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Article *in* Veterinary Anaesthesia and Analgesia · March 2018

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Veterinary Anaesthesia and Analgesia 2018, **xxx**, 1–8 https://doi.org/10.1016/j.vaa.2018.02.008

RESEARCH PAPER

Effective thiafentanil immobilization and physiological responses of free-ranging moose (*Alces alces*) in northern Sweden

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Abstract

Objective To evaluate clinical and physiological responses in moose to thiafentanil administration for immobilization.

Study design Cross-sectional clinical study.

Animals Eleven (six males and five females) free-ranging adult moose (*Alces alces*).

Methods Each moose was darted from a helicopter with 7.5 mg thiafentanil during March 2014 in northern Sweden. Physiological evaluation included vital signs and blood gases. Arterial blood was collected after induction and again after 10 minutes of intranasal oxygen administration and analyzed immediately with an i-STAT analyzer. A total of 10 mg naltrexone per milligram of thiafentanil was administered to all animals for reversal. Data were analyzed using descriptive statistics.

Results All moose were sufficiently immobilized with a single dart injection. Induction occurred within 3 minutes in 10 of 11 moose. One individual became recumbent while crossing a river and naltrexone was immediately administered. Animals maintained sternal recumbency with their head raised and vital signs were stable. Nine of 10 moose were hypoxemic before oxygen administration, with seven becoming markedly hypoxemic [partial pressure of arterial oxygen (PaO_2) between 40 and 59 mmHg (5.3–7.9 kPa)]. The PaO_2 increased significantly between samples, but six moose remained hypoxemic despite therapy. Hypercapnia was seen in all moose, with eight having marked hypercapnia [partial pressure of arterial carbon dioxide ($PaCO_2$) > 60 mmHg (>8.0 kPa)]. All moose were acidemic, with nine showing marked acidemia (pH < 7.20). The pH increased significantly with time and lactate decreased. Recoveries were rapid and uneventful, and all moose were living 6 months after capture.

Conclusions Thiafentanil provided rapid and sufficient immobilization of moose and its effects were rapidly reversed with naltrexone. As with other opioids, moose showed hypoxemia and varying degrees of respiratory and metabolic acidosis. Arterial oxygenation of moose improved following intranasal oxygen, but hypoxemia was not fully resolved despite therapy.

Clinical relevance Thiafentanil (7.5 mg per adult) is effective for immobilization of free-ranging moose. Supplemental oxygen may be of benefit when using this regimen; however, further investigation is required to confirm these results.

Keywords acid-base status, *Alces alces*, arterial blood gases, immobilization, thiafentanil.

Introduction

Free-ranging moose (*Alces alces*) are chemically captured and restrained for research and management purposes. Potent opioids, such as etorphine and carfentanil, have been the primary drugs used for capture of free-ranging moose, and are used alone or in combination with xylazine. Addition of xylazine

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has been discouraged because of an increased risk of regurgitation and aspiration pneumonia (Kreeger 2000; Kreeger & Arnemo 2012). Carfentanil has been widely used in moose and other cervids in North America, but is no longer commercially available. Thiafentanil is another potent opioid that is effective for immobilization of wild ungulates, including moose (McJames et al. 1994; Kreeger et al. 2005). Thiafentanil has potential advantages over both carfentanil and etorphine, including faster induction, shorter half-life and thus less potential for renarcotization (Lance & Kenny 2012). In Shiras moose (*Alces alces shirasi*), a standard dose of 10 mg thiafentanil per animal resulted in short recumbency times, minimal side effects, rapid and complete reversals with nalmefene or naltrexone and no resedation events (McJames et al. 1994). A mortality of 4% (three of 73 animals) within 30 days of capture was reported with the same protocol (Kreeger et al. 2005), though there was limited evidence to link the three deaths to drug effects. In addition to capture-related death, nonlethal adverse effects of chemical capture may have long-term implications for animal welfare and research results. Potent opioids are major respiratory depressants (Burroughs et al. 2012). Severe hypoxemia and respiratory acidosis have been previously reported in moose immobilized with an etorphine-based protocol (Evans et al. 2012; Lian et al. 2014), and can result in irreversible cell damage and subsequent dysfunction of vital organs (McDonell & Kerr 2015), emphasizing the need for physiological assessment of captures.

