Effect of forced-air heaters on perfusion and temperature distribution during and after open-heart surgery

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Abstract

Objectives: After cardiopulmonary bypass, patients often show redistribution hypothermia, also called afterdrop. Forced-air blankets help to reduce afterdrop. This study explores the effect of forced-air blankets on temperature distribution and peripheral perfusion. The blood perfusion data is used to explain the observed temperature effects and the reduction of the afterdrop. Methods: Fifteen patients were enrolled in a randomised study. In the test group (\(n = 8\)), forced-air warmers were used. In the control group (\(n = 7\)), only passive insulation was used. Core and skin temperatures and thigh temperatures at 0, 8, 18 and 38 mm depth were measured. Laser Doppler flowmetry (LDF) was used to record skin perfusion from the big toe. Blood flow through the femoral artery was determined with ultrasound. Results: Afterdrop in the test group was smaller than in the control group (1.2 \(\pm\) 0.2 \(^\circ\) C vs 1.8 \(\pm\) 0.7 \(^\circ\) C; \(P = 0.04\)) whilst no significant difference in mean tissue thigh temperature was found between the groups. Local skin temperature was 2.5—3.0 \(^\circ\) C higher when using forced-air heaters. However, skin perfusion was unaffected. Ultrasound measurements revealed that leg blood flow during the first hours after surgery was reduced to 24—70\% of pre- and peri-operative values. Conclusions: Forced-air blankets reduce afterdrop. However, they do not lead to clinical relevant changes in deep thigh temperature. LDF measurements show that forced-air heating does not improve toe perfusion. The extra heat especially favours core temperature. This is underlined by the decrease in postoperative leg blood flow, suggesting that the majority of the warmed blood leaving the heart flows to core organs and not to the periphery. © 2007 European Association for Cardio-Thoracic Surgery. Published by Elsevier B.V. All rights reserved.

Keywords: Cardiopulmonary bypass; Forced-air warmer; Afterdrop; Perfusion; Temperature; Hypothermia

1. Introduction

Whole body hypothermia is widely used to protect vital organs during open-heart surgery. During cardiac surgery the body is cooled by means of a heart—lungh machine. On nearing completion of the surgery the body is warmed with the help of the heart—lungh machine. Core body parts (trunk and head) rewarm more quickly than peripheral parts (extremities). After disconnecting the heart—lungh machine the body is allowed to self equilibrate. The normal vasoconstriction response is impaired by the administered anaesthesia\textsuperscript{[1]}. Hence, heat distribution takes place from the warm core to the colder periphery. This causes afterdrop\textsuperscript{[2]}: a decrease in the temperature of the core organs. Afterdrop may trigger shivering and increases the risk of post-operative complications\textsuperscript{[3—4]}. Forced-air warmers are often used to reduce hypothermia. Postoperative effectiveness is proven by Brauer\textsuperscript{[5]}, while Kim\textsuperscript{[6]} has shown its benefits during off-pump coronary bypass surgery. Tauchi\textsuperscript{[7]} used forced-air heating on the periphery to warm the core in anaesthetised hypothermic volunteers. Nowadays, forced-air heaters are also often used perioperatively during cardiac surgery. Heat is then transferred to the periphery while the core is rewarmed by the heat exchanger in the heart—lungh machine. Covering the inadequately warmed periphery with forced-air heaters during systemic rewarming reduces afterdrop\textsuperscript{[8—9]}. One explanation is that forced-air warming increases peripheral temperature. This reduces the core-periphery gradient and consequently reduces afterdrop. Measurements and analysis of Rajek\textsuperscript{[9]} did not support this explanation. Instead, they
think that forced-air heaters do not prevent redistribution, but preserve the total body heat content. It is of interest to assess how forced-air heaters change body temperature distribution.

