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Succinylcholine Chloride Immobilization of Black Bears

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INTRODUCTION

Succinylcholine chloride is a potent relaxant of voluntary striated muscle but has little direct effect on smooth muscle. It has no anesthetic or pain-obliterating properties; therefore, immobilized animals remain completely conscious although unable to move. The duration of effect is quite brief because succinylcholine is rapidly destroyed by non-specific cholinesterases in the blood plasma and liver. Immobilization lasts five to 12 minutes in man and horses and somewhat longer in other species, with ruminants generally requiring longest recovery periods (Stowe *et al.* 1958).

Since the discovery of its curariform properties in 1949, succinylcholine has been widely used for immobilization of animals and in human surgical procedures. Several workers have used succinylcholine on bears (Black 1958, Knudsen 1959, Craighead *et al.* 1960, Troyer *et al.* 1961, Hornocker 1962, Mundy 1963, Pearson *et al.* 1968, Jonkel and Cowan 1971 and Mundy and Flook 1973).

Despite its common usage, the disadvantages of this drug and the factors that modify its effects are not well known. Certain of these aspects were investigated in the course of population studies of black bears (*Ursus americanus*) in the Upper Peninsula of Michigan during 1966 through 1968 and in northeastern Minnesota during 1969.

METHODS AND MATERIALS

For preliminary studies, three bears which were to be killed were confined in

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pens and used for comparative study of the effects of injection into different tissues and for evaluation of the dose-effect relationship. One of them, a 194 kg animal, was given nine equal doses (117 mg each) over a period of two weeks by means of projectile syringes equipped with 2.5 cm needles. Each dose was injected into a different area of the body, and the time from injection to collapse was noted. Entry points were marked by shaving the areas around them. After the bear was sacrificed, the entry points were dissected to determine the types of tissue into which each dose had been injected. In this way, the relationship between the type of tissue at the point of injection and time to onset of effects were learned.

To determine how responses varied with dosage, the two other bears were given injections that ranged from ineffective dosages (< 0.10 mg/kg) to lethal dosages (> 2.0 mg/kg). These were administered intramuscularly at intervals of 24 hours or longer. Effects of each dose were recorded; following sacrifice of the bears, the entry points of the syringes were examined for subdermal tissue damage.

In field studies in Michigan, 191 immobilizations of black bears were accomplished by several methods. A pole-mounted syringe (Black *et al.* 1959) was used to inject 112 box-trapped and two treed bears. A syringe gun was used to inject 73 free-ranging bears (in garbage dumps or campgrounds) and four animals caught in leg-hold traps. Approximately twenty additional free-ranging bears escaped into heavy cover after being darted. Intramuscular injection was intended in all cases.

Doses generally were prepared from crystalline succinylcholine chloride (Anectine, Burroughs, Wellcome and Company) by dissolving 100 mg amounts into one ml of distilled water just prior to use, making a concentration of approximately 90.0 mg/ml. In a few cases, commercial solutions such as Sucostrin (E.R. Squibb and Sons Company, 20 mg/cc) or Quelicin (Abbott Laboratories, 25 mg/cc) were used. These solutions lose potency at a rate of about 3 percent per month at room temperature, so they were carried afield in an ice chest. A dosage of 0.75 mg of succinylcholine per kilogram of estimated bodyweight generally was given after experience showed that lower dosages often were insufficient. Immobilization was prolonged with sodium pentobarbital (Erickson 1957).

Data routinely recorded included sex, date, weight, dose, manner of delivery of drug, undesirable effects, and latent period. The terms *latency* or *latent period* were used to denote the time between injection and immobilization. Bears were considered to be immobilized when they were unable to stand. All bears were observed for at least an hour and then hidden in the brush. Recoveries were confirmed by examining release sites a day or two later.

The hearts of three bears (one from Michigan and two from Minnesota) that died during immobilization were examined macroscopically and compared with the hearts from six bears that were shot. The only data reported from the Minnesota study are mortality data.

