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A FIELD METHOD FOR IMMOBILIZING WEDDELL SEALS

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Studying the behavior of Weddell seals (*Leptonychotes weddellii*) at sea often requires fitting the seals with electronic monitoring devices. To ensure reliable attachment of the instruments, anesthetization of the animals is useful (see Kooyman et al. 1980, Bester 1988, Bengtson and Stewart 1992). However, there have been few studies on various anesthetics and dosages for Weddell seals.

Two principal methods used to induce narcosis are inhalation and injection. Inhaled anesthetics are easy to control, but require considerable equipment. This contradicts our intention of developing a practical field method. Injected anesthetics are more difficult to control, a disadvantage that is reflected in the relatively high mortality rates in Weddell seals (13–44%) reported by Gales and Burton (1988). The main problem in the field is estimating an adequate dose for every animal without knowing its condition, especially body mass. Therefore, the drug should have a wide safety margin between intended anesthetic depth and asphyxia. Although mortality rates of 20–28%

have been observed using ketamine hydrochloride (HCl) in Weddell seals (Hammond and Elsner 1977, Gales and Burton 1988), this drug has been recommended for seals because of its safety margin (Geraci 1973; Geraci et al. 1981; Parry et al. 1981; Gales and Burton 1987, 1988). Nevertheless, ketamine HCl does not induce complete narcosis, especially sufficient muscle relaxation, so it has to be used with other drugs such as diazepam (Gales and Burton 1987, 1988; Boyd et al. 1990) or xylazine HCl (Trillmich and Wiesner 1979, Gales and Burton 1987, Woods et al. 1989, Boyd et al. 1990). In addition, postnarcosis monitoring to prevent drowning can take hours. Rapid recovery is desirable when working on the ice. We tested the effectiveness of a combination of 4 chemicals to immobilize Weddell seals in the field.

METHODS

Our studies on Weddell seals were conducted at Drescher Inlet (72°52'S, 19°25'W), Riiser Larsen Ice Shelf, Antarctica, 1 January–22 February 1990. We used ketamine HCl, which can be administered using intravenous and intramuscular injection. It is a fast-acting analgetic with hypnotic properties and a large therapeutic range (Schmid 1980). It cannot be described as an anesthetic because it does not cause muscle relaxation. Rather, it induces a cataleptoid state. The main clinical effect is a fast onset of strong analgesia of the body periphery. The analgesia precedes and outlasts a moderately deep hypnosis. The swallowing reflex and breathing are not changed and the cardio-respiratory side effects of ketamine HCl are minor compared with those of other anesthetics (Booth 1988*a*). Xylazine HCl was used in combination with ketamine HCl to promote muscle relaxation. Xylazine HCl has various effects on the cardiovascular system including an initial increase in arterial blood pressure that is followed by a longer lasting decrease in blood pressure. Moreover, a decreased heart rate can be observed (Booth 1988*b*). Diazepam is administered in mixed anesthesia as a sedative and supplement to xylazine HCl. Convulsions that occur in dogs when xylazine HCl and ketamine HCl are administered alone can be avoided with diazepam (Schmidt et al. 1985). Diazepam has only minor effects on the circulatory system, does not lead to analgesia, and has no influence on body temperature (Booth 1988*c*). The galenic formulation is a solution of benzyl alcohol that cannot be administered together with other preparations in a mixed injection. Atropine sulfate was used for narcosis premedication (Baggot 1988) to counteract the xylazine HCl-induced bradycardia (Kolata and Rawlings 1982) and the ketamine HCl-induced salivation and bronchial secretion. Secretion in salivary glands and bronchi as well as contraction in bronchioles are reduced. Thus, the possibility of death caused by asphyxia is reduced. A further effect of atropine is dilation of the pupil, which may lead to retinal damage from exposure to the sun. This should be avoided by covering the seal's eyes during narcosis.

