Efficacy of intraoperative heat administration by ventilation with warm humidified gases and an oesophageal warming system

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Summary
We measured changes in body temperature in 12 hypothermic (mean aural temperature 34.4 (SD 1.0) °C) pigs during general anaesthesia with an open abdominal cavity and the effect of two warming systems: heating of inspired gases to 39 °C (intratracheal temperature) and oesophageal warming to 39 °C by a water perfused oesophageal heat exchanger. Each animal underwent both treatments and the control period in random sequence. Each condition was studied over 1 h. No additional protection against heat loss (draphes, blankets, i.v. fluids warming, etc.) was used. Anaesthesia, room temperature and relative humidity, amount and temperature of infusions and extension of exposed visceral surfaces were standardized. Mean decrease in body temperature was 1.0 (0.7) °C (P < 0.005) without warming and 0.6 (0.2) °C (P < 0.005) with heated inspired gases: this difference was not statistically significant. Oesophageal warming was very efficient as mean body temperature did not change significantly (-0.1 (0.2) °C; ns). (Br. J. Anaesth. 1996;77:530–533)

Key words

Mild hypothermia is relatively frequent during and after surgery under general anaesthesia. Several factors contribute to body heat loss: inhibition of the hypothalamus, peripheral vasodilatation induced by anaesthetic agents, paralysis, exposure of the viscera to the atmosphere and large amounts of i.v. infusions at room temperature. Mild hypothermia is not generally a problem during operation. However, in the immediate postoperative period, activation of some physiological mechanisms of thermogenesis (metabolic heat production, muscles activity, shivering) with a consequent increase in oxygen consumption and peripheral vasoconstriction are untoward and may even be harmful in patients with impaired cardiovascular reserve. Moreover, hypothermia has been implicated in some cases of delayed recovery from general anaesthesia.

Several methods are commonly used to reduce body heat loss. Heating inspired gases and oesophageal warming are easily applied during surgery and have been proposed for providing (central) warming, which is probably more "physiological" than other methods which deliver heat peripherally (warming the operating room, warm mattresses, thermal blankets, infrared heating lamps, forced air warmers). The aims of this study were: to evaluate, in an animal model, under strictly controlled conditions, changes in body temperature during general anaesthesia with an open abdominal cavity, with standardization of most confounding factors such as anaesthesia, room temperature, amount and temperature of infusions, and extension of exposed visceral surfaces; to evaluate, in the same controlled conditions, the efficacy of two warming systems (heating inspired gases and oesophageal warming) in reducing body heat loss.

Materials and methods
After obtaining approval from the Ethics Committee of our institution, we studied 12 female, 3-month-old White Landrace pigs, weighing 18.4 (SD 1.2) kg. These animals were chosen as, among non-primate laboratory animals, the pig is the most comparable with adult humans.

Each animal underwent three study periods (control; heating of inspired gases; oesophageal warming) in a randomized sequence according to a Latin square design. The sample size was based on the results of an unpublished pilot study, by assuming a type I error (α) of 0.05 and a type II error (β) of 0.2, taking into account the randomization design and the statistical analysis method (repeated-measures analysis of variance).

Anaesthesia was induced with thiopentone 5 mg kg⁻¹ and maintained with 1.5% enflurane and fentanyl 2–3 μg kg⁻¹ h⁻¹. Neuromuscular block was produced with pancuronium 50–60 μg kg⁻¹ h⁻¹.

Mechanical ventilation (Servo ventilator 900A, Elema, Schönander, Sweden) via a tracheostomy was set at 3.8–4.2 litre min⁻¹ to maintain a Pao₂ of 4.7–6.0 kPa. The total inspiratory time was 35% (25% inspiration + 10% inspiratory pause).

A carotid artery catheter and a 5-French gauge pulmonary artery catheter were inserted for haemodynamic monitoring and blood sampling. Arterial pressure (AP), pulmonary artery pressure (PAP), central venous pressure (CVP) and pulmonary...
wedge pressure (PWP) were measured with HP 1290A pressure transducers and a 78354A monitor (Hewlett Packard GmbH, Böblingen, Germany). Cardiac output (CO) was measured by thermodilution (mean of three measurements at expiratory pause) with a 9520A CO computer (Edwards, Santa Ana, CA, USA). Blood pH, P\textsubscript{CO\textsubscript{2}} and P\textsubscript{O\textsubscript{2}} were determined with an IL1302 analyser, and haemoglobin and oxygen saturation with an IL802 co-oximeter (Instrumentation Laboratory, Milano, Italy).

