

CARDIOPULMONARY RESPONSE OF ANESTHETIZED POLAR BEARS TO SUSPENSION BY NET AND SLING

Authors: Cattet, Marc R. L., Caulkett, Nigel A., Streib, Kurt A., Torske, Kristine E., and Ramsay, Malcolm A.

Source: Journal of Wildlife Diseases, 35(3) : 548-556

Published By: Wildlife Disease Association

URL: <https://doi.org/10.7589/0090-3558-35.3.548>

The BioOne Digital Library (<https://bioone.org/>) provides worldwide distribution for more than 580 journals and eBooks from BioOne's community of over 150 nonprofit societies, research institutions, and university presses in the biological, ecological, and environmental sciences. The BioOne Digital Library encompasses the flagship aggregation BioOne Complete (<https://bioone.org/subscribe>), the BioOne Complete Archive (<https://bioone.org/archive>), and the BioOne eBooks program offerings ESA eBook Collection (<https://bioone.org/esa-ebooks>) and CSIRO Publishing BioSelect Collection (<https://bioone.org/csiro-ebooks>).

Your use of this PDF, the BioOne Digital Library, and all posted and associated content indicates your acceptance of BioOne's Terms of Use, available at www.bioone.org/terms-of-use.

Usage of BioOne Digital Library content is strictly limited to personal, educational, and non-commercial use. Commercial inquiries or rights and permissions requests should be directed to the individual publisher as copyright holder.

BioOne is an innovative nonprofit that sees sustainable scholarly publishing as an inherently collaborative enterprise connecting authors, nonprofit publishers, academic institutions, research libraries, and research funders in the common goal of maximizing access to critical research.

CARDIOPULMONARY RESPONSE OF ANESTHETIZED POLAR BEARS TO SUSPENSION BY NET AND SLING

Marc R. L. Cattet,^{1,5} Nigel A. Caulkett,² Kurt A. Streib,³ Kristine E. Torske,² and Malcolm A. Ramsay⁴

¹ Department of Veterinary Pathology, Western College of Veterinary Medicine, University of Saskatchewan, 52 Campus Drive, Saskatoon, Saskatchewan, Canada S7N 5B4

² Department of Veterinary Anesthesiology, Radiology, and Surgery, Western College of Veterinary Medicine, University of Saskatchewan, 52 Campus Drive, Saskatoon, Saskatchewan, Canada S7N 5B4

³ Orleans Veterinary Hospital, 2000-10th Line Rd., Orleans, Ontario, Canada K1C 1T1

⁴ Department of Biology, University of Saskatchewan, 112 Science Place, Saskatoon, Saskatchewan, Canada S7N 5E2

⁵ Corresponding author (e-mail: cattet@skyway.usask.ca)

ABSTRACT: Since 1995, at least three polar bears (*Ursus maritimus*) have died in the area of Churchill (Manitoba, Canada) as a direct result of being suspended in a net during helicopter-assisted translocations. To assess and improve methods of suspending anesthetized polar bears, we conducted a study during November 1997 to determine the cardiopulmonary responses of eight captive polar bears to suspension by net and by sling. Each bear was anesthetized on two occasions in which the sequence of activities followed and the type of data collected was identical, with only the method of suspension differing. Control data obtained from 11 captive polar bears during 1995–96 was included in the statistical analyses of cardiopulmonary data to help clearly differentiate the cardiopulmonary effects of suspension from those of drug metabolism. Suspending polar bears above the ground by net caused acute hypertension (e.g., 17 to 49% increase in mean arterial pressure), possibly as a result of increased venous return due to body compression. Increased arousal (e.g., head, tongue, and limb movement) also occurred consistently during net-suspension and suggested a stress response. Surprisingly, most suspended bears showed little change in blood gas values, but at least one bear became hypoxemic (i.e., $P_aO_2 < 60$ mm Hg) with each method of suspension. Because of the potential health risks of hypertension and hypoxemia, we recommend modifying the method by which polar bears are suspended with the goal of reducing body compression.

Key words: Anesthesia, cardiopulmonary effect, hypertension, hypoxemia, polar bear, suspension, Telazol®, *Ursus maritimus*.

INTRODUCTION

On 15 November 1973, “The International Agreement on the Conservation of Polar Bears and Their Habitat” was signed in Oslo, Norway, by representatives of Canada, Denmark, Norway, the former Union of Soviet Socialist Republics, and the United States of America. In compliance with this international agreement to manage and conserve polar bears (*Ursus maritimus*), researchers and wildlife managers have been intensively studying polar bear populations in Canada for the past 25 yr. Because chemical immobilization is often employed in research and management programs, considerable effort has been devoted towards developing and improving drug protocols so as to reduce handling mortality (Schweinsburg et al., 1982; Haigh et al., 1985; Ramsay et al.,

1985; Stirling et al., 1989; Cattet et al., 1997). However, other aspects of handling also may cause significant risk to anesthetized polar bears, as some die each year as a direct result of handling without evidence of adverse drug effect. For example, 13 (or 2.6%) of approximately 500 polar bears handled in western Hudson Bay, the most intensively researched population in Canada, died during 1995–97 (Polar Bear Technical Committee, unpubl. data). The cause of death in most cases was not determined.