RESULTS

Penned Animals

The effectiveness of succinylcholine injections in the 194 kg bear mentioned above varied with the vascularity of the tissue at the site of the injection (Table 1). Injections into muscle produced immobilization in approximately two

TABLE 1. SUMMARY OF NINE IMMOBILIZATION ATTEMPTS USING EQUIPONDERANT DOSES OF SUCCINYLCHOLINE INJECTED INTO DIFFERENT AREAS OF A 194 kg BEAR.*

Injection number	Date	Site of Injection	Tissue Type	Response
1	8-18	rump	Entry hole not discernable	Latency 8 minutes
2	8-21	left shoulder	muscle	Latency 2 minutes, duration 38 min.
3	8-23	high on rump on midline	10 cm of fat under skin	No visible effects
4	8-24	high on rump on midline	10 cm of fat under skin	No visible effects
5	8-26	middle of side near last rib	2.4 cm of fat under skin and covering 2 cm of muscle covering 7th rib	Latency 13 minutes, duration 73 min.
6	8-28	high on neck	0.6 cm of skin and 0.9 cm of fat covering muscle	Latency 4 minutes, duration 100 min.
7	8-29	posterior side of upper hind leg	Dart bounced out upon discharging	No visible effects. Blood and drug ran out of entry hole.
8	8-30	side of chest behind shoulder	skin 0.5 cm, fat 1.2 cm, muscle 0.3 cm, fat 3 cm	Latency 8½ minutes, duration 52 min.
9	9-1	upper part of hind leg	5 to 8 cm of fat	No visible effects. Dart bounced out and exact point of entry could not be found.

*All injections were of 117 mg. (0.6 mg/kg) and were given by means of projectile syringes equipped with 2.5 cm needles.

minutes (Injection 2, Table 1), but injections of the same dosage into fat resulted in prolonged latent periods (Injections 5, 6 and 8, Table 1) or no visible effect (Injections 3 and 4, Table 1) depending upon the thickness of the fat. One dose (Injection 7, Table 1) that was injected into vascular tissue was washed out of the entry hole by blood, and no visible effects ensued.

Increasing the dosage in the other two penned bears resulted in only slightly and inconsistently reduced latent periods. The median latent period was two minutes and ranged from one to four minutes in 37 of the 40 successful immobilizations. Six injections failed to produce immobilization because they were injected into fat or were otherwise faulty. Two of these failures resulted when blood and drug ran out of holes that remained in the skin after projectile syringes discharged and fell away. In general, it appeared that latency was affected less by dosage than it was by the vascularity of the tissue into which the drug was injected. Peak immobilization and paralysis occurred within fifteen minutes of collapse at all dosages.

Dissection of punctures from projectile syringes revealed pockets in subcutaneous fat. These pockets, which varied in size according to the amount of solution injected, apparently were created by the explosive entry of drugs expelled from syringes by powder charges.

Wild Bears in the Field

Extensive field studies in Michigan involved 191 immobilizations of 186 wild bears of both sexes representing various age and weight groups. Data from penned bears were not combined with data from wild bears.

General Reactions to Injections

Most free-ranging bears ran for heavy cover after being struck by projectile syringes. They usually collapsed within $2\frac{1}{2}$ minutes but still were able to move their heads and bite for another minute or so. Respiratory muscles were the last to be affected and the first to recover.

Many bears in box traps already were lying down when succinylcholine chloride took effect. In these animals, transient muscle fasciculation, which often appeared as a wave-like rippling under the skin, and dropping of the head were taken as signs of adequate immobilization. Approximately 76 percent (84 of 111) of the trapped bears were immoblized with initial injections.

During peak immobilization, the thoracic component of respiration often was depressed; and respiration appeared to be accomplished mainly by abdominal movements which appeared to be diaphragmatic. In 21 (11 percent) of the 191 immobilizations, respiration was depressed to the point that artificial respiration was required to prevent death. Spontaneous respiration usually resumed within fifteen minutes of collapse; but in one case, artificial respiration was necessary for 55 minutes.

