Predicting the efficacy of convection warming in anaesthetized children

R. Stanger¹, K. Colyvas², J. G. Cassey³*, I. A. Robinson⁴ and P. Armstrong⁵

¹Department of Chemical Engineering, University of Newcastle, Newcastle, Australia. ²School of Mathematical and Physical Sciences, University of Newcastle, Newcastle, Australia. ³John Hunter Hospital, Newcastle, Australia. ⁴University of Newcastle, Newcastle, Australia

*Corresponding author: 23 Veronica Street, Cardiff 2285, NSW, Australia. E-mail: jcpaed@iinet.net.au

Background. We previously described a convection warming technique (Cassey J, Armstrong P, Smith GE, Farrell PT. Paediatr Anaesth 2006; 16: 654–62). This study further analyses the children in that original study with three aims: (i) to investigate factors purported to influence children’s heating rates, (ii) to describe the most effective usage of this warming technique, and (iii) to understand better the physiology of convection warming.

Methods. Children having anaesthesia for elective surgery lasting longer than 90 min in ambient temperature 21 °C were warmed by a ‘Bair Hugger’ attached to a custom-built heat dissipation unit. Relationships between child and procedure characteristics and various thermal measures were analysed, and a thermodynamic model was evaluated.

Results. Thirty-nine children (aged 2 days to 12.5 yr) were studied. There were statistically significant correlations between a number of factors (e.g. height and weight) and heating efficacy. Our model demonstrated the impact of changing patient characteristics on temperature profiles. Neither the morphological characteristics nor our model could predict an individual’s Tcore behaviour.

Conclusions. (i) Although the effectiveness of this warming technique is influenced by patient/procedure characteristics, these do not predict normothermia (uncertainty ± 28 min). Effectiveness is independent of simple thermal measures. (ii) Previously described measures of vasoconstriction are not valid in children. (iii) Our model shows children’s thermal properties change with their Tcore. However, key factors are unknown for an individual and our model does not predict heating efficacy. (iv) To minimize the risk of hyperthermia, we recommend continuous measurement of Tcore during convection heating. The device air temperature should be turned to medium (38 °C) as Tcore approaches 37 °C.

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Core body temperature (Tcore) decreases in all children having general anaesthesia (GA) for surgical procedures.¹ The practice of raising ambient temperature (Tambient) in paediatric theatres for elective surgery is not necessary, given the availability of effective means of maintaining normothermia at room temperatures of 21°C.² Our previously reported results have all been based on a Bair Hugger unit combined with a purpose-designed heat dissipation unit and draping technique.² ³ Despite close temperature monitoring, Tcore in 25% of our clinical group increased above 37.9°C. We analysed the factors commonly purported to impact on the heating rate in an effort to:

(i) provide guidelines to the most effective usage of this convective warming technique;
(ii) predict the likely time to normothermia for an individual;
(iii) develop a thermodynamic model to help us understand the physiological processes in convection

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warming and determine if it could explain the outcomes.

Methods

Clinical

The clinical aspects and methodology of the study have been described previously.2 A brief summary follows.

The study was approved by the Hunter Area Health Service ethics committee and informed written consent was obtained by the principal author from the parents/guardians (and potential participants if able to give their own consent). The general inclusion criterion was any child having GA for an elective surgical procedure where the total time from induction of anaesthesia to transfer to the recovery room was \( \geq 90 \) min. The ambient of the induction room and operating theatre was set to \( 21 ± 0.2 ^\circ C \). Eleven Mon-a-therm 400 series thermistors (Mallinckrodt Inc., Missouri, MO, USA) were placed as in Figure 1. The thermistors were attached to an NI 4350-USB data-logger (National Instruments Corp., TX, USA) with each thermistor logged to disk at 5 s intervals. Real-time display was on a laptop using a custom-built program (developed by Matrix Consultants, Newcastle, Australia) using LabView (National Instruments Corp.). Heating was achieved by the technique previously described.2 That study, performed between 2003 and 2005, used a ‘Bair Hugger’ model 505 (Augustine Medical, Minnesota, MN, USA) with the outlet hose modified to fit the rear of a custom-designed heat dissipation unit manufactured by Trident Engineering, Newcastle, Australia. We currently use the same device. We do not know whether the manufacturer’s current ‘505 Bair Hugger’ is any different internally than the unit we use(d), but its specifications and external appearance are the same. The unit produces turbulent airflow from multiple holes in its outlet face. The Bair Hugger temperature was initially set to \( 43 ± 0.5 ^\circ C \). The set temperature was turned down by the anaesthetist, if the patient’s \( T_{\text{core}} \) was \( >37 ^\circ C \). No i.v. fluid warming, active heating/humidifying of inspired gases, or warming blankets were used. Several other pieces of data were collected in the original study, including procedure and child characteristics, various temperatures, and time intervals. Table 1 lists those used in this analysis.