In this study, we evaluated a lower than previously used dose of thiafentanil (McJames et al. 1994; Kreeger et al. 2005) for aerial darting of free-ranging moose in northern Sweden, and assessed the animals' clinical and physiological responses to capture based on vital signs, arterial blood gases and acid-base status. In addition, we evaluated intranasal oxygen for correction of hypoxemia during immobilization.

Materials and methods

Eleven free-ranging moose (six males and five females, 2−7 years old based on tooth wear; Ericsson & Wallin 2001) were chemically captured for deployment of tracking collars and sampling during March 2014 in Nikkaluokta, northern Sweden (67°52′N, 18°66′E), as part of a long-term ecological research project. Captures were approved by the Ethical Committee on Animal Experiments, Umeå, Sweden (#A 50/12). All personnel were certified according to the standards by the Swedish Board of Agriculture.

Captures occurred at ambient temperatures from −1 to 3 °C and altitudes from 460 to 638 m above sea level. Moose were located in the study area by direct observations and darted from a helicopter in the gluteal or brachial muscles using a carbon dioxide-powered rifle (Dan-Inject ApS, Denmark). Only animals appearing healthy and in relatively open areas were darted. A total dose of 7.5 mg of thiafentanil (thiafentanil oxalate 10 mg mL^{-1}, Thianil; Wildlife Pharmaceuticals Pty Ltd, South Africa) was administered to each moose, using 1.5 mL darts with 2.0 × 40 mm barbed needles (Dan-Inject). Animals were not weighed. Darts were fitted with a tracking device (RECCO AB; Lidingö, Sweden) to ensure that lost darts could be recovered. Time from sighting to successful darting (chase time) and time from darting to recumbency (induction time) were recorded. At the end of the capture, the effects of thiafentanil were antagonized with 10 mg of naltrexone (naltrexone hydrochloride 50 mg mL^{-1}, Trenoxil; Wildlife Pharmaceuticals Pty Ltd) per milligram of thiafentanil, half intramuscularly and half intravenously. Time from darting to administration of antagonist (handling time) and times from administration of antagonist to standing (standing time) and walking away (walking time) were recorded. Induction and recovery quality were assessed subjectively, according to whether or not it occurred in a calm and uneventful manner.

Once immobilized, moose were approached quietly, corrected for positioning if needed and blindfolded to reduce stimuli during handling. Heart rate (by auscultation), respiratory rate (by counting breaths), rectal temperature (using a digital thermometer), capillary refill time (CRT; time to capillary refill after blanching an area of the gum) and mucous membrane color were recorded shortly after recumbency and approximately 15 minutes later.

When opioids are used alone, moose are conscious and responsive to stimuli, but usually immobilized sufficiently to be safely approached and handled (Kreeger & Arnemo 2012). The degree of immobilization was classified as: level 1 (slightly sedated, does not lie down); level 2 (incompletely immobilized, lies down but rises when approached or handled); level 3 (immobilized, remains down, intact reflexes, raised head, responds to stimuli); level 4 (completely immobilized, unable to lift head, depressed reflexes, less or nonresponsive to stimuli); and level 5 (unconscious, absent reflexes). This evaluation was made

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at approach and along with the monitoring described earlier.

As soon as possible after induction, an arterial blood sample was collected anaerobically from the auricular artery using self-filling preheparinized syringes (PICO70; Radiometer Copenhagen, Denmark) and analyzed immediately with an i-STAT 1 Portable Clinical Analyzer (Abbott Laboratories, IL, USA) and i-STAT CG8+ cartridges, except one animal on which CG4+ cartridges were used instead. The analyzer was stored in an insulated box with a warm water bottle to maintain operating temperatures (16−30 °C). Measured parameters included pH, partial pressure of arterial oxygen (PaO_2), partial pressure of arterial carbon dioxide ($PaCO_2$) and blood concentrations of sodium (Na), potassium (K), ionized calcium (iCa) and glucose. PaO_2, $PaCO_2$ and pH were corrected based on rectal temperatures. Calculated parameters included total carbon dioxide (TCO_2), bicarbonate (HCO_3^-), base excess (BE), hemoglobin oxygen saturation (SaO_2), hematocrit (Hct) and hemoglobin (Hb). Blood concentrations of lactate were measured with Lactate Pro 2 (ARKRAY Factory, Inc., Japan).