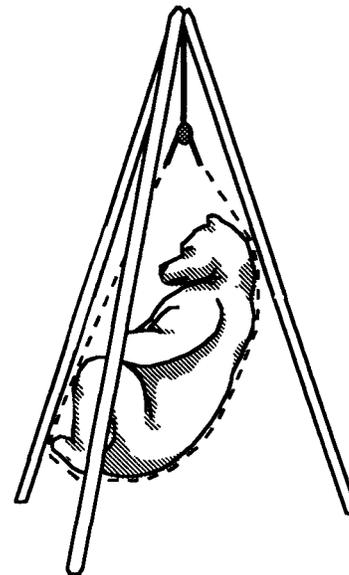
An aspect of handling which may impart excessive physiologic stress is the suspension of anesthetized polar bears above the ground. That is, each year in Churchill (Manitoba, Canada) polar bears are translocated away from the townsite while suspended in a cargo net beneath a helicop-

ter. Three of 13 polar bear deaths over the past 3 yr have occurred while bears were suspended in nets. Although necropsies were not conducted, compromised respiratory function due to inappropriate body positioning in the net was assumed to have resulted in fatal hypoxia. In effort to assess and improve methods of suspending anesthetized polar bears, we designed and conducted a study to determine the effect of suspension on the cardiopulmonary function of anesthetized polar bears. Within the context of this primary objective, we compared two methods of suspending anesthetized polar bears; one by net as employed in helicopter-assisted translocations, and the other by sling as used to determine body mass of individual bears.

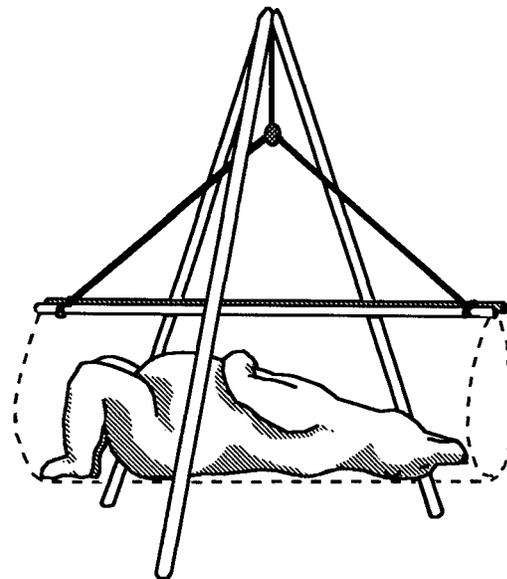
MATERIALS AND METHODS

We conducted our study with eight captive polar bears at Churchill (Manitoba, Canada; 58°45'N, 94°06'W) during November, 1997. Each bear was initially captured by Manitoba Department of Natural Resources personnel, with baited culvert traps or chemical immobilization (Telazol®, i.m. at 6 to 8 mg/kg), and maintained captive in a holding facility for 5 to 35 days prior to our study. These captures were conducted as part of an established wildlife management program designed to reduce the incidence of polar bears ranging into the vicinity of Churchill.

For this study, eight bears were anesthetized on two occasions each, with the sequence of activities followed and the type of data collected identical during each anesthesia event. Only the method of suspension differed between events; the sequence being determined randomly for each bear, e.g., net followed by sling or vice versa. Each anesthetic event was subdivided into three phases in effort to crudely represent the sequence of activities undertaken during research and translocation programs, in which polar bears are anesthetized and maintained on the ground for 20 to 30 min, then suspended for 5 to 20 min, and finally returned to the ground to recover. Phase 1 lasted 10 min and began immediately after physiologic equipment was attached to the anesthetized bear and calibrated, usually within 20 min of anesthesia induction. Throughout Phase 1, the bear was positioned in dorsal recumbency on the ground. Prior to the next phase, the bear was suspended by net or sling (Fig. 1), its body mass recorded, and the physiologic equipment



Net Suspension



Sling Suspension

FIGURE 1. Body position assumed by polar bears during two methods of suspension used in this study. Broken line represents outline of net and sling, respectively.

re-calibrated. Phase 2 lasted 15 min during which time the bear remained suspended. Afterwards, the anesthetized bear was lowered to the ground, re-positioned in dorsal recumbency, and the physiologic equipment recalibrated. Phase 3 was then initiated and lasted 10 min. During each of the three phases, physiologic data was recorded every 5 min, whereas blood gas data was recorded at the start and end of each phase.

