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Summary: Surface-induced hypothermia is used to protect tissues from ischemic events during surgery. In a review of 19 clinical cases in dogs, the technique was used to enable intracardiac surgery (4 dogs) and to facilitate removal of extensive thoracic or abdominal masses (15 dogs). For 16 dogs (84%), anesthesia was induced with an opioid/benzodiazepine combination and maintained with a balanced technique by use of an opioid, a neuromuscular blocking agent, and isoflurane in oxygen. Dogs were cooled in an ice bath to a mean esophageal temperature of 27.8 ± 1.4 °C. Mean anesthesia time was 4.04 ± 1.37 hours. Hypothermic-induced adverse effects, such as increased blood viscosity, increased myocardial irritability, and shivering, were managed by hemodilution, manipulation of acid-base balance, and administration of opioid and neuromuscular blocking agents. Complications requiring treatment included severe hypotension (74%), arrhythmias (47%), hypoxemia (42%), and acidemia (58%). Six dogs (32%) went into cardiac arrest and all were successfully resuscitated once the surgical procedure was completed. One dog was euthanized during surgery, another died after surgery, and the 17 remaining dogs (90%) were discharged from the hospital to their owners. The technique appears beneficial in selected cases to decrease the morbidity and mortality associated with the risk of prolonged ischemia and life-threatening hemorrhage.

Surface-induced hypothermia was originally developed to protect body tissues from ischemia during circulatory arrest in people undergoing intracardiac surgery. The technique has largely been replaced by cardiopulmonary bypass, but is occasionally used for pediatric patients because of size-related technical difficulties associated with bypass. Cardiopulmonary bypass is rarely performed in veterinary medicine because it is technically challenging and prohibitively expensive. In selected cases, where circulatory arrest of short duration is required either to facilitate intracardiac surgery or avoid life-threatening hemorrhage, surface-induced hypothermia may be a potential alternative to prevent ischemia-induced complications.

The technique of surface-induced hypothermia has been reported only a few times in veterinary practice. In 1971, and in 1984, it was successfully used to enable surgical correction of interventricular septal defects in 2 dogs. In 1973, the technique was used in 25 dogs undergoing deliberate cardiac arrest for various types of intracardiac surgery. In that study, only 9 dogs had cardiovascular disease and 4 of them died within 24 hours after surgery.

The purpose of the study reported here was to document the anesthetic management, hypothermic protocol, complications, and outcome in 19 dogs with clinical cases for which surface-induced hypothermia was used to protect against prolonged periods of tissue ischemia. In 4 dogs, it was used in the traditional manner, to enable intracardiac surgery. In the remaining 15 dogs, it was used to facilitate removal of large thoracic or abdominal masses.

Materials and Methods

Medical records from the veterinary medical teaching hospital, for the period September 1987 to December 1989 were reviewed, and the records of all cases in which surface-induced hypothermia was used were retrieved. Nineteen cases were found, and the age, weight, primary disease, and secondary diseases of the dogs at time of admission were recorded. A thorough medical evaluation including a CBC and biochemical analysis prior to surgery was done on all dogs. Twelve hours before surgery, food and water were withheld, and 20 minutes prior to induction, preanesthetic medication (meperidine hydrochloride, oxymorphone hydrochloride, atropine sulfate, or glycopyrrolate) was administered SC (Table 1). The surgical site was clipped, and 2 to 3 peripheral venous catheters...
Table 1—Anesthetics and fluid requirements during surgery of 19 dogs undergoing deliberate surface-induced hypothermia

