ORIGINAL ARTICLE

Body temperature, cutaneous heat loss and skin blood flow during epidural anaesthesia for emergency caesarean section.*

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Summary

It is not clear how converting epidural analgesia for labour to epidural anaesthesia for emergency caesarean section affects either cutaneous vasomotor tone or mean body temperature. We hypothesised that topping-up a labour epidural blocks active cutaneous vasodilation (cutaneous heat loss and skin blood flow decrease), and that as a result mean body temperature increases. Twenty women in established labour had body temperature, cutaneous heat loss and skin blood flow recorded before and after epidural top-up for emergency caesarean section. Changes over time were analysed with repeated measures ANOVA. Mean (SD) mean body temperature was 36.8 (0.5)°C at epidural top-up and 36.9 (0.6)°C at delivery. Between epidural top-up and delivery, the mean (SD) rate of increase in mean body temperature was 0.5 (0.5) °C.h^{-1}. Following epidural top-up, chest ($p < 0.001$) and forearm ($p = 0.004$) heat loss decreased, but head ($p = 0.05$), thigh ($p = 0.79$) and calf ($p = 1.00$) heat loss did not change. The mean (SD) decrease in heat loss was 15 (19)% ($p < 0.001$). Neither arm ($p = 0.06$) nor thigh ($p = 0.10$) skin blood flow changed following epidural top-up. Despite the lack of change in skin blood flow, the most plausible explanation for the reduction in heat loss and the increase in mean body temperature is blockade of active cutaneous vasodilation. It is possible that a similar mechanism is responsible for the hyperthermia associated with labour epidural analgesia.

Introduction

Maintenance of normal maternal body temperature in the peripartum period is paramount, as both hypo- (surgical site infection, coagulopathy [1]) and hyperthermia (neonatal neurological dysfunction [2]) are associated with adverse outcomes. Neuraxial blockade interrupts the sympathetic supply to the cutaneous vasculature, and as a result the ability to regulate body temperature is decreased [3]. A concentrated local anaesthetic top-up of an epidural sited for labour analgesia is a common method of providing anaesthesia for emergency caesarean section [4]. However, because of the unique dual sympathetic supply of the cutaneous circulation [5], the effect of this method upon body temperature is not clear.

Cutaneous vasomotor tone is regulated by two branches of the sympathetic nervous system, the noradrenergic 'active vasoconstriction' pathway and the cholinergic 'active vasodilation' pathway [5]. In normothermic and hypothermic conditions, skin blood flow is exclusively regulated by noradrenergic active vasoconstriction, but in hyperthermic conditions cholinergic active vasodilation is responsible for up to 80% of the increase in skin blood flow [5]. Body temperature prior to neural blockade is therefore the factor that determines whether sympatholysis results in vasodilation or vasoconstriction [3, 5]. In normothermic, non-pregnant individuals epidural anaesthesia blocks active vasoconstriction, and as a result cutaneous heat loss increases and mean body temperature decreases [3]. A similar pattern of temperature change accompanies epidural anaesthesia for elective caesarean section [6]. However, during labour heat production is increased [7] and epidural analgesia for labour is associated with hyperthermia [8]. It is possible therefore that topping-up a labour epidural for an emergency caesarean section blocks active cutaneous vasodilation, and as a result heat loss is limited and mean body temperature rises.

Cutaneous heat loss and skin blood flow are two indices of cutaneous vasomotor tone, both of which may be used to infer cutaneous sympathetic nerve activity [9]. Following neuraxial blockade there are four potential explanations for a reduction in cutaneous heat loss or skin blood flow:

thermoregulatory vasoconstriction (a physiological response to a reduction in body temperature) [5]; baroreceptor mediated reflex vasoconstriction (a physiological response to a reduction in mean blood pressure) [10]; non-thermoregulatory vasoconstriction (a mechanism of increasing body temperature to an elevated set-point during fever) [11]; and blockade of active cutaneous vasodilation [5]. The explanations are distinguished by the area of the body surface in which the changes are observed, and the associated variation in body temperature and blood pressure.

This mechanistic observational study aimed to determine how topping-up epidural anaesthesia for emergency caesarean section affects cutaneous vasomotor tone and mean body temperature. It was hypothesised that active cutaneous vasodilation is blocked (cutaneous heat loss and skin blood flow decrease), and as a result mean body temperature increases.

