Effect of prewarming on the body temperature of small dogs undergoing inhalation anesthesia

Clara F Rigotti, DVM, PhD; Colette T. Jolliffe, BVetMed; Elizabeth A. Leece, BVSc

Objective—To investigate whether prewarming affects body temperature of small dogs (weighing < 10 kg [22 lb]) undergoing inhalation anesthesia.

Design—Prospective, randomized, blinded clinical trial.

Animals—20 dogs weighing < 10 kg with American Society of Anesthesiologists physical status I or II.

Procedures—Baseline rectal temperature was recorded. Before IM administration of buprenorphine hydrochloride and acepromazine maleate, dogs were randomly assigned to be placed in a pediatric incubator at 33°C (91.4°F) for approximately 30 to 60 minutes (prewarming group) or to receive no prewarming (control group); subsequently, dogs underwent inhalation anesthesia with isoflurane in oxygen. Rectal, esophageal, and ambient temperatures were measured every 5 minutes from induction of anesthesia (IOA) for > 1 hour by an observer who was unaware of treatment. If a dog became hypothermic (esophageal temperature < 36°C [96.8°F]), it was withdrawn from the study. Variables of interest relating to dogs, anesthesia, temperatures, hypothermia, and study withdrawal were compared between groups.

Results—1 dog was excluded from the prewarming group after becoming excessively excited in the incubator. Between groups, age, weight, body condition score, degree of preanesthesia sedation, interval from sedation to IOA, duration of anesthesia, baseline rectal temperature, rectal temperatures immediately prior to IOA, esophageal temperature following IOA, ambient temperature during the first 70 minutes of anesthesia, esophageal or rectal temperature during the first 90 minutes of anesthesia, and incidence of hypothermia and study withdrawal (5 dogs/group) did not differ significantly.

Conclusions and Clinical Relevance—Prewarming in an incubator prior to IOA failed to improve or maintain body temperature of dogs weighing < 10 kg during inhalation anesthesia.


Perioperative hypothermia is common, occurring in 60% to 80% of human surgical patients. Redondo et al demonstrated that postanesthetic hypothermia (< 36.4°C [97.5°F]) occurs in up to 32% of canine surgical patients.

In humans, it is known that intraoperative hypothermia has negative consequences on organ function, including excessive sympathetic stimulation, interference with drug metabolism, alteration of platelet activity, inhibition of the immune system and impaired wound healing, and increased postoperative breakdown of muscle proteins. In dogs, hypothermia has been associated with a delay in clot formation, prolonged QT interval on ECG tracings, bradycardia, and prolonged recovery from anesthesia.

Core to peripheral redistribution of heat is the main cause of hypothermia (redistribution hypothermia) in humans during the first few hours of a period of anesthesia, accounting for 81% of the heat loss during the first hour and 65% of the heat loss during the first 3 hours. Prevention may be the best strategy to minimize redistribution hypothermia. Short prewarming periods have been investigated in human surgical patients, and 10 minutes of forced air warming has been found to be effective in preventing hypothermia. To our knowledge, there are no reports of clinical studies investigating the effect of prewarming in dogs and cats, and 1 experimental study found no difference between the effects of prewarming in combination with warming after IOA and those of only warming after IOA in dogs.

The objective of the study reported here was to investigate whether prewarming in an incubator after premedication affects body temperature of dogs undergoing inhalation anesthesia. The hypothesis was that prewarming would reduce the incidence or severity of hypothermia (or both) in dogs undergoing inhalation anesthesia.

Materials and Methods

Dogs—Dogs weighing < 10 kg (22 lb) with American Society of Anesthesiologists physical status I or II undergoing diagnostic imaging or minor surgical procedures...
cures (ocular surgeries, eyelid surgeries, or small cutaneous mass excision) involving inhalation anesthesia were considered for recruitment in the study. Dogs undergoing more invasive procedures involving opening of body cavities or large skin incisions, aggressive dogs, or those < 6 months old were excluded. This study was approved by the Animal Health Trust Clinical Research Ethics Committee (study No. AH08_09); study-specific owner consent was not considered necessary by the institution’s ethics committee because consent to participate in the study was considered covered by the clinic’s general client consent form.

A sample size calculation was performed following recruitment of the first 10 dogs. Seven dogs in each group (prewarming and control groups) were required for the study to have an 80% power to detect a difference (α = 0.05) in mean esophageal temperature of 1°C (1.8°F) at 30 minutes following IOA.