<table>
<thead>
<tr>
<th>Stage or requirement of anesthesia</th>
<th>Drug/therapy</th>
<th>No. of dogs</th>
<th>Dose range (mg/kg of body weight + route)</th>
<th>Dose mean ± SD (mg/kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preanesthetic medication</td>
<td>Procainemethine</td>
<td>19</td>
<td>5 to 10 min</td>
<td>NC</td>
</tr>
<tr>
<td></td>
<td>Oxymorphone</td>
<td>11</td>
<td>0.03 to 0.07, SC</td>
<td>0.05 ± 0.01</td>
</tr>
<tr>
<td></td>
<td>Midazolam</td>
<td>9</td>
<td>0.01 to 0.04, SC</td>
<td>0.03 ± 0.01</td>
</tr>
<tr>
<td>Induction</td>
<td>Oxymorphone</td>
<td>9</td>
<td>0.06 to 0.20</td>
<td>0.14 ± 0.05</td>
</tr>
<tr>
<td></td>
<td>Fentanyl</td>
<td>7</td>
<td>0.005 to 0.025</td>
<td>0.018 ± 0.006</td>
</tr>
<tr>
<td></td>
<td>Ketamine</td>
<td>1</td>
<td>4.6</td>
<td>ND</td>
</tr>
<tr>
<td></td>
<td>Midazolam</td>
<td>1</td>
<td>3.9</td>
<td>ND</td>
</tr>
<tr>
<td></td>
<td>Diazepam</td>
<td>17</td>
<td>0.091 to 0.09</td>
<td>0.25 ± 0.14</td>
</tr>
<tr>
<td>Maintenance</td>
<td>Isoflurane</td>
<td>19</td>
<td>ND</td>
<td>ND</td>
</tr>
<tr>
<td></td>
<td>Fentanyl infusion</td>
<td>7</td>
<td>0.6 to 0.7 µg/kg/min</td>
<td>0.7 ± 0.0</td>
</tr>
<tr>
<td></td>
<td>Oxymorphone bolus</td>
<td>10</td>
<td>0.025 to 0.5</td>
<td>NC</td>
</tr>
<tr>
<td></td>
<td>Atropine</td>
<td>8</td>
<td>0.05, then 0.12</td>
<td>NC</td>
</tr>
<tr>
<td></td>
<td>Dopamine infusion</td>
<td>5</td>
<td>2.5 to 10 µg/kg/min</td>
<td>NC</td>
</tr>
<tr>
<td></td>
<td>Epinephrine infusion</td>
<td>1</td>
<td>NC</td>
<td>ND</td>
</tr>
<tr>
<td></td>
<td>Nitroglyceride infusion</td>
<td>1</td>
<td>NC</td>
<td>ND</td>
</tr>
<tr>
<td></td>
<td>Phenylephrine infusion</td>
<td>1</td>
<td>NC</td>
<td>ND</td>
</tr>
<tr>
<td></td>
<td>CaCl₂ bolus for hypertension</td>
<td>1</td>
<td>NC</td>
<td>ND</td>
</tr>
<tr>
<td></td>
<td>Lidocaine infusion</td>
<td>3</td>
<td>NC</td>
<td>ND</td>
</tr>
<tr>
<td></td>
<td>Midazolam bolus</td>
<td>1</td>
<td>NC</td>
<td>ND</td>
</tr>
<tr>
<td>Fluids</td>
<td>Lactated Ringer's solution</td>
<td>19</td>
<td>0.94 to 15 ml/kg/h</td>
<td>5.37 ± 4.22</td>
</tr>
<tr>
<td></td>
<td>Fresh blood</td>
<td>13</td>
<td>1.0 to 57</td>
<td>16.0 ± 17.80</td>
</tr>
<tr>
<td></td>
<td>Autotransfusion</td>
<td>3</td>
<td>ND</td>
<td>ND</td>
</tr>
<tr>
<td></td>
<td>Fresh frozen plasma</td>
<td>4</td>
<td>3.3 to 20</td>
<td>2.26 ± 5.58</td>
</tr>
<tr>
<td></td>
<td>Dextran 70</td>
<td>16</td>
<td>100 mg/kg/CC ≤ 18 C</td>
<td>3.52 ± 4.71 ml/min</td>
</tr>
<tr>
<td></td>
<td>Sodium chloride</td>
<td>9</td>
<td>0.17 to 1.29</td>
<td>0.58 ± 1.09</td>
</tr>
<tr>
<td></td>
<td>Packed RBCs</td>
<td>1</td>
<td>150 ml/6.4 kg</td>
<td>ND</td>
</tr>
</tbody>
</table>

*Route—ei unless otherwise stated.