Body temperature distribution depends strongly on blood-borne heat transfer. Under anaesthesia thermoregulation is impaired. This leads to alterations in blood flow, with corresponding changes in temperature distribution [1]. Only a few animal studies have been performed that assess perfusion during extracorporeal circulation [10]. To our knowledge, no study on peripheral heat flow during human extracorporeal circulation has been published. Therefore, we simultaneously measured perfusion and temperature distribution during and after cardiac surgery. In this way, information is provided about the amount of convective heat flow to the periphery. Ultimately, this will help explain how forced-air heaters favour core temperature.

This paper focuses on the changes in peripheral perfusion and temperature distribution during and first hours after surgery, with and without using forced-air heaters. Furthermore, we analyse whether the data on blood perfusion can explain the observed temperature effects and the reduction of the afterdrop.

2. Materials and methods

2.1. Patients

With approval from the AMC medical ethical committee and written informed patient consent, we studied 15 patients undergoing elective aortic valve replacement. Patients were followed during surgery and the first 3 h after surgery at the intensive care unit (ICU). We enrolled patients aged between 40 and 80 without severe left ventricular dysfunction. Patients were randomly assigned into one of two protocols: a test group (n = 8) that was rewarmed with forced-air heaters and a control group (n = 7) that was rewarmed with only passive insulation. The inlet air temperature of the forced-air blankets (The Surgical Company, Mistral-air model: tube) was set to a maximal temperature of 43.3 °C.

2.2. Anaesthesia

Calcium channel blockers and long acting nitrates were given until the evening before surgery. Beta-adrenoceptor blocking agents were continued until the morning of surgery. Lorazepam 2–3 mg was given for premedication 2 h before surgery. Anaesthesia was induced with sufentanil 3 μg kg⁻¹ (Sufenta®, Janssen-Cilag, Tilburg, The Netherlands) and propofol 50–100 mg (Fresenius Kabi, Den Bosch, The Netherlands). Pancuronium bromide 0.1 mg kg⁻¹ (Pavulon®, Organon, Oss, The Netherlands) was given for muscle relaxation. Morphine 10–30 mg was given as a slow bolus injection. Anaesthesia was maintained with a continuous infusion of propofol 2–5 mg kg⁻¹ h⁻¹ and/or isoflurane at 0.5 MAC. The lungs were ventilated with air/oxygen (FiO₂ = 0.5). Following the induction of anaesthesia, a flow directed pulmonary artery catheter (Edwards Lifesciences, Irvine, CA, USA) was inserted into the right internal jugular vein. Dexamethasone and alpha 2-adrenoceptor agonists were not used in any of the participating patients. Incidentally, vasoactive medication was administered to a few patients (viz. metaraminol, dobutamine, milrinone and nitroglycerine).

2.3. Protocol

Temperature in the operating theatre was kept near 20 °C. In the test group the blanket was mounted along the body by sticking the blanket on the surgery table with tape. The sterile cover was positioned over the patient and covered also the forced-air heater. Forced-air heaters in the test group were used after fastening the aortic valve (28 ± 18 min after the onset of rearming) and active heating was continued until skin closure. All patients were cooled to a nasopharyngeal temperature of 30 °C. Thereafter, patients were rewarmed to a nasopharyngeal temperature of 33 °C and depending on the state of the surgery, fully rewarmed (when the aortic valve was already fastened) or kept for a few minutes at this temperature until the aortic valve was fastened. Patients were rewarmed to a nasopharyngeal temperature of 37–37.5 °C and a rectal temperature of minimally 36.3 °C. Non-pulsatile bypass flow was kept at 2.4 l min⁻¹ m⁻². Arterial inflow temperature was maximum 4 °C warmer than the venous return temperature. Subsequently the heart–lung machine was decoupled and the patients were transferred to the ICU.

2.4. Morphometric measurements

Before surgery, the patient’s body characteristics were measured: height, weight and fat percentage. Fat percentage was determined by measuring skinfold thickness at four positions: at the m. biceps brachii, m. triceps brachii, subscapular and suprailliacal (Durnin [11]). Body surface area (BSA) was calculated according to DuBois [12]:

\[
\text{BSA} = 0.007184 \times W^{0.425} H^{0.725}
\]

with weight W in kg and height H in cm.

