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ORIGINAL RESEARCH

Core temperature following pre-hospital induction of anaesthesia in trauma patients

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Abstract

Introduction: Hypothermia is a well-recognised finding in trauma patients, which can occur even in warmer climates. It is an independent predictor of increased morbidity and mortality. It is associated with pre-hospital intubation, although the reasons for this are likely to be multifactorial. Core temperature drop after induction of anaesthesia is a well-known phenomenon in the context of elective surgery, and the mechanisms of this are well established.

Methods: We conducted a prospective observational study to examine the behaviour of core temperature in patients undergoing pre-hospital anaesthesia for traumatic injuries.

Results: Between 2017 and 2021 data were collected on 48 patients. The data from 40 of these were included in the final analysis.

Discussion: Our data do not show a decrease in the core temperatures of patients who receive pre-hospital anaesthesia, unlike patients who are anaesthetised without pre-warming, in operating theatres. The lack of a change could relate to patient, anaesthetic or environmental factors.

Key words: *anaesthesia, pre-hospital, temperature.*

Key findings

- Our data do not demonstrate a significant decrease in the core temperatures of patients following induction of pre-hospital anaesthesia.
- There is no evidence of heat redistribution in patients receiving pre-hospital anaesthesia within a well governed pre-hospital medicine service.
- Even in temperate climates there is a significant rate of preexisting hypothermia in critically ill trauma patients requiring pre-hospital anaesthesia.

Introduction

Hypothermia is a well-recognised finding in trauma patients,[1,2] which can occur even in warmer climates.[3] It is an independent predictor of increased morbidity and mortality[2–8]; any potential protective effects of hypothermia in traumatic brain injury are overwhelmed by its negative effects.[9] It inhibits blood coagulation, and is associated with increased transfusion requirements.[10,11] In non-anaesthetised patients hypothermia increases cardiac and metabolic demand. Along with acidaemia and coagulopathy, it is a member of the 'lethal triad'[12] or, more recently with the addition of hypocalcaemia, the 'lethal diamond' of risk factors in trauma.[13,14] It is associated with pre-hospital intubation,[2,15,16] but the relationship with pre-hospital anaesthesia is less clear. Previous studies have included a variety of patients in the intubation group: non-drug-assisted intubations, muscle relaxant only intubations and some true pre-hospital anaesthesia (the administration of an induction dose of hypnotic and a muscle relaxant to facilitate intubation). Pre-hospital interventions include the prevention and treatment of hypothermia as major therapeutic priorities.

Core temperature drop following induction of anaesthesia is well recognised in the context of elective surgery, where hypothermia is associated with adverse outcomes, including increased blood loss, wound infection and myocardial ischaemia and infarction.[17] Hypothermia during anaesthesia is due to multiple mechanisms. Firstly, there is redistribution of heat from the patient's core to their periphery,[18] consequent to peripheral vasodilation in arteriovenous shunts caused by anaesthetic drugs that lower the temperature threshold for vasoconstriction,[19] and also have a direct vasodilatory effect, propofol in particular.[20] This redistribution is mainly responsible for the initial core temperature decrease, which can be 1.0–1.5°C, in the 30–40 min following induction of anaesthesia. Secondly, a drop in metabolic heat production contributes to a second, slower phase

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of core temperature decrease. Finally, a state of dynamic equilibrium is reached wherein heat generation by the body matches ongoing heat loss to the environment, which is principally (90%) by radiation and convection.[21] There are significant differences between elective in-hospital and pre-hospital anaesthesia that may affect redistribution. Choice of induction drug, a greater degree of shock and a reduced depth of maintenance may limit the degree of vasodilation and mitigate temperature change due to redistribution in pre-hospital patients. Conversely, we would expect a proportion of pre-hospital patients to have significantly colder peripheral compartments than in-hospital patients, which would be expected to exacerbate the early temperature change.

Pre-warming of patients, even for 10–20 min, prior to induction of anaesthesia prevents the core temperature drop due to heat redistribution, and is far superior to attempting to warm the patient after induction, when redistribution has already taken place.[22–25] Methods include use of a forced-air warming blanket,[23,26,27] electric resistive under-body warmers,[28,29] circulating-water devices[30] or chemically heated blankets.[31] Chemical warming blankets are a compact and convenient device for use in the pre-hospital environment,[32,33] which is necessarily austere and where the patient may have been exposed to low ambient temperatures.

