IMMOBILIZATION OF MOOSE WITH CARFENTANIL

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Abstract: From March 1983 through March 1984, 92 adult moose (Alces alces) were immobilized using carfentanil. The concentration of the drug (10 mg/ml) allowed the use of small volumes for injection, alleviating some problems associated with large volume dosages. Total dosage per moose varied from 2.5 to 5 mg carfentanil (0.006 to 0.014 mg/Kg). Mean induction time for moose receiving at least 3 mg was 5.0 minutes (SD = 2.1, n = 75). Diprenorphine (M50-50) was used as the antagonist. During 1983, generally 14 mg were given IV and 6 mg IM. In 1984, dosage was increased and generally 20 mg were given IV and 10 to 20 IM, and 3 moose were given 30 mg IM only. Mean recovery time was 4.2 minutes (SD = 1.9, n = 52), excluding the IM-onlyosed moose. Hyperthermia, acute capture myopathy and/or narcotic recycling were attributed to 6 mortalities (8.5%) directly associated with immobilization. Causes of mortality and ways to minimize it are discussed.

Immobilization of moose (Alces alces) in North America with a synthetic opiate (etorphine hydrochloride, Lenmon Co., Sellersville, PA) has been the drug of choice in recent years (Franzmann 1982). However, the drug is marketed in the United States in a solution of only 1 mg/ml which required injecting large volumes into moose for immobilization creating a series of potential problems (Franzmann 1982). In the late 1970's, an extremely potent synthetic opiate, carfentanil (Janssen Pharmaceutica, Beerse, Belgium) was developed and tested with success on 217 free-ranging African herbivores of 20 different species (DeVos 1978). More recently, carfentanil
was successfully used to immobilize 58 wapiti (*Cervus elaphus*) and 3 moose (Meulman et al. 1984) and polar bears (*Ursus maritimus*) (Haigh et al. 1983). Haigh et al. (1982) listed carfentanil as the immobilizing agent for moose in Saskatchewan.

Carfentanil is the most recent drug we have tested at the Moose Research Center (MRC). Our experiences using other immobilizing drugs for moose at the MRC were reported (Franzmann et al. 1982). We had more difficulties with the use of carfentanil than anticipated based upon previous reports, but the promise for the drug remains. We have outlined in this paper our experiences and from them we have provided our assessment for minimizing these problems.

METHODS

We immobilized 92 adult moose (30 males, 62 females) with carfentanil. Sixty-seven moose were injected by firing standard 2 or 3 ml Cap-Chur darts (Cap-Chur, Palmer Chemical Co., Douglasville, GA) from a helicopter, 20 moose were injected with Cap-Chur darts while in traps (LeReshe and Lynch 1973) at the MRC (Figure 1), and 5 moose were injected with a hand-held disposable 3 ml syringe.

Immobilized moose were processed which included measuring, bleeding, collecting hair, ear tagging, radio-collaring, and weighing when possible. Tame experimental animals at the MRC were weighed on a platform scale prior to immobilizing (injections were given on the scale). Moose that were caught in MRC traps were weighed using our tripod device mounted on the front of a 4-wheel drive vehicle (Arneson and Franzmann 1975). Moose that were immobilized via helicopter and moved to other MRC enclosures by slinging from the helicopter were weighed using a scale suspended from the helicopter. We estimated weights of moose not scale weighed by applying the regression equation from total length measurements (weight in Kg = 239.7 + 2.07 x total length in cm) (Franzmann et al. 1978). Radio-collars equipped with mortality sensors (Telonic, Mesa, AZ) permitted us to monitor the moose subsequent to immobilization.

Moose were kept immobilized from 15 to 90 minutes depending upon conditions and whether or not they were translocated. Eight moose were transported between MRC enclosures on a snowmachine trailer and 3 moose were moved in a sling from a helicopter.

