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EFFECT OF ACTIVE COOLING AND α-2 ADRENOCEPTOR ANTAGONISM ON CORE TEMPERATURE IN ANESTHETIZED BROWN BEARS (*URSUS ARCTOS*)

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Abstract: Hyperthermia is a common complication during anesthesia of bears, and it can be life threatening. The objective of this study was to evaluate the effectiveness of active cooling on core body temperature for treatment of hyperthermia in anesthetized brown bears (*Ursus arctos*). In addition, body temperature after reversal with atipamezole was also evaluated. Twenty-five adult and subadult brown bears were captured with a combination of zolazepam-tiletamine and xylazine or medetomidine. A core temperature capsule was inserted into the bears' stomach or 15 cm into their rectum or a combination of both. In six bears with gastric temperatures ≥40.0°C, an active cooling protocol was performed, and the temperature change over 30 min was analyzed. The cooling protocol consisted of enemas with 2 L of water at approximately 5°C/100 kg of body weight every 10 min, 1 L of intravenous fluids at ambient temperature, water or snow on the paws or the inguinal area, intranasal oxygen supplementation, and removing the bear from direct sunlight or providing shade. Nine bears with body temperature >39.0°C that were not cooled served as control for the treated animals. Their body temperatures were recorded for 30 min, prior to administration of reversal. At the end of the anesthetic procedure, all bears received an intramuscular dose of atipamezole. In 10 bears, deep rectal temperature change over 30 min after administration of atipamezole was evaluated. The active cooling protocol used in hyperthermic bears significantly decreased their body temperatures within 10 min, and it produced a significantly greater decrease in their temperature than that recorded in the control group.

Key words: α-2 antagonist, brown bears, cooling, hyperthermia, *Ursus arctos.*

INTRODUCTION

Hyperthermia is a common complication during anesthesia of free-ranging bears.[20,25,27] Forty-six percent of free-ranging brown bears (*Ursus arctos*) captured following helicopter pursuit, via darting with medetomidine-zolazepam-tiletamine (MZT), developed hyperthermia.[12] In contrast, none of six captive brown bears anesthetized with the same drug combination developed hyperthermia.[12] The authors related the hyperthermia in free-ranging brown bears to physical exertion and stress following helicopter pursuit.[12] Hyperthermia has also been documented in physically exerted brown bears captured by helicopter darting or leg snares and anesthetized with a combination of xylazine-zolazepam-tiletamine.[7] During immobilization of polar bears (*Ursus maritimus*) located from a helicopter and darted with MZT, mildly hyperthermic animals were treated with cold water enemas to maintain their rectal temperatures below 39°C.[6] After intravenous (IV) administration of atipamezole, a 1-yr-old polar bear developed prolonged intense convulsions, marked hyperthermia (42.6°C), and died 25 min after reversal.[6] Two black bears (*Ursus americanus*), anesthetized with a combination of ketamine-xylazine, were found dead 2.5 days after anesthesia.[18] During anesthesia, rectal temperatures up to 43°C were recorded in these black bears, and hyperthermia was considered the probable cause of death.[18]

Treatment methods for hyperthermia reported in humans and animals include external cooling, cold water enemas, IV fluid administration, and intranasal oxygen supplementation.[2,11,12,28] Immer-

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sion in a bath with cold water and ice is considered the best method for cooling hyperthermic human athletes,[4,11] but this method may not be feasible for a large animal, such as a bear, captured in the field, unless capture occurs close to a natural water source. Other methods that were developed specifically for human use, such as circulating cold water or ice pack vests,[11] are not anatomically compatible with brown bears. Cooling methods reported for hyperthermic bears include cold water enemas,[6] external cooling with water,[18] intranasal oxygen supplementation,[13] and placement of snow or wet moss on the bears' foot pads and in the inguinal area.[26] To our knowledge, no studies have yet evaluated the effectiveness of these treatment methods for bears, and there are few publications on cooling methods in other wild mammalian species.[24]

