Tissue Heat Content and Distribution During and After Cardiopulmonary Bypass at 17°C

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We measured afterdrop and peripheral tissue temperature distribution in eight patients cooled to approximately 17°C during cardiopulmonary bypass and subsequently rewarmed to 36.5°C. A nasopharyngeal probe evaluated trunk and head temperature and heat content. Peripheral tissue temperature (arm and leg temperature) and heat content were estimated using fourth-order regressions and integration over volume from 30 tissue and skin temperatures. Peripheral tissue temperature decreased to 19.7 ± 0.9°C during bypass and subsequently increased to 34.3 ± 0.7°C during 104 ± 18 min of rewarming. The core-to-peripheral tissue temperature gradient was −5.9 ± 0.9°C at the end of cooling and 4.7 ± 1.5°C at the end of rewarming. The core-temperature afterdrop was 2.2 ± 0.4°C and lasted 89 ± 15 min. It was associated with 1.1 ± 0.7°C peripheral warming. At the end of cooling, temperatures at the center of the upper and lower thigh were (respectively) 8.0 ± 2.2°C and 7.3 ± 4.2°C cooler than skin temperature. On completion of rewarming, tissue at the center of the upper and lower thigh were (respectively) 7.0 ± 2.2°C and 6.4 ± 2.3°C warmer than the skin. When estimated systemic heat loss was included in the calculation, redistribution accounted for 73% of the afterdrop, which is similar to the contribution observed previously in nonsurgical volunteers. Implications: Temperature afterdrop after bypass at 17°C was 2.2 ± 0.4°C, with approximately 73% of the decrease in core temperature resulting from core-to-peripheral redistribution of body heat. Cooling and rewarming were associated with large radial tissue temperature gradients in the thigh.

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A rapid reduction in core temperature is common after cardiopulmonary bypass (CPB) (1). This core hypothermia (afterdrop) is believed to result from a redistribution of body heat from core-to-peripheral tissues (2). It is reasonable to assume that the core-to-peripheral temperature gradient, and therefore the potential for redistribution, would be magnified by lower bypass temperatures. Consistent with this theory, the core-to-peripheral tissue temperature gradient was 3.5 ± 1.8°C at the end of rewarming and the afterdrop was 1.5 ± 0.4°C when bypass was conducted at 31°C, whereas the gradient was 4.6 ± 1.9°C and afterdrop was 2.3 ± 0.9°C at a bypass temperature of 27°C (3).

When prolonged brain ischemia is anticipated in patients undergoing circulatory arrest, core temperature is reduced to 16–18°C because such low temperatures provide additional protection (4). Unless rewarming time is prolonged substantially, a natural consequence of rapid and large core-temperature perturbations is marked exaggeration of the core-to-peripheral tissue temperature gradient.

Rapid induction of profound hypothermia and subsequent rewarming is also likely to produce abnormal tissue-temperature gradients within the peripheral thermal compartment. Quantifying local temperature distribution is important for several reasons. Tissue temperatures, for example, indicate the time-dependent efficacy of regional rewarming and indicate the extent to which

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prolonging warming moderates local temperature gradients and increases peripheral tissue heat content. Furthermore, redistribution of heat is hardly restricted to the core-to-peripheral axis: heat can also be redistributed within discrete tissue volumes depending on final bypass temperatures and the ability of vasomotion and tissue insulation to maintain local temperature. Finally, regional tissue temperature distribution after major thermal perturbations (such as bypass) can help to determine the extent to which vasoactive drugs and thermo-regulatory vasodilation facilitates homogeneous tissue heat distribution. We therefore determined the time-dependent temperature distribution in six extremity segments by fitting numerous skin and tissue temperatures to fourth-order radial regressions.

Methods

With approval from the Ethics Committee of the University of Vienna and written, informed consent, we studied eight patients undergoing thoracic aorta graft procedures. Three of the procedures were elective, but the others were urgent. All patients had left-ventricular ejection fractions exceeding 50%. We enrolled only patients aged 40–80 yr for whom we did not anticipate needing large amounts of intra- or postoperative vasoactive medications. Potential patients were excluded when they had a body mass index exceeding 30 kg/m² or an ASA physical status of IV. Three of eight patients took vasoactive medications before surgery (i.e., nitroglycerin, calcium-channel antagonists, β-blockers, and/or inhibitors of angiotensin-converting enzyme).