When processing and handling was completed, we used the antagonist diprenorphine hydrochloride (MSO-50, Lemmon Co., Sellersville, PA) to neutralize the effects of carfentanil. Nearly all moose during 1983 received 14 mg diprenorphine intravenously (IV) and 6 mg intramuscularly (IM). Only 4 animals were given additional antagonist IM when response to the antagonist was poor. In 1984, the antagonist was increased and 20 mg were given IV and 10 to 20 IM.
Eleven moose were given xylazine hydrochloride (Rompun, Haver-Lockhart Laboratories, Shawnee, KS); 8 received 100 mg and 4 received 150 mg. The xylazine was mixed with the carfentanil.

RESULTS

MRC Trapped Moose (1983)

Eighteen adult moose and 2 yearling males were captured in MRC traps and immobilized with total dosages of carfentanil from 3 to 4 mg (\(\bar{x} = 3.73\)). Dosage per Kg varied from 0.009 to 0.012 (\(\bar{x} = 0.011, n = 20\)). Induction times varied from 1.5 to 5 minutes (\(\bar{x} = 3.8, \text{s.d.} = 1.0\); excluding 1 male moose that was darted in a testicle with a resulting induction time of 19 minutes. Each moose received 14 mg diprenorphine IV and 6 IM. Recovery times ranged from 2 to 8 minutes (\(\bar{x} = 4.4, \text{s.d.} = 1.6\) (Table 1). Eleven moose were given the tranquilizer xylazine with the carfentanil (8 given 100 mg, 2 given 150 mg). No significant differences (P >0.1) were detected in induction time for moose receiving xylazine (\(\bar{x} = 3.98, n = 10\)) to those not receiving it (\(\bar{x} = 3.49, n = 9\)) or in recovery for those receiving xylazine (\(\bar{x} = 5.0, n = 8\)) versus not receiving it (\(\bar{x} = 4.1, n = 9\)).

Mean recovery times for the eight moose trapped and translocated with the snowmobile trailer was 5 minutes (s.d. = 1.5) which was slightly longer than those not moved (\(\bar{x} = 3.9, \text{s.d.} = 1.6, n = 9\)); however, the differences were not significant (P >0.1).

One mortality was directly attributed to capture and immobilization. A mature male (1-83) was captured in December and given 4 mg carfentanil. Induction time was 3 minutes (Table 1). Difficulties were experienced in weighing the animal due to equipment failure and he was handled longer than usual (40 min.) When the antagonist was given the operator was not certain if the initial 14 mg were injected into the vein. The moose was slow to respond and did not get up for several hours. He was found dead 300 m from the trap 48 hours after capture. This moose was captured earlier (September) and was the moose that was darted in the testicle at that time (Table 1). No effects of the dart in the testicle were noted at capture in December.

Three additional moose died subsequent to capture and immobilization in MRC traps, but we were uncertain as to the causes of death. One moose may have died soon after we released her in May. She was not found until September, but was not seen during the intervening time in the MRC #1 enclosure (Figure 1). When captured in May, she was in extremely poor condition (Grade of 4, Franzmann 1977). Moose 22-83 died 20 days post-immobilization and moose 25-83 died 99 days post-immobilization. Both moose were in enclosure #3 at the MRC and their carcasses were well consumed when found. Wolf (Canis lupus) tracks surrounded the carcasses. We do not know if wolves killed these moose, but a lone wolf was known to reside in the enclosure during the time.
MRC Tame Moose (1983)

Five moose were immobilized at the MRC using a hand-held syringe and injecting them as they were weighed on the scale. Injections for immobilization given by hand-held syringe required less dosage of other drugs used in the past at the MRC (Franzmann et al. 1982) and we adjusted our dosage of carfentanil accordingly. The 2 mature bulls (Rodney, Chief) each received 4 mg, the 2-year old bulls (Charlie, Joker) each received 3 mg and the mature cow received 3.5 mg of carfentanil, but had to be given an additional 2 mg to complete induction (Table 2). The mature bulls each received 0.006 mg/Kg of carfentanil and the 2-year old bulls received 0.008 mg/Kg. Induction times were 4 to 5 minutes and recovery time ranged from 1 to 9 minutes (Table 2).