Analysis

General

The original individual patient data sets were averaged into 1 min epochs. Since rectal (\( T_{\text{rectum}} \)) and oesophageal (\( T_{\text{oesophagus}} \)) temperatures followed similar trends, we performed analysis of \( T_{\text{core}} \) using \( T_{\text{oesophagus}} \) alone. We followed a stepwise, increasingly complex methodology in analysis of the data, ultimately requiring application of thermodynamic theory and a simple model to examine each individual’s response to heating. The reader is referred to Supplementary Appendix (available online) for a complete description of this.

Thermal measures

We used a variety of thermal measures in order to best understand the dynamics of the situation: markers of vasoconstriction/heat deficit, simple temperature measurements, and thermodynamic calculations. Each of these thermal measures was calculated for each individual.

(1) Markers of vasoconstriction and heat deficit. Since there was a range in both child characteristics and ‘lag time’ intervals between induction and commencement of heating, it seems logical that there may have been a range of ‘heat deficits’ among the children. In order to take this into account when analysing the response to warming, we used:

(a) markers of vasoconstriction: we examined whether temperature of left toe (\( T_{\text{LT}} \)) or the temperature difference between toe and oesophagus (\( T_{\text{LT}} - T_{\text{oesophagus}} \)) might be useful. Other markers have been proposed in the literature,4–6 temperature differences between right calf and right toe (\( T_{\text{Cal}} - T_{\text{RT}} \)), and forearm and fingertip (\( T_{\text{forearm}} - T_{\text{finger tip}} \)). They were all calculated from measurements recorded immediately before commencement of convection warming.
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Table 1 Child, procedure, temperature, and time factors evaluated for relationship with various thermal measures. *Patients were stratified into three groups: peripheral (e.g. herniotomy, orchiopexy, and urethroplasty), open body cavity group 1 (e.g. extraperitoneal minimal incision renal), and open body cavity group 2 (major laparotomy)

<table>
<thead>
<tr>
<th>Child characteristics</th>
<th>Age, gender, weight, height, BSA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Procedure characteristics</td>
<td>Stratification of procedure (peripheral, open1, open2)*</td>
</tr>
<tr>
<td>%BSA uncovered</td>
<td>Position (supine, lateral, prone)</td>
</tr>
<tr>
<td>Temperatures</td>
<td>Toesophagus, Tleft toe, Tright toe, Tforearm, Tfinger tip</td>
</tr>
<tr>
<td>‘Lag time’</td>
<td>This was the time interval (min) between induction of anaesthesia (time ‘0’) and ‘time heating began’ (time at which convection warming started)</td>
</tr>
</tbody>
</table>

(b) Other indicators of ‘heat deficit’: (i) $T_{oesophagus}$ just before heating began and (ii) the ratio of the ‘lag time’ to the time taken to achieve $T_{LT} - T_{oesophagus} > 0$.

We make no claims that these measure heat deficit directly but rather than they reflect the individual’s thermal response to heat lost during the ‘lag time’ before heating commenced.

(2) Simple temperature measurements.

(a) $T_{oesophagus}$ ($^\circ$C) measured using a mid-oesophageal probe placed within a few minutes of induction of anaesthesia. As discussed above, we used $T_{oesophagus}$ in all analyses involving $T_{core}$.

(b) Raw heating rate ($^\circ$C min$^{-1}$) defined as $T_{oesophagus} - T_{oesophagus}$ at time heating began / time between the two temperatures.

(3) Thermodynamic calculations.