Dosage

Dosage data suitable for analysis were obtained from 177 immobilization attempts. Nine attempts involved cubs and will be considered separately (see below). Data from the remaining 168 bears were divided into the following four response groups:

    I.   Bears not immobilized (n = 27; median dosage 0.55 mg/kg)

II. Bears immobilized and breathing adequately (n = 118; median dosage 0.80 mg/kg)

III. Bears requiring artificial respiration and recovering (n = 14; median dosage 1.0 mg/kg)

IV. Bears that died (n = 9; median dosage 1.2 mg/kg).

Data from males (112) and females (56) were subjected to Duncan's Multiple Range Test which indicated no significant differences in response due to sex.

Application of the same test showed that the dosages of groups I, II and III were significantly different from one another (P < 0.05), indicating that response was at least partially dependent upon dosage. Dosage differences between bears that required artificial respiration and recovered (Group III) and those that died (Group IV) were not significant after the datum from one bear accidentally given a triple dose (2.4 mg/kg) was deleted from Group IV. Groups II, III and IV include data from both trapped and free-ranging bears, but Group I includes data only from trapped bears.

Data for the 168 bears were used to construct dose-response curves (Marsh 1951) in which dosages were plotted against percentages of animals that were immobilized (Curve A, Fig. 1) or that died or experienced prolonged respiratory paralysis (Curve B, Fig. 1). (Death from apnea was prevented by artificial respiration, and 14 of the 23 animals comprising Curve B were revived.) The 20 free-ranging bears that escaped were not considered in construction of the dose-response curves; hence, the percentages indicated in Curve A should be regarded as maxima and actually could be as much as 9 percent lower if all of the 20 failed to become immobilized. Similarly, Curve B would be too low if
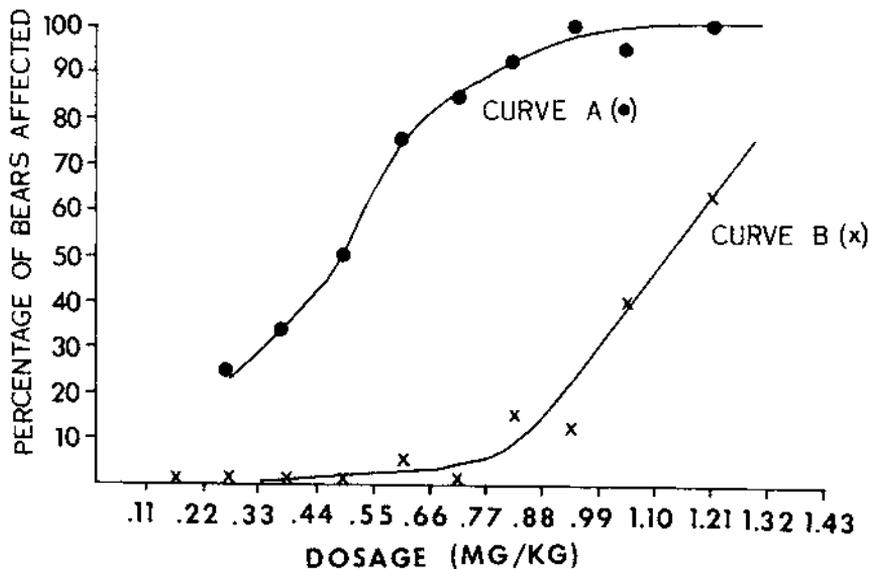

Fig. 1 Dose-response relationship of 168 wild bears to succinyl-choline chloride. Curve A shows percentage of animals immobilized at each dosage. Curve B shows percentage that required artificial respiration or died at each dosage. A concentration of 90 mg/cc was used in most cases.

TABLE 2. RELATIONSHIP BETWEEN SUCCINYLCHOLINE DOSAGE AND NUMBER OF SUCCESSFUL IMMOBILIZATIONS WITH LATENCY LESS THAN 1 AND LESS THAN 2.5 MINUTES.