Room, oesophageal, tracheal and aural temperatures, and four cutaneous temperatures (anterior leg (A), nipple (B), thigh (C) and calf (D)) were monitored with an MC9200 electronic thermometer (Exacon, Roskilde, Denmark) using site-specific probes (Exacon, Roskilde, Denmark). Relative humidity of the room was measured with a standard hygrometer.

Room temperature and relative humidity were measured with a hygrometer.

I.v. infusions (at room temperature) were standardized at 10 ml kg\textsuperscript{-1} h\textsuperscript{-1}. A laparotomy was performed to obtain an oesophageal temperature of 39 ± 0.5 °C. The tracheal temperature probe also allowed measurement of end-expiratory gas temperature. The inspiratory branch of the ventilator circuit was wrapped in aluminium sheets. The oesophageal heat exchanger was not in place. The 60-min study started as soon as the inspiratory tracheal temperature was stabilized at 39 ± 0.5 °C for at least 5 min.

(2) The oesophageal warmer is composed of a water perfused oesophageal heat exchanger and a water heating pump (water flow 4 litre min\textsuperscript{-1}) with a built-in thermostat adjustable up to 42 °C (Thermal Therapy System (TTS) TT8200, Exacon, Roskilde, Denmark). The oesophageal heat exchanger consists of a light rigid inner supply tube, surrounded by a 50-cm long, non-elastic and thin-walled outer tube (surface area ~0.05 m\textsuperscript{2}). Distilled water is circulated through the inner tube to the tip, fills up the entire volume of the outer tube and returns to the pump through the proximal end of the oesophageal tube. After the oesophageal heat exchanger was placed, the system thermostat was set to obtain an oesophageal temperature of 39 ± 0.5 °C. A Kontron Pearl Humidifier 4150 (Kontron Instruments, Milan, Italy) at room temperature was used in the ventilator circuit. The 60-min study started as soon as oesophageal temperature was stabilized at 39 ± 0.5 °C for at least 5 min.

(3) No warming systems were used. The oesophageal heat exchanger was not in place and the Kontron humidifier was at room temperature.

Temperature, haemodynamic measurements and blood samples were obtained at the beginning (pre) and end (post) of each 60-min study period. The following variables were calculated:

Mean skin temperature (MBT) according to Ram-athanath’s formula:\textsuperscript{21}

\[ \text{MBT} = 39°C - 0.3 \times (TA + T\textsuperscript{B}) + 0.2 \times (TC + TD) \]

Mean body temperature (MBT) according to Colin and colleagues’ formula:\textsuperscript{22}

\[ \text{MBT} = 0.66 \times T\text{aural} + 0.34 \times \text{MST} \]

Total body heat (TBH) relative to 0 °C according to Burton’s formula:\textsuperscript{23}

\[ \text{TBH} = \text{MBT} 	imes 3.47 \times \text{body weight (kg)} \]

Oxygen consumption (\(\dot{V}_\text{O}_2\)) according to Fick’s equation:

\[ \dot{V}_\text{O}_2 = 10 \times (\text{Ca}_\text{O}_2 - \text{Cv}_\text{O}_2) \times \text{CO} \]

Right to left shunt fraction (\(Q\text{va}/Qt\)) according to the following equation:

\[ Q\text{va}/Qt = (\text{Ce}_\text{O}_2 - \text{Ca}_\text{O}_2)/(\text{Ce}_\text{O}_2 - \text{Cv}_\text{O}_2) \]

where \(\text{Ca}_\text{O}_2\) (ml 100 ml\textsuperscript{-1}) = 1.34 \times Hb (g 100 ml\textsuperscript{-1}) + 0.003 × (P\text{ao} (mm Hg) - P\text{co} (mm Hg)); \(\text{Cv}_\text{O}_2\) (ml 100 ml\textsuperscript{-1}) = 1.34 × Hb × Sv\text{O}_2 (mm Hg); and \(\text{Ce}_\text{O}_2\) (ml 100 ml\textsuperscript{-1}) = 1.34 × Hb × S\text{O}_2 (frac) + 0.003 × P\text{O}_2 (mm Hg).