As soon as the first arterial sample was collected, intranasal oxygen insufflation from a portable oxygen cylinder at a flow rate of 3 L minute^{-1} was administered. A nasal catheter was inserted about 10 cm into one of the nostrils and fixated with adhesive tape. Ten minutes after starting oxygen supplementation, a second arterial sample was collected and analyzed as described.

Hypoxemia was defined based on partial pressure of oxygen as mild (60−79 mmHg), marked (40−59 mmHg) and severe (<40 mmHg). Hypercapnia was defined based on partial pressure of carbon dioxide as mild (45−60 mmHg) and marked (>60 mmHg). Hypocapnia was defined as a $PaCO_2 < 35$ mmHg. Acidemia was defined as a pH < 7.35 and marked acidemia if pH < 7.20.

Data analysis

All statistical analyses were performed with the software R version 3.3.1 (R Core Team 2017). Descriptive statistics were used to report median (range) values for the physiological variables, including heart rate, respiratory rate, rectal temperature and arterial blood gases, measured before and after oxygen supplementation. Results from first and second measurement were compared using a Wilcoxon signed-rank test. Time variables, including chase time, induction time, handling time and standing and walking times, were also reported as median (range) and compared between sexes using a Wilcoxon rank-sum test. A p value <0.05 was considered significant in all analyses. We chose to apply nonparametric tests, as normality of data distribution could not be reliably assumed, given the small number of observations.

Results

All animals became sufficiently immobilized with one dart injection and no supplemental doses were required. There was no significant difference in the recorded times between sexes (Wilcoxon rank-sum test; $p > 0.05$), and data were therefore pooled together (Table 1). Ten of 11 moose became recumbent in ≤3 minutes after successful darting. One female moose, chased for about 7 minutes, moved into a shallow open river within several minutes after darting and the drug effect was reversed immediately to prevent drowning. This animal was excluded from further analyses. For the rest, inductions were calm, with no relevant intercurrences.

Immobilizations were mostly characterized as level 3, with moose remaining in sternal recumbency, with head raised, slight muscle rigidity and moderately responsive to touch and sound. In four animals, degree of immobilization varied between levels 3 and 4 during the handling period.

Vital signs and arterial blood-gases results from immobilized moose, before and after oxygen treatment, are summarized in Table 2. Mucous membranes were pale in two of nine assessed animals and the CRT was 2.5−4.0 seconds long in six of nine, shortly after induction. At the second assessment, all moose showed a pink oral mucosa and the CRT had normalized to ≤2 seconds in most. Heart rate, respiratory rate and rectal temperature did not change significantly between samples (Table 2). Nine of 10

Table 1 Time variables (in minutes) recorded for free-ranging moose (*Alces alces*) chemically immobilized with thiafentanil by darting from helicopter, in northern Sweden.

Variable	*n*	Median	Range
Chase time	11	3.0	1.4−7.0
Induction time	11	2.5	1.3−5.0
Handling time	10	43.3	39.0−54.0
Standing time	10	1.6	0.8−3.3
Walking time	10	2.7	0.8−5.7

Chase time, time from sighting to successful darting; handling time, time from darting to reversal; induction time, time from darting to recumbency; standing time, time from reversal to standing; walking time, time from reversal to walking away.

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Table 2 Physiological variables from moose (Alces alces) chemically immobilized with thiafentanil by darting from a helicopter in northern Sweden. Results of two measurements, before and after a median oxygen supplementation time of 15 (10–16) minutes, were compared using a Wilcoxon signed-rank test. Statistically significant results at a level of $p < 0.05$ are presented in bold.