Mg/kg	Number of Immobilizations	Number with latency less than 2.5 minutes	Number with latency less than 1 minute
0.33	10	2 (20%)	1 (10%)
0.50	15	6 (40%)	2 (12%)
0.66	31	18 (60%)	4 (13%)
0.75	32	29 (90%)	14 (45%)
0.9	30	28 (92%)	12 (40%)
1.0	22	17 (80%)	11 (50%)
1.1	9	8 (88%)	6 (67%)
1.2	3	3 (100%)	2 (67%)
1.3	4	4 (100%)	4 (100%)

TABLE 3. RESULTS OF ADMINISTERING SUCCINYLCHOLINE CHLORIDE TO BLACK BEAR CUBS

Date	Sex	Weight in kg	Dose (mg)	Mg/kg	Response
6/29	male	7	18	2.6	slightly ataxic
6/29	female	7	18	2.6	slightly ataxic
7/28	female	9	18	2.0	required artificial respiration for 6 min.
7/28	female	9	14	1.6	no effect
8/9	male	10	10	1.0	immobilized in 3 minutes
8/9	male	11	12	1.1	no effect
8/12	male	14	4	0.3	no effect
8/14	female	11	18	1.6	immobilized in 15 min.— injected into body cavity.
8/14	male	14	18	1.3	immobilized in 85 seconds

any of the 20 bears died. However, bears that were able to run long enough to move beyond the area in which search efforts were concentrated probably did not die because long latent periods generally were associated with an absence of undesirable effects (see below).

Figure 1 indicates that respiratory paralysis and cardiac arrest were uncommon at dosages less than 0.75 mg/kg but were common among bears that received higher dosages. At a dosage of 1.1 mg/kg, approximately half of the animals required artificial respiration or died.

TABLE 4. DEATHS OF BLACK BEARS FROM DRUG(S)

Bear*	Date	Body weight (kg)	Succinyl-choline Chloride (mg/kg)	Latent period in sec.	Sodium Pento-barbital (mg/kg)	Time until death in min.	Sex
1 FR	7-12	114	1.0	90	none	< 10	male
2 FR	7-27	31	2.3	30	none	< 10	male
3 FR	7-28	48	0.86	30	none	< 15	female
4 FR	7-30	34	0.95	60	none	< 10	male
5 B	8-21	43	1.0	40	none	< 15	male
6 FR	7-17	210	0.86	70	10.0	< 15	male
7 B	6-20	88	1.2	60	37.0	15	male
8 B	7-19	34	1.3	45	32.0	40-60	female
9†FR	8-17	130	0.84	55	23.0	< 20	male

*B = box-trapped; FR = free-ranging.

†Necropsy showed large fresh hemorrhagic areas in both ventricles of the heart.

Latency

Latent periods tended to become shorter as dosage was increased (Table 2). However, latent periods varied considerably at all dosages suggesting that other factors such as supply of blood to injected tissues also were important, as was observed in the penned bears.

Seventy-one bears became immobilized in less than 75 seconds, and 22 (31 percent) of these suffered undesirable affects. By comparison, only one (2 percent) of 58 bears with longer latent periods died or required artificial respiration.

Most (21 of 23) cases of respiratory paralysis or death occurred when dosages greater than 0.75 mg/kg were injected into tissue vascular enough to permit immobilization in less than 75 seconds. Undesirable effects occurred in 40 percent (21 of 54) of the cases in which both high dosage and rapid immobilization occurred.

Age

Nine cubs, which weighed 7 to 14 kg, were less sensitive to succinylcholine than were adults. Several of them were affected little by dosages up to 2.6 mg/kg, which probably would have been lethal for adults. However, one cub required artificial respiration after receiving a high dosage (2.0 mg/kg) (Table 3). The sensitivity of cubs seemed to increase through the summer; and by the time they were a year old, their sensitivity differed little from older bears.