No = not determined; and NC = not calculated.

were placed for fluid and drug administration. A Doppler crystal was placed over the median artery, together with a forelimb inatable cuff, provided indirect measurement of blood pressure during the awake state and immediately after induction. Limb leads were placed to continuously display a lead-II ECG during induction. Oxygen was administered via face mask to all dogs for 5 minutes and then anesthesia was induced (Table 1; oxymorphone and diazepam, fentanyl citrate and diazepam, fentanyl and midazolam hydrochloride, ketamine and diazepam, thiamyl sodium and diazepam, or isoflurane). The trachea was intubated and anesthesia was maintained with isoflurane in 100% oxygen. After induction, an esophageal stethoscope and an esophageal ECG were placed and ECG limb leads were removed. The dorsal pedal artery was catheterized for continuous direct monitoring of arterial blood pressure, using a pressure transducer, and to allow collection of blood samples for arterial blood gas, pH, PCV, total plasma protein (TPP), plasma sodium, potassium, and ionized calcium determinations. Rectal and esophageal temperatures were measured simultaneously via a thermistor. Ventilation was controlled by use of pressure-limited ventilator and tidal volume measured with a respirometer. In some dogs, the following measurements were made: central venous pressure from a jugular central venous catheter and a pressure transducer, lingual arterial oxygen saturation by pulse oximetry, and end-tidal carbon dioxide tension by capnography. A high-frequency jet ventilator was used on 1 dog when standard mechanical ventilation was inadequate.

Following induction, instrumentation, and preparation of the surgical site, dogs were transported to the surgical suite and placed directly into a crushed ice-water immersion bath. The ice water slurry was continuously poured over the body of the dog, but head and limbs were not submerged. Potential maximal tissue ischemic time from vascular occlusion or arrest, as estimated by the surgeon, was used to determine the target temperature for each dog. The approximate safe circulatory occlusion times, in minutes, for temperatures 37 to 32 C, 32 to 28 C, 28 to 18 C, and 18 to 4 C are reported as 4 to 10, 10 to 16, 16 to 60, and 60 to 90, respectively. During cooling, tidal volume was maintained at pre-immersion values by adjusting the inspiratory pressure on the ventilator. At this time, most dogs received a plasma volume expand-

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6Parks Medical Electronics Inc, Cherry Hill, NJ.
7Vittec, Spacelabs, Hillsborough, Ore.
8Transpak II, Sorenson Research, Salt Lake City, Utah.
9ABL 30 blood gas analyzer, Radiometer, Copenhagen, Denmark.
10Synergy temperature monitor, American Pharmaceul Co, American Hospital Supply Corp, Valencia, Calif.
11Bird Mark 7, Bird Corp, Palm Springs, Calif.
12Mark 14, Medical Marketing Caremed Inc, San Jose, Calif.
13Model 503, Criticare Systems Inc, Waunakee, Wis.
14CO₂ monitor, Puritan-Bennett Corp, Los Angeles, Calif.
15SAV 6, Silver Medical Co, Tokyo, Japan.
er at 100 mg/kg of body weight, IV, for each degree the target temperature was below 37 C. Dogs were removed from the ice bath when core temperature was 2 to 3 degrees higher than target temperature and surgical site was disinfected. A temperature-controlled circulating water pad was placed under the dog and adjusted to maintain target temperature. Some of the dogs were paralyzed with a muscle relaxant, atracurium besylate (Table 1), and degree of neuromuscular blockade was monitored by a peripheral nerve stimulator placed over the facial or ulnar nerve. An opioid, a benzodiazepine, or both, were often administered to supplement the inhalant anesthetic agent (Table 1). During surgery, an IV balanced electrolyte solution was also administered and, in some cases, compatible cross-matched fresh blood and/or fresh frozen plasma (FFP) were also administered (Table 1). Management of intraoperative complications included administration of atropine, dopamine hydrochloride, phenylephrine hydrochloride, epinephrine, sodium nitroprusside, lidocaine hydrochloride, furosemide, mannitol, calcium chloride, potassium chloride, and sodium bicarbonate (Table 1).

The decision for vascular occlusion was usually made during surgery, and if it was decided to induce cardiac arrest, a cardioplegic solution was used to arrest the heart. Following either deliberate or noninduced arrest, all fluid and anesthetic administration was stopped and controlled ventilation discontinued. Continuous positive airway pressure was administered at 2 to 5 cm H2O during the arrest, if it did not limit surgical visibility. Dogs were resuscitated by use of standard cardiopulmonary resuscitation, including ventricular defibrillation when required.

After completion of most of the surgical procedure, dogs were slowly rewarmed by increasing the temperature of the water circulating pad and flushing warm saline solution into the exposed body cavity. Dogs were not moved to the intensive care unit (ICU) until esophageal temperature reached a minimum of 32 C.

