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CHEMICAL RESTRAINT OF WEDDELL SEALS (*LEPTONYCHOTES WEDDELLII*) WITH A COMBINATION OF TILETAMINE AND ZOLAZEPAM

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ABSTRACT: A 1:1 combination by weight of tiletamine hydrochoride and zolazepam hydrochloride was administered to 30 adult Weddell seals (*Leptonychotes weddellii*) in doses varying from 100 to 300 mg. Full immobilization was achieved in 16 seals, moderate sedation in seven and light sedation in seven. Three animals died; two were fully immobilized and one was moderately sedated prior to death. The drug combination was considered satisfactory, although its usefulness was limited by the lack of chemical antagonists when complications were encountered in immobilized animals.

Key words: Weddell seal, *Leptonychotes weddellii*, tiletamine hydrochloride, zolazepam hydrochloride, immobilization.

INTRODUCTION

The use of chemical restraint in Weddell seals (*Leptonychotes weddellii*) has been reported infrequently (see review by Gales, 1989). Most authors cited by Gales (1989) reported complications or fatalities associated with the use of a wide range of agents, and fatality rates in the range of 13–44% have been recorded (Gales and Burton, 1988). Hammond and Elsner (1977) used a combination of tiletamine hydrochloride and zolazepam hydrochloride in a ratio of 2:1 by weight in Weddell seals and reported no fatalities. They suggested, after trying a 1:1 mixture in harbor seals (*Phoca vitulina*), that this was safer and more useful.

The combination of tiletamine and zolazepam in a 1:1 ratio by weight has been used successfully to immobilize a wide range of domestic and wild mammalian species (Gray et al., 1974; Boever et al., 1977; Haigh et al., 1985; Schobert, 1987; Stirling et al., 1989). In general, these studies were confined to species other than pinnipeds. Gray et al. (1974) reported a drug trial on northern elephant seals (*Mirounga angustirostris*) (18 episodes) with satisfactory results but reported two mortalities in five trials with California sea lions (*Zalophus californianus*). The use of tiletamine and zolazepam mixtures in pinnipeds has been reported by Stirling and Sjare (1988) on Atlantic walruses (*Odobenus rosmarus*), Loughlin and Spraker (1989) on northern sea lions (*Eumetopias jubatus*) and Baker et al. (1990) on both gray seals (*Halichoerus grypus*) and southern elephant seals (*Mirounga leonina*). Hammond and Elsner (1977) also refer to the use of a 1:1 combination of tiletamine and zolazepam in southern elephant seals at a dose rate of 0.5 mg/kg. The effect was described only as immobilization, and the number of animals treated was not given. Results from these studies indicate that safe and reliable immobilization can be expected in phocid seals and walruses but difficulties are more common in otarids. These studies, especially when viewed in association with the difficulties experienced previously with other chemical agents used on Weddell seals, led us to the conclusion that a mixture of tiletamine and zolazepam might be the most appropriate agent for field immobilization of *L. weddellii*. The aim of this study was to assess the use of tiletamine hydrochloride and zolazepam hydrochloride, in a 1:1 ratio by weight, in order to find a safe method of chemical restraint for Weddell seals for scientific procedures in the field.

MATERIALS AND METHODS

The study was conducted in the Vestfold Hills (68°30'S, 78°00'E) during December 1989. The study group totalled 30 animals, all but one of which were of breeding age; 19 were males and 11 were females.

The combination of tiletamine hydrochloride and zolazepam hydrochloride in a 1:1 ratio by weight (Zoletil® Virbac, Sydney, Australia) was reconstituted to a 100 mg/ml solution. The dose chosen for each animal was based on visual assessment of the individual's size and girth. Precise weights and consequent dose rates were estimated retrospectively. Three ml of atropine sulphate (Atrosine Mitis® 0.65 mg/ml, Parnell, Sydney, Australia) was administered concurrently to each animal. The Zoletil® solution was drawn into a 20 ml syringe, the atropine sulphate was then added and the final volume made up to 10 ml with Water for Injection B.P.