Rodney resisted the narcotizing effects of the drug and when released from the scale began pushing strenuously against the fence with his antlers. He began to overheat and by the time he collapsed (5 min.) his body temperature rose to 40.5°C (40.2°C considered critical point-Franzmann et al. 1974). We attempted to cool him with water, but could not bring his temperature to normal. We administered the antagonist (20 mg IV and 10 mg IM of diprenorphine) but he did not get up for 60 minutes. His respirations were laborious the remainder of that day and the next. He died the following morning. Necropsy revealed acute lung congestion and congestion of the small intestine and liver. Spraker (1982) described this as the acute death syndrome of capture myopathy.

Other tame moose recovered in a seemingly uneventful manner, but we noted a decrease in appetite for several days. Chief's body weight (BW) dropped in 9 days from 632 to 558 Kg (7% loss of BW) and Jezebel's dropped from 510 to 468 Kg (8% loss of BW). We had no follow-up weights on Charlie and Joker until well into the rut. We also observed similar loss of appetite when moose were immobilized with etorphine. During January 1981, we immobilized moose with etorphine that were on a measure intake and their food intake dropped dramatically. We had to abort the feeding trail and the moose did not get back on full feed for 2 weeks.

Helicopter Captured Moose (1983)

Forty-six adult moose were immobilized with carfentanil with a Cap-Chur dart fired from a helicopter during 1983; 1 group in March (n = 32) and 1 in November-December (n = 14). Eight moose were given a total dosage of 2.5 mg carfentanil (\( \bar{X} = 0.007 \) mg/Kg). Mean induction time was 7.8 minutes and recovery time was 6.3 minutes (Table 3). Nine moose were given a total dosage of 3 mg carfentanil (\( \bar{X} = 0.008 \) mg/Kg). Mean induction and recovery times were both 5.3 minutes (Table 3). The remaining 15 moose immobilized in March were given a total dosage of 4 mg carfentanil (\( \bar{X} = 0.011 \) mg/Kg). Mean induction time was 4.7 minutes and mean recovery time was 4.9 minutes (Table 3). Most moose immobilized in November-December were given a total dosage of 4 mg (2 received 5 mg) (\( \bar{X} = 0.011 \) mg/Kg). Mean induction time was 5.7 minutes and mean recovery time was 3.2 minutes. However, recovery time data were obtained from only 9 of 14 moose.
Three 1983 helicopter darted moose died soon after capture; 2 in the Beluga population and 1 at the MRC. Moose 88 was dead 7 days following capture and she had not moved from the site. Moose 938 died in 3 days, but had moved 8 miles from the capture site. The moose was given 2 doses due to poor response to the first and did not respond readily to the antagonist. Moose 32-83 was darted first in the fatty rump patch. She partially responded to this, but had to be given an additional 2 mg on the ground. She responded to the antagonist is 3.5 minutes, but was found dead 200 m from capture site in 4 days. Narcotic recycling, capture stress, and capture myopathy were likely contributors to all 3 mortalities.

In the Beluga population, 4 delayed mortalities were attributed to brown bear (*Ursus arctos*) predation. Two delayed mortalities occurred in the MRC helicopter captured group. One was a mature male (31-83) that died 40 days post-immobilization from causes unknown. This moose had jumped into the MRC enclosure a week prior to immobilizing him. Moose 30-83 died 103 days post-immobilization from malnutrition/starvation. She was 16 years old, had pearls for incisors and was moved from enclosure 1 to 4 at the MRC while immobilized (Figure 1). Higher mortality rates have been recorded for introduced versus resident moose at the MRC (Bailey and Franzmann 1983), and we believe the factors identified previously were important adjuncts to the causes of death of these 2 moose.