In the ICU, dogs were warmed to normothermia with heating pads and heat lamps. Dogs remained in the ICU for a minimum of 24 hours, with ventilatory and cardiovascular support given as required.

Results

Dog status—All results are reported as mean ± SD. Dogs in this study were 7.5 ± 4.1 years old and weighed 23 ± 11 kg. Surgery in-

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[Table 1: Unavailable due to formatting issues]

Anesthetic duration—Mean anesthetic time, from induction to the end of surgery, was 4.04 ± 1.37 hours (range, 2.00 to 6.58 hours).

Cooling—Mean time to reach target temperature was 28.47 ± 9.88 minutes. Mean esophageal temperature during surgery was 27.8 ± 1.4 C.

Intraoperative physiologic values—Hypothermia induced bradycardia in all dogs, relative to their normothermic heart rate. The mean intraoperative heart rate was 86.1 ± 16.9 beats/min with 42% of the dogs having a mean heart rate ≤ 80 beats/min.

Mean arterial blood pressure during hypothermia ranged from 45 to 90 mm of Hg (69.7 ± 11.9 mm of Hg). Only 2 dogs had a mean blood pressure > 85 mm of Hg. One other dog, with pheochromocytoma, had a mean blood pressure of 77 mm of Hg, with episodic bouts of hypertension (mean, 224 mm of Hg) and hypotension (mean, 46 mm of Hg).

Minute ventilation was adjusted to achieve an arterial carbon dioxide partial pressure ≤ 40 mm of Hg and an arterial pH ≥ 7.40. Mean individual intraoperative ventilatory rates ranged from 6 to 40 breaths/min (12.4 ± 3.1 breaths/min).

Surgical manipulation and cardiac arrest—Aorta and vena cava were cross-clamped in 21% (4/19) of the dogs, whereas only the caudal vena cava was cross-clamped in an additional 21% (4/19) of the dogs. One dog required aortic occlusion only. For these 9 dogs, mean aortic occlusion time was 20 ± 8 minutes, and mean vena cava occlusion time was 31 ± 42 minutes.

Six dogs (32%) had cardiac arrest. In 1 dog, sinoatrial arrest was induced by use of cardioplegic solution following vascular occlusion. Noninduced cardiac arrest (ventricular fibrillation in 2 dogs and sinoatrial arrest in 3 dogs) developed in 5 dogs. In 1 dog, it occurred during surgery, whereas in the...
other 4 dogs, it occurred following vascular occlusion. Mean arrest time, from initial arrest to start of resuscitation, was 35 ± 17 minutes.

Resuscitation—All 6 dogs that had cardiac arrest were successfully resuscitated. Time from first resuscitation attempt to successful cardioversion ranged from 10 to 110 minutes (35.7 ± 37 minutes). Before resuscitation, all air was removed from opened vessels and heart chambers to reduce aspiration of air emboli. Intracardiac or central venous epinephrine injections were administered in 4 dogs (67%) and atropine was administered to 1 dog (17%). Calcium chloride was administered to 2 dogs (33%) for inotropic support. Sodium bicarbonate was administered to correct metabolic acidosis whenever plasma bicarbonate concentration was ≤ 18 mmol/L. All dogs required defibrillation (3 ± 1 times). Esophageal temperature at the start of resuscitation was 26.2 ± 3.1 C.

After resuscitation, 1 dog required an epinephrine infusion to prevent recurrence of sinoatrial arrest and to provide vasopressor support. One other dog was given a dopamine infusion for inotropic support and a lidocaine infusion for treatment of ventricular tachycardia. The remaining dogs that had cardiac arrest did not have any hemodynamic complications following resuscitation.

Complications—Excluding the 6 dogs that had cardiac arrest, the most common complications during surgery included hypotension (74%), arrhythmias (42%), hypoxemia (42%), and acidemia (58%). Treatment of these complications varied depending on the proposed cause, the severity of the problem, and the response of the dog to treatment.

Of the dogs treated for hypotension, 26% had an improvement in blood pressure when the delivery of anesthetic concentration and/or the inspiratory pressure were decreased, continuous positive airway pressure was discontinued, or a fluid bolus was given. The remainder of the dogs treated for hypotension were unresponsive to these manipulations and were treated with a dopamine infusion. Twenty-one percent of the dogs were further treated with calcium chloride boluses when dopamine did not improve blood pressure. Hypotension and hypertension were observed in the dog with pheochromocytoma. Hypotension was treated with a phenylephrine infusion, and episodes of hypertension were controlled with a nitropussu infusion.