Patients were premedicated with oral diazepam 10 mg. A central venous catheter was inserted into the superior vena cava, and a catheter was inserted into a radial artery. Anesthesia was induced by IV administration of etomidate 0.25 mg/kg, midazolam 0.1 mg/kg, fentanyl 5 μg/kg, and pancuronium 0.1 mg/kg. The patients’ tracheas were intubated, and mechanical ventilation was adjusted to maintain PETCO₂ near 35 mm Hg. Anesthesia was maintained with fentanyl 0.3 mg/h and midazolam 4 μg/h, and patients’ lungs were ventilated with oxygen and air.

The arterial cannulation site during cooling was the femoral artery in six patients and the ascending aorta in two patients. During rewarming, the arterial cannulation site was moved to the ascending aorta in all patients. The venous cannulation site was the right atrium. Topical cardiac cooling was not used. The bypass pump was primed with 2100 mL of lactated Ringer’s solution, and bypass flow was maintained at 2.5 L·min⁻¹·m⁻². A membrane oxygenator was used in all cases.

Patients were cooled during CPB to a minimal nasopharyngeal temperature of 16.8 ± 1.1°C. They were rewarmed on completion of surgery to a nasopharyngeal temperature near 36.5°C and a rectal temperature near 36°C. Rewarming was accomplished using the heat exchanger on the CPB machine at a flow of 2.8–3.0 L·min⁻¹·m⁻²; the water bath temperature was maintained approximately 8°C greater than core temperature. CPB was subsequently discontinued, and patients were transferred, intubated and sedated, to the intensive care unit. Patients were covered by standard surgical draping; no active surface warming was used during the study period.

Blood pressures and heart rates were recorded every 5-min, along with arterial oxygenhemoglobin saturation and PETCO₂. Core temperatures were recorded from the nasopharynx. All measurements were continued until at least 1 h after CPB.

Arm and leg tissue temperatures were determined as previously described (5). Briefly, right leg muscle temperatures were recorded using 8-, 18-, and 38-mm, 21-gauge needle thermocouples inserted perpendicular to the skin surface. Needles were inserted into the upper and lower thigh, upper and lower calf, upper arm, and forearm. Skin surface temperatures were recorded immediately adjacent to each set of needles and directly posterior to each leg set. Temperatures were recorded from thermocouples connected to calibrated 16-channel electronic thermometers with an accuracy of 0.1°C and a precision of 0.01°C. Temperatures were measured at 5-min intervals, starting approximately 30 min after the induction of anesthesia. Subcutaneous temperature was measured on the ball of the foot and palm of the hand using a Coretemp® “deep tissue” thermometer (Terumo Medical Corp., Tokyo, Japan) (6). This device estimates tissue temperature approximately 1 cm below the skin surface.

Arm and anterior leg tissue temperatures, as a function of radial distance from the center of the leg segment, were calculated using skin-surface and muscle temperatures using fourth-order regressions. Temperatures at the center of the upper and lower thigh were set to the core temperature. In contrast, temperatures at the center of the arm and upper and lower calf segments were estimated from the regression equation with no similar assumption. Individual values at 0.5-cm intervals were computed, then averaged among the patients to produce mean radial temperature distributions at various study times. Average anterior radial temperature distributions were calculated from the forth-order regressions by integration of volume.

Anterior limb heat content was estimated from these temperatures, using a fourth-order modification of a previously described formula (7):

\[
Q_{0 \rightarrow r} = \frac{2(\pi r^2)Ls}{a_0 + a_2r^2 + a_4r^4/3},
\]

where \(Q_{0 \rightarrow r}\) (cal) is heat content of the leg segment from the center to radius \(r\). \(L\) (cm) is the length of the
Results

Six patients received grafts of the ascending aorta, one had a combined ascending and arch graft, and one was given a combined arch and descending graft. Deep hypothermic circulatory arrest was used in all patients and lasted 28 ± 7 min. Five of the eight patients were male. The average age of the patients was 59 ± 11 yr, they were 172 ± 7 cm tall, and they weighed 84 ± 17 kg. Seven of eight patients required small amounts of nitroglycerin during bypass. None of our patients received sodium nitroprusside during the procedure. Estimated masses of the legs and arms were 30 ± 6 kg and 10 ± 3 kg, respectively. Consequently, arms and legs represented 48% ± 4% of the patients’ total mass.