Each animal was restrained by placing a canvas bag over its head (after Stirling, 1966). Time was allowed for the animal to become relaxed and the mixture was injected into the dorsal gluteal or lumbar musculature using a 90 mm, 19 gauge spinal needle attached directly to a hand-held syringe. After injection, the head bag was kept in place except where those females with pups continued to struggle excessively. During induction, respiration was monitored and the bag was repositioned if necessary. The bag was removed when a reasonable level of sedation was achieved, at which time it was possible to introduce a spinal needle (90 mm, 18 gauge) into the midlumbar extradural intravertebral vein to enable intravenous injection of further drugs to effect if necessary. Degree of immobilization was assessed and standard length and axillary girth were measured. On completion of these measurements the animal was rolled onto its side to minimize respiratory difficulties. The degree of immobilization was assessed by monitoring caudal flipper response to pressure and head response to approach and touch. In both cases the response was judged to be in one of three categories: (1) light sedation where the animal was still able to move and react purposefully, but was noticeably slower than normal; (2) moderate sedation where the animal was resting quietly unless substantially aroused, but was still capable of reduced head and flipper movement; and (3) full immobilization with the complete absence of head and flipper movement in spite of repeated stimulation. For these animals, induction time and time to recovery (see Table 1) refer to the intervals from initial injection to full effect (i.e., immobilization) and to recovery of normal locomotory functions respectively.

Heart and respiration rates were monitored regularly (usually every five minutes), and body temperature was measured per rectum using a 30 cm laboratory thermometer (range from 10 to 100 C). Animals were observed continuously, and prevented from entering the water until normal locomotory and defensive actions were restored.

In the event of respiratory arrest, intravenous doxapram hydrochloride, 20 mg/ml (Dopram® A.H. Robins, Sydney, Australia) was administered. A portable oxygen cylinder ('C' size, CIG, Melbourne, Australia), a Hudson Demand Valve (model number 5090, Hudson Oxygen Therapy Sales Company, Temecula, California 92390, USA) and a cuffed endotracheal tube were used for mechanical ventilation (at a rate of 2 to 3 breaths/min) in one animal (number 10) after a period of apnea unresponsive to repeated doses of doxapram.

RESULTS

Total doses of Zoletil® ranged from 100 mg to 300 mg, with dose rates between 0.29 and 1.08 mg/kg (Table 1). These figures include additional doses given in 10 cases, eight of which were given intravenously and two intramuscularly, at times from 12 to 38 min after the initial dose. Estimated masses of seals ranged from 212 kg to 440 kg (Table 1). Of the 30 animals immobilized, 16 were fully immobilized, seven moderately immobilized and seven lightly sedated. One animal (number 25) was insufficiently sedated for standard measurements to be made. The mean dose of Zoletil® for fully immobilized animals (0.78 ± 0.14 mg/kg, $n = 16$) was significantly greater (Scheffe F-test, $P < 0.05$) than for moderately sedated animals (0.58 ± 0.13 mg/kg, $n = 7$). There was no significant difference between the dose rate for moderately sedated animals and lightly sedated animals (0.51 ± 0.23 mg/kg, $n = 7$).

For cases where full immobilization was achieved ($n = 16$), induction time, period of immobilization and time to recovery are recorded (Table 1). Seven animals required additional intravenous doses of Zoletil® ranging from 50 to 100 mg to achieve full immobilization which then occurred within 2 min. Animal number 24 also re-

TABLE 1. Sex and measurements of Weddell seals immobilized, together with dose rates and effect (I, immobilized, with time immobilized in parentheses; M, moderate sedation; L, light sedation).

Animal	Sex	Standard length (cm)	Axillary girth (cm)	Dose (mg)	Estimated wt. (kg)[2]	Dose rate[2] (mg/kg)	Effect (time immob min)	Induction time (I) and time to recovery (R) (minutes)	
								I	R
1	F	216	152	250[1]	231.0	1.08	I (19)[4]	21	71
2	F	228	165	300	287.3	1.04	I (24)[5]	1	100
3	F	227	165	200	286.1	0.70	I (40)	15	95
4	F[6]	230	148	200[1]	233.2	0.86	I (18)[3]	15	70
5	F[6]	231	152	200	247.0	0.81	I (48)	12	80
6	M	255	193	300[1]	439.7	0.68	I (23)[3]	24	100
7	M	216	160	150[1]	256.0	0.59	I (12)[3]	28	120
8	M	223	155	200[1]	248.0	0.81	I (15)[3]	15	100
9	M	237	179	200[1]	351.5	0.57	I (15)[3]	15	60
10	M	247	161	210[1]	296.4	0.71	I (Died)[3]	13	
11	M	222	158	200	256.5	0.78	I (15)	15	75
12	M	236	155	200	262.4	0.76	I (19)	8	90
13	M	244	173	200	338.0	0.59	I (30)[3]	20	73
14	M	234	157	200	267.0	0.75	I (20)	10	60
15	M	226	160	200	267.8	0.75	I (20)	10	70
16	M	240	161	250	288.0	0.87	I (Died)	12	
17	F[6]	227	163	150	279.2	0.54	M		
18	M	227	178	150	332.9	0.45	M		
19	M	230	151	200	242.7	0.82	M		
20	M	240	172	200	328.7	0.61	M		
21	M	226	191	200	381.6	0.52	M		
22	M	250	181	200	379.1	0.53	M		
23	F[6]	253	172	100	346.5	0.29	L		
24	F[6]	237	160	125[1]	280.8	0.45	L		
25	F	not measured		200[1]	not calculated		L		
26	F	234	180	300	351.0	0.85	L		
27	F[6]	215	146	150	212.1	0.71	L		
28	F	256	190	200	427.8	0.47	L (Died)		
29	M	225	172	100	308.1	0.32	L		
30	M	200	157	100	228.2	0.44	L		

[1] These doses include any additional top-ups.