Study animals were anesthetized with zolazepam and tiletamine, in a 1:1 combination by weight (Telazol[®], Fort Dodge Laboratories, Inc., Fort Dodge, Iowa, USA), at an estimated total dose of 8 to 10 mg/kg (Stirling et al., 1989). The drug was prepared in solution (227 mg/ml), by adding 1.8 ml of sterile water to 500 mg of lyophilized Telazol[®], and administered as two portions. The first portion, or induction dose (estimated 6 to 8 mg/kg), was delivered intramuscularly by pole syringe or blow gun. The second portion, or top-up dose (estimated 2 to 4 mg/kg), was delivered by hand injection into the muscles of the neck approximately 5 min prior to Phase 1. We administered a top-up dose to avoid the necessity of administering additional drug while collecting data and potentially causing drug-induced physiologic change.

Anesthetized bears were prepared for cardiopulmonary measurements by clipping the hair and aseptically preparing the skin overlying their femoral artery. The prepared skin was incised and the artery cannulated with an 18 ga × 10 cm intra-arterial catheter (Arrow Medical Products, Ltd., Mississauga, Ontario, Canada). The catheter was subsequently secured in the artery with subcutaneous ligatures and connected to a pressure transducer (Uniflow, Baxter Healthcare Corp., Irvine, California, USA), via non-compliant plastic tubing filled with heparinized saline. The transducer was, in turn, connected to a physiological monitor (Propaq 400, Protocal Systems, Inc., Beaverton, Oregon, USA) used to measure arterial pressures and heart rate. Percent saturation of hemoglobin with oxygen (SpO₂) was monitored using a reflectance probe inserted in the rectum, and connected to a pulse oximeter (4402 Vet/Ox[™] pulse oximeter system, Sensor Devices Inc., Waukesha, Wisconsin, USA).

Physiologic parameters measured at 5 min intervals during each phase were arterial pressures, heart rate, respiratory rate, and SpO₂. We also collected arterial blood samples in 3 ml sterile glass tubes containing anticoagulant (K₃) EDTA (Vacutainer[®], Becton Dickinson & Co., Franklin Lakes, New Jersey, USA) to determine hemoglobin concentration, and in 3 ml heparinized plastic syringes (Monoject[®], Sher-

wood Medical Co., St. Louis, Missouri, USA) for blood gas analyses. Blood samples were chilled in ice water immediately following collection. Within 3 hr of collection, hemoglobin and blood gas analyses were completed using an automated blood counter (Coulter Electronics Inc., Hialeah, Florida, USA) and blood gas analyzer (238 pH/Blood Gas Analyzer, Chiron Diagnostics Inc., Halstead, Essex, UK). Blood gas parameters were corrected for body temperature and hemoglobin concentration. When measurements were completed on each bear at the conclusion of Phase 3, physiologic equipment was disconnected, the intra-arterial catheter removed, and the skin incision closed using absorbable suture (PDS II, Ethicon[®], Ethicon, Inc., Somerville, New Jersey, USA). All bears were administered long-acting penicillin (Dual-Pen[®], TechAmerica Veterinary Products, Kansas City, Missouri, USA) for prophylaxis (10 ml per 50 kg i.m.).

Anesthetized bears were suspended during Phase 2 from a 3 m high tripod constructed of telescoping aluminum poles (Fig. 1). A square cargo net constructed of interlaced braided cord (10 cm mesh size) with braided nylon rope (2.5 cm diameter) attached along the margins was used for net suspension. In this, bears assumed a semi-reclined position with head resting forward on the chest; the position adopted during helicopter translocations (Fig. 1). A rectangular sling constructed of nylon webbing, woven in a criss-cross pattern, with two metal poles (each 4 cm diameter × 2.2 m length) attached along its length was used for sling suspension. In this, bears remained in dorsal recumbency with their neck and head supported and extended, and their forelimbs crossed over their chest (Fig. 1). We determined the body mass of suspended bears by linking an electronic load scale (Senstek[®], Norac Systems International, Inc., Saskatoon, Saskatchewan, Canada) between the net, or sling, and tripod. To avoid causing severe physiologic stress to suspended bears, acceptable ranges for heart rate (40 to 140 beats/min) and SpO₂ (≥60%) were established based on data collected during previous studies (M. Cattet, unpubl.). If a bear was to exhibit values outside of these ranges, it would be lowered to the ground immediately, supportive therapy would be instituted, and data collection would cease.

All data were analyzed statistically using SPSS software (SPSS Inc., Chicago, Illinois, USA) and statistical test values were regarded as significant when the probability (*P*) of Type I error < 5 percent. To compare drug doses and handling times between treatments or anesthesia events, we used *t*-tests for paired comparisons (Zar, 1996). Preliminary examination

TABLE 1. Comparison^a of drug doses and handling times for eight polar bears suspended by net and sling.