Premature ventricular contractions or paroxysmal ventricular tachycardia were the most common arrhythmias observed (70%). Forty percent of dogs with these arrhythmias were treated with lidocaine boluses or infusion when hemodynamic depression was considerable.

Seventy-eight percent of the hypoxemic dogs were treated with continuous positive airway pressure. One dog became severely hypoxemic when major airways were transected while a large pulmonary tumor was removed. High-frequency ventilation of one bronchus and simultaneous endobronchial intubation of the other bronchus with rigorous manual hyperventilation were attempted to increase oxygenation.

For those dogs with metabolic acidosis, 80% were treated by administration of sodium bicarbonate. Severe respiratory acidosis was found in 1 dog when the thorax was closed without proper evacuation of the trapped air. This dog responded after surgery to oxygen cage therapy to alleviate hypoxemia.

Other complications were also observed. Hypocalcemia in 1 dog was treated with calcium chloride given iv and hypokalemia in 1 dog was treated with potassium chloride given iv. Potential renal insufficiency, as evaluated by observing a small bladder size and low urine production during surgery, was found in 2 dogs (11%). One was treated with mannitol and the other was treated with furosemide.

Extensive hemorrhage during surgery was a complication in 2 dogs. One dog had massive, temporarily uncontrollable blood loss during removal of a large tumor involving the liver and spleen and required 57 ml of blood/kg and 4.6 ml of FFP/kg to maintain blood pressure. The other dog was given 50 ml of blood/kg and 20 ml of FFP/kg during the removal of a heart base tumor to maintain adequate PCV and TFP attributable to intermittent but extensive blood loss.

Rewarming—Warm saline solution flushes into the open body cavity speeded rewarming, but complications developed in 3 dogs (16%). In 2 dogs, ventricular arrhythmias developed but stopped when the flushes were discontinued. In the third dog, ventricular fibrillation occurred when the warm flush was administered, so mechanical defibrillation was required.

Postoperative intensive care—Mean duration in ICU was 2.4 ± 1.9 days (range, 1 to 9 days). Immediate postoperative care included rewarming to normothermia, respiratory support, and cardiovascular monitoring. Time to normothermia (38 C) ranged from 1 to 12.8 hours (4.8 ± 3.4 hours).

All dogs required some type of respiratory support after surgery. Sixty-one percent of dogs received assisted or controlled mechanical ventilation to prevent hyperventilation. In those dogs, the inspired oxygen concentration was altered as necessary to prevent hypoxemia. Tracheostomies were performed in 2 dogs because of anticipated prolonged mechanical ventilation. One of those dogs (interventricular septal defect repair) was mechanically ventilated for 20.7 hours. Excluding this dog, the mean time for ventilatory support was 3.2 ± 2.0 hours. The remaining 39% of dogs were breathing spontaneously on arrival in the ICU, but required supplemental oxygen via a Bain circuit, if
still intubated, or nasal insufflation to maintain adequate oxygenation.

Blood pressure and ECG were continuously monitored in the ICU. Fifty percent of dogs had postoperative arrhythmias. Premature ventricular contractions were the most common abnormality (67%) and rarely required treatment. The dog that required prolonged ventilatory support also required vasoressor support. Two other dogs were given low doses of dopamine to improve renal blood flow.

**Outcome**—One dog was euthanized during surgery because of an inoperable abdominal tumor with widespread metastases. The dog that required ventilatory and circulatory support developed severe arrhythmias and multiple organ failure, and had cardiac arrest 3 days after surgery. The remaining 17 dogs (90%) were discharged to their owners.

One dog that had massive hemorrhage requiring blood transfusion during surgery was euthanatized 6 days after surgery because of uncontrollable autoimmune hemolytic anemia. One dog died 5 days after surgery because of unknown causes, and another dog that was doing well at the 1 month follow-up evaluation was euthanatized 6 months later for unknown reasons. One dog that had no visible lesions during surgery died of primary liver disease 7 months after surgery.

Thirteen dogs (72%) are potentially still alive, although 7 of these dogs have been lost to follow-up evaluation. Follow-up evaluation of the remaining 6 dogs ranges from 5 to 26 months (13 ± 8.9 months).