[2] Masses and associated Dose Rates calculated from Castellini and Kooyman (1990) where M = 1.31 × (S.L. × GIRTH × GIRTH)/(2.83 × 10^4). M = Body Mass in kilograms; S.L. = Standard Length in centimeters (as measured in a straight line from tip of nose to tip of tail); GIRTH = Axillary Girth as measured immediately caudal to the pectoral flippers.

[3] Animal number 30 was a sub-adult, all others were adults.

[4] Indicates full immobilization was achieved as a result of an additional intravenous dosage.

[5] Indicates possible inadvertent intravenous administration of initial dose.

[6] Indicates female with pup.

ceived 25 mg of Zoletil® intravenously, 13 min after the initial dose of 100 mg but only light sedation was achieved.

Excessive salivation was not evident in any animal. Change in normal body temperature was recorded in only one animal, where body temperature dropped from 36.5 to 35.5 C until the animal was insulated from a cold wind, by covering it with wind-proof material. This animal recovered normally. Hyperthermia was not noted in any animal, even on calm sunny days with no available shade.

Doxapram hydrochloride (200 to 600 mg) was administered into the extradural venous sinus of six animals when periods of apnea in excess of 2 or 3 min were recorded. Observations of untreated resting animals indicated the normal respiratory pattern to consist of six to eight

breaths per minute with occasional periods of apnea generally lasting no more than 1 min. Two animals (numbers 4 and 5) showed marked respiratory improvement (from nil to 12 breaths/min) within 2 min of administration of 200 mg of doxapram hydrochloride. Both of these animals had been apneic for a period of 5 min prior to drug administration. Animal number 14 required an initial dose of 400 mg followed by a further dose of 200 mg 5 min after the first before respiration gradually increased to 10 breaths/min over the next 15 min. These three animals maintained a normal respiratory pattern throughout their subsequent recovery from immobilization. The remaining three animals (two immobilized and one moderately sedated) subsequently died. Transient respiratory improvement was noted even in one of the fatalities; this animal (number 10) was given 200 mg of doxapram hydrochloride after an apneic period of eight min. Two breaths were then taken almost immediately but further doses failed to maintain the response. The three animals which died differed from those that recovered in that heart rates (normally 70 to 80 beats/min) halved in a very short time period. In two animals, the observed decline of respiratory and then cardiac function appeared to be related to administration of the immobilizing agent. In the third animal, these effects appeared to occur secondarily to a period of respiratory distress.

DISCUSSION

Excluding the animal ($n = 1$) that was induced in 1 min (and therefore possibly accidentally injected intravenously), the mean time to immobilization in this study (15.5 ± 5.5 min) was shorter than the 24.6 ± 17.2 min reported by Gales and Burton (1988) but not significantly so ($t = 1.952$, $P = 0.06$). Gales and Burton (1988) used a mixture of ketamine and diazepam and the techniques for injection varied in these two studies; Gales and Burton (1988) used a remote method of injection after Ryding (1982), compared with our use of hand-held syringes. We chose physical restraint and direct injection, due to the tractability of the species and the greater assurance of intramuscular drug administration.

The duration of full immobilization with Zoletil® (mean 22.7 ± 10.2 min, range 12 to 48 min, $n = 14$) was significantly shorter ($t = 16.81$, $P < 0.001$) than that of the ketamine/diazepam combination used by Gales and Burton (1988) (mean 127.9 ± 20.7 min, range 95 to 160 min, $n = 12$). The animals which died while fully immobilized were excluded from these calculations. In all other fully immobilized animals, recovery was smooth and judged to be complete within 60 to 120 min of initial injection. Of the two drug combinations at the two different doses, Zoletil® gave a shorter duration of full immobilization and may be more appropriate than ketamine/diazepam for use in Weddell seals when a short duration of immobilization is required.

