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IMMOBILIZATION OF POLAR BEARS WITH CARFENTANIL

J. C. Haigh,¹ L. J. Lee,² and R. E. Schweinsburg²

ABSTRACT: Sixty-four free-ranging polar bears (*Ursus maritimus*) were immobilized using carfentanil at doses ranging from 1.0–38.0 µg/kg (mean 20 ± 8 µg/kg). Induction was rapid (5.0 min) ($n = 46$) and the bears showed good muscle relaxation. Respiratory rate was depressed (mean 3.8 bpm). The mean arousal after the administration of narcotic antagonists was longer than 5 min. Recurrence of narcotic effects—so called recycling—was seen in some bears and in a separate black bear trial was consistently observed in animals given doses of 10 µg/kg or more of carfentanil. The rapid induction, low drug volume and excellent muscle relaxation related to carfentanil immobilization make this a potentially useful drug for polar bear immobilization.

INTRODUCTION

When immobilizing free-ranging animals, it is important to be able to use an agent that suits the species and the environment. The drug should be one that allows small doses, has a short induction time, a wide margin of safety, as few side effects as possible, and allows for rapid recovery. Synthetic opiates such as etorphine hydrochloride (M99, D.M. Pharmaceuticals, Rockville, Maryland 20850, USA) and fentanyl citrate (Janssen Pharmaceuticals, Beerse, Belgium) have many of these qualities. A host of species, including all North American bears, have been successfully immobilized with these drugs (Flyger, 1967; Wallach et al., 1967; Miller and Will, 1974; Haigh, 1977; Patenaude, 1979; Hebert et al., 1980). Recently another extremely potent narcotic, carfentanil (Janssen Pharmaceuticals, Beerse, Belgium) has become available in North America on an experimental basis. Carfentanil has been used with great success to immobilize 217 free-ranging African herbivores of 20 different species (de Vos, 1978). Carfentanil has also been used in wapiti (*Cervus elaphus*) (Willard et al., 1982; Haigh, 1983) and moose (*Alces alces*) (Haigh et al., 1982). This paper reports results obtained using carfentanil to immobilize polar bears.

MATERIALS AND METHODS

This study was conducted in conjunction with a Northwest Territories Wildlife Service polar bear

mark-recapture project. During May and August 1980 and May 1981, 64 polar bears were located and immobilized from a helicopter as described by Lentfer (1968) and Larsen (1971). Carfentanil was supplied by the manufacturer in sealed vials containing 10 mg/ml.

Darts (Palmer Chemical Co., Douglasville, Georgia 30134, USA) were preloaded with two standard doses: 5 mg for large males, and 3 mg for females and smaller bears. During the second year the 3 mg dose was used for almost all bears except cubs of the year (COY). No yearlings were encountered. Cubs of the year were injected from the ground using a blow gun at doses of approximately 0.1 mg. Dart needles of 4.5 cm were used for adult bears while 3-cm needles were used on smaller animals. Most injections were intramuscular (i.m.) into the hind quarters of the animal. Mean heart rates, respiratory rates, rectal temperatures, and induction times were measured. They are reported only for those bears which were immobilized with one injection. Induction time, considered to be the period from injection to the time the bear was tractable, was measured. Changes in respiratory rates were examined with a Mann-Whitney *U*-test at a significance level of 0.05. Differences in induction times and dosages between sexes were examined with a 2-tailed *t*-test at a significance level of 0.05.

RESULTS AND DISCUSSION

A total of 64 polar bears was successfully immobilized. Fifty became tractable with one injection and 14 required a second dose (Table 1). Most of the bears requiring a second dose had been hit in either the flank or lower leg, sites often considered unsatisfactory for good immobilization (Harthoorn, 1976). After receiving the second dose the bears became tractable quickly.