Variable	Unit	n	Median (range)		*p*
			Before O_2	After O_2	
			T1: 9–25 minutes	T2: 28–41 minutes	
Heart rate	Beats minute^{-1}	10	60 (40–116)	62 (52–116)	0.622
Respiratory rate	Breaths minute^{-1}	10	28 (20–58)	22 (20–46)	0.291
Rectal temp.	°C	10	38.3 (36.9–40.3)	38.7 (36.6–40.8)	0.271
PaO_2	mmHg	10	52 (36–73)	69.5 (49–83)	**0.011**
PaO_2	kPa	10	6.9 (4.8–9.7)	9.3 (6.5–11.1)	**0.011**
PaO_2^*	mmHg	10	57 (42–85)	76.5 (55–87)	**0.014**
PaO_2^*	kPa	10	7.6 (5.6–11.3)	10.2 (7.3–11.6)	**0.014**
$PaCO_2$	mmHg	10	60.3 (53.8–79.8)	54.6 (39.4–77)	0.131
$PaCO_2$	kPa	10	8.0 (7.2–10.6)	7.3 (5.3–10.3)	0.131
$PaCO_2^*$	mmHg	10	64.1 (56.7–79.5)	58.7 (42.2–76.7)	0.160
$PaCO_2^*$	kPa	10	8.5 (7.6–10.6)	7.8 (5.6–10.2)	0.160
SaO_2	%	10	73 (44–88)	89 (76–94)	**0.011**
pH		10	7.10 (6.99–7.21)	7.21 (7.12–7.32)	**0.006**
pH*		10	7.09 (6.95–7.20)	7.19 (7.08–7.30)	**0.006**
BE	mmol L^{-1}	10	−11 (−16–(−5))	−6 (−15–(−2))	**0.009**
HCO_3^-	mmol L^{-1}	10	18.7 (16.4–24.6)	23.2 (14–27.6)	0.066
TCO_2	mmol L^{-1}	10	20.5 (18–27)	24.5 (15–30)	0.167
Hct	% PCV	9	44 (37–50)	43 (36–49)	**0.031**
Hb	g dL^{-1}	9	15 (13–17)	14.6 (12.2–16.7)	**0.022**
Lactate	mmol L^{-1}	10	11.3 (6.9–16.9)	5.2 (3.1–13.8)	**0.002**
Glucose	mg dL^{-1}	10	132 (110–179)	142 (106–163)	0.575
Na	mmol L^{-1}	9	141 (137–143)	140 (136–142)	0.143
K	mmol L^{-1}	9	3.8 (3.6–4.6)	3.5 (3.1–4.2)	**0.018**
iCa	mmol L^{-1}	9	1.25 (1.18–1.28)	1.24 (1.17–1.31)	0.400

BE, base excess; Hb, hemoglobin; HCO_3^-, bicarbonate; Hct, hematocrit; iCa, ionized calcium; K, potassium; Na, sodium; $PaCO_2$, partial pressure of arterial carbon dioxide; PaO_2, partial pressure of arterial oxygen; SaO_2, arterial oxygen saturation; T1, time range from darting to collection of first sample; T2, time range from darting to collection of second sample; TCO_2, total carbon dioxide. *PaO_2, $PaCO_2$ and pH are temperature-corrected values.

animals were hypoxemic at the first measurement, based on the temperature-corrected values. Two of the moose showed mild hypoxemia and seven were markedly hypoxemic. The PaO_2 significantly increased between samples (Table 2), but it remained below 80 mmHg in six animals. Hypercapnia was noted in all immobilized moose, with eight having marked hypercapnia. All moose were acidemic, with nine animals showing marked acidemia.

Recoveries following naltrexone administration were calm, and all moose stood up within approximately 3 minutes (Table 1) and walked away in a coordinated manner. All animals were alive 12 months after immobilizations, except for one male moose, which was shot after 6 months.

Discussion

With the thiafentanil regimen used in this study, moose became rapidly immobilized after a single dart injection and level of the immobilization was sufficient for a safe approach and handling.

A quick induction is essential in chemical capture of free-ranging wildlife, so that animals can be readily located, handled and monitored after drug administration, mitigating problems with injury, overheating and escapes. In addition, costs with capture are minimized, as helicopter flight duration is reduced with shortened induction times. Mean induction time for the 11 moose immobilized in our study was about 2 minutes shorter than the 4.4 ± 2.6 minutes reported for moose darted from a helicopter with the same dose of etorphine (7.5 mg per animal) in Norway (Haga et al. 2009).

