Limb Tourniquets and Central Temperature in Anesthetized Children

Edmond C. Bloch, MB, ChB, FFARCS, Brian Ginsberg, MB, ChB, FFASA, Robert A. Binner, Jr., MD, and Daniel I. Sessler, MD

Departments of Anesthesiology and Pediatrics, Duke University Medical Center, Durham, North Carolina, and Department of Anesthesia, University of California, San Francisco, California

We have observed an association between the use of tourniquets for limb surgery and a progressive increase in body temperature in pediatric patients. Consequently, we evaluated the effect of leg tourniquet(s) on intraoperative nasopharyngeal temperature in pediatric patients. We measured central temperature in three groups of children anesthetized with halothane and nitrous oxide: those with unilateral tourniquets (n = 15), those with bilateral tourniquets (n = 8), and a control group not requiring tourniquets (n = 24). Intraoperative ambient temperatures were maintained near 23°C, respiratory gases were actively heated and humidified, and skin was warmed using a circulating water blanket set at 38°C. The control patients remained normothermic during anesthesia and surgery. In contrast, central temperature increased 1.0 ± 0.6°C in 90 min in those with one tourniquet and 1.7 ± 0.6°C in those with bilateral tourniquets. The tourniquet-induced hyperthermia appeared to result from decreased effective heat loss from distal skin and from constraint of metabolic heat to the central thermal compartment. These data suggest that pediatric patients requiring intraoperative tourniquets should not be aggressively warmed during surgery.


It has been noted by us (1) and others (2) that central temperature progressively increases in pediatric patients during anesthesia for limb surgery. It has been our impression that this hyperthermia was associated with inflated limb tourniquet(s). Some years ago, we reviewed the anesthetic records of pediatric patients who had undergone operations requiring the application of unilateral and bilateral limb tourniquets. We compared them with the anesthetic records of patients who had undergone procedures during which they were similarly draped, but were not subject to tourniquet application (1). Although this retrospective review confirmed our impressions, it lacked the validity of a prospective investigation. We therefore prospectively tested the hypothesis that unilateral and bilateral leg tourniquet application would increase intraoperative central temperature compared with temperatures in patients not requiring tourniquets.

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Address correspondence to Dr. Bloch, Department of Anesthesiology, Duke University Medical Center, Durham, NC 27710.

Methods

With approval of the Institutional Review Board of Duke University Medical Center, we studied 47 pediatric patients. Fifteen patients had a single leg tourniquet applied to facilitate orthopedic surgery; eight patients required bilateral leg tourniquets. The control group (n = 24) were similarly draped and anesthetized for peripheral surgical procedures such as hypospadias repair and orchidopexy, but differed in not requiring tourniquets.

Patients were healthy (ASA physical status I and II), had no evidence of infection, had no personal or family history of malignant hyperthermia, had no evidence of increased intracranial pressure, and were not taking any medications known to affect thermoregulation (e.g., antipyretics). They were not given blood or blood products during the study.

Anesthesia was induced by intravenous administration of thiopental or inhalation of a mixture of nitrous oxide, oxygen, and halothane. Atropine (0.01 mg/kg) was administered intravenously immediately after induction. Atracurium or vecuronium was used to facilitate tracheal intubation and the subsequent control of ventilation. Anesthesia was maintained with nitrous oxide, oxygen, and halo-
thene (0.5%-1.5%) via a Mapleson F breathing system with a MR450 Servo airway heater and humidifier (Fisher and Paykel, Auckland, New Zealand). All patients were warmed by a circulating water blanket (Cincinnati Sub-Zero, Blanketrol HL, model 200HL) set to 38°C. Ambient temperature was thermostatically controlled near 23°C. Intravenous fluids were infused at maintenance rates and were not warmed. All the patients in this study were anesthetized in the same operating room.

Ambient temperatures were monitored using a Mallinckrodt (St. Louis, Mo.) model 8200 electronic thermometer located near the head of the operating table at a site free from drafts or heat from electronic equipment. Central temperatures were measured using a nasopharyngeal probe (YSI series 700, Mallinckrodt) inserted via a nostril to a depth equal to the distance from the tragus of the ear to the ala nasi (3). Patients' nasopharyngeal temperatures were measured as soon as possible after induction of anesthesia (elapsed time zero) and at 15-min intervals for 90 min. Data are presented only for patients completing the entire 90-min protocol. The same thermometers were used for each patient in the study and they were calibrated and maintained according to the manufacturer's instructions.