Experimental procedures—Twenty dogs scheduled to undergo inhalation anesthesia were randomly assigned into 2 groups (10 dogs/group) with a computer-generated randomization system. Before premedication with acepromazine maleate (0.02 mg/kg [0.009 mg/lb], IM) and buprenorphine hydrochloride (0.02 mg/kg [0.009 mg/lb], IM) and before IOA, dogs either were placed in a pediatric incubator at 33°C (91.4°F) for approximately 30 to 60 minutes (prewarming group) or were kept in their kennel for a period of similar duration (control group).

Agreement between the esophageal and rectal thermometers was tested by immersing them in a warm water bath (33.6°C [92.5°F]) at the beginning of the study. A 20- or 22-gauge IV catheter was placed in a peripheral vein of each dog, and baseline rectal temperature was recorded with a digital thermometer before premedication.

Immediately before IOA, rectal temperature was recorded and the degree of sedation was scored with a 5-point simple descriptive scale (1 to 5) by the anesthetist in charge of the case (who was unaware of each dog’s treatment), as follows: 1 = no sedation; 2 = mild sedation; 3 = moderate sedation, with possible recumbency but animal could still be aroused; 4 = heavy sedation, with the animal recumbent and difficult to arouse; and 5 = profound sedation and lateral recumbency, with no possible arousal. Precision of and agreement between the rectal and esophageal probes were checked by ensuring that they were registering the same ambient temperature before proceeding with each experiment.

Anesthesia was induced with alfaxalone injected IV to effect by an anesthetist who was unaware of each dog’s treatment. Each dog was orotracheally intubated, and anesthesia was maintained by inhalation of isoflurane in oxygen at 500 mL/kg/min (227.3 mL/lb/min) via a Mapleson D T-piece or a Humphrey ADE breathing system in the E mode. A heat and moisture exchanger was placed between the endotracheal tube and the breathing system. Hartmann solution was administered IV at a rate of 5 mL/kg/h (2.3 mL/lb/h). An esophageal temperature probe was inserted to the level of the heart (estimated by previously holding the probe along the side of the dog and measuring the distance from the tip of the nose to the point of the elbow). A rectal temperature probe was inserted approximately 4 cm into the rectum. The 2 temperature probes were connected to multivariable monitors. Anesthetic depth was assessed by evaluation of the palpebral reflexes, jaw tone, and response to surgical stimulation.

All dogs were isolated from the table surface by an absorbent pad, which consisted of a soft nonwoven cover, cellulose absorbent layer, and waterproof backing. The position of each dog varied depending on the procedure performed. No additional body warming systems were applied, although some dogs were covered by surgical drapes during the procedure at the discretion of the surgeon. Ambient, esophageal, and rectal temperatures were recorded every 5 minutes until the esophageal temperature decreased to < 36.0°C (96.8°F [hypothermia]) or until the end of the period of anesthesia (maximum duration of anesthesia, 150 minutes). At this point, the recording of the temperatures was discontinued and the dog was actively rewarmed. The interval from induction to reaching the esophageal temperature cutoff for hypothermia was recorded. During anesthesia, the following variables were monitored every 5 minutes: heart rate, respiratory rate, arterial blood pressure (noninvasive oscillometric method), end-tidal carbon dioxide tension, and arterial hemoglobin oxygen saturation (measured by pulse oximetry). Neuromuscular blockade was effected by IV administration of atracurium besylate (0.1 to 0.2 mg/kg [0.045 to 0.09 mg/lb]) for intraocular surgeries, and monitored with a peripheral nerve stimulator; intermittent positive pressure ventilation was provided.

Statistical analysis—After normality testing with the D’Agostino and Pearson omnibus test, data for the prewarming and control groups were compared by means of a t test (for normally distributed data) or Mann-Whitney U test (for nonnormally distributed data). For all comparisons, values of P ≤ 0.05 were considered significant. Rectal, esophageal, and ambient temperatures recorded during the first 90 minutes of anesthesia were compared by ANOVA; however, some data regarding ambient temperature were lost, and therefore ambient temperatures recorded during the first 70 minutes of anesthesia for each group were compared. A Fisher exact test was used to assess the proportion of dogs in each group withdrawn from the study because of hypothermia. For dogs that became hypothermic, any data collected after the point of withdrawal from the study were not analyzed. A Kaplan-Meier outcome event analysis was also used to compare withdrawal rate between the study groups. Parametric data are reported as mean ± SD, and nonparametric data are reported as median (range).