	Drug dose (mg/kg)			Time following induction (min) ^b			
	Induction	Top-up	Total	Phase 1	Phase 2	Phase 3	Total
Net	6.6 ± 0.73	2.9 ± 0.59	9.5 ± 0.81	34.3 ± 3.13	49.6 ± 4.12	71.3 ± 3.76	92.6 ± 4.50
Sling	7.0 ± 0.69	3.7 ± 0.70	10.7 ± 0.77	43.1 ± 8.77	53.8 ± 8.28	74.6 ± 8.53	94.4 ± 8.59

^a All values expressed as mean ± SE. Means for all parameters were compared using *t*-tests for paired comparisons and all differences between means were non-significant, i.e., $P > 0.05$.

^b Values represent mean duration of time elapsed between induction and beginning of Phase 1, 2, and 3, and conclusion of experiment, i.e., total.

of the descriptive statistics calculated from the physiologic data revealed that most bears showed considerable change over time in many parameters of cardiopulmonary function. Although some change was expected as a result of drug metabolism, large variation over time was not anticipated. This finding raised concern that we might not be able to distinguish between the physiologic effects of suspension and those of drug metabolism. Therefore, data collected from 11 polar bears during previous studies (Caulkett et al., 1996, 1998) were included to serve as a control group in the statistical analyses of cardiopulmonary data. Validity for this additional 'control' data was established on the basis that (1) the control group was similar in body mass to the experimental group (*t*-test, $P = 0.20$); (2) the total dose of Telazol[®] administered in both groups was similar (*t*-test, $P = 0.13$); (3) there were few significant differences in physiologic parameters (with the exception of values for SpO₂) over the initial 30 min following drug administration, i.e., period of time equivalent to anesthesia induction and Phase 1 in this study; and (4) both groups were handled and measured in similar ways, except during Phase 2 (i.e., 45 to 60 min following drug administration) where the control group remained on the ground in dorsal recumbency.

Control and experimental (i.e., net- and sling-suspension) data were compared during Phase 2 to identify cardiopulmonary effects of suspension and differences between methods of suspension. Comparison was made using two-way ANOVA for repeated measures (Zar, 1996) where treatment and time were factors. Tukey's Honestly Significant Difference (HSD) test was used to make multiple comparisons among treatments when significant differences were identified (Zar, 1996). Within experimental groups, one-way ANOVA for repeated measures (Zar, 1996) and Tukey's HSD test were used to compare cardiopulmonary function among phases. Drug metabolism rather than suspension was considered as a potential cause when significant difference occurred within an experimental group among phases, but did not

occur between the experimental and control group during Phase 2.

RESULTS

Five male and three female polar bears weighing 169.4 ± 19.4 kg (i.e., values reported as mean ± SE) were each anesthetized on two occasions. The length of time elapsed between anesthesia events was 63.0 ± 3.1 hr. Neither drug doses nor handling times differed significantly between treatments (Table 1), or between anesthesia events (paired *t*-tests, all $P > 0.25$).

Although all bears maintained heart rate and SpO₂ within the pre-defined acceptable range while suspended by net or sling, some parameters differed significantly among treatments during Phase 2 (Figs. 2, 3). Respiratory rate was significantly less in sling-suspended bears than control bears, whereas the rates for net-suspended bears were intermediate (Fig. 2). Heart rates were similar in all groups. Mean arterial pressure was significantly greater in net-suspended bears than in control and sling-suspended bears. It is important to note arterial pressures were measured at the end of expiration only, because the magnitude of respiratory waves in the arterial pressures of suspended bears was as much as 30 to 50 mm Hg. Also important to note is bears consistently showed signs of increased arousal (e.g. head, tongue, and limb movement) while suspended by net. Correlation between SpO₂ and hemoglobin oxygen saturation (SaO₂) calculated from a human hemoglobin oxygen dissociation curve was not significant ($r = -0.10$, $P = 0.35$), nor was