Mortality

Twelve (6 percent) bears died in 191 succinylcholine chloride immobilization

in Michigan. All of the deaths but one occurred within 20 minutes of immobilization. Two bears died as a result of falling from trees while immobilized, one cub died from hemorrhage caused by a projectile syringe, and nine bears (4.5 percent) died from the effects of succinylcholine or pentobarbital (Table 4). For purposes of constructing dose-response curves, the latter nine deaths may have been a factor in two of them (Bears 7 and 8, Table 4).

In the Minnesota study, three of 18 succinylcholine-injected bears died. Terminal symptoms and necropsy data from these suggest that cardiac damage may be responsible for a significant percentage of the deaths from succinylcholine.

Fig. 2   Heart of an old 142 kg black bear that died 5-10 minutes after an intramuscular injection of 0.53 mg/kg of succinylcholine chloride. Note the hemorrhagic area (see arrows) on the left ventricle (below center) and on the right ventricle (upper left).

Fig. 3   Same heart as in Fig. 2 showing a cut made through a hemorrhagic area in the right ventricle. Blood draining into the heart from the hemorrhagic area indicates that the lesion was fresh.

Each of the three respired adequately and spontaneously for the first several minutes of immobilization. Then respiration became labored and gasping even though there was no flaccid paralysis of the thoracic muscles, and death ensued in one to three minutes. Palpation of thoraxes prior to death revealed no detectable heartbeat or only a grossly irregular beat. Terminal signs preceding death included jerking and twitching of individual muscles rather than the fasciculation that commonly accompanied immobilization. All three had been given succinylcholine only; one had received a single injection of only 0.53 mg/kg but the other two had received multiple injections. Two of the bears were necropsied immediately, and extensive, fresh endo- and epicardial hemorrhages were found (Figs. 2 and 3). The single bear from Michigan that was necropsied after immobilization (Bear 9, Table 4) also showed extensive, fresh endocardial hemorrhage. No such hemorrhages were found on the hearts of six bears shot by hunters.

DISCUSSION

Factors that influence the effectiveness of succinylcholine chloride were discussed by Rogers (1970). One such factor is the concentration of succinylcholine chloride solution.

Vanderveen *et al.* (1963) found in a controlled study of man that intramuscular doses of 100 mg/cc concentrations were less effective than equiponderant doses of 20 mg/cc concentrations. The differences may be due to an osmotic impediment in the absorption of hypertonic concentrations (a concentration of approximately 38 mg/cc is isotonic). Data in Table 5 suggest a correlation between high concentration and high dosage requirement. Possibly the relatively high concentration (90 mg/cc) used in this study was responsible in part for the relatively large dosages that were necessary to achieve immobilization. However, Harthoorn (1965) did not consider high concentrations to be less effective than equiponderant ones of lower concentration.

The effects of succinylcholine also may be influenced by body temperature. In summer, body temperatures of bears immobilized with succinylcholine range from 36.8 to 41.8°C, depending largely upon amount of exertion prior to immobilization (personal observations, Rogers). Knudsen (1959) reported that bears overheated in traps sometimes required additional doses to achieve immobilization. Similarly, Bigland *et al.* (1958) and Zaimis *et al.* (1958) showed that when body temperature was lowered experimentally, susceptibility to succinylcholine in cats increased. Susceptibility decreased when subjects were rewarmed. These effects of body temperature may be related to increased enzymatic destruction of the drug at higher temperatures (Foldes 1959).

The body temperature of bears during winter denning are from 3 to 7 °C lower than the usual summer body temperature of 38 °C (Hock 1951, Irving and Krog 1954, Hock 1957, Rausch 1961, Erickson and Youatt 1961), so prolonged immobilization would be expected in denned bears. However, to achieve immobilization in three denned bears, Jonkel and Cowan (1971) found that reduced dosages were insufficient.