(a) $\Delta T_1$ ($^\circ$C), defined as $T_{LT} - T_{oesophagus}$. The time at which $T_{LT}$ becomes warmer than $T_{oesophagus}$. Time $\Delta T_1 > 0$ defines a point when the convective warming has halted the body from further cooling or from a heat transfer viewpoint, the heat flux has changed from negative to positive.

The six skin sites were analysed for their ability to predict the time interval for when skin temperature ($T_{skin}$) reached $T_{oesophagus}$. All gave similar results. We picked $T_{LT}$ to do this skin flux analysis since, although the maximum skin temperatures recorded here were marginally higher than elsewhere (presumably because the air temperature was highest here), it was the furthest from the core and overall took the longest to equal $T_{oesophagus}$.

(b) $\Delta T_2$ ($^\circ$C) defined as $T_{forearm} - T_{oesophagus}$. This measure was similar to $\Delta T_1$. However, while $T_{LT}$ was chosen for the skin flux assessment, $T_{forearm}$ was chosen for a more detailed thermodynamic analysis. Although initially based on prior research, our subsequent analysis confirmed $T_{forearm}$ to be the most stable skin site. We suspect that the relatively large amount of muscle in the forearm makes it less likely to have artifact from temperature changes in peripheral and core compartments than a toe where the blood supply is more reactive. Each child’s $\Delta T_2$ was integrated over time and graphed against $T_{oesophagus}$. The slopes of these lines reflect changes in the body’s response to the convective heating, for example, when the slope is downward directed, the body is still cooling, whereas when it turns upwards, the core is starting to warm. Changes in slope represent changes in the rate at which heat moves from the periphery to the core (from a thermodynamic viewpoint, a change in thermal resistance, $R_{thermal}$). A steeper slope corresponds to a higher thermal resistance and hence a longer time for the applied warmth to be reflected in $T_{core}$. We determined the slopes for different phases of heating and the relative change in slopes and assessed these against the ‘child characteristics’ (Table 1). Measuring the relative change in slope [by cancelling out other individual constants such as body mass and body surface area (BSA)] enabled comparison of phases between individuals, despite a relatively large difference in actual slope. Equation 5 in the Supplementary Appendix (available online) describes the relationship between the body’s apparent thermal properties and $T_{oesophagus}$.

The relationship between child and procedure characteristics and the thermal measures described above was analysed. We also analysed the relationship between the various vasoconstrictive and heat deficit markers and examined their validity and predictive value by looking at their impact on heating (using raw heating rates and time $\Delta T_1 > 0$). Predictive value was also analysed using the ‘child characteristics’. Statistical analysis was carried out with JMP version 7 (SAS Institute, NC, USA) using correlations, t-tests, ANOVA, and multiple linear regression, with significance assessed at the 0.05 level.

Modelling

The model is detailed in the Supplementary Appendix (available online) along with the relevant references. We devised a simple thermodynamic model to examine some of the mechanisms behind core body heating. We conceptualized a ‘infinite slab’ with a certain wall thickness composed of skin, fat, and muscle (periphery) and a liquid
centre (core)—each with its own thermal properties. We simplified the body’s total thermal resistance into a single number and changed this proportionally at certain temperatures to approximate the observed cooling, heating, and overheating phases. The model was designed in C++ programming language. It was never intended to represent the body’s response to heating. Rather, it is best understood as a way of attaching biological, physiological, and anatomical terms to thermodynamic ones and was primarily designed to illustrate the effect of heating on the body’s model’s thermal properties.

**Results**

Forty children were originally enrolled into the study. One child was heated from the head rather than foot end and since this could have affected ΔT1 and ΔT2 (and the calculations based on it), we excluded this child from the present analysis—even though raw heating rates were not different from the rest of the group. Patient data are summarized in Table 2. All had an inhalation induction of anaesthesia; GA was maintained with a variety of volatile agents using either spontaneous or positive pressure ventilation. The convective technique was used in a variety of ways to cool the 26 children who became hyperthermic (turned off, blowing ambient air, or blowing at ‘low’). We did not further analyse the cooling data as there were too many variations. Although all actions resulted in an immediate decrease in air temperature, leaving the Bair Hugger running but with no heating (i.e. blowing air at ambient) was the most effective cooling technique, result-
ing in decreases in T
skin of ~0.2–0.5°C min⁻¹ and T
oesophagus of 0.03–0.08°C min⁻¹.