MedSTAR is the pre-hospital and medical retrieval service of the South Australian Ambulance Service (SAAS). Together with the SAAS Special Operations Team (SOT), it attends pre-hospital ('primary') trauma scenes, which are generally reached by road vehicle or helicopter. For patients requiring pre-hospital anaesthesia, standard induction drugs include ketamine and rocuronium, with or without fentanyl; maintenance is either with boluses or infusion of ketamine, or boluses or infusion of propofol or fentanyl/midazolam, depending on circumstance.

Usually, the trauma scene is initially attended by a SAAS road crew, pending arrival of the MedSTAR/SOT team. There is a potential advantage to supplying these crews with chemically powered warming blankets for pre-warming the patient, in anticipation of the need for pre-hospital anaesthesia. However, these blankets also comprise a financial cost, and their application adds to the cognitive demand on the first crew of managing a critically injured patient, as well as potentially affecting access to the patient for assessment and treatment.

We therefore wished to know if our trauma patients undergoing pre-hospital anaesthesia exhibited the same core temperature drop as elective surgical patients. We undertook a prospective, observational study examining core temperature behaviour in adult, primary trauma patients receiving pre-hospital anaesthesia, aiming to replicate the in-hospital studies to see if the core temperature behaves consistently with redistribution associated with induction of anaesthesia. The study was planned as a pilot for a follow-up study looking at the impact of pre-warming critically injured adults prior to pre-hospital anaesthesia.

Materials and methods

Approval for the present study was granted by the Human Research and Ethics Committee of the Southern Adelaide Local Health Network, according to the criteria set out in the National Statement on Ethical Conduct in Human Research, of the National Health and Medical Research Council. The requirement for informed consent was waived, consistent with section 2.3.10 of this statement.

We recorded temperature data between 2017 and 2021. Patients were adult (16 years or older) primary trauma patients attended by MedSTAR, and who needed pre-hospital induction of anaesthesia. The only exclusion criterion would be an absolute contraindication to core temperature monitoring; however, we include this here as a formality: we are not aware of any such contraindications in practice, and in fact temperature monitoring is considered a normal standard of care for trauma patients.

There were no changes to our standard operating procedure (SOP) for pre-hospital anaesthesia, which follows a rapid sequence induction. Preoxygenation is through a bag/mask, with apnoeic oxygenation through nasal cannulae. The standard induction agent is ketamine at 1–2 mg/kg, with or without fentanyl at 1–2 μg/kg; paralysis is with rocuronium at 1–1.5 mg/kg. Use of a video laryngoscope and bougie is standard. Following induction, maintenance of anaesthesia may employ ketamine boluses or infusion, propofol or fentanyl/midazolam. Temperatures were recorded using a Zoll X series monitor (ZOLL Medical Corporation, Chelmsford, MA, USA); the monitors used were maintained and calibrated for clinical use. A temperature probe was placed in the patient's nasopharynx or oropharynx, and connected to the T1 jack of the Zoll monitor. A second temperature probe was connected to the T2 jack to measure ambient temperature. Times of intubation, departure from the scene itself, closing of the aircraft doors where applicable and arrival at the receiving trauma centre are recorded as part of our standard procedures.

After completion of the mission, data recorded by the monitor were printed out and entered into a spreadsheet (Microsoft Excel, version 2108), before being analysed and plotted using RStudio (version 1.1.456). We analysed data from patients for whom at least five temperature readings were taken in the first 45 min, and for whom the first reading was taken no later than 25 min post-intubation. We chose this interval because it covers the time of greatest temperature drop following induction of anaesthesia, as seen in previous, in-hospital studies. We calculated descriptive statistics for the core temperature at 5 min intervals from induction, and the gradient of the plot of temperature *versus* time, as calculated by the least-squares method. For patients who had not had a temperature recorded at induction we took the calculated y-axis ($t = 0$) intercept of the temperature *versus* time graph as the starting temperature. The first recorded temperature was not included if the subsequent temperature was more than 1°C higher, to ensure that the temperature probe had reached