Methods

Ethical approval was obtained from the London – Fulham NRES Committee. All women gave their written informed consent in accordance with the declaration of Helsinki. Women were identified in the delivery suite following epidural insertion for labour analgesia. Following identification, women were provided with the patient information sheet and had the opportunity to discuss study participation with a researcher. Consent was obtained after the duty obstetrician had obtained consent for emergency (category 2 or 3 [12]) caesarean section. Women had a minimum of 1 h to consider study participation before recruitment, and were pain free throughout the consent process. The inclusion criteria were age ≥ 18 years, established labour (regular contractions resulting in cervical dilation of ≥ 4 cm), and effective epidural analgesia (boluses of 0.1% levobupivacaine plus 2 $\mu g.ml^{-1}$ fentanyl, administered by a midwife). Women were not included if they had cardiovascular or neurological disease, pre-eclampsia, suspected intrapartum infection treated with antibiotics, or haemorrhage > 1500 ml / requiring blood transfusion.

Before surgery women dressed in a hospital gown and knee height thromboembolic deterrent stockings. The temperature of the operating theatre air conditioning system (eTCP, Starkstrom, Ruislip, UK) was set at 22°C. Following transfer to theatre women lay supine with 15° left lateral table tilt whilst the study apparatus was attached. Core temperature was recorded with a tympanic T-type thermocouple (TTS-TC; Smiths Medical, Ashford, UK). Skin temperature was recorded at seven sites on the woman's left-hand side (head, chest, forearm, index finger, thigh, calf and great toe) with T-type thermocouples (Sable Systems, Las Vegas, NV, USA). All thermocouples were connected to a self-calibrating thermocouple meter (TC 2000; Sable Systems). Cutaneous heat loss was recorded at five sites (head, chest, forearm, thigh and calf) on the woman's left-hand side with heat flux sensors (FR-025-T-6; Concept Engineering, Old Saybrook, CT, USA). Heat flux signals were DC amplified (x100, ISO-DAM bioamplifiers; World Precision Instruments, Stevenage, UK). Positive cutaneous heat loss values indicate heat loss. Skin blood flow was recorded at two sites on the woman's left-hand side (arm and thigh) with laser Doppler flowmetry probes (413 integrating probe; Perimed Instruments, Järfälla, Sweden). All data were sampled at 2 kHz by a data acquisition interface and then 1-min averaged (1401+ and Spike2 software; Cambridge Electronic Design, Cambridge, UK). Body temperatures, cutaneous heat loss and skin blood flow were recorded continuously. Mean blood pressure was recorded from the right calf in order not to interfere with either i.v. fluid administration or skin temperature measurement in the contralateral arm; this was performed with an electronic oscillometric cuff (IntelliVue patient monitor; Philips, Amsterdam, Netherlands) at 1-min intervals until delivery and then at 5-min intervals.

Following application of the recording apparatus, the woman remained motionless whilst baseline data were recorded for 5 min. After these recordings, a 3 ml test dose of lidocaine-bicarbonate-adrenaline mixture (final concentrations 1.8%, 0.8% and 1:200 000 [13]) was administered, followed after 3 min by a main dose of 12 ml. While the epidural was being topped-up, women were given 500 ml crystalloid (Plasmalyte™) co-load followed by a maintenance infusion at 125 $ml.h^{-1}$. Hypotension was defined as a decrease in systolic blood pressure > 10% of baseline, and was treated

with 50 µg boluses of phenylephrine. Phenylephrine was also given if the woman became lightheaded or nauseated. The distribution of sensory blockade was assessed 5 min after the end of the top-up using ethyl chloride spray; if there was a lack of anaesthesia (i.e. there was sensation of the spray on the skin) up to the T5 dermatome, a further 5 ml bolus was administered. After the initial 5 min period, sensory block was assessed at 1-min intervals and surgery was permitted when block of light touch from the spray reached T5 bilaterally [13]. At this time motor block was assessed with a four point score (1, full movement of legs and feet; 2, no hip flexion but movement of the knee and feet; 3, no knee flexion but movement of feet; 4, no movement of legs or feet [13]). After delivery, the tilt on the operating table was removed and 5 IU of oxytocin administered i.v. Subsequently 40 IU of oxytocin diluted in 500ml 0.9% saline were administered i.v. over 4 h. All drugs and i.v. fluids were administered at room temperature.