Cooling required 64 ± 16 min, and bypass lasted 195 ± 45 min. Core temperature at the onset of bypass, approximately 1 h after the induction of anesthesia, was 35.3 ± 0.9°C. Core temperature decreased to a minimum of 16.8 ± 1.1°C during bypass, then increased to 36.5 ± 0.3°C. The peripheral tissue temperature at the onset of bypass was 33.7 ± 0.7°C; peripheral tissue temperature decreased to a minimum of 22.6 ± 2.1°C during bypass and subsequently increased to 31.8 ± 1.5°C during 104 ± 18 min of rewarming. The core-to-peripheral tissue temperature gradient was 1.6 ± 0.8°C at the onset of bypass, −5.9 ± 2.9°C at the end of cooling, and 4.7 ± 1.5°C at the end of rewarming. The core-temperature afterdrop was 2.2 ± 0.4°C and lasted 89 ± 15 min after bypass (Fig. 1). It was associated with a 1.1 ± 0.7°C increase in peripheral tissue temperature.

Peripheral (arm and leg) tissue heat content at the onset of pump cooling was 1199 ± 233 kcal, whereas core (trunk and head) heat content was 1284 ± 352 kcal. Peripheral and core heat contents decreased 378 ± 66 and 670 ± 175 kcal, respectively, during bypass cooling. Total body heat content thus decreased 1057 ± 215 kcal. Even after pump rewarming, peripheral heat content remained 73 ± 75 kcal below precooling values. In contrast, core heat content increased to 41 ± 39 kcal above precooling values. Body heat content at the end of rewarming was thus 32 ± 104 kcal less than at the onset of cooling. During the first hour after discontinuation of bypass, core heat content decreased 73 ± 18 kcal, whereas peripheral heat content increased 35 ± 47 kcal (Fig. 2).

Longitudinal convection of heat along the extremities was insufficient to equilibrate peripheral and core tissue temperatures during the rapid thermal perturbations associated with hypothermic bypass. For example, temperature in the central axis of the leg after cooling was 6.0 ± 4.3°C greater than core temperature at the upper calf, 7.1 ± 4.2°C greater than core temperature at the lower calf, and 6.3 ± 4.1°C greater than core temperature in the foot. At the end of rewarming, the situation was reversed, with the upper calf being 2.9°C less than core temperatures. At that time, temperature at the center of the lower calf exceeded core temperature by 4.9 ± 2.0°C, whereas the foot exceeded core temperature by 6.7 ± 3.4°C.

Temperatures at the center of thigh, calf, and arm segments were roughly 2°C greater than skin temperature before active cooling started. At the end of cooling, temperatures at the center of the upper and lower thigh were (respectively) 8.0 ± 5.2°C and 7.3 ± 4.2°C cooler than skin temperature. There was thus a substantial (and nonlinear) radial temperature gradient through the thigh. On completion of rewarming, we
again observed a substantial temperature gradient, but now tissue at the center of the upper and lower thigh was 7.0 ± 2.2°C and 6.4 ± 2.3°C warmer than skin temperature. One hour after rewarming (when redistribution was largely complete), the thigh temperature patterns were again similar to those before cooling started (Fig. 3).

Active cooling also substantially reduced tissue temperature in the calves. However, there was relatively little radial or longitudinal temperature gradient, and tissue temperatures were similar throughout the region. The calves thus differed markedly from the thighs in that temperature at the center of these distal segments remained well above core temperature even at the end of cooling. Radial inequality was apparent at the end of rewarming; however, the gradient between skin and the center of the extremity was considerably smaller in the calf than in the thigh. As in the thigh, the radial temperature distribution in the arms better resembled the relatively flat pattern of the calf than the steep gradient in the thigh (Fig. 5).

Discussion

Patient core temperature in this study decreased roughly twice as much as the 27°C bypass group in our previous study of heat balance during and after cardiac surgery (20 vs 10°C) (3). As might be expected from such profound and rapid cooling, peripheral tissues remained considerably warmer than the core at the end of cooling. The core-to-peripheral temperature gradient was thus 5.9 ± 2.9°C when rewarming started. However, the rewarming period was considerably longer than that in our previous study: 104 ± 18 vs 66 ± 24 min. As a result, the final core-to-peripheral tissue temperature gradient was 4.7 ± 1.5°C, which
was virtually identical to the gradient we observed previously after bypass at 27°C. Magnitude of the afterdrop, 2.2°C, was also virtually identical to that we observed previously.