Time-dependent data within each group were analyzed using repeated-measures analysis of variance and Dunnett's tests for comparison to values recorded immediately after induction of anesthesia. Comparisons between groups (at each time) were made using one-way analysis of variance and Student-Newman-Keuls tests. Values are expressed as mean ± standard deviation (sd); P < 0.05 identified statistically significant differences.

Results

There were no statistically significant differences between the groups regarding age, weight, height, body surface area, or ambient temperature (Table 1). Tourniquets in the unilateral group were inflated for 103 ± 26 min and those in the bilateral group for 115 ± 27 min (P = NS). Anesthesia lasted 161 ± 51 min in the patients not requiring tourniquets. Initial temperatures after induction of anesthesia (elapsed time zero) were 36.6 ± 0.4°C in the unilateral tourniquet group, 36.4 ± 0.4°C in the bilateral group, and 36.5 ± 0.6°C in the control group. Patients in the control group remained nearly normothermic. In contrast, central temperatures increased 1.0 ± 0.6°C in 90 min in patients with unilateral tourniquets and 1.7 ± 0.6°C in those with bilateral tourniquets. Central temperatures in each treatment group were significantly greater than values at time zero after 15 elapsed minutes. Central temperature increases in both tourniquet groups significantly exceeded values in control patients at all times after each 15-min interval. Increases in the bilateral tourniquet group were significantly greater than those in the unilateral group at 75 and 90 elapsed minutes (4).

Discussion

Nasopharyngeal temperature increased 1.0 ± 0.6°C in patients with unilateral tourniquets (relative to

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**Table 1. Morphometric Characteristics and Ambient Temperature**

<table>
<thead>
<tr>
<th></th>
<th>Unilateral</th>
<th>Bilateral</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number</td>
<td>15</td>
<td>8</td>
<td>24</td>
</tr>
<tr>
<td>Age (yr)</td>
<td>3.0 ± 2.5</td>
<td>3.1 ± 3.0</td>
<td>2.6 ± 2.9</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>15 ± 7</td>
<td>16 ± 11</td>
<td>15 ± 13</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>93 ± 21</td>
<td>93 ± 25</td>
<td>88 ± 25</td>
</tr>
<tr>
<td>Surface area (m²)</td>
<td>0.6 ± 0.2</td>
<td>0.6 ± 0.3</td>
<td>0.6 ± 0.3</td>
</tr>
<tr>
<td>Ambient temperature (°C)</td>
<td>23.5 ± 1.6</td>
<td>23.3 ± 1.4</td>
<td>23.2 ± 1.5</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Time (min)</th>
<th>ΔTemp (°C)</th>
</tr>
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<tbody>
<tr>
<td>15</td>
<td>1.5</td>
</tr>
<tr>
<td>30</td>
<td>1.0</td>
</tr>
<tr>
<td>45</td>
<td>0.5</td>
</tr>
<tr>
<td>60</td>
<td>0.0</td>
</tr>
<tr>
<td>75</td>
<td>-0.5</td>
</tr>
<tr>
<td>90</td>
<td>-1.0</td>
</tr>
</tbody>
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Figure 1. The change in nasopharyngeal temperature in patients given unilateral leg tourniquets, bilateral leg tourniquets, or no tourniquets (control) during 90 elapsed minutes. Time zero was the time as soon after induction of anesthesia as possible at which the first central temperature was recorded. Initial temperatures after induction of anesthesia (elapsed time zero) were 36.6 ± 0.4°C in the unilateral tourniquet group, 36.4 ± 0.4°C in the bilateral group, and 36.5 ± 0.6°C in the control group. Patients in the control group remained nearly normothermic. In contrast, central temperatures increased 1.0 ± 0.6°C in 90 min in patients with unilateral tourniquets and 1.7 ± 0.6°C in those with bilateral tourniquets. Central temperatures in each treatment group were significantly greater than values at time zero after 15 elapsed minutes. Central temperature increases in both tourniquet groups significantly exceeded values in control patients at all times after each 15-min interval. Increases in the bilateral tourniquet group were significantly greater than those in the unilateral group at 75 and 90 elapsed minutes (4).
control patients, over 90 min) and 1.7 ± 0.6°C in patients with bilateral tourniquets. Central temperature increases when (a) metabolic heat production increases, (b) heat loss to the environment decreases, or (c) the internal distribution of body heat is altered. In paralyzed patients, the observed increase in central temperature is unlikely to result from increased metabolic heat production. Nonshivering thermogenesis is triggered by hypothermia and consequently would not be anticipated in hyperthermic patients. Furthermore, the average age of the patients in this study was ~3 yr, and nonshivering thermogenesis is unimportant in unanesthetized (4-6) or anesthetized (7 and Bissonnette and Sessler, unpublished data) humans over 2 yr of age.