Effective dosages for bears other than COY ranged from 1.0 to 38 µg/kg with a mean of 20 ± 8 µg/kg. These small dosages reflect the potency of carfentanil. The lowest effective dose recorded was 1.0 µg/kg to immobilize a 560-kg male. It is probable that a dose lower than the

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TABLE 1. Observations on polar bears after single intramuscular injections of carfentanil.

	Bears >1 yr				Cubs of the year			
	Mean	SD	Range	n	Mean	SD	Range	n
Dosage carfentanil (g/kg)	20	8.0	1.0–38.0	46	4.5	1.7	3.0–7.0	4
Induction time (min)	5.0	2.4	2.0–12.0	46	2.7	1.5	1.0–4.0	3
Arousal time after antagonist (min)	7.0	3.89	1.0–22.0	51	7.3	4.5	2.0–11.0	4
Heart rate (bpm)	59.0	18.6	27.0–123.0	33	102.5	5.0	100–110	4
Rectal temperature (C)	39.4	0.98	38.0–41.9	37	38.8	0.79	38.1–39.9	4
Respiratory rate (bpm)	3.8	1.8	1.0–12.0	37	5.5	0.6	5–6	4

mean of 20 $\mu\text{g/kg}$ would be effective in immobilizing most bears if a good injection occurred. During the later parts of the study, lower doses proved effective. When using narcotics to immobilize free-ranging animals, it is usually better to dose heavily and reverse quickly. Underdosing tends to prolong the induction time exposing the animal to a higher degree of stress and possibly injury. Effective dosages for COY ($n = 4$) ranged from 3.0 to 7.0 $\mu\text{g/kg}$ with a mean of $4.5 \pm 1.7 \mu\text{g/kg}$. The mean time to recumbency was 2.7 min (± 1.5). These doses are in the same range as those quoted by de Vos (1978) for a number of African herbivores.

Induction times for both adults and COY (Table 1) were relatively short in comparison to those obtained with other drugs (Lentfer, 1968; Seal and Erickson, 1969; Larsen, 1971; Lee et al., 1981). De Vos (1978) reports a mean induction time with carfentanil of 6.3 min ($n = 217$) over 20 different African herbivores. A rapid induction is very desirable with polar bears as they have a tendency to head for open water when harassed. They also tend to overheat under continued physical exertion. A swift immobilization minimizes these problems.

Induction of polar bears was characterized by a slowing of gait with some ataxia, quickly followed by a dropping or lowering of the head and neck until the nose was pointed straight down. Observations of tracks indicated a shortening of stride and a failure to pick up the feet. Stumbling for only a short distance (15–30 m) was evident in animals which received an effective dose. Once the head was pointed down most bears would stand with their body sway-

ing slightly for about 30 sec before collapsing completely. In short inductions, there was often little seen except for a sudden collapse to sternal recumbency.

Lowering of the head and neck was also observed in ungulates (de Vos, 1978). We found this sign to be a good indicator that the animal was successfully under the effect of the drug. In conditions of wet snow, if bears collapsed nose down, they were in danger of drowning. If an animal was to collapse this way it became necessary to immediately “buzz” the animal with the helicopter in an attempt to get it to lift its head. This was generally successful if done before the animal was unable to respond. In three instances it was necessary to land, jump out quickly and carefully pull the head to one side and then retreat and allow the animal to become totally immobilized. Six bears were closely approached within 3 or 4 min of going down and responded by moving a maximum of 10 m before again going down. It became standard practice to fly past the animals at about 50 m to check for head position. If the bears were satisfactorily positioned they were left alone for a full 5 min before being approached again.

Underdose, or perhaps more correctly, poor injection, simply prolonged induction. Bears would continue walking in a slow stumbling manner. Of the 14 bears that required a second injection, seven fell down after the first injection, but got up when approached by the helicopter and had to be left for extra time before being approached again. With the dosages used, no second doses were required while working

on the animal. No abnormal excitement was noticed during the induction phase or in the case of underdose such as is common with morphine-like analgesics (Harthoorn, 1976). De Vos (1978) reported that in most species immobilized with carfentanil "very little excitement was noticed during the induction phase" if external stimuli were kept at a minimum.