Data were analysed with SigmaStat (Systat, CA, USA) software. Maternal mass and height at the first antenatal appointment were used to calculate body mass index and body surface area [14]. Area-weighted mean skin temperature and cutaneous heat loss [15] and volume-weighted mean body temperature [16] were estimated. Total heat loss was estimated by multiplying area-weighted mean cutaneous heat loss by body surface area and augmenting this value by 10% to account for insensible heat loss due to evaporation [3]. Heat production was derived from total heat loss and mean body temperature by direct calorimetry [17].

A sample size of 20 was chosen based on previous investigations of thermoregulation during neuraxial blockade, where eight to 20 subjects were studied [18]. A formal sample size calculation was not performed as body temperature, cutaneous heat loss and skin blood flow have not been recorded previously during emergency caesarean section. Normality of data and equality of variance were examined with Shapiro-Wilk and Brown-Forsythe tests respectively. Mean body temperature (both absolute value and rate of change), core temperature, skin temperature, cutaneous heat loss (individual recording sites) and mean blood pressure changes were compared using one-way repeated measures ANOVA. Heat balance (heat production and heat loss) and skin blood flow

changes were compared with two-way repeated measures ANOVA. Post hoc analysis was conducted with Holm-Sidak methodology. The presented p-values are multiplicity adjusted.

Results

Twenty women were recruited between January - July 2016. The data for one woman were excluded from the final analysis as an attempt at instrumental delivery was made before the caesarean section. Recordings on a second woman were terminated after 75 min when blood loss exceeded 1.5 l, but data were included until this point. Maternal and neonatal clinical data are presented in Table 1. All women were ASA physical status 2.

The median (IQR [range]) intervals between starting the epidural top-up and achieving a T5 block were 11 (9 – 13 [5 – 18]) min; from T5 block till delivery were 20 (11 – 25 [7 – 42]) min; and from delivery till the end of the operation were 40 (30 – 47 [23 – 56]) min. The occasional protracted time intervals between T5 block until delivery were due to delays in the obstetrician's arrival in theatre, and were unrelated to the conduct of the study.

Body temperatures over the study period are shown in Fig. 1. In the 5 min before epidural top-up the mean (SD) rate of mean body temperature increase was 0.1 (0.5) °C.h^{-1}. Between achieving T5 block and delivery, the rate of rise of mean body temperature increased to 0.5 (0.5) °C.h^{-1} ($p = 0.04$). Mean body temperature was greater at delivery than at epidural top-up ($p = 0.03$), and it peaked 3 min after delivery. Between delivery and the end of the operation, the rate of mean body temperature decrease was 0.2 (0.4) °C.h^{-1}. At the end of the operation, mean body temperature was not different to that at epidural top-up ($p = 0.75$). Core temperature did not change between epidural top-up and delivery ($p = 0.38$). Following delivery core temperature decreased, and at the end of the operation it was less than at epidural top-up ($p < 0.001$). Mean skin temperature increased after T5 block was achieved, and was greater at delivery ($p < 0.001$) and at the end of the operation ($p < 0.001$) than at epidural top-up.

Heat balance over the study period is shown in Fig. 2. In the 5 min before epidural top-up mean (SD) heat loss was 95 (24) W. After T5 block was achieved mean (SD) heat loss was 15 (19)% less than before epidural top-up ($p < 0.001$). After T5 block heat loss decreased in the chest ($p < 0.001$) and

forearm ($p = 0.004$) but did not change in the head ($p = 0.05$), thigh ($p = 0.79$) or calf ($p = 1.00$). Before epidural top-up mean (SD) heat production was 141 (51) W. Heat production did not change after T5 block ($p = 0.92$) but following delivery the mean (SD) decrease in heat production was 57 (18)% ($p < 0.001$). Skin blood flow before and after epidural top-up is displayed in Fig. 3.

Discussion

This study assessed cutaneous vasomotor tone and body temperature before and after conversion of epidural analgesia for labour to epidural anaesthesia for emergency caesarean section. To our knowledge, this is the first study to record body temperature, cutaneous heat loss and skin blood flow during emergency caesarean section. The primary novel findings are firstly, heat production exceeded heat loss until delivery and so mean body temperature increased, and secondly, following epidural top-up cutaneous heat loss decreased but skin blood flow did not change. A secondary finding is that, following delivery, heat production decreased and mean body temperature fell.