In the absence of insulation (8), cutaneous heat loss is roughly proportional to surface area over the entire body (9). The surface area of each leg constitutes ~18% of the total body surface area (10). Based on the expected metabolic rate during anesthesia and the specific heat of humans (11), decreased effective heat loss from the skin to the environment would increase mean body temperature (relative to control patients) by ~0.2 and ~0.4°C, respectively, in the unilateral and bilateral tourniquet groups. The observed hyperthermia was considerably greater than predicted simply from the reduction in effective surface area, suggesting that distribution of heat within the body also was altered.

There are several indications that altered distribution of heat within the body contributes significantly to clinical thermal perturbations. For example, the precipitous decrease in central temperature after induction of general (12) or epidural (13) anesthesia does not result from increased heat loss to the environment or decreased metabolic heat production. Instead, it is caused by redistribution of central heat to peripheral tissues. Similarly, the central temperature plateau frequently observed after 3–4 h of anesthesia in patients sufficiently hypothermic to trigger peripheral thermoregulatory vasoconstriction (14,15) cannot be explained by the resulting small decrease in cutaneous heat loss (16) or nonshivering thermogenesis (7). Prevention of further central hypothermia in these patients apparently results when metabolic heat (most of which is centrally produced) is constrained by thermoregulatory vasoconstriction to the central compartment. Similar hypothermia prevention has been observed in volunteers immersed in water at 15°C (17).

Tourniquets similarly constrain centrally generated metabolic heat to the central thermal compartment. It is thus likely that central hyperthermia resulted in our patients, in part, because a similar amount of metabolic heat was distributed to a smaller tissue mass after tourniquet inflation. After tourniquet inflation (and prevention of metabolic heat transfer from the core), heat loss from the skin to the environment would continue in distal tissues, causing regional hypothermia. Release of the tourniquet(s) would allow mixing of heat in the warm central and cool peripheral tissues, precipitously decreasing central temperature. This “redistribution hyperthermia” is mechanistically similar to that observed during induction of anesthesia (12). A rapid decrease in central temperature after tourniquet release has previously been described in adult patients (18,19).

Most pediatric patients are aggressively warmed to prevent intraoperative hypothermia. However, warming can be excessive, and two major pediatric anesthesia textbooks assert that intraoperative hyperthermia may now be more common than hypothermia in infants and children (20,21). Our data indicate that hyperthermia is particularly likely in patients requiring intraoperative tourniquets. Hyperthermia itself increases blood pressure and heart rate, both of which may be further increased by tourniquet pain (22). The combination of hyperthermia, hypertension, and tachycardia may suggest malignant hyperthermia, particularly in children with musculoskeletal deformities who are especially prone to the syndrome (23). Ironically, induced hyperthermia facilitates triggering of malignant hyperthermia (24). Our results suggest that patients requiring tourniquets should not be aggressively warmed and that causes of hyperthermia other than malignant hyperthermia should be considered in these patients.

Our three study groups were not completely comparable in that the control patients required slightly different operations. However, surgical exposure, draping, age, and weight were similar: it is unlikely that the large temperature differences between the groups resulted simply from patient selection. Although tourniquet pain contributed to stimulation of surgery in the treatment groups, there is no obvious mechanism by which sympathetic stimulation itself would have caused hyperthermia in patients with tourniquets. Intraoperative temperature changes are determined by a variety of factors, including ambient temperature (25,26), cutaneous insulation (8), active skin-surface warming (9), airway heating and humidification (27-29), size of the surgical incision (30), and surgical skin preparation. Warming was sufficient in our patients to maintain the control group near-normothermic. Had warming been less aggressive, it is likely they would have become hypothermic; nonetheless, observed differences between the control and tourniquet groups would have persisted.

In summary, we evaluated the effect of leg tourniquet(s) on intraoperative nasopharyngeal temperature in pediatric patients. We measured central tem-
perature in three groups of children: those with unilateral tourniquets, those with bilateral tourniquets, and a control group requiring no tourniquets. The control patients remained nearly normothermic during anesthesia and surgery. In contrast, central temperature increased $1.0 \pm 0.6^\circ C$ in 90 min in those with one tourniquet and $1.7 \pm 0.6^\circ C$ in patients with bilateral tourniquets. Tourniquet-induced hyperthermia appears to result from decreased effective heat loss from distal skin and from constraint of metabolic heat to the central thermal compartment. These data suggest that pediatric patients requiring intraoperative tourniquets should not be aggressively warmed during surgery.

References