Immobilization of polar bears was normally quite complete with excellent muscle relaxation. Four bears in the early stages of narcosis were able to move their heads in response to having their mouths pried open, but had little strength or control. This stage was only noticed if the bears were approached soon after going down and it passed quickly as the animals became totally relaxed. No urination, excess salivation, tetany, or convulsions were observed. Bears' eyes remained closed during the immobilization period. Heart rates, respiratory rates and rectal temperatures are summarized in Table 1. Capillary refill times for bears immobilized with carfentanil were less than 1 sec.

Non-medicated resting polar bears exhibit heart rates between 40–65 bpm (Oritsland, 1970; Best, 1975), a somewhat narrower range than the 27–123 bpm observed in this study. It is difficult to assess the effect of carfentanil on pulse rate as each animal was captured under different conditions, however, the range and mean of heart rates for bears in this study were similar to bears immobilized with ketamine HCl and xylazine HCl (36–90 bpm, 62.3 ± 14.3 bpm, $n = 21$) (Lee et al., 1981).

Moderately active polar bears are reported to have respiratory rates of between 10–20 (Best, 1975), substantially higher than the range of 2–12 ($\bar{x} 3.8 \pm 1.8$, $n = 37$) for bears in this study. Although de Vos (1978) noted that respiratory depression was rare in ungulates administered carfentanil, morphine-related compounds are known to produce respiratory depression in some animals and bears seem particularly sensitive (Larsen, 1971; Miller and Will, 1974; Haigh, 1977; Hebert et al., 1980). During 1981 we administered azaperone concurrent with carfentanil in an attempt to increase the respiratory frequency. Although the mean frequency was slightly higher (4.3 bpm) with azaperone than without (3.6 bpm) there was no significant difference. Respiration did, however, appear to be deeper and more regular with azaperone. The intravenous administra-

tion of azaperone (0.5–1.0 mg/kg) to carfentanil immobilized black bears (*Ursus americanus*) has caused a marked rise in respiratory rates, which may assist in cooling (Haigh, 1983). This effect, first reported 13 yr ago (Marsboom, 1969) has also been produced in dogs, pigs and rats (Jageneau, pers. comm.) and will be further tested.

Administration of a partial dose of narcotic antagonist has been successful in raising the respiratory frequency in polar bears (Taylor et al., 1982) and black bears (Miller and Will, 1974) immobilized with M99. Although this method is effective, it is not recommended for polar bears unless the researchers desire, and are prepared for, rapid and complete recovery of the animal.

Rectal temperatures appeared to be elevated in polar bears immobilized with carfentanil. During this study, temperatures ranged from 38.0–41.0 C with a mean of 39.4 C which is considerably higher than reported for unmedicated bears (36.9 C) at rest (Best, 1975). The rapid induction obtained with carfentanil reduces the degree of overheating. Bears immobilized with ketamine HCl and xylazine HCl had a lower range (36.5–40.2 C) and mean (38.9 C, $n = 17$) rectal temperatures but yet had considerably longer chase time (Lee et al., 1981). The apparent elevation in body temperature observed when using carfentanil is probably related to the relatively low respiratory rate and consequent reduced heat exchange capability.

The narcotic antagonists used were naloxone hydrochloride (Endo Labs Ltd., Baie d'Urfe, Quebec) and diprenorphine hydrochloride (M5050, Cyanamid of Canada, and Revivon, Reckitt and Sons, Hull, England). In the spring of 1980, naloxone was administered alone, 60 mg intravenously (i.v.), 40 mg intramuscular (i.m.) and 20 mg subcutaneous (s.c.). The mean recovery (to standing) time was 8.75 min (± 6.5 , $n = 19$). Although no bears were subsequently found recumbent, some tracks as late as the next day indicated some degrees of gait incoordination.