2.5. Temperature measurements

Core temperatures were measured at regular intervals from the nasopharynx, in the rectum (oesophageal/rectal temperature probe, Philips 21090A), and from the pulmonary artery (Thermodilution Paceport Catheter: 931HF75, Edwards Lifesciences). In the operating room, the nasopharyngeal temperature was used as an estimator for the core temperature. At the ICU the pulmonary artery temperature was used to assess core temperature. Transient thigh temperature was determined at the skin and at deeper thigh tissue with a needle thermocouple (MT Needle microprobe 23 ga. Physitemp Instruments Inc., New Jersey) containing three sensors. Tissue thigh temperature was measured at 8-, 18-, and 38-mm depth. The needle was inserted perpendicular to the skin surface slightly lateral from the anterior mid-upper right thigh. Skin temperature measurements were performed using wireless thermistors (iButton [13]) at the forehead, lower arm, fingertip, foot dorsum, lower leg, upper leg and abdomen. Because chest
skin temperature could not be measured we modified the seven-point system of Hardy–Dubois [14] to a six-point system according to

\[
T_{\text{skin}} = \frac{0.07T_{\text{forehead}} + 0.14T_{\text{posterior, forearm}} + 0.05T_{\text{hand}} + 0.19T_{\text{anterior, thigh}} + 0.13T_{\text{anterior, calf}} + 0.07T_{\text{foot}}}{0.65}
\]  

Eq. (2) does not provide a mean skin temperature that can be compared to other studies, but can be used to identify and compare changes in transient mean skin temperatures of the two groups in this study.

### 2.6. Skin perfusion measurements

Skin perfusion was continuously measured by laser Doppler flowmetry (LDF) (Perimed PF4001, with a 780 nm laser diode, 0.25 mm fibre separation) under the right big toe. The maximum sensitivity of the LDF signal is obtained at a depth of 0.6—0.8 mm [15]. Sample rate was set to 4 Hz using a time constant of 0.2 s. All measurements were performed with a standard probe (Probe 408, Perimed AB), which was calibrated with PF 1000 calibration equipment (Perimed AB). Laser Doppler results were analysed as a running average of 50 s epochs, to filter out motion artefacts.

In order to make individual data comparable and to easily see the effect of forced-air heating on perfusion we present the perfusion results together with local temperature data, as explained below. Temperature and perfusion are often coupled to the \(Q_{10}\) relation of thermal physiology, which states that for every 10 °C decrease in temperature, metabolism is reduced by a factor 2—3 [16—18]. Perfusion is linearly related to metabolism [19]. Under current study circumstances the relation is expected to deviate from \(Q_{10}\) because in extremities, like the skin of the toe, variations in blood flow can be larger than in other parts [19] due to vasoactive responses of arteriovenous anastomoses. As a starting point for establishing a relationship, we will take a similar shape as the \(Q_{10}\) relationship:

\[
\frac{W_b}{W_{b,0}} = C(T-T_0/10^\circ \text{C})
\]  

where \(C\) is a constant that will be determined by fitting Eq. (3) to local toe perfusion and temperature data. \(W_b\) is the current perfusion value, \(W_{b,0}\) a reference perfusion value, and \(T\) and \(T_0\) are current and base line temperatures, respectively.

For each experiment a baseline value for \(T_0\) and \(W_{b,0}\) was determined. Because the period at the ICU was relatively stable, the mean perfusion value during the last 30 data points of that measurement period was used as base line value \(W_{b,0}\). Subsequently, all perfusion values \((W_b)\) were divided by \(W_{b,0}\). From all temperatures \((T)\) the base line temperature was subtracted \((T_0)\).

### 2.7. Leg blood flow measurements

Leg blood flow was determined in 12 patients by ultrasound measurements (Siemens Sonoline Antares) in the right femoral artery 1 cm before the bifurcation in the groin area. The femoral artery was visualised in B-mode. The diameter of the vessel was measured in systole and diastole. Subsequently, Doppler mode was used to record centreline velocity in the vessel during two heartbeats. Measurements were performed at four defined points in time, and repeated two times at these time points: (1) just before entering the surgery room, (2) at the coolest point during the bypass cooling, (3) just after surgery as the patient has arrived at the ICU and (4) about 3 h after surgery.