**Thermal measures**

At the ‘time heating began’, there was no significant correlation between any heat deficit/vasoconstrictive marker and any child or procedure characteristic. In other words, there was no reason to suspect that any particular child or procedure characteristic would be associated with a greater heat deficit before heating. When all the child and procedure characteristics were analysed against thermal measures, the only significant correlations were between ‘raw heating rates’ and ‘child characteristics’. Significant univariate relationships were found for age, height, weight, and BSA. These correlated negatively with heating rate (i.e. younger children tended to warm faster) with r ranging from −0.52 to −0.59 (all P<0.001). These correlations were clearly statistically significant and moderately large. We explored fitting a statistical model with all four significant size-related variables using a stepwise approach to multiple linear regression. Once height entered the model as the first term (P<0.001), the remaining variables were no longer significant as potential second terms for the model, the strongest P-value among the remaining variables being 0.61 for age. This effect is due to the other three variables being strongly correlated with height—age and weight both with r=0.90 and BSA r=0.96, indicating they all represent body size in similar ways. It is unlikely that height was truly a better predictor than age, weight, and BSA as their univariate correlations were in a narrow range, well within the range of uncertainty expected for a correlation coefficient with a sample size of 39 [e.g. r=−0.60, 95% CI (−0.77, −0.35)].

**Predictive value**

On the basis of this, we used height alone to develop an equation which predicted the raw heating rate for an individual. We then used this to predict the time for an individual’s T
oesophagus (‘at time heating began’) to reach 37°C. The estimated uncertainty for this was ±28 min (95% prediction interval for an individual observation) at an average time of 60 min after heating began. This was considered too great to provide a reliable guide as to when the Bair Hugger air temperature should be reduced. Addition of the ‘heat deficit’ markers to this predictive model did not improve its accuracy.

**Markers of vasoconstriction/heat deficit**

None of these correlated significantly with raw heating rates or lag times. It is important to remember that the vasoconstrictive markers (T
LT−T
oesophagus, T
LT, T
Calft−T
RT, and T
forearm−T
finger tip) were calculated from measurements recorded immediately before commencing convective warming. Although time to ΔT1 >0 correlated with both (T
LT−T
oesophagus) (r=−0.79) and (T
Calft−T
RT) (r=0.50), only (T
Calft−T
RT) and (T
LT−T
oesophagus) correlated with each other (r=−0.80). T
oesophagus at ‘time heating began’ did not correlate significantly with time to ΔT1 >0, although, not surprisingly, it did correlate with the actual time to reach 37°C. Irrespective of the T
oesophagus ‘at time heating began’, it took a minimum of 40 min for a child’s T
oesophagus to reach 37°C. Only three children had forearm–fingertip differentials of ≥4°C—none of whose T
oesophagus (35.2–35.6°C) differed significantly from the group. Of those children with T
oesophagus <35°C before warming, no child demonstrated a forearm–fingertip or calf–toe differential of >2°C.

<table>
<thead>
<tr>
<th>Table 2 Patient data</th>
<th>Range</th>
<th>Mean</th>
<th>Inter-quartile range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>2 days–12.5 yr</td>
<td>2.5 yr</td>
<td>0.6–3.5 yr</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>33.5–153</td>
<td>82.8</td>
<td>68–94</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>2.1–72.7</td>
<td>14.7</td>
<td>8.2–15.2</td>
</tr>
<tr>
<td>BSA (m²)</td>
<td>0.15–1.8</td>
<td>0.57</td>
<td>0.41–0.64</td>
</tr>
</tbody>
</table>
| T
oesophagus (°C)     | 35.4–38.5 | 37.2 | 36.6–37.9 |
| Anaesthesia duration (min) | 91–590 | 187 | 140–190 |
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Thermodynamic modelling

When warming starts, the wall is cool and the temperature difference between wall and air is the most important factor. However, as the wall begins to warm, the ability to transfer that heat through the liquid centre (the ‘internal heat resistance’) becomes dominant. Hence, the rate-limiting step varies according to the relative temperatures of the wall (periphery) and liquid centre (core). The thermal properties represented by $R_{\text{thermal}}$ (blood flow and conduction through the skin) affected heating rate, whereas the time from commencement of heating until core temperature increases was significantly affected by differences in tissue thickness (e.g. skin, muscle, and fat).