Before epidural top-up both core temperature and heat production were greater than the normal term non-labouring values of 36.7°C [19] and 85 W [20]. Additionally, as core temperature was greater than the typical active vasodilation threshold of 37.1°C [21, 22], it is likely that the non-anaesthetised segment of the cutaneous vasculature was actively vasodilated. This was expected as heat production increases during labour [23], and labour epidural analgesia is associated with hyperthermia [8]. Even before epidural top-up, heat production exceeded heat loss and during this period mean body temperature increased at $0.1°C.h^{-1}$, which is similar to the rate of core temperature rise associated with labour epidural analgesia [8]. Following epidural top-up, heat loss decreased by 15%. Heat production was unaffected, and so the rate of mean body temperature rise increased to $0.5°C.h^{-1}$. Although predicted, the reduction in cutaneous heat loss is a novel result. Epidural anaesthesia in normothermic individuals increases cutaneous heat loss secondary to blockade of active vasoconstriction [3, 24]. By contrast, skin blood flow did not change following

epidural top-up. Although this was unexpected, a similar result has been observed previously [3]. In normothermic healthy volunteers epidural anaesthesia elicited no change in skin blood flow despite other methodologies (cutaneous heat loss, skin temperature gradient, volume plethysmography) indicating a decrease in cutaneous vasomotor tone [3]. Following delivery, cutaneous heat loss did not change but heat production decreased by 57%. As a result, mean body temperature peaked 3 min after delivery and then decreased at 0.2 °C.h^{-1}. A consistent distribution of anaesthesia is required throughout caesarean section and so a constant rate of heat loss might be anticipated. The effect of delivery upon maternal heat production has not previously been recorded, but the results are also plausible. At delivery the fetoplacental unit separates from the mother [19], and soon afterwards uterine contractions cease [7]. Both are major sources of heat, and so a reduction in heat production after delivery is expected. Skin blood flow has not previously been recorded at the time of delivery, and so the increase in blood flow following delivery was unpredicted. Skin blood flow is however a function of cardiac output, and following delivery cardiac output increases [25]. It is possible therefore that the increase in skin blood flow was unrelated to variation in cutaneous vasomotor tone.

It is likely that heat loss limitation was responsible for the rise in mean body temperature before and after epidural top-up, with heat production and heat loss becoming uncoupled. Mean body temperature (T_B) is an index of total body heat content and is related to the rate of heat production (Q_P) and the rate of heat loss (Q_{Sk}) as follows:

$$Q_P = Q_{Sk} + \frac{(cm\Delta T_B)}{t}$$

where c is the specific heat capacity of human tissue, m is body mass and t is time. As such, mean body temperature only remains constant when the rate of heat loss matches the rate of heat production. Ordinarily heat production and heat loss are tightly coupled, with an increase or decrease in heat production being quickly followed by a reciprocal change in heat loss, and thus mean body temperature is maintained within a narrow window [26]. A consistently rising mean body

temperature, also known as ‘uncontrolled hyperthermia’, occurs either when a massive increase in heat production overwhelms the body’s normal heat loss mechanisms (e.g. with malignant hyperthermia), or when an intervention limits heat loss [11]. During exercise it is possible to generate a rate of heat loss tenfold greater than the recorded rate of heat production, and so in the present scenario it is most likely that heat loss was limited [26].

Of the four potential explanations for the reduction in cutaneous heat loss (thermoregulatory vasoconstriction, baroreceptor mediated reflex vasoconstriction, non-thermoregulatory vasoconstriction and blockade of active cutaneous vasodilation), blockade of active cutaneous vasodilation is the most plausible. Neither body temperature nor mean blood pressure decreased until after delivery and so it is unlikely that topping-up the epidural stimulated either the thermoregulatory [5] or the baroreceptor [10] reflexes. Non-thermoregulatory vasoconstriction is a possibility, as epidural analgesia for labour is associated with maternal inflammation [8]. However, given that the reduction in heat loss occurred in the chest and arms, the expected distribution of sympathetic block extension of T2–8 [27], blockade of active vasodilation is the more likely explanation. The lack of change in skin blood flow following epidural top-up was unexpected, but it does not necessarily refute this explanation of blockade of active vasodilation. Laser Doppler flowmetry is the gold standard for the measurement of skin blood flow [28], but it is not able to determine the relative quantities of blood flow at different depths within the skin. Cutaneous heat loss is dependent upon both factors [29], and so heat loss variation may occur without reciprocal changes in skin blood flow. The skin blood flow response to sympatholysis in an already heat stressed population is more complex than that of cutaneous heat loss [30]. During heat stress, skin blood flow is regulated by competing thermoregulatory (vasodilator) and non-thermoregulatory (vasoconstrictor) reflexes [22, 30], and so the response following sympatholysis is dependent upon the amount of thermoregulatory and non-thermoregulatory activity prior to neural blockade. The lack of change suggests that equal quantities of vasodilator (thermoregulatory) and vasoconstrictor (non-thermoregulatory) activity were present before epidural top-up.