Methods for calculating the leg blood flow are outlined in Appendix A. The flow at the first measurement period was considered to be the ‘neutral state’ \(Q_0\). For each patient the flow at each measurement moment was normalised with the individual base line value \(Q_0\). Leg blood flow results are thus

| Table 1 (a) Subject characteristics and (b) temperature and afterdrop characteristics |
|---------------------------------------------|-----------------------------|
| Control group | Test group | \(P\) |
| Gender (M/F) | 5/2 | 2/6 | 0.13 |
| Weight (kg) | 80.8 ± 10.9 | 78.6 ± 12.0 | 0.72 |
| Height (cm) | 172 ± 4 | 167 ± 11 | 0.28 |
| Age | 69.1 ± 15 | 57.9 ± 11 | 0.12 |
| BMI | 27.3 ± 3.0 | 28.2 ± 3.6 | 0.61 |
| Fat% | 31.6 ± 5.9 | 36.0 ± 5.4 | 0.17 |
| BSA (m²) | 1.93 ± 0.14 | 1.94 ± 0.16 | 0.90 |
| Nasopharyngeal temperature at the coolest point (°C) | 29.4 ± 0.6 | 29.7 ± 1.4 | 0.61 |
| Nasopharyngeal temperature at the end of rewarming (°C) | 37.2 ± 0.4 | 37.4 ± 0.5 | 0.41 |
| Rectal temperature at end of rewarming (°C) | 36.6 ± 0.2 | 36.5 ± 0.5 | 0.63 |
| Afterdrop* (°C) | 1.8 ± 0.7 | 1.2 ± 0.2 | 0.04* |
| Afterdrop duration (min) | 73 ± 24 | 59 ± 14 | 0.21 |
| Cooling time (min) | 38 ± 16 | 38 ± 17 | 1.0 |
| Warming time (min) | 64 ± 10 | 66 ± 19 | 0.81 |
| Bypass time (min) | 102 ± 19 | 105 ± 30 | 0.82 |

Values are given as mean values with standard deviation.

* Afterdrop is defined as: the nose temperature at the end of rewarming with the heart–lung machine minus the lowest nose temperature after decoupling the heart–lung machine.

* Significant difference \((P < 0.05)\).
presented as relative changes in relation to the individual neutral state \( \frac{Q}{Q_0} \).

At the same points in time when leg blood flow was measured cardiac output was also determined with thermodilution measurements in the pulmonary artery. During bypass, pump flow rate was used instead of cardiac output.

2.8. Statistics

Subject, temperature and afterdrop characteristics, as given in Table 1, are presented as mean with standard deviation. In the figures, results are presented as means with standard deviation of means. No adjustments for multiple comparisons were performed. Differences between the groups at different time points were analyzed with an unpaired t-test. For determining the effect of using forced-air heaters on afterdrop, a summary measure, which reveals the real clinical relevance, is used [20]. As summary measure for defining afterdrop, the core temperature of the moment of going off-pump minus the minimal postbypass core temperature, was used. For comparison of the gender and vasoactive medication between the two groups, Fisher’s exact test was used with two-tailed probability. Leg blood at the four time points are compared with a paired t-test. Differences were considered statistically significant when \( P < 0.05 \).

For fitting Eq. (3), linear regression after log-transformation was used.

2.9. Data analysis

The beginning of the bypass was the designated time zero for cooling. As the duration of cooling/warming differed among patients, the start of fully warming was defined as the designated time zero of warming, and the moment of decoupling the heart–lung machine was defined as the zero time for the postbypass period.

One needle did not work properly in one patient in the control group, so deep tissue temperature in that group are based on maximal \( n = 6 \). Four patients of the control group were covered with forced-air heaters at the ICU (24, 30, 60 and 64 min after arrival at the ICU). Two persons in the test group were given a heating screen at the ICU (62 and 200 min after arrival at the ICU). Only statistical differences before giving additional heating equipment at the ICU were studied.