Reversal is recommended in case of capture-related hyperthermia in field situations in which other treatments are not as effective.[1] The α-2 adrenoceptor agonist clonidine impairs thermoregulation in guinea pigs (*Cavia porcellus*) by promoting central thermogenesis, which increases the body heat production.[15] The α-2 adrenoceptor agonists, in general, also promote peripheral vasoconstriction,[21] which hampers loss of the excessive body heat.[16] In bears, combinations of α-2 adrenoceptor agonists and zolazepam-tiletamine are effective for immobilization, have analgesic properties, and enable the use of low-impact darting systems due to lower volume of drugs.[8] Another advantage is that the effects of the α-2 adrenoceptor agonists can be antagonized.[5,6,9] In one hyperthermic polar bear, however, rectal temperature did not decrease after one dose of atipamezole, and the animal died after intense convulsions.[6] The main objective of this study was to evaluate the effectiveness of active cooling on core body temperature for treatment of hyperthermia in anesthetized brown bears. In addition, body temperature after reversal with atipamezole was also evaluated.

MATERIALS AND METHODS

Captures

This study was approved by the Animal Care Committee of the University of Calgary, Canada (Protocol No. SHC11R-06) and by the Ethical Committee on Animal Experiments, Uppsala, Sweden (Application No. 07/12). The study included 25 adult and subadult brown bears that were captured for ongoing studies in Canada within the Foothills Research Institute Grizzly Bear Program (FGBP; $n = 13$), and in Sweden through the Scandinavian Brown Bear Research Project (SBBRP; $n = 12$). Within the FGBP, one bear was darted from a helicopter, and 12 bears were captured using culvert traps; all were anesthetized with a combination of a mean ± SD (range) dose of 5.4 ± 2.3 mg/kg (3.2–9.9 mg/kg) zolazepam-tiletamine (Telazol®, Fort Dodge Animal Health, Fort Dodge, Iowa 50501, USA) and 3.6 ± 1.5 mg/kg (2.1–6.5 mg/kg) xylazine (xylazine, 300 mg/ml; Bow Valley Research Inc., Calgary, Alberta T2N 4G3, Canada) administered intramuscularly. For captures within the SBBRP, all bears were darted from a helicopter with a combination of 4.0 ± 0.9 mg/kg (2.5–5.4 mg/kg) zolazepam-tiletamine (Zoletil Forte vet., Virbac S.A., Carros 06510, France) and 0.08 ± 0.02 mg/kg (0.05–0.11 mg/kg) medetomidine (Domitor® vet., 1 mg/ml, or Zalopine, 10 mg/ml; Orion Pharma Animal Health, Espoo FI-02101, Finland).

At the end of the anesthetic procedure, atipamezole was administered i.m. at five times the dose of medetomidine or at 0.2 mg/kg to bears anesthetized with xylazine (Antisedan® vet., 5 mg/ml; Orion Pharma Animal Health or Novartis Animal Health Canada, Inc., Mississauga, Ontario L5N 1V9, Canada). Within the FGBP, data for the present study were collected during captures in the province of Alberta, Canada, in June 2011, May and June 2012, and September 2012. Within the SBBRP, data for the present study were collected during captures in the county of Dalarna, Sweden, in April and June 2011 and April and August 2012.

Instrumentation

In bears anesthetized within the SBBRP, one core temperature capsule (Capsule Sensor, Mini-Mitter Company, Inc., Bend, Oregon 97701, USA) was inserted into the bear's stomach via an orally inserted gastric tube, as previously described.[22] Another capsule was inserted 15 cm deep into the animal's rectum with a standard applicator. Within FGBP, one core temperature capsule was inserted 15 cm into the rectum of the bears. All capsules were activated with the VitalSense® monitor (VS; VitalSense® Monitor, Mini Mitter Company, Inc.) and data transmission began approximately 1 min after activation and continued remotely every 15 sec thereafter. In all bears, rectal temperature was measured every 5–10 min with a handheld digital thermometer (Duoflex/PXR®, Rexall Brands Corp., Mississauga, Ontario L5M 0R4, Canada) inserted 8 cm into

the rectum. The tip of the handheld digital thermometer was directed to the rectal mucosa to ensure contact, and the temperature was recorded after the device produced an audible beep.