The present study is not the first to show an association between obstetric neuraxial blockade and an increase in body temperature [8]. Hyperthermia is also a recognised complication of epidural analgesia for labour [8]. Epidural hyperthermia (or fever) is a harmful condition which is associated with adverse neonatal neurological outcomes [2], an increased risk of operative delivery [31] and an increased risk of maternal and neonatal sepsis evaluation and treatment [31, 32]. Its underlying mechanism remains unclear. An association has been demonstrated with maternal inflammation, but no causal link has been uncovered [8]. An alternative explanation is that labour epidural analgesia limits evaporative heat loss by blocking the cholinergic sympathetic pathway responsible for sweating [32], but this hypothesis has not been tested. The results of the present study reveal a potential third explanation: limitation of cutaneous heat loss via blockade of active cutaneous vasodilation. Indeed, the rise in mean body temperature before epidural top-up and the lack of change in thigh and calf heat loss following epidural top-up could be interpreted as evidence of pre-existing heat loss limitation secondary to sympathetic blockade in the leg.

The main limitation of this study is that cutaneous sympathetic nerve activity was not tested directly. As a result the conclusions are reached by a process of elimination, rather than by a quantitative examination of neuronal function. However, direct testing of sympathetic nerve activity is not appropriate during an emergency caesarean section, as it is time-consuming and requires intradermal bretylium tosylate and botulinum toxin administration [33], neither of which are without risk. Consequently, in this population passive observation of skin blood flow and cutaneous heat loss are the optimal techniques.

A clinical implication of this study is that the use of active warming during emergency caesarean section should be reviewed. Even though no active warming measures were utilised in this study, women were hyperthermic throughout. Epidural hyperthermia is associated with adverse neonatal neurological outcomes [2] and so further heating could potentially be deleterious to the fetus. However, due to the inclusion and exclusion criteria, the findings of this study are only applicable to emergency caesarean section in specific circumstances, that is in labouring women having epidural

top-up, and in the absence of major haemorrhage. Consequently, before any changes in clinical practice are recommended, a larger study should fully investigate the indications for active warming during emergency caesarean section.

In summary, this study is the first to investigate the changes in cutaneous vasomotor tone and body temperature associated with conversion of epidural analgesia for labour to epidural anaesthesia for emergency caesarean section. Epidural top-up limited cutaneous heat loss, and as a result mean body temperature increased until delivery. The most plausible explanation for the heat loss limitation is blockade of active cutaneous vasodilation. It is possible that epidural hyperthermia is also a consequence of sympatholysis, and this is a potential subject for future investigations.

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Table 1 Maternal and neonatal clinical data from 19 women. Values are mean (SD), median (IQR [range]) or number (proportion).

Maternal	
Age; years	33.2 (4.7)
Body mass index; $kg.m^{-2}$	26.3 (4.9)
Parity	0 (0 – 0 [0 – 3])
Labour duration; min	812 (361)
Indication for caesarean section	
Failure to progress	15 (79%)
Fetal tachycardia	4 (21%)
Epidural duration until caesarean; min	755 (308)
Epidural volume until caesarean; ml	141.5 (73.3)
Epidural top-up volume; ml	20 (15 – 20 [15 – 20])
Upper level of sensory block	
Cold	T3 (T2 – T3 [C6 – T3])
Light touch	T4 (T4 – T5 [C6 – T5])
Motor block	3 (3 – 3 [2 – 4])
Mean blood pressure; mmHg	
- Baseline	88.5 (13.7)
- T5	85.4 (14.7)
- Delivery	81.6 (12.7)
- End of operation	75.5 (12.1)*
Phenylephrine dose; µg	150 (38 – 400 [0 – 1300])
Sepsis evaluation	6 (30%)
Positive bacterial cultures	0 (0%)
Antibiotic prescription	5 (25%)
Neonatal	
APGAR score	
1 min	9 (9 – 9 [4 – 9])
5 min	10 (10 – 10 [8 – 10])
Umbilical artery pH < 7.2	0 (0%)
Sepsis evaluation	6 (30%)
Positive bacterial cultures	0 (0%)
Antibiotic prescription	6 (30%)