**Discussion**

Surface-induced hypothermia was used in these 19 dogs for its protective effects against ischemic damage. In dogs, the technique enabled intracardiac surgery to be undertaken without the need of cardiopulmonary bypass, and in 15 dogs, it facilitated removal of extensive thoracic or abdominal masses. In these dogs, potential was high for ischemic damage because of vascular occlusion or hemorrhage. The CNS will tolerate an ischemic episode for only 5 to 6 minutes at normothermia before irreversible damage occurs, but this tolerance time will double for each 5°C the tissues are cooled. Approximate safe periods of total circulatory arrest at various temperatures have been reported, but these values are still disputed, and tissue damage may occur at any temperature.

Tissue protection from ischemia increases as temperature decreases because of a proportional decline in cellular metabolism and oxygen consumption. During periods of low flow or cardiac arrest, this lowered oxygen requirement allows more tissue to survive even though minimal oxygen is available. However, it is important to maximize oxygen delivery to the tissues prior to the ischemic episode by manipulations of perfusion and oxygen content.

**Anesthetic protocol**—Opioids were chosen for premedication because they provided adequate sedation with minimal cardiovascular depression, allowed tolerance of the oxygen mask, and decreased both the stress of instrumentation and the amount of induction agent required. Anticholinergics were used to decrease bradycardia that was observed during opioid induction techniques except in cases where the dog's cardiovascular status contraindicated their use, such as dogs with tachycardia.

An opioid-benzodiazepine combination was the most common induction protocol. Opioids were used to maximize cardiovascular stability and analgesia, decrease shivering, and allow a smooth transition into maintenance anesthesia. Shivering must be prevented during the cooling and rewarming phases of hypothermia because it increases metabolic oxygen requirements, increases myocardial work, prolongs the cooling procedure, and creates temperature gradients. Temperature gradients potentiate ischemic damage, lead to cardiovascular instability and fibrillation, and are the most common cause of postoperative organ dysfunction.

The benzodiazepines, midazolam or diazepam, were used for muscle relaxation, to shorten the induction time and decrease the amount of opioid required for intubation. Decreasing the amount of opioid reduced the potential for bradycardia and respiratory depression commonly observed with those agents.

Other induction agents were used in a few cases, on the basis of dog cooperation and the anesthesiologist's discretion. Thiopental is often advocated in people with hypothermia for its cerebral sparing effect and decreasing intracranial pressure. Most of our dogs, however, had poor cardiovascular status, questionable organ perfusion, and altered liver function. Thiopental was, therefore, used in only 1 dog; the overwhelming beneficial effects of the opioids made them the best induction agents.

Hypothermia will prolong drug action by slowing liver metabolism and by decreasing rate of elimination. In addition, anesthetic requirements are lowered as the dog cools. Thus, minimal amounts of balanced anesthesia were required, and anesthesia in most dogs was maintained by use of an inhalational anesthetic, an opioid, and a neuromuscular blocker.

Isoflurane was chosen because it has less arrhythmogenic potential, less direct myocardial depressant effects, and more peripheral vasodilating effects than other inhalational anesthetics. These cardiovascular effects are important because hypothermia decreases cardiac output, increases myocardial irritability, and causes vasoinstriction.
Opioids were used during surgery for their potent analgesic properties and cardiovascular stability. They decreased the amount of isoflurane needed for anesthesia, and this was beneficial in dogs sensitive to isoflurane's vasodilating and myocardial depressant effects. Fentanyl infusions provided a more even plane of anesthesia than the intermittent oxymorphone boluses, although both methods were acceptable. The opioids augmented bradycardia normally observed during hypothermia, and during surgery, this provided easier surgical manipulation of the heart.

Postoperative advantages for using opioids during surgery also existed. Their slower metabolism prolonged their beneficial effects and therefore provided both postoperative analgesia and sedation and also made dogs more tolerant of the support and monitoring equipment. Also, opioids given during surgery decreased the likelihood of postoperative shivering during rewarming.

Atracurium was considered the ideal neuromuscular blocking agent because recovery of neuromuscular function is independent of dose or biotransformation and depends only on temperature and pH. During hypothermia, atracurium's half-life is twice as long at 25 °C than at 37 °C, so infusion rates must be slowed or discontinued to retain the first twitch in the train-of-four peripheral nerve stimulation. Boluses were given in either smaller amounts or with longer intervals between injections. On rewarming of the dog, atracurium was rarely reversed but allowed to degrade.