Craighead *et al.* (1960) may have been the first to consider cardiac disturbance a possible cause of death for bears treated with succinylcholine. It is becoming increasingly clear that succinylcholine is a high risk drug when used under field conditions. There is no antagonist to succinylcholine, and some bears experience cardiac arrest (or apnea, Pearson *et al.* 1968) at dosages lower than those required to achieve immobilization consistently in bears. Cardiac dis-

TABLE 5. SUMMARY OF SUCCINYLCHOLINE CHLORIDE DOSAGE DATA IN BEARS

Species	Number of Bears	Concentration in mg/cc	Recommended dosage in mg/kg	Range of dosages used successfully	Source
U. americanus	—	20-50	.60	—	Black (1958)
U. americanus	35	20-50	.55	.22 to .88	Black et al. (1959)
U. americanus	30	20	.44	.31 to .53	Knudsen (1958)
U. arctos horribilis	30	'high potency solutions'	.55 to .73*	.48 to 1.1**	Craighead et al. (1960)
U. arctos middendorffi	65	20	.44	.26 to .70	Troyer (1961)
U. americanus	—	not reported	.44	.20 to .79	Jonkel (1960)
U. arctos horribilis	20	variable	.40	.26 to .55	Pearson et al. (1968)
U. americanus	20	variable	.42	.20 to .55	Pearson et al. (1968)
U. americanus	191	90	.70	.31 to 1.6**	This paper

*based on lean weight

**considering only single initial doses

turbances can occur even though a subject is not fully paralyzed and is re-spiring spontaneously (see also Bullough 1959). Death from respiratory para-lysis can be prevented by artificial respiration, but there is presently no field procedure for preventing death from cardiac arrest. Barbiturates administered before succinylcholine apparently have reduced the incidence of cardiac dis-turbances in man (Schoenstadt and Whitcher 1963) and horses (Hansson 1956, 1957, Larsen 1958, Hofmeyr 1960, Tavernor 1960), but such premedication is not feasible with wild bears.

The use of multiple doses to achieve or prolong immobilization appears to be particularly dangerous. Choline, produced by the hydrolysis of succinylcholine to succinylmonocholine and choline, can sensitize subjects to subsequent doses of succinylcholine; following sensitization, cardiac disturbances can be pro-duced by the entire succinylcholine molecule (Williams *et al.* 1961, Schoenstadt and Whitcher 1963). Pearson *et al.* (1968) abandoned the practice of prolonging immobilization with additional doses after two of five bears died. Two of the three bears from Minnesota that apparently died from myocardial injury were administered multiple injections to achieve immobilization. Repeated doses of succinylcholine also increase the likelihood of cardiac arrest in man (Bullough 1959, Craythorne *et al.* 1960, Lupprian and Churchill-Davidson 1960, Williams *et al.* 1961, Williams and Crain 1962, Schoenstadt and Whitcher 1963).

Larsen *et al.* (1959) stated that the severity of cardiac damage appears to be related to dosage. They observed endocardial hemorrhages in 10 of 15 horses killed by rifle fire following recovery from succinylcholine immobilization. Damage was found primarily in the right ventricles and was more common in horses treated with higher dosages. No cardiac damage was found in the hearts of seven additional horses which had not been given the muscle relaxant and were killed by shooting. Electrocardiograms recorded for six of the immobi-lized horses were compatible with a diagnosis of sudden myocardial injury. It is significant that all of the fifteen immobilized horses recovered from the paralytic effects of the drug and were able to stand even though 10 of them had suffered myocardial injury. It seems probable that some bears that recovered and were released in this study had similar injuries.

Cardiac arrests following treatment with succinylcholine have also caused mortalities among horses and zebras with deaths occurring from within ten seconds of intravenous injection to 30 minutes after recovery (Hansson 1957, Tavernor 1959, Larsen *et al.* 1959, Lock and Harthoorn 1959, Hofmeyr 1960). In one case, electrocardiograms showed that ventricular fibrillation began 45 seconds after the intravenous injection of succinylcholine chloride and that death occurred 135 seconds later (Hofmeyr 1960). Postmortem findings, where reported, indicated gross vascular damage to the larger arterial trunks or to the myocardium as we observed in black bears.