Helicopter Captured Moose (1984)

In March 1984, 21 adult moose were immobilized in the Melchina Basin using a helicopter. Thirteen received total dosages of 5 mg carfentanil and 8 received 4 mg (Table 4). The mean induction time was 6.3 minutes (S.D. 4.2 = n = 19) and mean recovery time was 3.6 (S.D. = 1.3, n = 5). The dosage of the antagonist was increased based upon our previous experiences. Eighteen moose received 20 mg M50-50 IV and from 10 to 20 mg IM ($\overline{x}$ = 15.1), while 3 other moose received 30 mg M50-50 IM only. Three moose did not respond to the initial dosage; and 2 moose still were recumbent with 24 hrs. and 1 within 48 hrs. Moose 694 was given an additional 20 mg M50-50 IM and recovered. Moose 628 was also given 20 mg IM in 24 hrs but did not get up and died within 1 week. One moose that was recumbent at 48 hrs was given 10 mg IM and was up in 5 minutes (Table 4). All moose except #628 were alive and well 3 weeks post-capture.

The cause of the single mortality in this group could not be determined. Narcotic recycling occurred in at least 2 other animal which recovered.

The mean induction time for all the groups combined and excluding double dosed animals was 5.0 minutes (S.D. = 2.1, n = 75). Mean recovery time was 4.2 minutes (S.D. 1.9, n = 52).
DISCUSSION

Carfentanil proved to be an effective immobilizing drug for moose with several advantages over etorphine; the current drug of choice for immobilizing moose (Franzmann 1982). One advantage was its concentration (supplied in 1 ml ampoules containing 10 mg carfentanil). We reconstituted the drug to a solution of 2 mg/ml for easier handling. This allowed us to use 3 ml Cap-Chur darts instead of 10 or 15 ml darts.

Ten criteria were outlined that an ideal immobilizing drug should possess (Franzmann 1982): (1) rapid absorption and action, (2) concentrated form, (3) wide range of tolerance, (4) safe for handler, (5) reversible, (6) no side effects, (7) effective anesthesia level, (8) not subject to controlled substance registration, (9) cleared for use on animals for food, and (10) low cost. Carfentanil qualifies positively for most of the criteria, but it is dangerous for the handler (Parker and Haigh 1982), subject to Dangerous Drug licensing, not cleared for food animals, and it may be expensive when commercially available (Wildlife Laboratories, 217 Bradley Drive, Fort Collins, CO 80524).

Recurrence of narcotic effects or narcotic recycling was evident in most of the moose we could follow (10%). We had similar recycling with etorphine as well, but not as prominent. Perhaps, this was because we were giving a dosage of narcotic that was greater per effect due to its concentration. We also may have been underdosing the antagonist. Haigh et al. (1983) reported similar experiences with polar bears and black bears (Ursus americanus) which they attributed to carfentanil dosages over 10 microgram/Kg (0.01 mg/Kg). Moose receiving over 0.01 mg/Kg carfentanil had the best response (shortest induction and recovery times, Table 3). We believe the amount and method of administering the antagonist may be a factor as well. Jessup et al. (1984) reported that in mule deer (Odocoileus hemionus) diprenorphine at the rate of 8 mg/1mg of carfentanil was necessary to reverse narcotic effects of carfentanil. However, Meuleman et al. (1984) reported successful (IM) reversal in elk (Cervus elaphus) with a ratio of 3 to 4 mg diprenorphine/1 mg carfentanil. Our ratios for moose during 1983 ranged from 5 to 8 mg diprenorphine/1mg carfentanil; mostly around 5 to 6 mg. In 1984 we used 8 to 10 mg diprenorphine/1mg of carfentanil and we still experienced recycling. Total dosage for most adult Alaskan moose was 4 mg and we presently recommend 20 mg diprenorphine IV, 10 mg IM, and an additional 10 mg SQ if difficulties were experienced during immobilization or handling. At this time, we consider the dosage range of 0.009 to 0.011 mg/Kg ideal for moose. More information is needed on antagonist dosage and method of dosage. Three moose given 30 mg M50-50 IM only recovered (Table 4). From Meuleman et al. (1984) experiences with elk and the few moose that we reversed IM, it appears the IM-only route may be best.