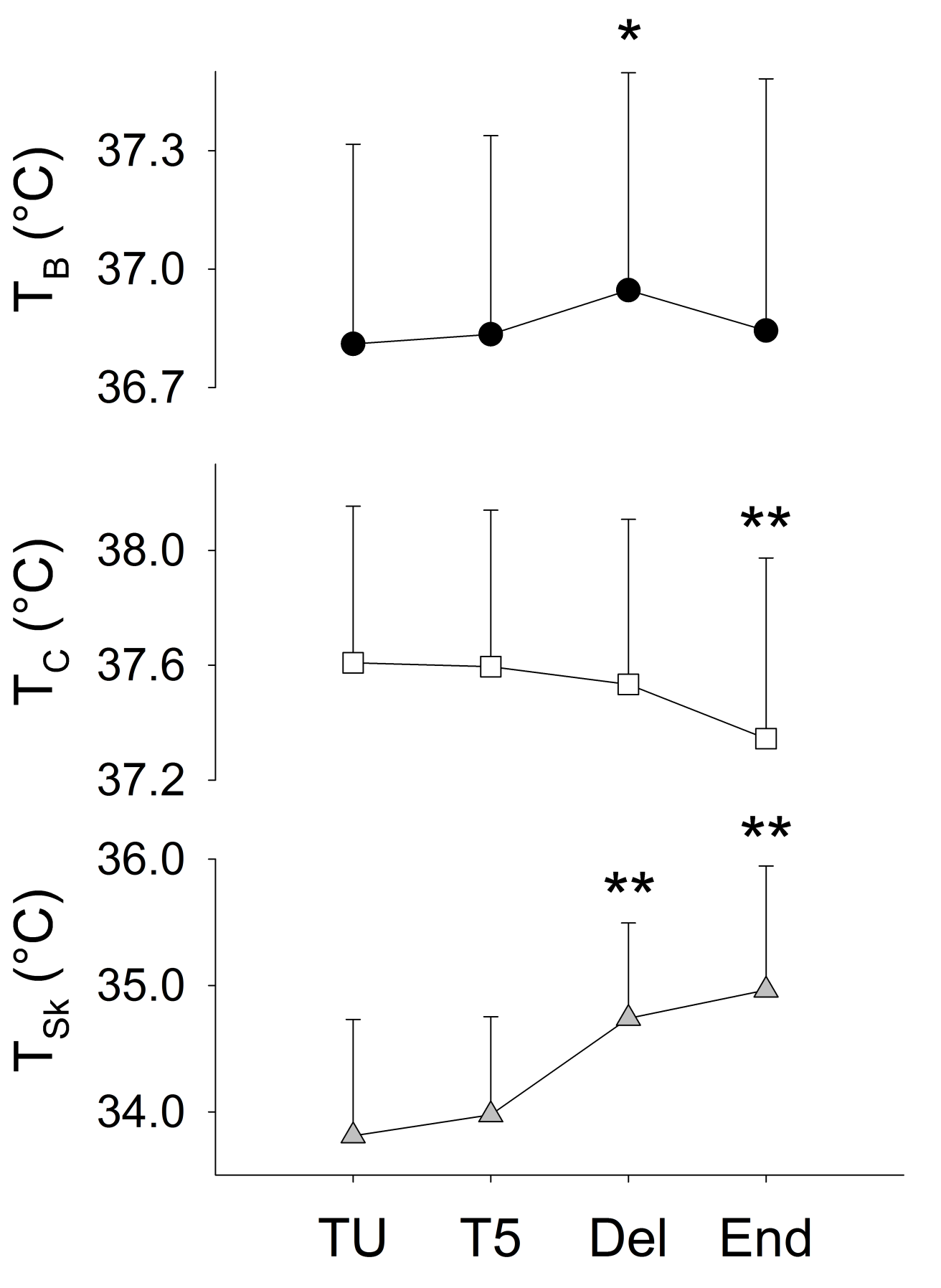
* p = 0.001 vs. baseline

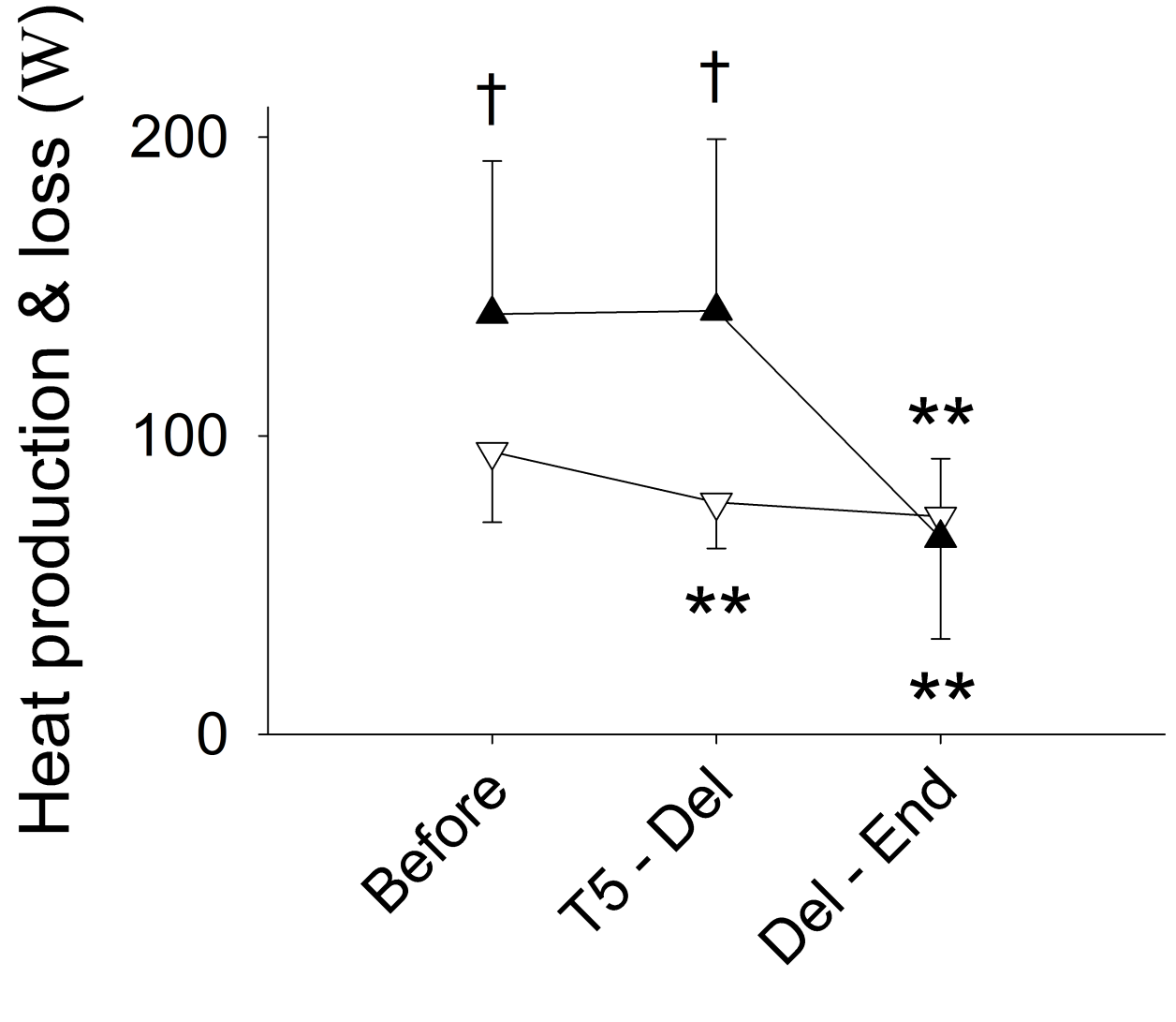
Captions for figures

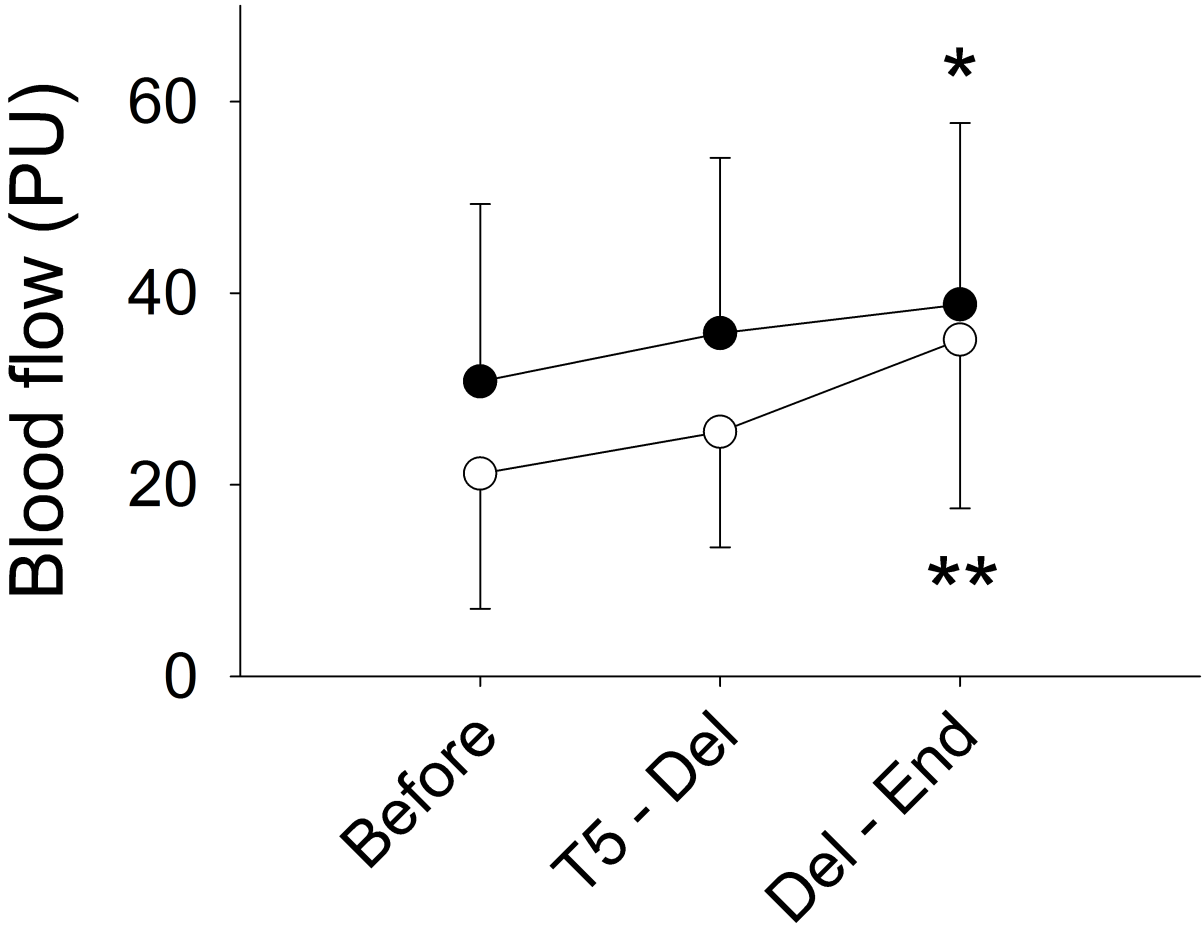
Figure 1 Mean (SD) temperatures in 19 women. T_B, mean body; T_C, core; T_{Sk}, skin. TU, epidural top-up; T5, T5 block achieved; Del, delivery; End, end of surgery. * $p < 0.05$ **, $p < 0.001$ vs. baseline.

Figure 2 Mean (SD) heat production (black triangles) and heat loss (white triangles) in 19 women. Before, before epidural top-up; T5 – Del, between T5 block achieved and delivery; Del – End, between delivery and the end of surgery. * $p < 0.05$ **, $p < 0.001$ vs. baseline. † indicates heat production was greater than heat loss during the same time interval ($p < 0.001$).

Figure 3 Mean (SD) arm (black circles) and thigh (white circles) skin blood flow in 19 women. Before, before epidural top-up; T5 – Del, between T5 block achieved and delivery; Del – End, between delivery and the end of surgery. PU, perfusion units. * $p < 0.05$ **, $p < 0.001$ vs. baseline.


*
37.3
37.0
36.7
T_B (°C)
**
38.0
37.6
37.2
T_C (°C)
**
**
36.0
35.0
34.0
T_{Sk} (°C)
TU
T5
Del
End


Heat production & loss (W)
200
100
0
†
†
**
**
**
Before
T5 - Del
Del - End


Blood flow (PU)
60
40
20
0
*
**
Before
T5 - Del
Del - End