At the end of the experiment, specimens of tracheal and oesophageal wall were obtained to detect possible heat-related damage.\textsuperscript{24}

Statistical analysis was performed using the SAS/GLM procedure, release 6.03 (SAS Institute Inc., Cary, NC, USA) on a AST Bravo LC 3/33s personal computer (AST Research, Inc., Irvine, CA, USA).

**Results**

Each animal completed both treatments and the control period.

Data for all measured variables at the beginning (pre) of each 60-min condition, and differences between the beginning and final values (post–pre) are shown in table 1. Pre values were not significantly different between the three conditions.

Room temperature and relative humidity were monitored continuously and did not change significantly during the experiment. All measured temperatures and derived variables (MST, MBT and TBH) decreased significantly under both control (\(P<0.005\)) and oesophageal warming, only cutaneous temperature A (anterior leg) showed significant variations.

As shown by the variation in all measured body temperatures and derived variables, heat loss was significantly greater during the control period than during oesophageal warming, while the difference between the control period and that during warming of inspired gases was not statistically significant, except for nipple temperature (B) and MBT (table 1).

\(\dot{V}_\text{O}_2\) decreased significantly (\(P<0.001\)) by 8–15% during the control period and during the two treatments (table 1), but there were no significant differences between the three conditions. \(Q\text{va}/Qt\) did not change significantly and there were no differences between the three conditions.

The animals were haemodynamically stable throughout the experiment.

There were no pathological changes at the histological examination of both tracheal and oesophageal walls at the end of the study.
**Table 1** Temperatures and haemodynamic variables measured at the beginning (pre) (mean (SEM)) and differences between the beginning and final values (post-pre) (mean (SEM)) during control conditions, warming of inspired gases (Hum) and oesophageal warming (TTS).

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>Hum</th>
<th>TTS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Aural temp. (°C)</strong></td>
<td>34.6 (0.7)</td>
<td>34.4 (0.9)</td>
<td>34.1 (1.2)</td>
</tr>
<tr>
<td><strong>Skin temp. A (°C)</strong></td>
<td>33.0 (0.9)</td>
<td>32.9 (0.8)</td>
<td>32.7 (1.1)</td>
</tr>
<tr>
<td><strong>Skin temp. B (°C)</strong></td>
<td>33.2 (0.7)</td>
<td>33.0 (0.9)</td>
<td>32.7 (1.4)</td>
</tr>
<tr>
<td><strong>Skin temp. C (°C)</strong></td>
<td>34.0 (0.9)</td>
<td>33.8 (0.7)</td>
<td>33.9 (1.0)</td>
</tr>
<tr>
<td><strong>Skin temp. D (°C)</strong></td>
<td>34.1 (0.8)</td>
<td>33.8 (0.9)</td>
<td>33.9 (0.9)</td>
</tr>
<tr>
<td><strong>Mean skin temp. (°C)</strong></td>
<td>33.7 (0.8)</td>
<td>33.5 (0.8)</td>
<td>33.4 (1.0)</td>
</tr>
<tr>
<td><strong>Mean body temp. (°C)</strong></td>
<td>34.2 (0.7)</td>
<td>34.1 (0.8)</td>
<td>33.9 (1.1)</td>
</tr>
<tr>
<td><strong>Total body heat (kJ)</strong></td>
<td>2187 (151)</td>
<td>2175 (136)</td>
<td>2163 (147)</td>
</tr>
<tr>
<td><strong>Room temp. (°C)</strong></td>
<td>24.1 (0.5)</td>
<td>23.9 (0.7)</td>
<td>23.8 (0.3)</td>
</tr>
<tr>
<td><strong>Relative humidity (%)</strong></td>
<td>48 (3)</td>
<td>49 (3)</td>
<td>48 (2)</td>
</tr>
<tr>
<td><strong>HR (beat min⁻¹)</strong></td>
<td>130 (25)</td>
<td>125 (22)</td>
<td>120 (21)</td>
</tr>
<tr>
<td><strong>CO (litre min⁻¹)</strong></td>
<td>2.7 (0.6)</td>
<td>2.4 (0.6)</td>
<td>2.1 (0.4)†††</td>
</tr>
<tr>
<td><strong>MAP (mm Hg)</strong></td>
<td>88 (9)</td>
<td>82 (5)</td>
<td>70 (9)†††</td>
</tr>
<tr>
<td><strong>MPAP (mm Hg)</strong></td>
<td>19 (4)</td>
<td>18 (3)</td>
<td>17 (5)</td>
</tr>
<tr>
<td><strong>PWP (mm Hg)</strong></td>
<td>5 (3)</td>
<td>5 (3)</td>
<td>7 (2)</td>
</tr>
<tr>
<td><strong>CVP (mm Hg)</strong></td>
<td>4 (3)</td>
<td>4 (3)</td>
<td>5 (3)</td>
</tr>
<tr>
<td><strong>TSR (dyn s min⁻¹)</strong></td>
<td>2640 (576)</td>
<td>2762 (656)</td>
<td>2585 (475)</td>
</tr>
<tr>
<td><strong>TPR (dyn s min⁻¹)</strong></td>
<td>429 (149)</td>
<td>438 (115)</td>
<td>438 (172)</td>
</tr>
<tr>
<td><strong>VO₂ (ml min⁻¹)</strong></td>
<td>79 (23)</td>
<td>78 (22)</td>
<td>74 (17)</td>
</tr>
<tr>
<td><strong>Qva/Qt</strong></td>
<td>0.17 (0.11)</td>
<td>0.14 (0.04)</td>
<td>0.14 (0.07)</td>
</tr>
<tr>
<td><strong>Pre values; † † †</strong></td>
<td><strong>P &lt; 0.05</strong></td>
<td><strong>P &lt; 0.05</strong></td>
<td><strong>P &lt; 0.001</strong></td>
</tr>
</tbody>
</table>