Groups

In six bears within the SBBRP that developed hyperthermia, defined as gastric temperatures ≥40°C, an active cooling protocol (group active cooling [AC]) was performed until gastric temperature was <40°C or until the end of the anesthetic procedure. The active cooling protocol consisted of applying all of the following: enemas with 2 L of water at approximately 5°C/100 kg of body weight every 10 min, 1 L of IV fluids at ambient temperature, water or snow on the paws, the inguinal area, or both and intranasal oxygen supplementation at a flow rate of 1–3 L/min,[13] and removing the bear from direct sunlight or providing shade. The active cooling protocol was adapted from what was currently used within SBBRP and from recommendations published as guidelines for different species.[2,10,17]

The effect of active cooling on the core temperature was determined by analyzing the temperature change measured by a gastric temperature capsule every 5 min for 30 min after the start of the active cooling protocol (time 0). The temperature change was calculated by subtracting the gastric temperatures at times 5, 10, 15, 20, 25, and 30 min after the start of active cooling from the gastric temperature at time 0. Data were also analyzed graphically to observe the difference in the temperatures measured with the handheld digital thermometer and the deep rectal capsules in comparison to core temperature measured with gastric capsules during the active cooling process, considering that the animals' recta were flushed with cold water. Animals included in group AC had abdominal surgery, with 5- to 10-cm-long surgical incisions, for implantation of different devices for study purposes within the SBBRP.

In the control group (group C), nine bears with initial temperatures >39.0°C that did not receive active cooling treatment, served as control animals for group AC. Bears included in this group were captured within both FGBP and SBBRP. The bears captured within the FGBP ($n = 3$) had one core temperature capsule inserted 15 cm deep into the rectum, and the temperatures were analyzed for 30 min after stabilization of the readings. That is, whenever the capsule recorded three consecutive measurements that did not vary more than 0.05°C. The bears captured within the SBBRP ($n = 6$) had a core temperature capsule inserted into the stomach, and the readings were analyzed for 30 min after stabilization of readings, as previously described. The temperature change was calculated by subtracting the deep rectal or gastric temperatures at times 5, 10, 15, 20, 25, and 30 min after beginning the monitoring (time 0) from those at time 0.

In 10 bears anesthetized with xylazine-tiletamine-zolazepam, the change in temperature recorded by deep rectal capsules was analyzed every 5 min for 30 min after administration of atipamezole (group A). The VS monitor was encased in a protective case and placed within 1 m of the bear to allow continuous monitoring of deep rectal temperature until the animal left the area. The temperature change was calculated by subtracting the deep rectal temperatures at times 5, 10, 15, 20, 25, and 30 min after administration of atipamezole (time 0) from the deep rectal temperature at time 0. The bears included in this group were captured within ongoing studies of the FGBP only, for logistical purposes, to enable retrieval of the VS monitor 24 hr after the end of the procedure, where it had been left by the recovering bear.

Data analysis

To compare the differences in temperature change between groups AC and C, the area under the curve (AUC) of temperature change over time was calculated for each individual bear. The AUCs obtained were analyzed with one-way analysis of variance (ANOVA). To evaluate the body temperature trend over time within each group, the change in temperature for each time point was compared with time 0 with a general linear model for repeated measures with the Tukey post hoc test.