With the use of thiafentanil as the sole agent, all moose were able to maintain sternal recumbency upon induction, which is desirable to reduce the risk of regurgitation and subsequent aspiration of rumen contents. These complications have been associated

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with carfentanil plus xylazine immobilization resulting in lateral recumbency and esophageal sphincter relaxation (Seal et al. 1985; Kreeger 2000). In addition, the sternal posture with head raised aids in collar placement and blood sampling from the jugular vein. By contrast, the use of thiafentanil alone, as seen with other opioids, results in some degree of muscle rigidity, which may hinder procedures such as opening the mouth.

Naltrexone rapidly reversed the effects of thiafentanil in moose, as previously reported (Kreeger et al. 2005), with no side effects noted. It is highly desirable that an effective reversal agent is available to shorten the recoveries in wild animals, especially large ruminants, which may develop bloat and regurgitation while recumbent. Other risks associated with prolonged recumbency include an extended period of hypoxemia and hypercapnia and compromised limb perfusion, especially in heavy animals placed in sternal recumbency. In addition, a prolonged recovery increases the chance of life-threatening complications, such as physical injury, hyperthermia and predation. Regardless of whether or not antagonist is administered, personnel should observe the animal until it can walk in a coordinated manner as well as assess possible hazards in the recovery area (Kreeger & Arnemo 2012).

Rectal temperature remained within acceptable ranges (38.4−38.9 °C; Franzmann et al. 1984) in most moose throughout immobilization. Clinically relevant hyperthermia was observed in one female, with rectal temperatures between 40.3 and 40.8 °C. This rise in body temperature may have resulted from physical exertion and/or excessive stress during helicopter pursuit, with 6 minute duration. Other factors, such an opioid-induced effect or a pre-existing pathological condition, cannot be ruled out.

Immobilization of wild animals with potent opioids is often associated with increases in body temperature (Cooper et al. 2005; Meyer et al. 2008b; Lian et al. 2016). While it is likely that opioids may interfere with normal thermoregulation as in domestic species (Lamont & Grimm 2014), the observed hyperthermia appears to be predominantly caused by the stress response to capture (Meyer et al. 2008a). Minimizing duration of intensive helicopter pursuit prior to darting helps to prevent hyperthermia associated with stress and physical strain. In addition, it is recommended that moose captures occur in winter. It is difficult to actively cool large animals such as moose under the capture circumstances reported, so antagonism of immobilizing agent to allow the animal's normal thermoregulatory mechanisms to recover should be considered (Arnemo et al. 2014).

Franzmann et al. (1984) reported safe expected ranges for heart and respiratory rates for chemically immobilized moose to be 70−91 beats minute^{-1} and 13−40 breaths minute^{-1}, respectively. Half of the animals in our study had heart rates ≤60 beats minute^{-1}. Tachycardia was observed in two animals, with heart rates ranging between 112 and 116 beats minute^{-1}. Heart rates remained relatively unchanged during immobilization. Differences in drug dosage (mg kg^{-1}), stress of capture, animal's age and health condition, among other uncontrolled factors, may account for the variation in observed cardiac responses. In addition, our results should be interpreted with caution given the small number of animals in this study.

Decreases in heart rate have been previously documented in moose immobilized with etorphine protocols, although more pronounced when combined with an alpha-2 agonist (Haga et al. 2009; Evans et al. 2012; Lian et al. 2014). The exact mechanisms behind effects of opioids on heart rate remain unknown; however, a baroreceptor-mediated decrease in heart rate as a result of opioid-induced systemic hypertension has been proposed (Heard et al. 1996). Bradycardia may also occur as result of an opioid-induced medullary vagal stimulation effect (Lamont & Grimm 2014).

Tachycardia was not noted in previous studies of moose administered opioids (Haga et al. 2009; Evans et al. 2012; Lian et al. 2014). However, the effects on heart rate appear to vary with the specific opioid used (Haigh 1990). The observed tachycardia did not improve with time, and the animals showed no signs of excitement associated with induction. A dose-dependent effect of thiafentanil, possible individual responses to the drug and/or a response to marked hypoxemia and acidemia may have played a role.