Results

Ten client-owned dogs were enrolled into each group. One dog in the prewarming group was excluded from the study because it became excessively excited after placement in the incubator.

Thus, there were 9 dogs in the prewarming group and 10 dogs in the control group from which data were collected and analyzed. Among those 2 groups of dogs, there were no differences with regard to age, weight, body condition score (assessed on a scale of 1 to 9), sedation score, time from sedation to IOA, or duration of anesthesia (Table 1). In the prewarming group, the
During the first 70 minutes of anesthesia, there was no significant difference between groups. Similarly, there was no significant difference in the elapsed time from IOA to becoming hypothermic; in the prewarming group, 5 dogs became hypothermic (at 25, 60, 65, 75, and 105 minutes after IOA), whereas in the control group, 3 dogs became hypothermic at 30, 55, and 60 minutes after IOA. The data of these dogs was included in the study until the time of withdrawal. When Kaplan-Meier outcome event analysis was performed with the point of withdrawal from the study due to hypothermia (esophageal temperature < 36.0°C) as the event, no significant (P = 0.8) differences were found between the 2 groups (Figure 3).

### Table 1—Comparison of study data including baseline rectal temperature, rectal temperature immediately prior to IOA, and esophageal and rectal temperatures immediately following IOA obtained from dogs weighing < 10 kg (22 lb) with American Society of Anesthesiologists physical status I or II that were (n = 10) or were not (10 controls) prewarmed in a pediatric incubator set at 33°C (91.4°F) for at least 30 minutes following premedication with acepromazine maleate (0.02 mg/kg [0.009 mg/lb], IM) and buprenorphine hydrochloride (0.02 mg/kg, IM).

<table>
<thead>
<tr>
<th>Variable</th>
<th>Prewarming group (n = 10)</th>
<th>Control group (n = 10)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (mo)</td>
<td>51.4 ± 24.5</td>
<td>44.7 ± 30.9</td>
<td>0.6</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>7.0 ± 2.0</td>
<td>7.5 ± 2.5</td>
<td>0.7</td>
</tr>
<tr>
<td>Body condition score*</td>
<td>5.5 (5–6)</td>
<td>6 (5–7)</td>
<td>0.6</td>
</tr>
<tr>
<td>Sedation score†</td>
<td>2 (2–3)</td>
<td>2 (2–3)</td>
<td>0.3</td>
</tr>
<tr>
<td>Time from premedication to IOA (min)</td>
<td>48 (32–45)</td>
<td>52.5 (30–60)</td>
<td>1.0</td>
</tr>
<tr>
<td>Duration of anesthesia (min)</td>
<td>110.6 ± 51.4</td>
<td>102.5 ± 57.8</td>
<td>0.8</td>
</tr>
<tr>
<td>Baseline rectal temperature (°C)</td>
<td>38.4 ± 0.7</td>
<td>38.5 ± 0.4</td>
<td>0.7</td>
</tr>
<tr>
<td>Rectal temperature prior to IOA (°C)</td>
<td>38.4 ± 0.5</td>
<td>38.3 ± 0.5</td>
<td>0.7</td>
</tr>
<tr>
<td>Rectal temperature following IOA (°C)</td>
<td>38.0 ± 0.5</td>
<td>37.4 ± 0.6</td>
<td>0.08</td>
</tr>
<tr>
<td>Esophageal temperature following IOA (°C)</td>
<td>37.8 ± 0.6</td>
<td>36.4 ± 3.1</td>
<td>0.2</td>
</tr>
</tbody>
</table>

*Body condition score was assessed on a scale of 1 to 9. †Degree of sedation was scored with a simple descriptive scale (1 to 5) by the anesthetist in charge of the case (who was unaware of each dog’s treatment). On this scale, 1 represented no sedation and 5 represented profound sedation.

Data are reported as mean ± SD or median (range).