equilibrium with the patient. Thereafter, isolated temperatures more than 2°C different from the mean were disregarded as they were considered physiologically implausible and likely to represent artefacts. We calculated the mean and standard error both for the individual time points, and for the slope of the temperature–time plot. We also calculated 95% confidence intervals (CIs), based on Student's *t* distribution. In addition to calculating temperature change for the patient sample overall, we compared the rate of change between patient subgroups determined by the presence or absence of (i) active warming; (ii) propofol administration; (iii) hypothermia (<35°C); and (iv) shock (shock index [SI] ≥ 1), using a two-tailed Welch's *t* test, where $P < 0.05$ was considered significant.

Results

We collected data from 48 patients. Eight patients were removed from the study sample based on these criteria: four because fewer than five

TABLE 1. *Summary of baseline characteristics*

	Hypothermic ($n = 15$)	Normothermic ($n = 25$)	Overall ($n = 40$)
Temperature (°C)			
Mean (SD)	34.2 (0.826)	36.0 (0.682)	35.3 (1.15)
Median (min, max]	34.5 (32.3, 34.9)	36.0 (35.1, 37.5)	35.4 (32.3, 37.5)
Age			
Mean (SD)	40.4 (21.0)	40.6 (16.3)	40.5 (18.0)
Median (min, max)	31.0 (16.0, 76.0)	34.0 (19.0, 77.0)	33.0 (16.0, 77.0)
Sex			
Female	5 (33.3%)	8 (32.0%)	13 (32.5%)
Male	10 (66.7%)	17 (68.0%)	27 (67.5%)
Ambient temp. (°C)			
Mean (SD)	19.3 (6.91)	19.0 (4.94)	19.1 (5.69)
Median (min, max]	19.8 (6.80, 30.0)	19.9 (9.80, 25.8)	19.8 (6.80, 30.0)
Missing	0 (0%)	1 (4.0%)	1 (2.5%)
Initial shock index (HR/SBP)			
Mean (SD)	0.868 (0.530)	0.914 (0.383)	0.897 (0.438)
Median (min, max)	0.740 (0.30, 1.99)	0.840 (0.390, 1.75)	0.790 (0.300, 1.99)
Chemical warming blanket			
None	3 (20.0%)	12 (48.0%)	15 (37.5%)
Post-arrival	11 (73.3%)	12 (48.0%)	23 (57.5%)
Pre-arrival	1 (6.7%)	1 (4.0%)	2 (5.0%)
Mechanism			
Blunt	13 (86.7%)	25 (100%)	38 (95.0%)
Immersion	1 (6.7%)	0 (0%)	1 (2.5%)
Penetrating	1 (6.7%)	0 (0%)	1 (2.5%)
Platform			
Rotary wing	11 (73.3%)	12 (48.0%)	23 (57.5%)
Road	4 (26.7%)	13 (52.0%)	17 (42.5%)
Time			
Day	10 (66.7%)	15 (60.0%)	25 (62.5%)
Night	5 (33.3%)	10 (40.0%)	15 (37.5%)
Propofol			
No	10 (66.7%)	6 (24.0%)	16 (40.0%)
Yes	5 (33.3%)	19 (76.0%)	24 (60.0%)