For three patients \( W_b,0 \) was not determined as no stable baseline value for skin perfusion was observed. These datasets were excluded in the analysis.

Leg blood flow data of one patient taken at measurement moment four was excluded, because we observed an extreme increase in heart rate.

3. Results

3.1. Patient characteristics

Subject characteristics of the control and test group are shown in Table 1a. No significant differences were found in morphometric characteristics of the two groups.
3.3. Mean skin temperature

In Fig. 2, the mean skin temperature is shown. Mean skin temperature of the test group was significantly higher 30—80 min after decoupling from the heart—lung machine. Mean skin temperature of patients from the control group who obtained forced-air heaters at the ICU is indicated with a different symbol. An increase in mean skin temperature was clearly visible after that time point.

3.4. Thigh temperature

No significant difference was found in skin temperature of the thigh (Fig. 3a) between the two groups during anaesthesia, cooling and warming. In the postbypass stage there was a tendency that the skin temperature of the thigh in the test group was higher than in the control group. The thigh temperature at 8 mm depth (Fig. 3b) differed significantly prior to cooling and during the first 10 min of cooling. About 25 min before cooling $T_{\text{thigh}}$ at 18 mm depth was significantly lower in the test group (Fig. 3c) which lasted till the moment cooling started.

After approximately 50 min of rewarming, thigh temperature at 38 mm depth was warmer in the control group than in the test group (Fig. 3d). This difference was observed till approximately 10 min after going off-pump. The 95% confidence intervals at 25 min after decoupling the heart—lung machine (where temperature differences were largest) are $-1.26 \leq T_{\text{test}} - T_{\text{control}} \leq 0.26$ and $-1.436 \leq T_{\text{test}} - T_{\text{control}} \leq 0.09$ at 18 and 38 mm depth, respectively. This indicates that clinically relevant improvement in deep thigh temperature at that time point can be excluded. During the first 3 h at the ICU no significant differences were found in thigh temperature at 8, 18 and 38 mm (not shown). Finally, no difference in mean thigh temperature as calculated with the method of Belani [21] was found at the operating theatre and ICU. The method for calculating the mean thigh temperature is dilated in Appendix B.

3.5. Skin blood flow

A typical curve of skin temperature under the toe and skin perfusion is given in Fig. 4. Skin perfusion is given in dimensionless perfusion units (PU). In Fig. 5, average values of the sampling distribution of the relative perfusion ($W_{b}/W_{b,0}$) is plotted against the change in temperature ($\Delta T = T - T_{0}$). The data with $\Delta T < 7 \, ^\circ\mathrm{C}$ was used to fit the value of the unknown constant $C$ in Eq. (3). The resulting fit and the average values are given in Fig. 5. The best fit revealed a $C = 15.8$, which means that at 10 °C in local temperature coincides with a 16-fold increase in skin perfusion.

In Fig. 6, a typical example of a patient is shown before and after being covered with a forced-air blanket. The
The perfusion-temperature curve shows a shift to the right when forced-air heating is applied. This means the perfusion was the same as in the situation without forced-air heaters whilst the local skin temperature of the toe increased 2.5—3.8°C. This effect was also visible in patients in the control group to whom forced-air blankets were supplied at the ICU. Hence, we can conclude that under these circumstances the forced-air heater did not change the amount of skin perfusion. Only skin temperature was affected by forced-air blankets.

3.6. Leg blood flow

Arterial blood supply into the right femoral artery was determined for 12 patients at 4 defined points in time when no forced-air heating was used (Fig. 7). Before entering the surgical room and during bypass cooling, no significant difference was observed. Just after surgery the leg blood flow decreased significantly \((P = 0.02)\) and was still significantly reduced \((P = 0.02)\) when the patients were at the ICU for 3 h. No significant difference was observed between the last 2 measurement time points. From these results, it can be concluded that leg blood flow through the femoral artery after surgery reduced to ~70% of the pre-and perioperative value.