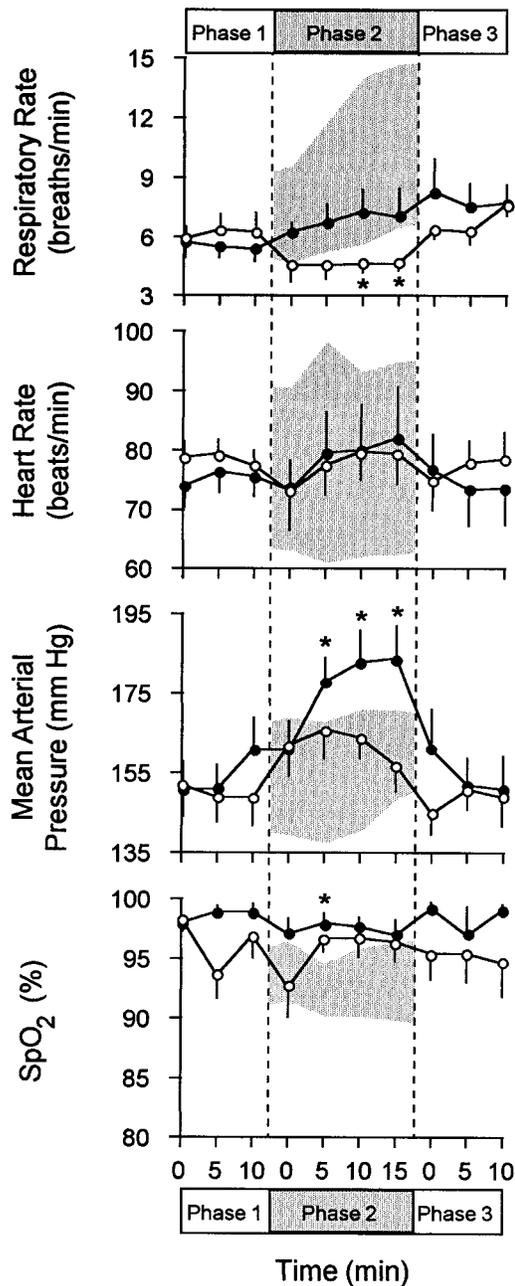


FIGURE 2. Comparison of cardiopulmonary responses of eight anesthetized polar bears between suspension by net (●) and sling (○). The mean and standard error for eight experimental bears is presented at each measurement time. Ninety-five percent confidence interval for control bears (□) is presented during Phase 2 only. Significant differences ($P < 0.05$) among treatments is indicated by an asterisk (*).

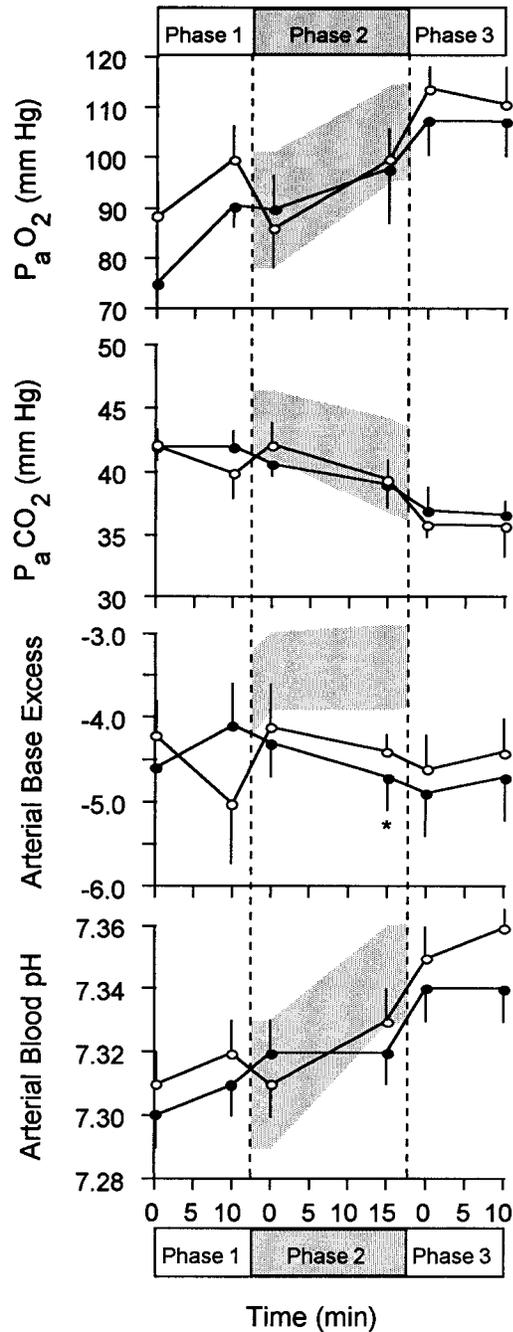


FIGURE 3. Comparison of blood gas responses of eight anesthetized polar bears between suspension by net (●) and sling (○). The mean and standard error for eight experimental bears is presented at each measurement time. Ninety-five percent confidence interval for control bears (□) is presented during Phase 2 only. Significant differences ($P < 0.05$) among treatments is indicated by an asterisk (*).

there consistent difference between SpO₂ and SaO₂ values (paired *t*-test, *P* = 0.73). Surprisingly, SpO₂ was significantly greater in experimental bears (i.e., both net- and sling-suspended) than control bears. However, similar differences also were observed between experimental and control bears during Phase 1. With regard to blood gas values, only arterial base excess differed significantly among treatments during Phase 2, being significantly greater in control than experimental bears (Fig. 3).