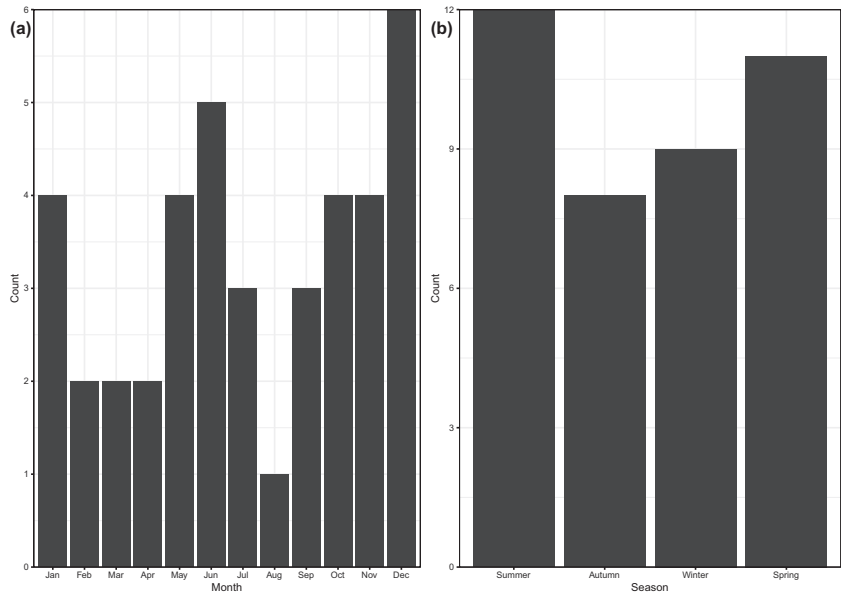

Figure 1. *Distribution of cases by time of year. (a) Grouped by month. (b) Grouped by season.*

temperature readings were recorded for them, and three because their temperature recordings started later than 25 min post-intubation. Another patient was excluded because of recurrent, excessive variability in the temperature readings (range 22.5–32.5°C), which was evidently the result of equipment malfunction. The patient characteristics for the remaining 40 patients are presented in Table 1. Figure 1 shows grouping by time of year. There was some variation in the number of cases recorded for each calendar month (Fig. 1a), but less when they were grouped by season (Fig. 1b).

For practical reasons relating to the workflow around intubation and resuscitation, there were differences between patients in the starting points for the temperature series, relative to intubation time, and the duration of data collection varied between patients depending on time to the receiving trauma centre. The crude plots of temperature *versus* time for these patients are shown in Figure 2a. The number of temperature readings at each 5-min point post-intubation are plotted in Figure 2b. There were a few outlying data points; we discarded four data points across the entire data set that differed by more than 2°C (lower) from the mean for that patient. These were most likely due to temporary probe dislodgement.

We calculated the temperature/time gradient for each patient as the slope of the least-squares line of best fit across the first 45 min from intubation. The mean value of this slope was 0.0092°C/min (95% CI 0.0026–0.0158). Therefore, on average, there was a slight rise of the patients' core temperatures following induction of anaesthesia. By determining the *y*-intercept of the temperature *versus* time graph for each patient, it was possible to calculate the change in temperature from this baseline for each 5-min time point. The very slight gradient of the temperature plot is reassuring that there would be very little error in the estimate of initial temperature. The mean changes at each point are plotted in Figure 3; again, there was a rise in core temperature which peaked at 0.47°C (95% CI 0.21–0.73) at 40 min post-intubation.

We compared the rate of temperature change between defined subgroups. There was no significant difference between patients who were actively warmed with a chemically powered blanket (25 patients) and those who received no active warming (15 patients) (Fig. 4a). Although all our patients received ketamine as the main induction agent, some subsequently received propofol as a bolus or as an infusion. Our data showed no significant difference between those who received propofol at some point in the retrieval (22 patients), and those who did not (18 patients) (Fig. 4b).

We found a significant difference between patients who were hypothermic (initial temperature, as determined by the *y*-intercept of their