These data indicate that there was sufficient time during rewarming for substantial amounts of core heat to move peripherally. In other words, blood-borne convection of heat and direct diffusion restricted the final core-to-peripheral tissue temperature gradient, and therefore, magnitude of the afterdrop. Presumably, the core-to-peripheral tissue gradient would have been reduced had rewarming been slower; similarly, the gradient may have been increased if rewarming had been faster. The rewarming protocol we used, however, is typical and likely applies to most patients managed with deep hypothermia.

The first postbypass hour was associated with a 35 ± 47-kcal increase in peripheral tissue heat content, which was insufficient to explain the observed 2.2°C afterdrop. However, we must also consider metabolic heat production and heat loss to the environment. Metabolic heat production during anesthesia is usually 40–55 kcal/h (5,9,10), which is considerably less than typical cutaneous losses in anesthetized, normothermic subjects (i.e., 80–100 kcal/h) (5,10). Actual systemic heat balance in our patient was not measured and can only be approximated because loss was surely increased by evaporation from within surgical incisions (11) but simultaneously reduced by insulating covers (12). Nonetheless, it seems likely that cutaneous loss exceeded metabolic rate by roughly 40 kcal/h during the postbypass period.

If it is assumed that the inequity between heat loss and production is evenly distributed between core and peripheral compartments, this will distribute systemic cooling in proportion to the relative masses of each compartment. Peripheral tissue heat content increased 35 ± 47 kcal in the first postbypass hour. This value, though, significantly underestimates redistribution from the core because the periphery’s share of the systemic heat loss (approximately 20 kcal) must be considered. Another approach is to consider that the total amount of heat redistributed from the core is the observed increase in peripheral tissues (35 kcal) plus the 20 kcal lost from the periphery into the environment. Core-to-peripheral redistribution thus accounted for roughly 1.6°C or 73% of the total. This value is similar to the directly measured 81% observed in volunteers not undergoing surgery (5). The remaining core hypothermia results from the other 20 kcal of systemic heat imbalance combined, perhaps, with a small amount of redistribution within the core thermal compartment.

It is thus apparent from these measurements and estimates that core-to-peripheral redistribution of body heat is responsible for most of the afterdrop, with an imbalance in systemic heat balance contributing the remainder. To the extent that environmental heat loss contributes, cutaneous insulation or active warming will ameliorate afterdrop. Measures that increase peripheral tissue warming including prolonged bypass (13), pulsatile flow (14), or nitroprusside administration (15), may also be helpful.

Large radial temperature gradients were apparent at the end of cooling and the end of rewarming—the periods of maximal thermal perturbation. Gradients were most pronounced in the thigh and sometimes
exceeded 0.5°C/cm. Previous investigators have estimated peripheral compartment temperature and the adequacy of rewarming during bypass from skin temperature (16) or a limited number of tissue measurements (17). Our data suggest that substantial errors are likely when peripheral temperature and heat content are extrapolated from just a few measurement sites. Errors are especially likely in the proximal segments because these regions seem to have the largest regional temperature gradients. In contrast, the extrapolation error will be reduced if measurements are restricted to relatively steady-state periods, such as before cooling or well after rewarming and redistribution is complete.

We evaluated a single group of patients, all of whom were cooled to approximately 17°C. This limitation is inherent in our clinical population because deep hypothermia is only indicated for specific conditions; we therefore could not randomly assign these patients to higher bypass temperatures. Comparison between our current and previous (3) results should thus be approached with some caution. Nonetheless, the protocol and methodology were similar in each protocol, and it is likely that our general conclusions about afterdrop magnitude and mechanism at each temperature are correct. We previously described the limitations of our regional tissue temperature estimates (5,7). All those limitations apply to this study, with the further caveat that tissue temperature estimates are least reliable during periods of rapid thermal perturbation.

In summary, afterdrop after bypass at 17°C was similar to that observed during a previous study conducted at a bypass temperature of 27°C. Afterdrop was presumably similar because longer rewarming in the patients cooled to 17°C resulted in a final core-to-peripheral tissue temperature gradient that was virtually identical to that in the previous patients who were cooled to only 27°C. When estimated systemic heat loss was included in the calculation, redistribution accounted for 73% of the afterdrop, which is similar to the contribution previously observed in nonsurgical volunteers. Large radial gradients in the thighs indicate that measurements restricted to a few sites will poorly describe the temperature of this tissue.

References