Within experimental groups, significant differences among phases occurred with some, but not all, parameters (Figs. 2, 3). Respiratory rate increased progressively over time in net-suspended bears, being significantly greater in Phase 3 than Phase 1 (Fig. 2). The pattern differed in sling-suspended bears where respiratory rate was significantly less during Phase 2, and did not differ between Phases 1 and 3. Mean arterial pressure was significantly greater in both experimental groups during Phase 2. The pattern of change among phases in blood gas values differed slightly between net- and sling-suspended bears (Fig. 3). In net-suspended bears, P_aCO₂ values progressively decreased over time with values in Phase 1 being significantly greater than values in Phase 3. P_aO₂ and arterial pH showed similar progressive change, but in the opposite direction, i.e., Phase 1 values < Phase 3 values. In sling-suspended bears, blood gas values in Phases 1 and 2 were similar, but differed significantly from those in Phase 3; P_aCO₂ being less, and P_aO₂ and arterial pH being greater, in the final phase. Parameters that did not differ significantly among phases were heart rate, SpO₂, and arterial base excess.

DISCUSSION

The physiologic effects of drug metabolism, and residual drug effects in individual bears between anesthesia events, could have potentially confounded data from this study. However, confidence in distinguishing the cardiopulmonary effects of suspen-

sion from those of drug metabolism was established by including control data in the statistical analysis of physiologic data. In regard to residual drug effects, Telazol® and its metabolites have been shown to be eliminated from the plasma of polar bears within 72 hr (Health and Welfare Canada, unpubl.). The duration of time between anesthesia events in our study (e.g., 2 to 3 days) was determined to meet the requirements of a concurrent wildlife management program, but was probably too short to ensure adequate drug clearance. Nevertheless, we did not observe significant differences between treatments or anesthesia events with respect to drug doses or handling times, nor were differences observed between treatment groups with respect to physiologic values during Phase 1. These findings suggest that drug residues from the first anesthesia event were not of sufficient quantity or activity to measurably affect the cardiopulmonary data recorded during the second anesthesia event.

Cardiopulmonary effects following Telazol® anesthesia the 11 polar bears used in our control group have been described previously (Caulkett et al., 1996, 1998), and are summarized as (1) initial mild decrease in respiratory rate, P_aO₂, arterial pH, and arterial base excess followed by progressive increase in all parameters; (2) initial mild increase in P_aCO₂ followed by progressive decrease; and (3) mild increase in heart rate and arterial pressures. However, in this study, effects on respiratory rate, arterial pressures, and arterial base excess differed significantly from those of control bears suggesting that suspension by net and sling caused measurable cardiopulmonary effect.

Sling suspension caused significant decrease in respiratory rate. This finding could have been attributed to measurement error as respiratory movements of sling-suspended bears were more difficult to observe than those of net-suspended bears. However, a comparative analysis of blood gas values suggests otherwise (see

Fig. 3). Normally, the mild respiratory acidosis and decrease in P_{aO_2} which immediately follows anesthesia induction with Telazol® improves progressively afterwards, as was observed with net-suspended bears. However, the blood gas values of sling-suspended bears deteriorated slightly (i.e., mild increase in P_{aCO_2} and mild decreases in P_{aO_2} and arterial pH) at the onset of Phase 2, suggesting mild respiratory depression occurred, possibly due to decreased compliance of the chest wall. Suspended bears appeared able to compensate as their blood gas values improved by the end of Phase 2 despite no change in respiratory rate.

Both methods of suspension caused increased arterial pressures. With sling suspension, increase was mild and transient. With net suspension, increase was marked and progressed throughout Phase 2, returning to baseline values only when bears were lowered to the ground. Acute hypertension could result, in part, from increased venous return as a consequence of compression by the net. Presumably, if body compression was sufficient, venous blood from skeletal muscle and, possibly, abdomen would be squeezed toward the heart. A similar mechanism underlies military anti-shock trousers, an inflatable garment used to combat shock and increase peripheral vascular resistance (Gaffney et al., 1981; Lee et al., 1983). Changes in venous return also may have contributed to generating the large respiratory waves observed in the arterial pressures of suspended bears.