Cooling time depended on the dog's size, ratio of dog's surface area to surrounding ice water, amount of hair shaved, and length and type of remaining hair. The correct size of water tub was essential to allow even cooling without the risk of completely submerging the dog under water. Core temperature lags behind the temperature of more superficial adipose tissue and muscle. When active cooling stops, temperature equilibration occurs with heat from the warmer core tissues being lost to the superficial layers. For this reason, dogs routinely cooled an additional 3 to 4 °C after being removed from the ice bath. This afterdrop must be taken into account to avoid letting the dog's temperature decrease below target temperature. Rectal and esophageal temperatures were monitored as a guide that even cooling was being provided.

**Management during surgery**—As the heart cools, myocardial irritability increases; arrhythmias are common below 28 °C, and ventricular fibrillation or asystole occurs below 20 °C.8,14,15 Thus, the heart is more susceptible to fibrillation because of factors such as hypoxia, hypercapnia, acidemia, or patient movement, and all these factors must be minimized. The heart may be protected from ventricular fibrillation if pH is slightly alkaline,16 and in our dogs, pH was maintained ≥ 7.45 by vigorous ventilation to maintain arterial carbon dioxide tension < 35 mm of Hg, and sodium bicarbonate administration if bicarbonate < 18 mmol/L.

Substantial vasoconstriction occurs with decreasing temperature, causing large increases in systemic vascular resistance and decreases in tissue blood flow. This decreases oxygen transport to the tissues. The PCV also increases because of a loss of plasma fluid into the extracellular space and causes an increase in blood viscosity.17,18 Increased blood viscosity promotes RBC sludging, further increases systemic vascular resistance, and decreases microcirculatory perfusion. Hemodilution is advocated to decrease viscosity, improve flow, and increase oxygen transport to the tissues.19 In our series, dogs were initially hemodiluted with dextrans during the cooling phase and were then administered crystalloids, blood or FFP to maintain the PCV between 18 and 24% and a TPP < 2.5 mg/dl. Dextrans were used as the initial hemodiluting fluid because they have been shown to provide oncotic pressure for better maintenance of plasma volume, decrease platelet aggregation for prevention of microthrombi, and improve survival.19

Hypothermia causes a decrease in cardiac output, heart rate, and systemic blood pressure,1,15,20 and an increase in systemic vascular resistance, central venous pressure,8,21 and pulmonary vascular resistance.22 The decrease in cardiac output and heart rate is proportional to the decrease in oxygen consumption.20,23 The decrease in cardiac output is primarily attributable to bradycardia and increased systemic vascular resistance, because stroke volume and myocardial contractility initially remain unchanged.21

Heart rate and blood pressure were, therefore, tolerated at lower values in our dogs than during normothermia, assuming that tissue oxygenation was still adequate. During hypothermia, mean systemic blood pressure remains unchanged until 30 °C, when it then gradually decreases as temperature decreases. Our mean arterial blood pressure appeared adequate (70 ± 12 mm of Hg), but treatment for hypotension was initiated whenever the mean blood pressure decreased < 40 mm of Hg. Fluid loading and decreasing anesthetic level were initial treatments, followed by administration of inotropic agents in nonresponsive dogs. Low doses of dopamine (3 to 5 μg/kg/min) were tried initially to increase preload and provide inotropic support. If blood pressure remained low, the dose was increased to also cause vasoconstriction (7 to 10 μg/kg/min). Because of the potential of already high systemic vascular resistance, vasoconstrictors were considered contraindicated as a first choice in treating hypotension because they might increase blood pressure at the expense of further decreasing tissue perfusion.