We documented blood-gas values describing varying degrees of hypoxemia, hypercapnia and acidemia in thiafentanil-immobilized moose. Our findings are consistent with those of previous blood-gas studies in moose immobilized with etorphine combined with xylazine and acepromazine (Evans et al. 2012; Lian et al. 2014).

Respiratory depression and hypoxemia are clinically important side effects of potent opioid administration in ungulate species (Howard et al. 2004; Meyer et al. 2010; Lian et al. 2016, 2017), and may be complicated by muscle rigidity, especially when opioids are used alone.

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Potent opioids cause a dose-dependent depression of the brain-stem respiratory centers, resulting in reduction of breathing frequency and decreased responsiveness to carbon dioxide. Ventilatory responses to hypoxic and hypercapnic peripheral chemoreceptor stimulation are also suppressed (McCrimmon & Alheid 2003; Pattinson 2008). As a result, individuals may be unable to maintain adequate ventilation and normal gas exchange to the alveoli, and potentially develop hypoxemia and hypercapnia.

The respiratory depression following thiafentanil administration was not accompanied by decreases in respiratory rates, which underscores the unreliability of assessing the effectiveness of ventilation based on respiratory rate alone. The observed respiratory rates, on the high side of normal for undisturbed moose (19 ± 8 breaths minute^{-1}, $n = 19$; Franzmann et al. 1984), were likely accompanied by decreased tidal volume, not measured in our study, such that adequate alveolar ventilation was not maintained and $PaCO_2$ rose.

While gas derangement following opioid administration may be in part attributed to opioid-induced hypoventilation, hypoxemia can also develop because of intrapulmonary causes, such as ventilation–perfusion mismatch, shunt or diffusion impairment. Evidence of impaired oxygen exchange, as demonstrated by increased alveolar–arterial oxygen partial pressure gradient (A–a gradient), has been reported in moose immobilized with etorphine-based protocols (Evans et al. 2012; Lian et al. 2014), suggesting that intrapulmonary alterations are likely contributing factors for the respiratory depression and hypoxemia in opioid-immobilized moose.

In a recent study, pulmonary hypertension was shown to play a significant role in altering pulmonary gas exchange, which is primarily responsible for the severe hypoxemia following etorphine administration in goats (Meyer et al. 2015). Whether similar pulmonary hypertension, decreasing pulmonary exchange, results from administration of thiafentanil still needs to be investigated.

In addition to drug-induced alterations, large ruminants often respond poorly to recumbency, because of the large rumen contents pushing forward the diaphragm, reducing lung volumes and thus further compromising ventilation and gas exchange (Buss et al. 2012). The degree of ventilation–perfusion mismatch is, however, more pronounced in dorsal and lateral positions.

Because our study was conducted at maximum elevations of about 600 m above sea level, low partial pressure of inspired oxygen (at high altitude and low barometric pressure) is not expected to be a significant contributing factor for the observed hypoxemia.

Evans et al. (2012) found no improvement in PaO_2 values in markedly hypoxemic, untreated moose during immobilization with etorphine–xylazine–acepromazine. Intranasal oxygen was shown to effectively improve arterial oxygenation in moose immobilized with the same protocol (Lian et al. 2014). Arterial oxygenation of moose in our study also improved following oxygen therapy, but hypoxemia persisted in six animals despite treatment. Most hypoxemic moose had been under oxygen supply for less than 15 minutes when the second arterial sample was collected, whereas the four nonhypoxemic moose had received oxygen for at least 15 minutes, which may have played a role. Intranasal oxygen was maintained until immediately before reversal, and perhaps hypoxemia was resolved by that time, but this could not be confirmed. Our oxygen flow rate was lower than that previously used in opioid-immobilized moose (Lian et al. 2014). Respiratory rates on the upper limit (or slightly increased) could be a sign of shallow breathing, which increases dead space ventilation. The insufficient response to supplemental oxygen could be also a result of nasal catheter incorrectly placed, kinked or blocked (though unlikely), or possible high shunt fractions (Fahlman 2014).