The significance of acute hypertension to the health of bears suspended in nets is not clear-cut. The fact that most polar bears translocated by helicopter from Churchill over the years have survived net suspension, and returned to Churchill in subsequent years, suggests hypertension is of little consequence. However, high survivorship does not reflect morbidity; a feature often too subtle to be recognized in wild animals. The pathologic consequence of acute hypertension in polar bears is not

known, but transient acute hypertension (e.g., <10 min duration) can cause reversible and irreversible organ damage, as well as death, in humans (Sideris et al., 1988), rats (Olivetti et al., 1985), rabbits (Lacombe and Seylaz, 1984), dogs (Lamping and Dole, 1987), and pigs (Bolande et al., 1996); largely as a result of widespread microvascular injury and hemorrhage (Ault and Ellrodt, 1985; Harrison et al., 1991). Although severe respiratory depression has been postulated to be the cause of the three polar bear deaths which have occurred in nets during helicopter-assisted translocations since 1995, it appears equally as likely that death may have resulted from hypertension. In the event of a future death, necropsy and histopathology will be valuable in ascertaining the pathologic significance of hypertension.

All bears became aroused while suspended in the net, despite receiving a dose of Telazol® (range = 5.9 to 13.3 mg/kg) equal to or greater than that reported to provide full immobilization in free-ranging polar bears (Haigh et al., 1985; Stirling et al., 1989). Arousal was temporary, however, and no longer evident after bears were lowered to the ground. Such findings are consistent with a stress response but, because plasma catecholamine and cortisol concentrations were not determined, this explanation remains tentative. It is important to note, however, if there was significant stress, increased sympathetic activity could have compounded hypertension resulting from body compression, and may help to explain differences in arterial pressure response observed between the two methods of suspension, i.e., mild and transient in sling vs marked and progressive in net.

Determination of SpO_2 in polar bears was not accurate, nor was it possible to determine a correction factor for the difference between SpO_2 and SaO_2 values because the difference was inconsistent and highly variable, e.g., ranged from -23 to +19%. The accuracy of SaO_2 values was also uncertain as they were calculated

based on a human hemoglobin oxygen dissociation curve and might not have been valid for polar bears. Differences in probe recording site were the most plausible explanation for significantly greater SpO₂ values in experimental bears than control bears. A reflectance probe was inserted rectally in experimental bears, whereas recordings from control bears were made using a clip-type probe attached to the tongue. The thick, and sometimes pigmented, skin of the tongue, and its frequent movement during Telazol® anesthesia, likely inhibited light transmission resulting in lower SpO₂ values (Allen, 1992).

Surprisingly, suspension had very little effect on the blood gas values of most polar bears. Arterial base excess was less in experimental bears, indicating they were more acidotic than control bears when suspended by net or sling. However, the difference was small and not clinically significant. Although we anticipated that head positioning of net-suspended bears (see Fig. 1) would increase airway resistance and cause some respiratory depression, P_aO₂ values remained high in most bears, e.g., 9 of 16 arterial samples collected from net-suspended bears had P_aO₂ values > 90 mm Hg. This was an unexpected finding in anesthetized animals not receiving supplement oxygen and maintained in less-than-ideal body position. Nevertheless, one bear became hypoxemic (P_aO₂ < 60 mm Hg) while suspended by net, despite excellent SpO₂ values, e.g., concurrent P_aO₂ and SpO₂ values during Phase 2 were 61 mm Hg and 99% at 0 min, and 55 mm Hg and 98% at 15 min. Similarly, another bear with consistently good SpO₂ values was later found by blood gas analysis to have been hypoxemic while suspended in the sling. These findings further underscore our conclusion that oximetry does not appear to be an accurate means of determining hemoglobin oxygen saturation in polar bears. More important to the objective of this study, however, is that this finding indicates hypoxemia could

threaten the health and survival of some bears while suspended by net or by sling.

The most important conclusions from this study are that suspending polar bears by net will cause acute hypertension in most bears, and hypoxemia in some bears. These adverse cardiopulmonary effects should not be life-threatening to most bears but could impair the health of some bears, or cause death in others with pre-existing health problems. Because suspending bears by sling only mildly improved cardiopulmonary response relative to net suspension, we recommend modifying the method by which bears are suspended with the goal of reducing body compression. We suggest a light-weight, flat tray upon which an anesthetized polar bear could be positioned and suspended within a net, without compression, might offer some solution.

ACKNOWLEDGEMENTS

We gratefully acknowledge N. Campbell, C. Cassidy, M. Dyck, C. Hutchins, and C. Morran for their assistance in collecting data and analyzing blood samples. We thank the Churchill employees of the Manitoba Department of Natural Resources, particularly W. Roberts and J. Batstone, for their cooperation and assistance. We also thank F. A. Leighton and two anonymous reviewers for helpful comments on a previous draft of this manuscript. This research was funded, in part, by operating grants from the Wildlife Health Fund of the Western College of Veterinary Medicine, the Manitoba Department of Natural Resources Research Fund, and the U.S. National Science Foundation. Further financial and logistical support was received from the Churchill Northern Studies Centre (1997 Northern Research Fund), the Churchill Regional Health Authority, the Medical Research Council of Canada, and the Keewatin Region Health Centre Laboratory.