![Figure 1—Mean ± SD ambient temperatures over a 70-minute period of anesthesia in dogs weighing < 10 kg (22 lb) with American Society of Anesthesiologists physical status I or II that were (n = 10; circles) or were not (10 [controls]; squares) prewarmed in a pediatric incubator set at 33°C (91.4°F) for at least 30 minutes following premedication with acepromazine maleate (0.02 mg/kg [0.009 mg/lb], IM) and buprenorphine hydrochloride (0.02 mg/kg, IM). Dogs were undergoing inhalation anesthesia for diagnostic imaging or minor surgical procedures; IOA was performed at 0 minutes. One dog was excluded from the study because it became overexcited during the prewarming period. The duration of prewarming ranged from 30 to 60 minutes; control dogs remained in their kennels for a period of similar duration before IOA.](image)
Discussion

In the present study, prewarming in an incubator for at least 30 minutes did not alter the body temperature of dogs < 10 kg undergoing inhalation anesthesia. This contrasts with prewarming of human patients, which has been shown to be effective in reducing the incidence of perioperative hypothermia. Reasons for the different results may include differences in pre-warming technique, temperature, or duration; the effect of drugs used; or the different methods used to measure temperature.

For the dogs of this report, the time spent in the incubator may not have been appropriate; it was decided to limit the duration of prewarming to at least 30 minutes because prolonged prewarming was considered clinically impractical. However, the duration of pre-warming of dogs ranged between 30 and 60 minutes, which may have affected the results. The time spent in the incubator may have been too short to reduce redistribution hypothermia, possibly because of the high surface area-to-volume ratio of small dogs, compared with that of humans. It is possible that starting the pre-warming procedure before premedication or continuing the procedure after IOA may be more useful for decreasing incidence of perioperative hypothermia in dogs weighing < 10 kg.

The dogs of the present study were prewarmed in an incubator set at 33°C. This incubator temperature might have been inadequate, but higher temperatures may be difficult to tolerate. One dog was excluded from the study because it became stressed during the pre-warming period. In humans, a rapid increase in skin temperature leads to discomfort and decreased tolerability of aggressive warming. In such situations, sweating by humans or panting by dogs decreases body heat content, which may reduce the efficacy of prewarming. The skin temperature causing thermal discomfort in dogs is unknown.

Owing to their small physical size and high surface area-to-volume ratio, dogs in the present study may have lost body heat very quickly once they were removed from the incubator. Insulation with blankets or bubble wrap or continuing the prewarming with heated blankets may have limited the dogs’ heat loss. Panting is often seen in dogs after administration of µ-opioid receptor agonists because these drugs act on the central thermoregulatory center to reset the body temperature to a lower level. Buprenorphine is a partial µ-opioid receptor agonist and may have this effect. Panting may have contributed to the development of hypothermia after premedication. Other drugs may also affect ther-

Figure 2—Mean ± SD esophageal (A) and rectal (B) temperatures over a 90-minute period of anesthesia in the dogs in Figure 1 that were (circles) or were not (controls [squares]) prewarmed in a pediatric incubator set at 33°C for at least 30 minutes following premedication prior to IOA performed at 0 minutes. In the prewarming group, 4 dogs became hypothermic (at 25, 60, 65, and 75 minutes after IOA), whereas in the control group, 4 dogs became hypothermic (at 20, 35 [2 dogs], and 40 minutes after IOA). 1 dog in each group became hypothermic after the 90-minute time point. The data from these dogs until the time of withdrawal were used for analysis. There was no significant (P > 0.05) difference between the groups with regard to esophageal or rectal temperature at any time point during the first 90 minutes of anesthesia. See Figure 1 for remainder of key.

Figure 3—Kaplan-Meier outcome event analysis curves for the dogs in Figure 1 that were (n = 9) or were not (controls; 10) prewarmed in a pediatric incubator set at 33°C for at least 30 minutes following premedication prior to IOA performed at 0 minutes. Dogs were undergoing inhalation anesthesia for diagnostic imaging or minor surgical procedures, the maximum duration of which was 150 minutes. For any dog, the point at which esophageal temperature decreased to < 36.0°C (96.8°F) and rewarming was initiated was defined as the outcome event. Hash marks indicate dogs withdrawn from the study because of hypothermia (5 dogs/group). The analysis revealed no significant (P = 0.8) difference between groups.