We administered doses (Table 1) well below those previously reported for Weddell seals (Erickson et al. 1974, Hammond and Elsner 1977, Gales and Burton 1988). To calculate the appropriate dose, we first made a conservative estimate of the animal's body mass. In cases where the dose was inadequate a second dose was given to complete immobilization. In order to be within the therapeutic range, the second dose amounted to one-third of the initial application of ketamine HCl, xylazine HCl, and diazepam. Ten ml of ketamine HCl (100 mg/ml) were introduced into a bottle containing 500 mg of dry substance xylazine HCl, which was dissolved in the ketamine HCl. Diazepam was administered with a separate syringe. We gave all injections intramuscularly into the gluteal region by hand using a 7-cm canula, and we used caution to avoid injury to the periosteum, particularly in lean animals. The delivery site was controlled by aspirating the syringe to ensure that the injection was not given intravenously. We prevented freezing of the injection solutions by carrying them in body contact while working on the ice.

Because of individual differences in the temperament of seals, we had to use different methods of application. When seals were calm, injections were given without restraint; agitated seals had to be caught in a bag as described by Stirling (1966).

The induction time of immobilization was defined as the period between first injection and failure of the animal to respond to stimuli. We subdivided this phase into 3 stages. The period of time between application of drugs and a markedly reduced awareness was designated stage 1. Stage 1 ended when our approach was first tolerated. In stage 2, the animal raised its head when we touched it. This stage ended when the head was no longer raised. Slight head movements in response to "touch-stimuli" were characteristic of stage 3. This stage ended with the failure of the animal to react to any stimuli. We used induction time duration as an indicator of a sufficient dosage. If the end of stage 3 was not reached within 25 minutes, we gave a second dose. The animals were then weighed using a tetrapod that supported a scale that could be lifted by a hoist. During narcosis, respiratory and heart rates, rectal temperature, and corneal reflex were monitored.

Yohimbine HCl is used as an antidote in the ketamine HCl/xylazine HCl narcosis (Booth 1988*d*). A dosage of 0.2–0.5 mg/kg has been reported for a variety of zoo animals (Göltenboth 1988) but not for seals. We used 0.5 mg/kg as an approximation for our experiments. We prepared the yohimbine HCl solution by dissolving 5 g of pure substance in 500 ml of sterile water at 80 C.

Table 1. Dosages of chemicals used to immobilize Weddell seals using intramuscular injection into the gluteal region, Antarctica, 1990.

		Atropine (mg/kg)	Ketamine (mg/kg)	Xylazine (mg/kg)	Diazepam (mg/kg)
Induction dose	$\bar{x}$	0.02	2.80	0.92	0.04
	SE	0.01	0.19	0.04	0.01
	n	2	18	18	18
Maintenance dose	$\bar{x}$		0.78	0.22	0.04
	SE		0.05	0.03	0.02
	n		7	2	2
Total dose	$\bar{x}$	0.02	3.11	0.94	0.04
	SE	0.01	0.19	0.04	0.01

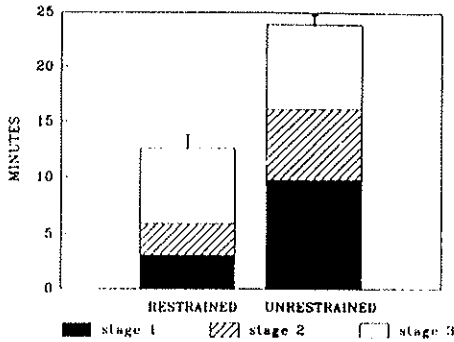

Fig. 1. Average duration of the 3 induction stages of narcosis in restrained Weddell seals caught in a bag (n = 9) and in seals that were drugged without restraint (n = 9), Antarctica, 1990. Standard error bars (T) are shown on total time. See text for explanation of induction stages.

RESULTS

We immobilized 14 Weddell seals. Except for 2 pups, all seals were adults. Within several days, 2 seals were anesthetized a second time and 1 seal was recaptured twice. Nine seals were drugged without restraint, and 9 seals (including 4 recaptures) were caught in a bag, as described by Stirling (1966). After injection, the bag was removed as soon as the animal did not respond to "touch-stimuli" as described above. Induction time ($\bar{x}$ = 14.3 min, SE = 2.66) and recovery time ($\bar{x}$ = 7.3 min, SE = 2.60) for seals repeatedly immobilized did not differ from those immobilized once (induction time $\bar{x}$ = 8.4 min, SE = 2.27, P = 0.16; recovery time $\bar{x}$ = 8.0 min, SE = 1.53, P = 0.8836).