Figure 3 demonstrates the large inter-individual variation in both magnitude and slope of a selection of the children’s heating curves. It also illustrates that some children have quite a large control over thermoregulation while under anaesthesia, and others do not. This suggests that while the basic thermal response for each child is similar, it remains unique to each individual. The reason for this most likely lies in individual characteristics such as skin thickness and composition, the subcutaneous blood flow and capillary network, and overall body mass, composition, and shape. The constants in our C++ thermodynamic model greatly influenced the predicted heating curves. When we varied these constants, we were able to approximate actual temperature profiles. Figure 4 exemplifies the result of this process (i.e. varying the constants)—the predicted and actual curves are from the same patient (Fig. 2). However, each patient required a different set of adjustments to the constants and they could not be predicted from knowledge of expected thermal properties, patient characteristic, or morphological data. It was clear that no average population values could be applied and an overall model could not be described because the variation between individuals was too great.

Discussion

There is no clear evidence for significant physiological harm from a decrease in $T_{\text{core}}$ to 35°C in children under anaesthesia, provided they can be rewarmed to normal within a few hours.\(^8\) Despite this, current clinical practice is to maintain normothermia in most clinical settings.\(^9\) The Bair Hugger system has been the most widely used for this. Our report, using a modification of the technique, remains the largest and most comprehensive to date in children.\(^2\) We are aware that other modifications of convection heaters without the manufacturer’s blankets are utilized. We caution about using techniques that have not been tested in a clinical trial. We identified hyperthermia as a risk using convective warming and attempted to develop guidelines to predict which children were more likely to become hyperthermic, when that was likely to happen, or both.

\(\Delta T_2\) — The body’s thermal resistance, was examined over the course of heating. Figure 2 shows the thermal integration of $\Delta T_2$ with respect to time for a 4-yr-old boy. The graph demonstrates three very different phases: initial cooling (where the slope is downwards directed), heating (where the slope increases), and overheating (where the gradient of the slope decreases). The initial slope (cooling phase) is the steepest of the three in the figure. This reflects the body’s high resistance to heat flow at this time. Despite being actively warmed by the system the entire time, initial cooling continues because $T_{\text{forearm}}$ is below $T_{\text{oesophagus}}$ and hence heat is still being transferred out of the body. As the body continues being warmed, $T_{\text{forearm}}$ increases and eventually equals $T_{\text{oesophagus}}$. The heat flux changes direction and $T_{\text{oesophagus}}$ now begins to increase. The cooling phase slope is lowered by ~75% when changing to the heating phase. The curve continues to increase in a linear fashion until $T_{\text{oesophagus}}$ reaches 37°C, at which time the slope decreases further. The lowered thermal resistance allows greater heat flow into the body and makes the body more likely to overheat. Not all children’s integrated curves were as well defined as this example, but the majority exhibited triphasic patterns and the remainder were biphasic. These patterns were clearly recognizable, although the slopes varied considerably between individuals, that is, the individual rates of cooling and warming were different.

The mean slopes for the three phases (cooling, heating, and overheating) were 3.7, 1.7, and 1.1, respectively. These were all statistically significantly different from each other $(P<0.001)$. When examined against ‘child’ and ‘procedure’ variables, only two relationships were identified: between the overheating phase and height ($r=–0.54$, $P=0.03$), and between the cooling phase and procedure stratification, the peripheral group having higher cooling rates than the two open groups $(P=0.02)$. No significant correlations were found between the relative changes in slopes and ‘child’ or ‘procedure’ variables.
Defining a starting point is difficult in clinical studies of this type. For example, having equal lag times does not guarantee the same heat deficit, despite the fact that $T_{oesophagus}$ may well be the same. Nevertheless, given the currently available measures of heat deficit, we could find no reason to suspect that any particular child or procedure characteristic would be associated with a greater heat deficit before heating. As expected, we found significant univariate relationships for age, height, weight, and BSA, with younger children tending to warm faster than older children. Since the other variables strongly correlated with height, we used height alone to predict the time for an individual’s $T_{oesophagus}$ to reach 37°C. However, the estimated uncertainty for this (±28 min) made it an unreliable guide as to when to change the Bair Hugger temperature. The ‘heat deficit’ markers did not correlate significantly with raw heating rates or lag times. It was not surprising, therefore, that they did not improve the accuracy when added to the predictive model.