The amount of xylazine we added to the carfentanil had no effect on induction or recovery times. Method of handling; i.e., helicopter capture, trapping and moving, or hand injection all were associated with some of the problem moose, and none could be singled out as a contributing factor. The
use of tranquilizers as adjunct drugs to carfentanil requires additional study. Jessup et al. (1984) considered them as useful to "potentiate and balance" the narcotic effect in mule deer. We did not use tranquilizers in most of these trials and we must be convinced of their value in future use for short-term handling procedures that are generally associated with radio-collaring and field sampling procedures. Nevertheless, they should continue to be investigated in light of our present problems.

The delayed deaths (9) we experienced were listed to keep the record complete. We do not know if immobilization procedures were involved in these deaths, but we are suspect of some. The loss of appetite and weight loss that our experimental animals experienced following immobilization could be significant to an animal in a negative energy balance. The narcotic recycling could also put the animals in jeopardy in bad terrain or where predators were prevalent. We must be aware that these animals receive a great amount of stress from immobilization, even when all appears to go well. In some cases, that additional stress may be critical to the animals’ survival.

There was no common denominator that could be identified with the causes of acute capture-related mortalities (Table 5). The circumstances associated with immobilization were different for each of the 6 animals (6.5% of total immobilized). We were concerned about the moose we translocated, but none of those animals died. We had at least 1 death associated with each location, sex, or method of capture. Some moose received 2 injections of carfentanil and some not; one received an adjunct tranquilizer. The dosage ranged from 0.006 to 0.012 mg/Kg carfentanil. The only factor that is somewhat consistent is that the recovery time in 4 of the 6 was prolonged. This would indicate that our problems were related to drug dosage and/or absorption, narcotic recycling, and/or inadequate antagonist effect and not necessarily procedural methods. Rodney's death was from the acute death syndrome of capture myopathy, but triggered by hyperthermia. Moose 1-83 also died from acute capture myopathy, but triggered by stress and incomplete antagonism of carfentanil. Moose 32-83 also died from acute capture myopathy. Her syndrome was triggered by prolonged induction time due to dart placement in rump fat (additional dose was needed) and perhaps narcotic recycling due to incomplete antagonism of carfentanil.

Some of the problems that haunt all immobilization procedures were not absent from these trails. We are aware of 2 darts which did not fire or inject, one incomplete injection, and poor placement of several darts. A few animals were stressed more than necessary and the antagonist dosage was perhaps too low on some. Nevertheless, we can conclude that we have an improved product for immobilizing moose; the concentration of the drug was ideal; induction times were reduced; and the level of immobilization was satisfactory. The problems we experienced were primarily related to the antagonist (diprenorphine and its method of administration). Naloxone hydrochloride needs additional testing as a carfentanil antagonist in moose.
ACKNOWLEDGEMENTS

We thank K. Schneider and S. Peterson who reviewed early drafts of the manuscript and D. Warning, M. Hubbert, C. Gardner, G. Del Frate, J. Whitman and D. Groves who assisted us in the field and all other Alaska Department of Fish and Game personnel who provided support. Janssen Pharmaceutica, Beerse, Belgium supplied the carfentanil for which we are most grateful and Kenai Air Service did an excellent job as usual with the helicopter work. This work was supported in part by Federal Aid in Wildlife Restoration Project W-21-R.

LITERATURE CITED


Table 2. Moose immobilized with carfentanil administered via hand-held syringe during September, 1983 at MRC.

<table>
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<th>Moose</th>
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<th>Dose per Kg of Carfentanil</th>
<th>Induction Time in IV mg</th>
<th>Recovery Time in IV mg</th>
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<td>510</td>
<td>3.5 * 2.0</td>
<td>---</td>
<td>---</td>
<td>14 6 2</td>
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<td>632</td>
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<td>14 6</td>
<td>9.0 7% BW lost in 2 days dead in 48 hrs, acute capture myopathy Hyperthermia</td>
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<td>4</td>
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*Weights are actual scale live weights.*
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Table 1. Summary of snow cover-related data of snow sampled with accessories in Alaska.