LITERATURE CITED

- ALLEN, J. L. 1992. Pulse oximetry: everyday uses in a zoological practice. *Veterinary Record* 130: 354-355.
- AULT, M. J., AND A. G. ELLRODT. 1985. Pathophysiological events leading to end-organ effects of acute hypertension. *American Journal of Emergency Medicine* 3: S10-S15.
- BOLANDE, R. P., LEISTIKOW, E. A., WARTMANN, F.

- S., III, AND T. M. LOUIS. 1996. The effects of acute norepinephrine-induced hypertension on the coronary arteries of newborn piglets. *Experimental and Molecular Pathology* 63: 87–100.
- CATTET, M. R. L., CAULKETT, N. A., POLISCHUK, S. C., AND M. A. RAMSAY. 1997. Reversible immobilization of free-ranging polar bears with medetomidine-zolazepam-tiletamine and atipamezole. *Journal of Wildlife Diseases* 33: 611–617.
- CAULKETT, N. A., CATTET, M. R. L., AND S. C. POLISCHUK. 1996. Comparative cardiopulmonary effects of medetomidine-ketamine and Telazol® in polar bears (*Ursus maritimus*). *Conference Proceedings of the American Association of Zoo Veterinarians* 1996: 394–400.
- , ———, CAULKETT, J. M., AND S. C. POLISCHUK. 1998. Comparative cardiopulmonary effects of medetomidine-zolazepam-tiletamine and Telazol® in polar bears (*Ursus maritimus*). *Conference Proceedings of the American Association of Zoo Veterinarians and American Association of Wildlife Veterinarians* 1998: 314–319.
- GAFFNEY, F. A., THAL, E. R., TAYLOR, W. F., BASTIAN, B. C., WEIGELT, J. A., ATKINS, J. M., AND C. G. BLOMQUIST. 1981. Hemodynamic effects of Medical Anti-Shock Trousers (MAST garment). *Journal of Trauma* 21: 931–937.
- HAIGH, J. C., STIRLING, I., AND E. BROUGHTON. 1985. Immobilization of polar bears (*Ursus maritimus* Phipps) with a mixture of tiletamine hydrochloride and zolazepam hydrochloride. *Journal of Wildlife Diseases* 21: 43–47.
- HARRISON, D. G., TREASURE, C. B., MUGGE, A., DELLSPERGER, K. C., AND K. G. LAMPING. 1991. Hypertension and the coronary circulation: with special attention to endothelial regulation. *American Journal of Hypertension* 4: 454S–459S.
- LACOMBE, P., AND J. SEYLAZ. 1984. Significance of the cerebrovascular effects of immobilization stress in the rabbit. *Journal of Cerebral Blood Flow and Metabolism* 4: 397–406.
- LAMPING, K. G., AND W. P. DOLE. 1987. Acute hypertension selectively potentiates constrictor responses of large coronary arteries to serotonin by altering endothelial function *in vivo*. *Circulation Research* 61: 904–913.
- LEE, H. R., BLANK, W. F., MASSION, W. H., DOWNS, P., AND R. J. WILDER. 1983. Venous return in hemorrhagic shock after application of military anti-shock trousers. *American Journal of Emergency Medicine* 1: 7–11.
- OLIVETTI, G., GIACOMELLI, F., AND J. WIENER. 1985. Morphometry of superficial glomeruli in acute hypertension in the rat. *Kidney International* 27: 31–38.
- RAMSAY, M. A., STIRLING, I., KNUDSEN, L. O., AND E. BROUGHTON. 1985. Use of yohimbine hydrochloride to reverse immobilization of polar bears by ketamine hydrochloride and xylazine hydrochloride. *Journal of Wildlife Diseases* 21: 396–400.
- SCHWEINSBURG, R. E., LEE, L. J., AND J. C. HAIGH. 1982. Capturing and handling polar bears in the Canadian arctic. *In* *Chemical immobilization of North American wildlife*, L. Nielsen, J. C. Haigh, and M. E. Fowler (eds.). Wisconsin Humane Society, Inc., Milwaukee, Wisconsin, pp. 267–289.
- SIDERIS, D. A., CHRYSOS, D. N., MALIARAS, G. K., MICHALIS, L. K., AND S. D. MOULOPOULOS. 1988. Effect of acute hypertension on the cardiac rhythm: experimental observations. *Journal of Electrocardiology* 21: 183–191.
- STIRLING, I., SPENCER, C., AND D. ANDRIASHEK. 1989. Immobilization of polar bears (*Ursus maritimus*) with Telazol® in the Canadian Arctic. *Journal of Wildlife Diseases* 25: 159–168.
- ZAR, J. H. 1996. *Biostatistical analysis*, 3rd Edition. Prentice Hall, Upper Saddle River, New Jersey, 662 pp.

Received for publication 7 October 1998.