Mean cardiac output or bypass flow was \(2.4 \pm 0.4 \text{ l min}^{-1} \text{ m}^{-2}\). Individual changes in cardiac output/bypass flow were small over the four measurement points (mean fluctuation was 6.8%).

4. Discussion

In this survey, the reduction in afterdrop by using forced-air heaters was confirmed. A new approach in explaining the effectiveness of forced-air heaters was the simultaneous assessment of transient temperature distribution and peripheral perfusion in patients undergoing cardiac surgery. Two groups were studied: patients who were perioperatively rewarmed with, and without forced-air blankets. New findings in this study are that by using forced-air blankets no clinically relevant improvements in deep tissue temperature in the thigh can be expected, but only in skin and superficial tissue temperatures. Usage of forced-air heaters results in higher local skin temperature, but nonetheless does not lead to higher peripheral perfusion rates. Our data shows that the extra heat from forced-air warming especially ends up in the core, and not in the deep periphery.

4.1. Perfusion

Local temperature and skin perfusion data was used to establish a relationship for skin temperature and perfusion in the toe during cardiac surgery, when no forced-air heating was used. Our data suggested that a 10°C increase in skin temperature of the toe leads to a 16-fold increase in skin perfusion.

Usage of forced-air heaters resulted in higher local toe skin temperature (temperature increase of typically 2.5–3°C), but did not accompany higher perfusion according to Eq. (3). Apparently perfusion cannot be improved by cutaneous heating. If Eq. (3) would have held, perfusion could be advanced by applying forced-air heaters. In that case, forced-air heaters had been even more effective in reducing afterdrop.

Ultrasound measurements pointed out that leg blood flow during the first hours after cardiac surgery was reduced to approximately 70% of the blood flow measured prior and during surgery. This decrease in postoperative blood flow coincided with the decrease in skin perfusion that was observed under the toe at the ICU. Low skin perfusion suggests a high resistance in the vascular bed of the periphery. This leads to a lower leg blood flow. At about equal cardiac output (fluctuation 6.8%) this means that less blood leaving the heart is distributed to the periphery compared to the core.
4.2. Temperature

Afterdrop magnitudes of 1.8 ± 0.7 °C were found in the control group, and 1.2 ± 0.2 °C in the test group. This was slightly larger than in previous studies by Rajek [9,22]. Rajek [9] reported an afterdrop of 1.2 ± 0.2 °C for the control group and 0.5 ± 0.2 °C for the test group. Patients in our study were cooled to lower temperatures (average 29.6 °C) than Rajek’s patients (31.8 °C) and rewarmed to higher nasopharyngeal temperatures (37.3 vs 37.1 °C). This might explain the larger afterdrop in our study. Rajek’s previous study [22] supports this explanation. Here afterdrop magnitudes of 1.5 ± 0.4 °C were found for patients with passive covers who were cooled till 30.8 ± 1 °C and were rewarmed till core temperatures of 37.5 °C. Also the fact that we used another type of heating blanket (tube model vs a cardiac blanket that covered the legs) could have caused the different outcomes.

Only marginal differences were observed between the thigh temperatures in the two groups. Skin temperature and thigh temperature at 8 mm depth of the test groups tended to be warmer in the postbypass stage. The results were unexpected, that at the end of the rewarming stage and beginning of the postbypass stage deep thigh temperature (at 18 and 38 mm depth) were higher in the group without additional forced-air heaters. The 95% confidence intervals, at 25 min after decoupling the heart—lung machine, show that clinically relevant improvement when using forced-air heaters can be excluded at the deep peripheral tissue. This already indicates that the extra heat of forced-air warmers neither penetrates to the deeper peripheral tissue by means of conduction, nor by heat redistribution by the blood. Thigh tissue temperature in the control group was slightly higher over the total measurement period. It is remarkable that forced-air heaters did not compensate, or reverse this. It is expected that heat provided by forced-air heaters only causes a clinical relevant temperature rise at the skin and at superficial situated tissue, but not at the deeper tissue. Average thigh temperatures as calculated according to Belani [21] did not show significant differences between the groups, which agreed with the results of Rajek [9].