Hypothermia decreases carbon dioxide production by slowing cellular metabolism. During spontaneous respiration, the minute respiratory
volume decreases and ventilation stops completely at about 23 C. Controversy has arisen concerning the most appropriate minute ventilation for patients because some believe that the pH and P\textsubscript{aCO\textsubscript{2}} at normothermia are also the appropriate pH and P\textsubscript{aCO\textsubscript{2}} during hypothermia. This implies that blood gas values should be corrected for the patient's temperature prior to interpretation. Overwhelming evidence, however, now indicates that regardless of the patient's temperature, a pH of 7.4 or slightly greater and a P\textsubscript{aCO\textsubscript{2}} of \leq 40 mm of Hg causes the least myocardial depression, the most hemodynamic stability, and improves surgical outcome after cardiac arrest. Our blood gas results were, therefore, interpreted at 37 C, directly from the machine, and analyzed without correcting for the dog's temperature. The desired pH and P\textsubscript{aCO\textsubscript{2}} were achieved by adjusting ventilation to maintain the dog's normothermic minute ventilation. Thoracic wall compliance and minute ventilation varied substantially during cooling because of the crushed ice and water alternately being poured onto the thorax and abdomen. When a pressure-limited ventilator was used, breath-to-breath adjustments of ventilator settings were needed at that time, as well as frequent monitoring during surgery, to maintain stable acid-base status during changing surgical manipulations. Carbon dioxide values also determine cerebral blood flow, but the effect of hypothermia on cerebral autoregulation has not yet been determined. In our study, no apparent neurologic defects developed, using the aforementioned criteria for P\textsubscript{aCO\textsubscript{2}} and acid-base regulation. Other investigators have also reported no adverse neurologic sequela when they used this method of acid-base regulation. Hypoxemia was a frequent complication requiring treatment. Lung function was compromised in dogs before surgery because of pulmonary edema, intrathoracic masses, or restrictive pulmonary disease. Further pulmonary dysfunction was likely during surgery when the lungs were packed to improve surgical visibility, and during intracardiac surgery, when they were allowed to collapse to provide an unobstructed surgical field. In addition, hypothermia has been reported to attenuate hypoxic pulmonary vasoconstriction. Oxygenation was maximized by using 100% inspired oxygen, and atelectasis and hypoxemia were minimized by use of continuous positive airway pressure whenever possible. The level of hypothermia for each dog was made on predicted occlusion or arrest times, but occasionally, the ischemic time extended beyond the “allowable” periods. In 1 dog with esophageal temperature of 26 C, sinoatrial arrest was extended to 62 minutes and this was undoubtedly the major cause of the multiple organ failure causing death on the third day after surgery. This dog would have benefited from cardiopulmonary bypass or deeper hypothermic conditions. All other dogs that had cardiac arrest tolerated the arrest and resuscitation without major complications. All dogs were rewarmed without major adverse effects. Care was required during rewarming not to bump or handle the heart or cause a rapid change in temperature because the lowered fibrillatory threshold causes a more irritable heart. In a few dogs that had thoracotomy, arrhythmias were noticed during the warm saline solution flushes but stopped when the flushes were discontinued. One dog that had thoracotomy had ventricular fibrillation during this period, requiring mechanical defibrillation, and this may have been prevented with cooler, more gentle saline solution flushes. As a further precaution in preventing cardiovascular decompensation attributable to handling, dogs were not moved out of the surgical suite until esophageal temperature reached a minimum of 32 C. As with cooling, shivering may commence during rewarming. It cannot be overemphasized that shivering in either situation is detrimental and may be an important cause of postoperative complications. Shivering may be prevented through the judicious use of opioids and occasionally must be further augmented with neuromuscular blockade. The prolonged effects of our induction and maintenance opioids usually prevented shivering in the postoperative phase. To provide some residual paralysis, the atracurium was not reversed. The atracurium was also not reversed because of the potentially detrimental cardiovascular effects of the atropine or edrophonium at a time when the cardiovascular system is most labile and susceptible to stresses. All dogs survived the immediate hypothermic surgical procedure. The postoperative time course of these cases was very short, considering the physiologic insult of hypothermia with extensive surgery. Most dogs required ICU monitoring for only 2.5 days, and all dogs leaving ICU were discharged to the owners a short time later. This attests to the protective effects of appropriately administered hypothermia. Surface-induced hypothermic anesthesia should be considered as an alternative to cardiopulmonary bypass for intracardiac surgery requiring a short arrest period of up to 30 to 45 minutes. Hypothermia appears to also be protective to tissues when vascular occlusion may be required to prevent hemorrhage or to provide surgical visibility. The major hazard with this technique is its complexity, but with organization, it may be performed with ease. The technique was used successfully in the 19 dogs reviewed here and should be considered for selected high-risk surgical procedures.

References
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