The mortality rate reported here (10%) is similar to that reported elsewhere for Weddell seals (Gales and Burton, 1988), but is higher than that recorded by Hammond and Elsner (1977), who reported no fatalities in 17 Weddell seals immobilized with tiletamine hydrochloride and zolazepam hydrochloride in a 2:1 ratio by weight. Of these animals, four adult females were dosed at 1.0 mg/kg; the remainder (10 pups and three adults) were given 1.5 mg/kg. The most important difference between the present study and that of Hammond and Elsner (1977) may be in the ratio of the two compounds; however, two other differences are important. First, 10 of the animals they treated were pups; second, all 13 of the animals given the higher dose had to be intubated and ventilated.

The anatomical features of the respiratory system of Weddell seals, as described by Hammond and Elsner (1977), and the associated difficulties in terms of artificial ventilation were apparent in three animals (numbers 10, 16 and 28). Attempts

at intubation in animal number 10 were hampered by spongy peripharyngeal tissue and an extremely flaccid soft palate which prevented visualization of the laryngeal glottis. Digital palpation was needed to locate and force apart the arytenoid cartilages (which appeared to be in spasm) before an endotracheal tube (11.0 mm I.D.) could be inserted. We were able to inflate the thorax of the intubated seal effectively with the animal positioned in lateral recumbency. Similarly, seals breathing unaided appeared to do so most easily while in lateral recumbency. Animals left in sternal recumbency appeared to have more difficulty achieving effective inspiration, and did not breathe as frequently as those animals in lateral recumbency. All animals were, therefore, maintained in lateral recumbency until they were capable of active resistance. It was noted in animal number 16 that laryngeal manipulation induced immediate and violent spasmodic head movement and it became impossible to intubate this animal.

In the case of animal number 28, a different problem arose. At no stage was this animal more than moderately sedated; at 25 minutes the animal began to show signs of labored respiration. The nostrils opened and closed rapidly, but there were no accompanying thoracic movements nor were the usual respiratory sounds (particularly those associated with expiration) detectable. No improvement could be obtained despite attempts to reposition the animal and the intravenous administration of repeated doses of doxapram. By 70 min, the animal had ceased all active movement and a decline in consciousness followed, terminating fatally by 90 min. The signs initially exhibited by this animal were consistent with upper respiratory tract obstruction. The anatomical features of the Weddell seal's upper respiratory tract (Hammond and Elsner, 1977) and the fact that the trachea of a Weddell seal is so compliant as to be capable of complete collapse when relaxed (Kooyman, 1981) could be expected to predispose to obstructive problems. Unlike animals number 10 and 16, animal number 28 was conscious at the time respiratory difficulty began. It would seem unlikely that the mortality in animal number 28 was directly attributable to the immobilizing agent. The acute distress consequent to the progressive dyspnea may have precipitated an irreversible cardiopulmonary decline similar to that observed in earlier fatalities.

The respiratory stimulant doxapram hydrochloride was used in six animals, three of which died. In the remaining three animals, there was clear evidence that respiration improved subsequent to administration of the drug. Two apneic animals returned to a normal pattern of respiration almost immediately and another showed a delayed but persistent improvement in its respiration. The transient nature of the respiratory improvement in animal number 10 may have been due to a delay in drug administration, possibly allowing the initiation of cardiovascular dysfunction thereby inhibiting the distribution and effectiveness of the drug. Extradural administration of doxapram after initiation of the observed decline in cardiopulmonary function appeared to have no significant effect.

The principal pathophysiological features exhibited by the animals which died were a prolonged period (>10 min) of apnea which was unresponsive to repeated doses of doxapram hydrochloride, quickly followed by profound bradycardia (heart rate < 30 beats/min) progressing to cardiac arrest within another 10 to 15 min. Gales and Burton (1988) suggest that such events are an inappropriate activation of the physiological changes associated with diving, often described as the dive response. The results of the present study suggest that to reliably counteract apnea, doxapram should be given at a time when cardiovascular function is uncompromised, thus ensuring delivery of the drug to its target organ (the carotid and aortic chemoreceptors). When apnea is associated with cardiovascular dysfunction, the

best method of drug administration may be intracardiac injection, as this ensures that the preferred chemical is incorporated immediately into the functional circulation. Difficulty of access could, however, prevent the routine use of such an approach. The sublingual vein may maintain circulation in apneic or unresponsive seals (Gales, 1989) and would provide a more accessible alternative than the intracardiac route.

Two key areas of future research are indicated; one is a full pharmacokinetic study of immobilizing agents in Weddell seals and the second is a simultaneous study of the induced cardiopulmonary dysfunction. Further study is required on the matters of which drugs are most appropriate for the induction and subsequent reversal of immobilization in Weddell seals, on agents capable of counteracting the pathophysiological changes which may occur with chemical immobilization and on the most effective route by which such agents can be administered.

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