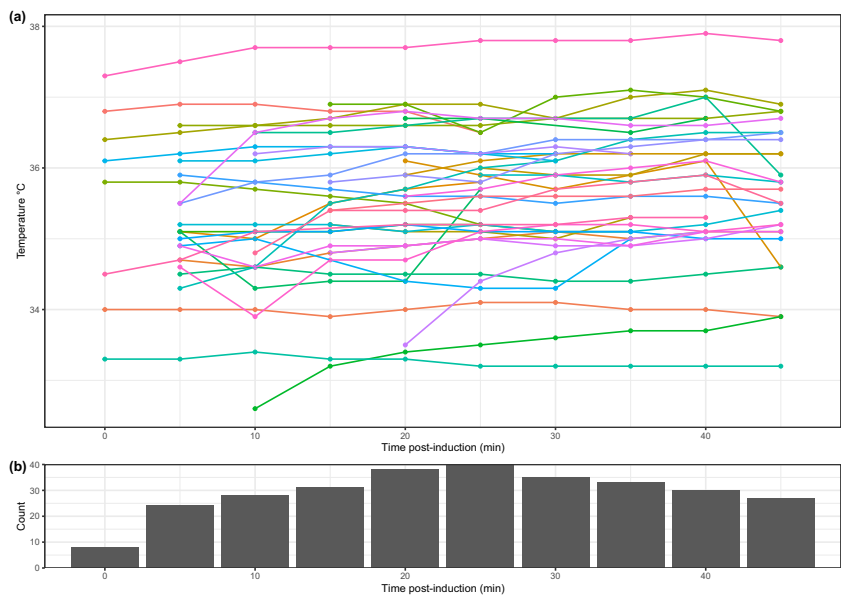

Figure 2. *Raw data. (a) Raw temperature data for individual patients. (b) Number of observations available for each 5-min time period post-induction.*

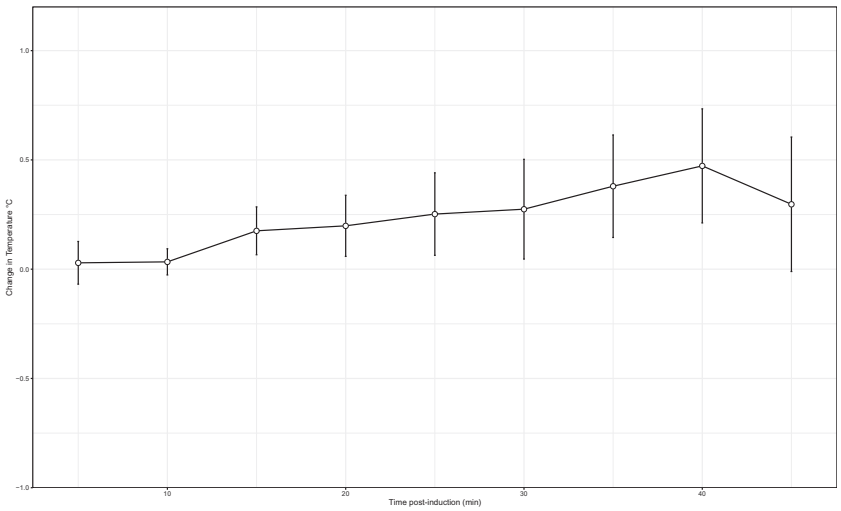

Figure 3. *Mean change in core temperature following induction of anaesthesia (error bars represent 95% confidence interval).*

temperature plot, less than 35°C) (15 patients), with those whose who were not (25 patients), with the former warming more ($P = 0.014$) (Fig. 4c).

Finally, we looked for any association of shock with temperature change post-induction, using SI as a discriminant. SI is calculated as the heart rate divided by the systolic BP; a higher ratio implies a greater degree of shock. We compared those with a SI greater than or equal to 1 (shocked, 12 patients) with those whose SI was less than 1 (not shocked, 28 patients). There was no significant difference between the two groups (Fig. 4d).