**Discussion**

There have been several clinical studies on the effects of different warming systems in preventing perioperative hypothermia with controversial results.

Both heating of inspired gases and oesophageal warming have been demonstrated as either effective and ineffective in different studies. The “within study” and “between studies” variability of the experimental conditions (patients, type of surgery, type of anaesthesia, fluid infusions, room temperature and relative humidity, etc.) makes it difficult to compare these studies.

In our study such confounding factors were standardized and controlled carefully. Moreover, in order to maximize the differences between treatment and control conditions, we chose to avoid any system used clinically to reduce heat loss (drapes, warming of i.v. fluids, etc): during the experiment our animals became hypothermic more quickly than usually seen in anaesthetic practice (approximately 0.5°C h⁻¹). This greater heat loss may also have resulted from the smaller body size and mass of our animals compared with an adult human.

The mean decrease in temperature (aural and the four cutaneous temperatures) was approximately 1°C h⁻¹ without warming. Mean body temperature (MBT), calculated according to the formula used in humans, reflects very well this heat loss. Some may argue that the derived variables (MST, MBT and TBH) may not be quantitatively accurate because they were computed using formulae developed for humans and which might not apply to pigs. However, as we were interested in evaluating variations rather than absolute values for each condition, we believe that our results are not invalidated by the use of human formulae.

Our study clearly showed the efficacy of oesophageal warming in attenuating the decrease in MBT and TBH. Warming of inspired gases was not as effective and the reduction in heat loss was not statistically significant. The variation in efficacy may be explained by important differences in the way the two systems work. Heat transport is much higher for oesophageal warming (~8 kJ h⁻¹) than for warming of inspired gases (~5000 kJ h⁻¹) at a water flow of 4 litre min⁻¹ than for warming of inspired gases (~8 kJ h⁻¹) at 34 to 39°C at a water flow of 4 litre min⁻¹ because of the low specific heat of air compared with water. This difference appears to be important in overcoming the larger heat exchange area during warming of inspired gases (trachea and large bronchi) and probably higher heat transfer efficiency of the respiratory system (as suggested by the small difference between the temperature of the expired gas and core temperature). The technical characteristics of oesophageal warming did not allow measurement of the temperature at the outlet of the oesophageal heat exchanger and consequently calculation of net heat transfer during oesophageal warming. However, the large amount of heat transported during oesophageal warming (~5000 kJ h⁻¹) would have resulted in a greater temperature change if efficiently transferred to the body.

There were no side effects in our study. Heat damage to the tracheal and oesophageal walls was not demonstrated at histological examination or by deterioration in respiratory function (Qva/Qt).

Oesophageal warming requires a specific device but it is easily used and has no complications. It is not only the most effective non-invasive core-warming system that can be used during operation, but it may also be used successfully in other non-surgical situa-
tions of severe accidental hypothermia (exposure, cold water immersion, etc.).

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