There are three mechanisms by which adding heat to peripheral skin can favour core temperature: (1) direct conduction through peripheral tissue, which would reduce the core-periphery gradient, (2) blood-borne convection of heat from the skin, which would result in a higher blood pool temperature and (3) blood-borne convection of heat from the deeper peripheral tissue.

The fact that forced-air warmers reduced afterdrop, but did not warm the deep peripheral tissue, indicates that the effectiveness of forced-air warmers in reducing afterdrop is especially explained by blood-borne convection from the skin. Forced-air heaters favour skin temperature by convective heat transfer. Moreover, heat loss from the patient’s body to the surrounding is reduced. We think that the major part of the extra heat of forced-air warming is transported from the capillary network in the skin to the venous system through the superficial veins of the lower extremities [23]. From there, the heat is transported via direct connections to the heart. This leads to higher mixed blood pool temperature in the test group than in the control group. The slightly warmer blood is redistributed over the body. Core organs are better perfused than peripheral organs under current circumstances. Indeed, our leg blood flow measurements underline this thought, as we observed that postoperative leg blood flow was reduced significantly compared to pre-and peri-operative flow. In this way, the decrease in core temperature drop and the unchanged deep peripheral temperature in the test group can be explained.

5. Conclusion

Our data confirm that afterdrop is reduced by using forced-air heating. Current measurements reveal that using forced-air heaters does not lead to clinically relevant improvements in deep peripheral temperature, but only in skin and superficial peripheral temperature. Laser Doppler flowmetry measurements point out that forced-air heating does not improve skin blood flow proportionally to the increase in skin temperature. We think that the increase in temperature of the venous blood that returns to the heart especially favours core organs. Indeed, the measured significant decrease in postoperative leg blood flow suggests that less warmed blood leaving the heart flows to the legs in the last stage of the surgery. We therefore conclude that the majority of extra heat from forced-air heaters is directly transported from the skin capillaries to the heart, and from there mainly redistributed to core organs, and not so much to the periphery.

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References

Flow rate before surgery, after arriving at the ICU and after 3 h at the ICU (measurement moment 1, 3 and 4) are estimated with help of inverse Womersley relations [24]. With this expression single harmonics of the centreline velocity ($\hat{u}_l$) can be used to express harmonics of the flow rate ($\hat{q}$) according to:

$$\hat{q}(t) = \pi R^2 \hat{u}_l \left[ \frac{J_3(\alpha R \beta - \alpha a \beta)}{J_3(\alpha R)} - \frac{J_1(\alpha R \beta - \alpha a \beta)}{J_1(\alpha R)} \right]$$

where $R$ is the vessel radius and $\alpha$ the Womersley number defining the ratio of the instationary inertia forces and the viscous forces. In the calculations, blood viscosity was kept constant at $3 \times 10^{-3}$ Pa s for simplification.

Eq. (4) is based on rigid, straight tube walls. Only small errors are made when using this equation in our situation because distension was small ($\approx 5\%$) and tapering angle of the femoral artery is small.

Flow rate during the non-pulsatile flow when connected to the heart lung machine (measurement moment 2) is calculated by considering the flow as a fully developed laminar flow. Then the flow rate is calculated by

$$q = \frac{1}{2} \pi R^2 \hat{u}_l$$

### Appendix B

Average tissue temperature in the thigh segment was calculated using parabolic regression as described by Belani [21]. This regression assumes that tissue temperature is radially symmetrical. We used the skin temperature at the posterior thigh ($T_{nosephen}$), thigh temperatures at 8, 18 and 38 mm below the skin. Similarly to Belani, we set the core temperature in the leg to nasopharyngeal temperature. The parabolic regression equation was formulated as

$$T(r) = a + br^2$$

with $T(r)$ temperature, $r$ the radial position and $a$ and $b$ regression constants. Average tissue temperature in the thigh was then calculated by

$$T_{\text{avg}} = \frac{1}{\pi R^2} \int_0^R 2\pi (a + br^2) r \, dr = a + \frac{1}{2} b R^2$$