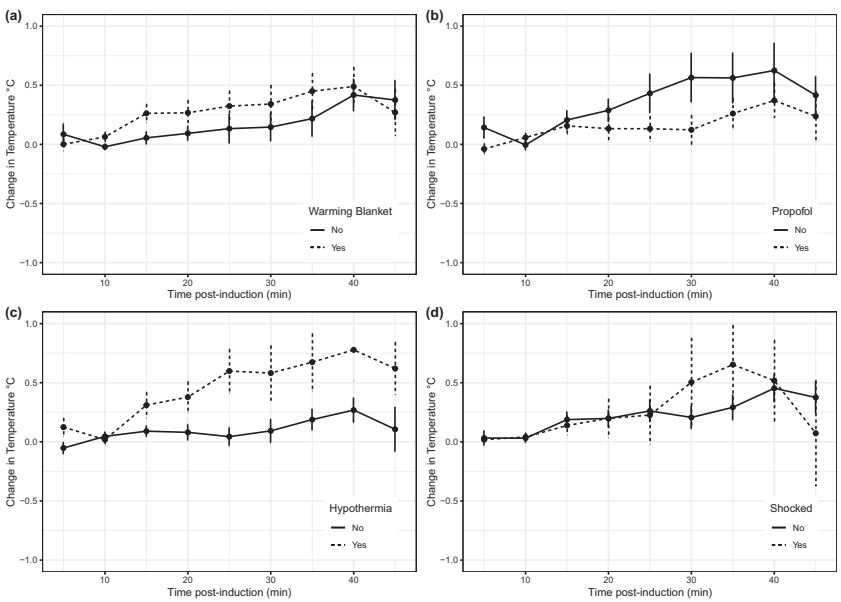

Figure 4. *Impact of different variables on temperature change. Error bars represent standard error of the mean (SEM) for all graphs. (a) Change in core temperature following induction, grouped by warming blanket use. (b) Change in core temperature following induction, grouped by propofol exposure. (c) Change in core temperature following induction, grouped by initial hypothermia. (d) Change in core temperature following induction, grouped by the presence of shock (shock index > 1) at retrieval team arrival.*

Discussion

Our data do not demonstrate a significant change overall in the core temperatures of patients following induction of pre-hospital anaesthesia, unlike patients who are electively anaesthetised without pre-warming, in operating theatres. They suggest that the post-induction heat redistribution, with ensuing core temperature drop, that is ubiquitous in elective patients without pre-warming-does not occur in pre-hospital trauma patients. The presence of propofol, shock, active warming or hypothermia made no difference to this finding. Patients who were initially hypothermic showed an increased rate of temperature rise compared to those who were normothermic; this does not appear to be explained by the use of active warming.

One explanation for the lack of heat redistribution is the difference in the type and doses of anaesthetic agents used for severely injured patients. Our SOP for induction of anaesthesia specifies the use of ketamine and rocuronium, with or without fentanyl depending on clinician judgement. Ketamine's activation of the sympathetic nervous system protects cardiac output and BP,[34] and it causes much less heat redistribution than propofol.[35] Ketamine, with or without fentanyl, was used as the induction agent for all the patients in our study. Twenty-four patients subsequently received propofol either as a bolus,[2] infusion[20] or both.[2] There was no difference in core temperature behaviour between these patients and those who never received propofol. Importantly, propofol, where used here for maintenance, was given at rates to maintain sedation rather than full general anaesthesia.

Based on the results of the present study, we would only advise the application of active warming prior to the arrival of a retrieval team where the patient was already hypothermic, or in danger of becoming so due to environmental factors, and not simply because we expected a need for anaesthesia.

Our study has limitations. Firstly, the sample size is small; this may reflect the complex and dynamic nature of patient management in the pre-hospital trauma setting, where more immediate priorities may supervene over temperature measurement; this was further complicated by the COVID-19 pandemic. Additionally, a short transfer time to hospital precluded, in some cases, the collection of an adequate set of time points for the purposes of the present study. However, we can identify no reasons why the sample that we did obtain should be unrepresentative of temperature behaviour in anaesthetised patients generally. We note that several of the original papers examining the behaviour of core temperature in individuals anaesthetised in a controlled environment used even smaller numbers of subjects, yet were still able to show a very consistent significant decrease in the 30 min following induction.[18,20,22–24] Secondly, where comparisons are involved (warming blanket, propofol use, hypothermia, shock) it is a retrospective study, with potential for confounding. Thirdly, again due to the cognitive and task demands associated with the environment and clinical situation, not all the temperature series begin immediately at induction of anaesthesia. Nevertheless, the series that we did obtain covered the interval of concern for heat redistribution. None of the patients showed a temperature drop; in fact, there was a tendency to a slight rise in temperature. Finally, the present study was conducted in the relatively temperate climate of South Australia; possibly, different temperature patterns would be observed in a colder location. Even so, 15 (37.5%) out of 40 of our patients were hypothermic.

Although we have found no evidence for a temperature drop following induction of anaesthesia in pre-hospital trauma patients, this does not eliminate the need for temperature monitoring and management in these patients. It is important to reiterate that the patients in the present study received active temperature management as described in the methods section. The avoidance and treatment of hypothermia is a well-accepted component of trauma management, and it will be important to determine the utility of active warming, applied by either first responders or retrieval teams, in this situation. Ideally, this should be addressed by a prospective, randomised, controlled clinical trial conducted in a multicentre setting to include areas with lower ambient temperatures.

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Competing interests

None declared.

Data availability statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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