Children demonstrate a three phase response to convective heating: cooling, warming, and overheating. The mean slopes for the three phases are significantly different from each other as are individual rates of cooling and warming. No significant correlations were found between the relative changes in slopes and ‘child’ or ‘procedure’ variables. The constants in our thermodynamic model greatly influenced the predicted heating curves. However, the constants for an individual could not be predicted from knowledge of expected thermal properties, patient characteristic, or morphological data.

We did not find support for usage of any of the vasoconstriction/heat deficit markers (either literature-derived or our putative measures). Rubinstein and Sessler\(^5\) defined vasoconstriction as 4°C difference between forearm and fingertip skin temperatures. Using this definition, several authors\(^4–6\) have designated ‘vasoconstriction’ at $T_{core}$ values ranging from 34.3°C to 36°C and ‘mean skin temperatures’ (derived from multiple weighted sites) ranging from 33.3°C to 34.9°C. At the onset of warming, our children had mean temperatures comparable with the above: $T_{oesophagus}$ 35.5°C, $T_{LT}$ 31°C, and $T_{forearm}$ 33.5°C. We accept that $T_{oesophagus}$ is not a reliable index of thermal balance (as suggested by Webb)\(^10\) and that its significance can only be inferred in the context of the clinical situation. Nevertheless, time to normothermia and raw heating rates were independent of these vasoconstrictive markers, despite ‘preheating’ temperatures similar to those in the literature and our children’s integrated heating graphs showing them to be in a cooling phase at that time. Since redistribution of blood flow in response to hypothermia occurs first at muscle level and thence skin,\(^11,12\) group morphological differences between the children in the above reports and our study might explain the discrepancies. Although possible, this seems unlikely. We believe that either these markers are poor indices of heat deficit or, alternatively, the combination of an effective

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**Fig 3** Inter-individual variation in both magnitude and slope of a selection of the children’s heating curves.

**Fig 4** The actual and model predicted temperature curves for the patient in Figure 2. Note that the modelled curves result from fitting thermodynamic theory to patient response.

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thermoregulatory response by the body and effective heating means they are superfluous. Nevertheless, accurate measurement of fat, lean body mass, and their distribution will be important in future studies.

Although younger children tended to have faster heating rates, neither age nor morphology has good predictive value for time when $T_{\text{oesophagus}}$ will return to 37°C. This is because the response to heating is not linear, and there are significant inter-individual variations in thermal behaviour. Hence, raw heating rates, although valuable, do not predict time to normothermia. Unfortunately, there are no child or thermal markers available to the anaesthetist at commencement of warming by which to set the ‘turn down time’ for this system. Ongoing calculation of $R_{\text{thermal}}$ is the obvious but impractical solution.

Previous literature has reported body morphology (height, weight, % body fat, etc.) to determine the rate of both warming and cooling. Szmuk and colleagues found that morphology significantly influenced warming. Our results were not quite so dramatic. It is possible that the differences in BSA/weight ratios of our children were not sufficiently different from each other to influence heating rates. This seems unlikely since they ranged from 0.025 to 0.079 with over half having a BSA/weight ratio higher than the highest recorded in the report of Szmuk and colleagues. Although Szmuk’s group were certainly colder than ours at start of rewarming, the most likely explanation for the difference in conclusions between Szmuk and ourselves is simply that we chose both correlation and predictive time to normothermia (as opposed to simple correlation). We did this, as our goal was to provide guidelines for when to reduce the heating rate to prevent hyperthermia. However, the uncertainty in the prediction (±28 min) was deemed too large to be reliable. There is conflicting evidence regarding morphology and cooling. Positive correlations between decreases in $T_{\text{core}}$ and low body fat have been found in adults. Unfortunately, the above studies only derived body fat from population-based estimates. Tikuisis and colleagues found that neither weight nor BSA was relevant in adults when body fat could be accurately measured. Other authors have found cooling to be more likely related to muscle mass and its distribution. Since we did not measure either fat or muscle mass, we cannot say if these would be correlated. This should be examined in future studies.