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moregulation and heat loss. Acepromazine causes vasodilation by antagonizing \( \alpha \)-adrenergic receptors and resets body temperature by affecting the thermoregulatory center. \(^{17}\) Vasodilation may increase the speed of heat gain while a dog is inside an incubator, but once the dog is removed from that unit, heat loss is more rapid because heat readily moves from the central compartment to the periphery. In the present study, the premedicants administered were standardized for all dogs to avoid any influence of drugs and anesthetic depth on body temperature. Variation in anesthetic depth may have influenced the dogs’ body temperature, with deep levels of anesthesia contributing to ongoing losses of temperature by depressing heat production and promoting heat loss. Oxygen delivery and IV fluid rates were also standardized for all dogs because cold fresh inhaled gases\(^{18}\) and fluids administered IV\(^{19}\) decrease body temperature in anesthetized animals. Atracurium is a nondepolarizing neuromuscular blocking agent that may contribute to the development of hypothermia by decreasing muscle tone; however, the proportion of dogs receiving atracurium in the prewarming and control groups of the present study did not differ. Ambient temperature is important in determining heat balance in an anesthetized patient because heat flow is dependent on the thermal gradient between the patient and the environment. Human infants become hypothermic when the ambient temperature is < 26°C (78.8°F). \(^{20}\) In animals, the ideal ambient temperature to maintain normothermia during anesthesia has not been described, to our knowledge. However, in 1 study, \(^{21}\) anesthetized dogs became hypothermic when the ambient temperature was < 27°C (80.6°F). In the present study, mean ambient temperature was < 24°C (75.2°F) for both groups, which could cause considerable heat loss from the dogs to the environment. During the period of anesthesia in the present study, active warming was started and study participation was discontinued when a dog's esophageal temperature decreased to < 36°C. It was considered ethically unacceptable to allow any dog's body temperature to decrease to < 36°C without active rewarming. This shortened study participation for 5 dogs in each study group, thereby reducing the amount of data available for analysis.

All dogs in the present study that were undergoing surgery underwent minor surgical procedures. The reason for excluding invasive surgeries was to limit the effects of surgical preparation and evaporation from the surgical field on body temperature. Clinical and experimental studies in humans\(^{22}\) and in dogs\(^{23}\) have revealed that esophageal temperature is a reliable indicator of mean body heat content, but esophageal temperature can vary markedly depending on the site at which the measurement is obtained. Whitby and Dunkin\(^{24}\) reported the lower fourth of the esophagus as both the warmest and most reliable measurement site in humans. In the present study, the esophageal temperature probe was inserted to the level of the heart, as estimated by holding the probe along the side of the dog. Position of the probe was not confirmed radiographically; hence, slight variations in probe position could have affected temperature measurements.

In the present study, rectal temperature at the time of premedication was measured with a digital rectal thermometer. After IOA, rectal and esophageal temperatures were measured with the same type of probe; however prior to IOA, a different type of probe was used, which may have affected the repeatability of measurements. Furthermore, there was no gold standard against which to compare the temperature probes. These factors may have contributed to the lack of significant differences between the 2 groups. However, based on the data obtained in this study, prewarming in an incubator for at least 30 minutes after premedication was not an adequate method to reduce the development of hypothermia in small dogs undergoing inhalation anesthesia.

References


From this month’s AJVR

Temporospatial and kinetic gait variables of Doberman Pinschers with and without cervical spondylomyelopathy

Carolina G. D. Lima et al

Objective—To characterize and compare gait variables in Doberman Pinschers with and without cervical spondylomyelopathy (CSM).

Animals—18 Doberman Pinschers (9 clinically normal dogs and 9 CSM-affected dogs).

Procedures—A neurologic examination was performed on all dogs. The diagnosis of CSM was confirmed with MRI. Temporospatial and kinetic gait variables were measured by use of a pressure-sensitive walkway. Temporospatial variables evaluated included stance phase duration, swing phase duration, gait cycle duration, stride length, and gait velocity. Kinetic variables evaluated included peak vertical force and vertical impulse. Random-effects linear regression was used to determine the difference between CSM-affected and clinically normal dogs for each of the 7 variables.

Results—Values for temporospatial variables were significantly smaller in the thoracic limbs of CSM-affected dogs, compared with values for the thoracic limbs of clinically normal dogs. For the kinetic variables, peak vertical force was significantly higher in the thoracic limbs than the pelvic limbs for all dogs. Vertical impulse values were higher in the thoracic limbs than the pelvic limbs. There were significant differences in mean vertical impulse between the thoracic and pelvic limbs for both groups.

Conclusions and Clinical Relevance—In this study, significant differences in temporospatial variables were identified between the thoracic limbs of clinically normal and CSM-affected dogs, with the values being smaller for the CSM-affected dogs than for the clinically normal dogs. A pressure-sensitive walkway may provide a valid, practical option for rapid, objective assessment of gait and response to treatment in dogs with CSM. (Am J Vet Res 2015;76:848–852)