In all bears, the following variables were recorded: heart rate (HR), determined through stethoscope auscultation; respiratory rate (RR), determined via observation of respiratory movements; ambient temperature; anesthetic time, defined as the time from darting until administration of the antagonist; and body mass in kilograms. To determine if body temperature at time 0, ambient temperature, anesthetic time, and body mass influenced the temperature variation among the groups, ANOVA with the Tukey post hoc test was performed. A difference was considered statistically significant for all analyses when $P < 0.05$. All statistical analyses were performed with

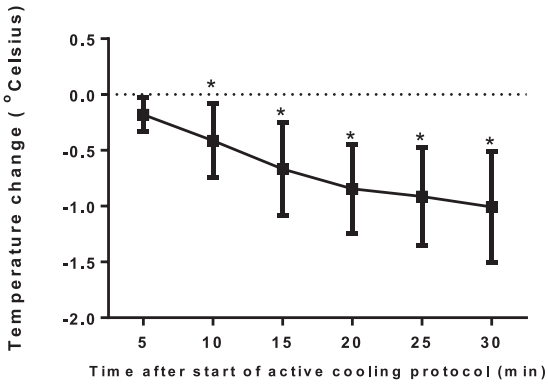

Figure 1. Gastric temperature change in anesthetized brown bears (Group AC, $n = 6$) with initial temperature of 40.59 ± 0.38°C (40.16–41.28°C), followed by treatment with an active cooling protocol with cold water enemas, intranasal oxygen supplementation, IV fluids, and external cooling. The asterisks represent the time points that were significantly different than time 0 (start of active cooling protocol).

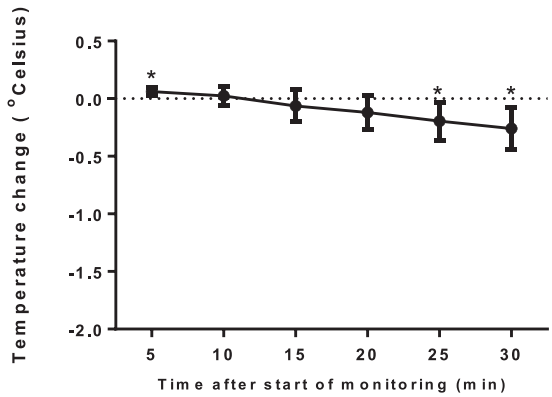

Figure 2. Core temperature change in anesthetized brown bears (Group C, $n = 9$) that served as controls for treated hyperthermic bears and had initial temperature of 39.97 ± 0.42 °C (39.18–40.51°C). The asterisks represent the time points significantly different than time 0 (start of monitoring).

IBM SPSS 20 (IBM SPSS® Statistics for Windows, Version 20.0, Armonk, New York 10504, USA) and GraphPad Prism 6 (GraphPad® Prism, Version 6 for Windows, GraphPad Software, La Jolla, California 92037, USA).

RESULTS

The active cooling protocol used in hyperthermic bears significantly decreased their body temperature within 10 min (Fig. 1). A significant decrease in temperature over time was observed in animals treated with the active cooling protocol in comparison to the control animals (mean difference = –12.84; $P = 0.002$). No statistical difference regarding initial temperature (Table 1) was observed between groups AC and C (mean difference = 0.61; $P = 0.41$). Temperatures within the control group decreased significantly after 25 min (Fig. 2).

Two of six bears in group AC remained hyperthermic 30 min after the start of the active cooling protocol. In one bear, stomach temperature decreased from 40.57°C before active cooling to 40.25°C 30 min after administration of two cold water enemas, placement of snow in its inguinal area, oxygen supplementation, and 1 L of IV fluids. At the end of the anesthetic procedure, the stomach temperature was 40.06°C, and it decreased further after atipamezole administration, reaching 38.94°C 30 min after reversal. The stomach temperature of the other bear decreased from 41.10°C before active cooling to 40.17°C 30 min after treatment. Its temperature steadily continued to decrease, and 30 min after administration of atipamezole, it reached 39.60°C. Temperatures measured by the handheld digital thermometer and the deep rectal capsules were markedly lower than the stomach temperature after the animals' recta had been flushed with cold water (Fig. 4).

In group A, temperature decreased significantly at times 5 (mean difference = 0.23; $P = 0.02$), 10 (mean difference = 0.39; $P = 0.01$), 15 (mean

Table 1. Mean ± SD and range of body temperature (time 0 and 30) measured with gastrically and deep rectally inserted core temperature capsules in anesthetized brown bears ($n = 25$) divided in groups AC (active cooling), A (atipamezole), and C (control).

