emptying time is generally <1 hour (with variations depending on food type, meal size, kibble shape, and water intake) and total gastrointestinal transit time is approximately 24 hours,[25,26] which suggests that cats have an even shorter window for GID following toxin ingestion.

If GID is warranted, cats pose several challenges. Using historically conventional emetics, emesis induction has been inconsistent clinically in cats and there are limited published data on the efficacy of emetics for decontamination. In this study, xylazine was the most commonly used emetic, but was only effective in 41% of cases in which it was used. Due to the receptor components of the chemoreceptor trigger zone, apomorphine rarely induces vomiting in cats and hydrogen peroxide is considered dangerous due to reports of hemorrhagic gastritis.[6] The emetic response to xylazine in the current study is similar to what was found in other recent studies looking specifically at emesis induction in cats.[27,28] Dexmedetomidine administration of 7–10 μg/kg IM has recently been reported to be more efficacious at producing emesis than previously used medications.[27,28]

The risk of GID also needs to be evaluated in each case. Emetics may have sedative effects, side effects of hemorrhagic gastritis, and risks of aspiration of emesis or activated charcoal. Although aspiration may be rarer in cats than in dogs, aspiration (especially of activated charcoal) can cause extensive pulmonary damage.[22] Activated charcoal administration has been associated with hypernatremia, lethargy, and vomiting in dogs[29] but has not been thoroughly examined in cats. More aggressive GID, such as gastric lavage (which is rarely performed in cats) under anesthesia or activated charcoal administration by orogastric tube, has its own set of risks such as esophageal rupture. Although complications of GID may be serious, they are typically infrequent, and no complications were noted in the medical records in this study.

Given the risks and benefits of GID in cats, this technique may not be warranted for potential exposure to ACR. Because the population is at low risk of intoxication, the clinician must carefully evaluate multiple factors such as the active ingredient, time since ingestion, preexisting diseases, estimated dosage, and baseline PT before deciding how to proceed with the management of an individual patient. Depending on the dose ingested and timing of ingestion in the patient with ACR exposure, or if non-anticoagulant rodenticide with a low margin of safety was ingested, the clinician may need to perform GID aggressively. More importantly, regardless

of GID efforts, a PT should be evaluted approximately 48 hours following ingestion of an ACR.

The current study is limited by the retrospective nature of the study and its limited sample size. Specifically, due to small sample size, the study is likely underpowered to detect the effect of GID, and the individual methods (ie, emesis, activated charcoal administration) of GID could not be evaluated separately. As a retrospective study, the dose of rodenticide ingested and the mechanism of action could not be verified if it was recorded at all. An additional limitation of this analysis is bias introduced by the inclusion criteria, since some included cats probably did not ingest any rodenticide. Even in those that ingested some rodenticide, it is highly unlikely that all of them ingested toxic doses; these factors meaningfully limit the comparison of GID efficacy between groups and prevents standardization between groups. Due to the limitations of this study, a definitive conclusion on GID in cats exposed to ACRs cannot be made; however, this clinical study serves to demonstrate some of the challenges associated with the medical management of cats with rodenticide exposure. These challenges underscore the importance of appropriately timed PT testing after known or suspected ACR exposure.

Although cats may be exposed to an ACR, the incidence of toxicosis appears to be low. It may then be more effective to monitor for the rare case of ACR toxicosis by measuring PT at 48 hours after ingestion, rather than subject the larger population to risks of attempted GID. Gastrointestinal decontamination should be attempted using dexmedetomidine as the emetic, if the exposure risk is definite and the timing is appropriate, particularly if the rodenticide has no antidote (eg, bromethalin or cholecalciferol). In conclusion, GID for ACR exposure in cats should be considered based on time since ingestion and estimated dose after considering risks, and PT should be performed approximately 48 hours following exposure if possible.

Footnotes

[a] SigmaPlot for Windows Version 11.0, Systat Software Inc, San Jose, CA.
[b] Compounded by Wedgewood Pharmacy, Swedesboro, NJ.
[c] Tranquived, Vedco, Saint Joseph, MO.

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