Two seals premedicated with atropine died during narcosis. Death was preceded by a 10-minute period of decreasing respiratory rate and increasing heart rate leading to respiratory arrest, and a period (9 min) of decreasing heart rate ending in cardiac arrest. Subsequently, we refrained from administering atropine premedication. Without this drug, no salivation or bradycardia was noted. Respiratory arrest of 3–9 minutes occurred in 7 cases, 3 of which occurred during induction. Because respiration commenced spontaneously, this was not assessed as a complication and was comparable to sleep-associated apnea described by Castellini et al. (1992). The expected correlation between decreased respiratory rate and increased heart rate was not observed in all cases. During narcosis, the corneal reflex was maintained in all nonlethal cases.

Mean induction time for seals caught and drugged in a bag was 12.3 minutes (SE = 4.00) compared to 23.5 minutes (SE = 3.52) for seals drugged without restraint (Fig. 1). When we used a bag, stages 1 and 2 together were reduced by nearly 10 minutes.

In 3 seals, a second dose was necessary for complete immobilization. For maintaining narcosis, supplemental injections of ketamine HCl were necessary in 7 seals, xylazine HCl in 2 seals, and diazepam in 2 seals (Table 1). We injected a maintenance dose of ketamine HCl when anesthetization had to be prolonged in order to attach electronic devices. In 2 trials that lasted 3 hours, additional xylazine HCl and diazepam were given to ensure sufficient muscle relaxation.

With yohimbine HCl dosage of 0.5 mg/kg (SE = 0.02), the animals recovered within 8 minutes (SE = 0.98) after injection. The mean duration of narcosis was 123 minutes (SE =

13.84). Further observation of seals ensured that they did not die because of late recurrence of narcosis.

DISCUSSION

Previous chemical immobilization of Weddell seals with ketamine HCl (Hammond and Elsner 1977, Gales and Burton 1988) was unsatisfactory because mortality rates exceeded 20%. One reason for high mortality may be the dive reflex triggered during narcosis (Gales and Burton 1988). The diving reflex may lead to a centralization of blood circulation to the heart, lung, and brain. Consequently, more anesthetic is transported to central organs, particularly the brain, and may lead to a lethal dose. Death of 2 seals in our study did not show such a pathogenesis. Bradycardia occurring with the dive reflex (Kooyman 1981) was not observed. In both cases a respiratory arrest preceded death. Respiratory depression has been observed in dogs anesthetized with a ketamine HCl–xylazine HCl combination (Kolata and Rawlings 1982) and was attributed to ketamine HCl. During respiratory arrest and decreasing heart rate, intramuscular injection of an antagonist probably would be too late because of the time required for its redistribution from the muscle. It is difficult to puncture the intravertebral supraspinous vein if access had not been previously gained. As an option, an antagonist or a strong respiratory analeptic like doxapram could be injected into the sublingual vein.

A new advance to come from our study was the use of the antagonist yohimbine HCl to reverse the immobilization. Recovery times between 45 minutes and 3.5 hours were described for seals drugged with ketamine HCl, combinations of ketamine HCl–diazepam or ketamine HCl–xylazine HCl (Geraci 1973, Geraci et al. 1981, Parry et al. 1981, Gales and Burton 1987). In our study, yohimbine HCl shortened the recovery phase in seals to an average of 8 minutes, thus reducing stress on the animals during narcosis and saving time during field work. The success of our procedure may be attributed to the distribution of the components of narcosis (analgesia, loss of consciousness, and skeletal muscle relaxation) to several drugs. This permits lower dosages of the individual drugs, which reduce side effects of the anesthetic episode. Moreover, by using an antagonist, potential complications arising during the seal's recovery phase can be reduced.

SUMMARY

We anesthetized 14 Weddell seals by using a combination of ketamine hydrochloride (HCl), xylazine HCl, and diazepam. Narcoses were terminated with yohimbine HCl. The mean total dosage/kg body mass was ketamine 3.11 mg (SE = 0.19), xylazine 0.94 mg (SE = 0.04), diazepam 0.04 mg (SE = 0.01), and yohimbine 0.5 mg (SE = 0.02). This drug combination enabled us to shorten the recovery time considerably and did not cause undesirable side effects, especially during recovery.

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