Group	*n*	Body temperature (°C) at time 0	Body temperature (°C) at time 30
AC	6	40.59 ± 0.38 (40.16–41.28)	39.58 ± 0.67 (38.65–40.39)
A	10	38.70 ± 1.52 (36.09–41.79)	38.14 ± 1.62 (35.24–41.21)
C	9	39.97 ± 0.42 (39.18–40.51)	39.71 ± 0.49 (38.78–40.35)

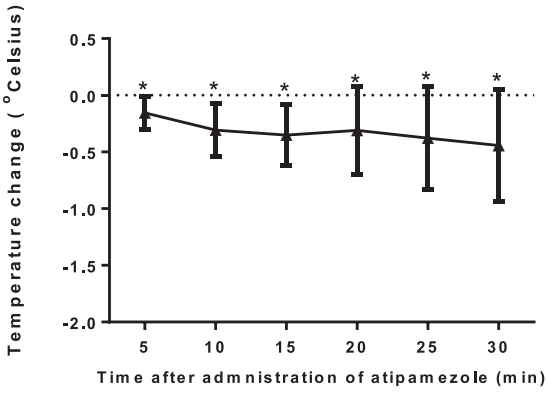

Figure 3. Deep rectal temperature change in brown bears (Group A, $n = 10$) with initial temperature of 38.70 ± 1.52°C (36.09–41.79°C), followed by reversal of anesthesia with atipamezole. The asterisks represent the time points that were significantly different than time 0 (administration of atipamezole).

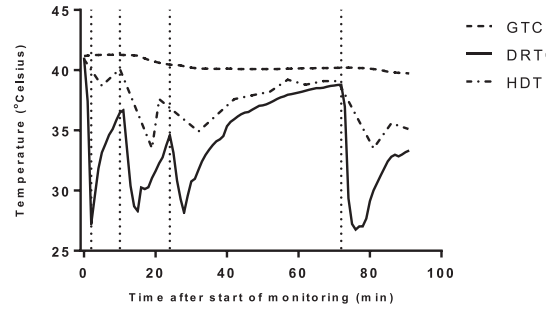

Figure 4. Temperatures measured by handheld digital thermometer (HDT), deep rectally (DRTC), and gastrically inserted (GTC) core temperature capsules in one anesthetized brown bear during active cooling with cold water enemas, intranasal oxygen supplementation, intravenous fluids, and external cooling. The vertical dash lines represent the time of administration of a cold water enema.

difference = 0.47; $P = 0.01$), 20 (mean difference = 53; $P = 0.01$), 25 (mean difference = 0.61; $P = 0.01$), and 30 (mean difference = 0.68; $P = 0.01$; Fig. 3). One bear in group A had a low deep rectal temperature (35.80°C) at the time of atipamezole administration, and 30 min after administration of atipamezole, the temperature had increased to 36.14°C. Initial temperatures in group A (Table 1) significantly differed from groups AC and C (mean difference = 2.45; $P < 0.0001$; mean difference = 1.83; $P = 0.0002$, respectively).

The ambient temperature during capture of the bears in group AC was 14.5 ± 8.6°C (5.0–26.2°C), in group A, 6.3 ± 3.9°C (3.0–11.0°C), and in group C, 9.1 ± 3.0°C (5.0–12.4°C). The anesthetic time in group AC was 149 ± 30 min (121–178 min), in group A, 144 ± 14 min (133–160 min), and in group C, 133 ± 27 min (100–165 min). The temperatures at time 0 and 30 in the three different groups are summarized in Table 1. The HR and RR were the following: in group C, 68 ± 12 beats/min (42–100 beats/min) and 8 ± 4 breaths/min (1–16 breaths/min); in group AC, 61 ± 18 beats/min (22–88 beats/min) and 13 ± 8 breaths/min (6–46 breaths/min); and in group A, 70 ± 10 beats/min (44–80 beats/min) and 11 ± 4 breaths/min (5–20 breaths/min). Ambient temperature did not differ significantly among groups AC and C ($F = 1.35$; $P = 0.31$). No significant differences were observed when comparing the anesthetic times among the groups ($F = 0.44$; $P = 0.66$).