Numerous studies have demonstrated a release of potassium from skeletal muscle into plasma following succinylcholine injection (Klupp and Kraupp 1954, Paton 1956, Mazze *et al.* 1969, Weintraub et al. 1969, Evers *et al.* 1969); subse-quently, much of the potassium is excreted through the kidney (Stevenson 1960). The induced temporary hyperkalemia long has been suspected as a cause of cardiac irregularities and arrest (Stevenson and Hall 1959, Allan *et al.* 1961, Galindo and Davis 1962, Dowdy and Fabian 1963, Belin and Karleen 1966, Surawicz 1967, Weintraub *et al.* 1969). Mazze *et al.* (1969) showed that hyper-kalemia in man is more pronounced and cardiac effects are more frequent in traumatized patients, particularly in burned patients. These workers monitored cardiac function and plasma potassium levels in 14 traumatized patients be-fore, during and after succinylcholine administration. In each case, succinyl-

chloline injection was followed by a significant rise in plasma potassium and a concomitant appearance of cardiac irregularities. Of the five patients showing the greatest rise in plasma potassium, three experienced ventricular fibrillation. Despite this evidence, however, hyperkalemia does not explain all cardiac disturbances following succinylcholine (Williams *et al.* 1961, Evers *et al.* 1969, Mazze *et al.* 1969).

Succinylcholine can stimulate the vagal nerve and produce bradycardia and arrythmia (Craythorne et al. 1960, Williams et al. 1961, Adams and Hall 1962, McCaughey 1962). There is also evidence of catecholamine release from adrenergic tissue capable of producing tachycardia and hypertension (Stevenson and Hall 1959, Williams *et al.* 1961, Galindo and Davis 1962, Katz and Katz 1966, Tavernor and Lees 1970). Furthermore, catecholamine release has been implicated in the pathogenesis of hemorrhagic areas in the heart (Reichenbach and Benditt 1970).

Succinylcholine has other undesirable effects, although none is as serious as its effects upon the respiratory muscles or heart. Muscle fasciculation occurs at the onset of immobilization and (in man) is transiently painful. Muscle pain also is common in man one or two days after recovery (Bennike and Nielson 1964). Several cases of myoglobinuria suggest damage to muscle cells (Airaksinen and Tammisto 1965) and potential toxicity to kidneys.

Drug-related mortality occurred in nine (4.5 percent) of 191 immobilizations in this study. The number of bears that survived but suffered myocardial injury is unknown. Although the high mortality rate may have been due in part to the relatively large dosages, there is a clear need for a more humane, reliable and safer immobilizing agent for bears. There are several possibilities, including the combination of phencyclidine (Sernylan) and promazine (Sparine) that Seal *et al.* (1970) reported to be effective, safe and possibly anesthetic. In our own experience with this combination, no fatalities have occurred in over 500 successful immobilizations of black bears in Minnesota.

SUMMARY

Succinylcholine chloride, a muscle relaxant commonly used in projectile syringes, was employed in 191 immobilizations of black bears in the Upper Peninsular of Michigan during the summers of 1966 through 1968. Dosages of 0.66 to 0.75 mg/kg were required to achieve immobilization consistently. These dosages, which are higher than those used for bears by other workers, may have been necessary because hypertonic solutions (90 mg/cc) were used; hypotonic solutions (less than 38 mg/cc) generally were used by others. There were twenty-three cases of respiratory paralysis or cardiac arrest. Twenty-one (91 percent) of these cases occurred when dosages greater than 0.75 mg/kg were injected into tissue vascular enough to facilitate immobilization in less than 75 seconds. Artificial respiration prevented suffocation, but field procedures were not available to prevent death from cardiac arrest. The latter was produced by dosages as low as 0.53 mg/kg. The administration of multiple injections to achieve or prolong immobilization seems particularly likely to cause myocardial injury and cardiac arrest. The effects of succinylcholine on the heart are discussed. The effectiveness of succinylcholine apparently is influenced by its concentration, the vascularity of the tissue into which it is injected, body temperature, and whether the bear is older or younger than about one year.

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