The respiratory acidosis observed in thiafentanil-immobilized moose, as indicated by hypercapnia and lower pH values, tended to clinically improve with time in most animals, though not significantly, contrary to previous findings (Lian et al. 2014). This improvement was unexpected as oxygen insufflation in opioid-immobilized animals tends to improve PaO_2 but, as ventilation remains unaltered, $PaCO_2$ levels increase over time. A decrease in $PaCO_2$ tends to suggest improved alveolar ventilation; if this occurred, the PaO_2 would have increased despite oxygen therapy. The interpretation of our results is limited by the fact we did not include a control group receiving no oxygen treatment.

The respiratory acidosis in these moose was apparently complicated by coexisting metabolic acidosis, as reflected by a decreased HCO_3^- concentration, when compared with domestic ruminants (Jackson & Cockcroft 2002), and a negative value for BE (i.e. base deficit). The metabolic part of the acidosis was likely caused by the severe lactacidemia

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observed. Median lactate concentration in the first sample was about five times higher than the 2.2 $mmol^{-1}$ upper limit for cattle (Jackson & Cockcroft 2002), indicating an increase in lactate levels associated with the immobilization procedure. The lactic acidosis improved with time, as reflected by significantly decreased lactate levels and increased pH and BE in the second sample. The plasma lactate concentrations in our study were in the range reported for immobilized moose (Haga et al. 2009; Evans et al. 2012), and for other chemically immobilized wild ruminants (Evans et al. 2013; Lian et al. 2016), which also showed a significant decrease in plasma concentrations over time.

In etorphine-immobilized moose, lactate concentrations were positively correlated with deep snow cover, likely resulting in higher physical exertion and short induction times, which may be related to a more profound opioid effect (Haga et al. 2009). Elevated lactate concentrations in captured moose are likely caused by increased oxygen demand resulting from physical strain and stress of capture, leading to increased production of lactate by skeletal muscle. The effects of thiafentanil, including respiratory depression and hypoxemia, may further compromise oxygen delivery to tissues. Lactate metabolism may be also decreased. The ability of the liver to consume lactate tends to decrease progressively as the plasma concentration of lactate increases. In addition, the hepatic uptake of lactate may be impaired because of several factors, including hypoxia, hypoperfusion (Tashkin et al. 1972) and severe acidosis (Lloyd et al. 1973). In addition, glucose levels were on the high side for a ruminant (Jackson & Cockcroft 2002), which is in part an expected result of stress and/or exertion associated with capture procedure (Rostal et al. 2012). An accelerated generation of pyruvate from glucose, being then converted into lactate, may have also played a role in the lactic acidemia.

Thiafentanil proved to be an effective immobilizing agent for free-ranging moose, under the described circumstances, providing short induction times, safe handling and rapid and uneventful recoveries with naltrexone administration. However, immobilized moose suffered hypoxemia and varying degrees of respiratory and metabolic acidosis. Arterial oxygenation of moose improved following oxygen insufflation, but hypoxemia was not resolved in all despite therapy. Our findings warrant further investigation on effectiveness and optimal flow rate of supplemental oxygen, as well as on other possible supportive measures, to improve respiratory function and blood oxygenation in thiafentanil-immobilized moose.

Acknowledgements

The Swedish University of Agricultural Sciences, the Swedish EPA and the program Beyond Moose and the Swedish Association of Hunting and Wildlife Management supported this project. The i-STAT analyzer and cartridges for the arterial blood-gas evaluation were provided by the Inland Norway University of Applied Sciences. Oliver Devineau from the Inland Norway University of Applied Sciences advised on the statistical analysis.

Conflict of interest statement

Authors declare no conflict of interest.

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Please cite this article in press as: Barros DSB, Evans AL, Arnemo JM et al. Effective thiafentanil immobilization and physiological responses of free-ranging moose (*Alces alces*) in northern Sweden, Veterinary Anaesthesia and Analgesia (2018), https://doi.org/10.1016/j.vaa.2018.02.008

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Received 9 May 2017; accepted 15 February 2018.

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Please cite this article in press as: Barros DSB, Evans AL, Arnemo JM et al. Effective thiafentanil immobilization and physiological responses of free-ranging moose (*Alces alces*) in northern Sweden, Veterinary Anaesthesia and Analgesia (2018), https://doi.org/10.1016/j.vaa.2018.02.008