In August 1980, diprenorphine was given at a dose of 2–3 mg s.c. followed by naloxone 50 mg i.v. The mean time to recovery was 5.59 min (± 2.06 , $n = 8$). This did not alleviate the apparent 'recycling' and one bear was found partially drugged in a snow bank 20 hr later.

During the spring of 1981 diprenorphine was

also given s.c. in conjunction with naloxone 30–50 mg i.v. and 30–40 mg i.m. No bears were subsequently seen in a drugged state, but adverse weather conditions prevented a proper assessment.

De Vos (1978) reported prolonged drowsiness in animals immobilized with high doses of carfentanil and it may be that this, rather than the amount of antagonist, lies at the root of the problem in bears effected some time after handling. This hypothesis was partially supported in a separate black bear trial (Haigh, unpubl. data) in which recycling occurred in COY given doses of 10 µg/kg or more of carfentanil even when very large doses of either antagonist were given. These cubs would appear to recover completely from carfentanil within 10 min of antagonist administration and remain normal for several hours. They became renarcotized from 8 to 20 hr later, and could only be aroused following the administration of further antagonist. Black bear COY given doses of 5 µg/kg or less of carfentanil showed no recycling after any of the antagonist regimes.

Arousal times, for bears other than COY, after injection of the antagonist ranged from 1 to 22 min with a mean of 7.0 ± 3.89 ($n = 51$) (Fig. 1). This is somewhat longer than reported (3.6 min, $n = 217$) by de Vos (1978) when using carfentanil to immobilize several African species. He found that a significantly higher dose of the antagonist was required to counteract the action of carfentanil as opposed to other narcotics. Two further bears had to be given second injections of antagonist by darting 55 min after the initial dose had been given only by the i.m. route. These animals were up and moving within 8 min of the second injection.

The first indication of revival was an abrupt and marked increase in respiratory frequency. Panting was noticed in several bears after respiration had begun to increase. The next noticeable sign was the lifting of the head, followed very quickly by the animal getting to its feet and moving off. The rate at which the bears became fully mobile varied. Although some animals got to their feet and moved away unsteadily, they gained full coordination after 50 to 100 m. Some bears did not get up after 12–15 min and were “buzzed” with the helicopter, after which they got to their feet and moved away.

One bear died during the August tagging op-

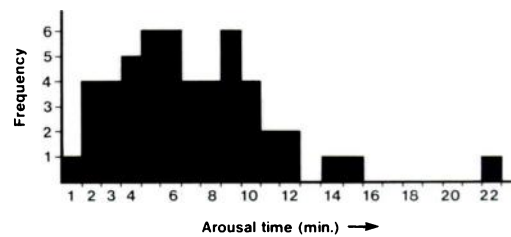


FIGURE 1. Frequency distribution of arousal times for 51 polar bears above 1 yr treated with narcotic antagonist (naloxone ± diprenorphine) following immobilization with carfentanil.

eration, but we do not feel this was drug related. The animal, a young female, was very fat, and ran 700–1,100 m before becoming immobile. It is likely the bear died from a combination of stress and overheating. An experienced hunter examining the carcass commented that young female bears chased hard in early fall had been known to succumb to chasing by hunters without a shot being fired. Post mortem examination did not reveal any specific histologic lesions. Extensive petechiation and ecchymosis indicated a shock-related death.

Carfentanil appears to have considerable potential as an immobilizing agent for polar bears. Its rapid action, high potency, and reversibility all make it very desirable. But, the problem of recycling needs to be addressed under controlled conditions to determine its frequency and ways to prevent it. Presently carfentanil is only available on an experimental basis and being a particularly potent narcotic, it is strictly controlled.

Carfentanil is potentially lethal to humans at the doses used in polar bears and only a small puncture by a used needle could lead to death. The drug should be handled with extreme care and researchers using it should be totally familiar with accident procedures (Parker and Haigh, 1982).

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