**Thermodynamic modelling**

All attempts at modelling the human thermoregulatory system have suffered from being insufficiently complex, confounded by the considerable variations among individuals or based on unknown or unquantifiable inter-relations. The rationale behind our reasonably simple model was to determine which thermal properties might have most impact on heating and cooling and hence direct our future research efforts. The model did not include control pathways and was therefore never envisaged as a model of a child’s response to heating. A complete discussion of the thermodynamic principles on which much of the subsequent discussion is based is described in the Supplementary Appendix (available online). Thermoregulation is made up of two main components: the physical structure (skin, muscle, fat, blood supply, etc.) and the body’s individual control of it. Our model demonstrates that the thermal properties represented by $R_{\text{thermal}}$ (e.g. blood flow and conduction through the skin) affect heating rate, whereas the time from commencement of heating until core temperature increases is significantly affected by differences (actual or functional) in tissue thickness (e.g. skin, muscle, and fat). Since skin thickness itself is not greatly variable at different ages, age-related and individual differences in circulation and tissue distribution/volume/thickness are likely to have a far greater impact on heat transfer rather than traditional indices.

A child with active thermoregulation is able to quickly identify when body heat is being lost or gained from their surrounds and manipulate their ‘apparent’ thermal properties relative to the environment. It remains unclear whether the trigger for these thermoregulatory changes is heat quantity or an actual temperature. Both human and animal studies have shown significant inter-individual variations in the threshold response to thermal manipulation. We have shown that, even under GA, the apparent thermal properties of the body (i.e. the internal thermal resistance) change with the body’s $T_{\text{core}}$.

Heat redistribution has been suggested as the mechanism for the initial decrease in $T_{\text{core}}$ after induction of anaesthesia. We did not completely explore this. However, our analysis shows that despite the absence of good vasoconstrictive markers, the body is actively increasing thermal resistance during initial cooling from induction. We have assumed this response results from both: (i) a reduction in blood supply to the peripheries (including surface capillaries), thereby increasing the effective thickness of the skin and muscle layer (an increased wall thickness in our model) and (ii) the reduced blood flow decreases the internal convection heat transfer coefficient. When heating is applied, the external heat transfer coefficient increases. The body senses a reversal of heat flow (from out to in) and begins to increase the blood flow back to the periphery, thus lowering thermal resistance. As the body reaches thermoneutrality, it actively tries to shed excess heat both by increasing peripheral blood flow further (thereby further lowering thermal resistance) and, possibly, sweating. This increased blood flow close to the skin surface directs more heat to the body core. When convection heating is maintained beyond thermoneutrality, the lowered $R_{\text{thermal}}$ results in the sudden overheating observed clinically.

In conclusion, our convection warming technique is effective in keeping children warm under anaesthesia without the need to increase ambient temperature above 21°C. However, there is no simple child characteristic or
Thermal measure (either singly or in combination), which predicts time to normothermia in an individual child using this technique. Previously described measures of vasoconstriction are not valid in children in an operating theatre environment and children’s apparent thermal properties change with their $T_{core}$, even under anaesthesia. Although we were able to identify some thermal properties which determined heating rate, we were unable to either quantify them or estimate their relative importance. Hence, we were unable to satisfactorily predict time to normothermia. Thermodynamic theory and our model explain the three broad changes in heating rates observed in children using our convective warming technique. Future research will need to include measurement of these properties in study design. We also conclude that the critical time to alter the temperature of the convection heater is during the heating phase, before thermoneutrality. Until we have a better understanding of the processes, we recommend that the Bair Hugger air temperature should be turned to high (43°C) at the outset and then to medium (38°C) when the $T_{oesophagus}$ approaches 37°C. Further study will be required to determine if even this would result in an overshoot. We recommend continuous measurement and close monitoring to determine if even this would result in an overshoot.

References


Supplementary material

Supplementary material is available at British Journal of Anaesthesia online.