DISCUSSION

The evaluated active cooling protocol effectively treated hyperthermia in four of six anesthetized brown bears. In the two bears that remained mildly hyperthermic, a more aggressive protocol with, for example, continuous cold water enema, cold water immersion, or both, may have been more effective. In humans, whole body immersion in a water-ice mixture produced a considerable decrease in body temperature.[23] However, this could lead to hypothermia, as the temperature continued to decrease in the human patients for up to 20 min after the end of the immersion, despite the use of active rewarming with forced warm air.[23]

This is the first study evaluating an active cooling protocol that includes administration of cold water enemas in wild animals. Other methods of active cooling have been evaluated in hyperthermic wild antelopes (*Damaliscus dorcas phillipsi*).[24] The treatment methods tested in that study were water dousing at different temperatures (4, 17, and 28°C), water dousing at 28°C with fanning, fine mist spray at 28°C, 1 L of IV saline at 4°C, and placement of ice packs on their skin.[24] The most effective method was considered water dousing, irrespective of the water temperature.[24]

In group A, a significant decrease in temperature over time was observed after administration of atipamezole. However, because no animals in group A were hyperthermic, in this study, it was not possible to determine if atipamezole would effectively treat hyperthermia in XZT anesthetized brown bears. It was also not possible to

attribute the decrease in temperature to the administration of atipamezole because there was no control group and because all animals received the reversal agent. Interestingly, in the hypothermic bear, the temperature increase after administration of atipamezole suggests that atipamezole might normalize thermoregulation impaired by α-2 adrenoceptor agonists. Further study on the influence of atipamezole on body temperature is warranted.

Because measurement of rectal temperature is not reliable in animals treated with cold water enemas, a new method for monitoring of core body temperature was used in this study by placement of gastric temperature capsules in the bears in group AC. Additionally, as previously reported in brown bears, deep rectally inserted temperature capsules accurately estimate core temperature measured by gastric capsules in bears that do not receive cold water enemas.[22] Thus, it was possible to compare temperature changes between groups AC and C by monitoring body temperature with either gastric or deep rectal capsules.

The ambient temperatures recorded in the present study did not differ significantly among the three groups; thus, it is not possible to determine how this variable might have impacted the temperature variation within each group. However, in the present study, ambient temperatures did not exceed previously published recommendations for polar bear capture (<20°C),[5] with the exception of one animal that was captured in 26.2°C. Rectal temperatures up to 43°C have been recorded when snare-capturing and anesthetizing black bears with ketamine and xylazine in ambient temperatures up to 35°C.[18] The average anesthetic time in the present study was similar to those previously reported in bears.[3,6]

Body condition can affect the ability of heat loss of an individual. In anesthetized human patients, higher total body fat content leads to a slower decrease in body temperature produced by active cooling.[19] In the present study, none of the captured bears had an extreme body condition; thus, it was assumed that heat loss variations due to body condition were not a variable influencing the study results. However, it is important to emphasize that bears captured in the fall could have a very high fat content that may affect the effectiveness of an active cooling for treatment of hyperthermia.

One limitation of this study was the occurrence of data loss on the VS monitor. This had little effect on the real-time monitoring of temperatures during the anesthetic procedure, but the missing data points did not allow the use of repeated measures ANOVA. To adjust the data set for these missing values, the AUC was calculated for each individual, and ANOVA was performed. In addition, a sample size of 11 bears per group would be necessary to obtain a statistical power of 0.9 and observe an effect size of 0.5 with repeated measures ANOVA.[14]

CONCLUSIONS

The active cooling protocol used in hyperthermic bears significantly decreased their body temperatures within 10 min, and it produced a significantly greater decrease in their temperature than that recorded in the control group. The data also suggested that atipamezole may normalize thermoregulation impaired by α-2 adrenoceptor agonists, as observed in the hypothermic bear in group A.

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