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Perioperative Heat Balance

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ANESTHESIA and surgery commonly cause substantial thermal perturbations. Hypothermia, the typical alteration, results from a combination of anesthetic-induced impairment of thermoregulatory control, a cool operating room environment, and factors unique to surgery that promote excessive heat loss. Available data suggest that inhibition of normal thermoregulatory defenses contributes more to hypothermia than does cold exposure *per se*. Furthermore, much core hypothermia results from altered distribution of body heat rather than from a systemic imbalance between metabolic heat production and heat loss.

General Features of Heat Production and Distribution

Tissue temperature is directly related to heat content by the specific heat of tissue, which is approximately

$0.83 \text{ kcal} \cdot \text{kg} \cdot ^{-1} \cdot ^\circ\text{C}^{-1}$.^{1,2} Body temperature perturbations are thus a direct consequence of alterations in tissue heat content. How heat is generated and distributed within the body is therefore of considerable interest.

Heat Production

Body tissues produce heat in proportion to their metabolic rates.^{3,4} The First Law of Thermodynamics specifies that energy generation by a chemical reaction is determined only by the substrates and products of the reaction. Nearly all of this energy is eventually converted to heat. The major substrates for human metabolism are glucose, protein, and fat; the major products of aerobic metabolism are generally carbon dioxide and water. Combustion of glucose and protein produces 4.1 kcal/kg, whereas fat releases 9.3 kcal/kg.

The brain and major organs in the trunk are the most metabolically active tissues and generate more metabolic heat than does muscle at rest.⁵⁻⁷ In contrast, heat production by skeletal muscle can, at least briefly, exceed the basal metabolic rate by a factor of 10. Metabolism is normally the only internal source of heat, although internal heating can result from drinking warm beverages, microwave radiation, or magnetic resonance gradient coils.⁸

Thermal Compartments

The human body can very roughly be described as having a core thermal compartment and a peripheral compartment.¹ The core is defined by well-perfused tissues in which temperature remains relatively uniform. In other words, distribution of heat within this compartment is fast compared with rates at which heat content normally changes. As a result, temperatures at various sites within the core compartment rarely differ by more than a few tenths of a degree centigrade. Physically, the core compartment consists of the trunk and head. Of course, the skin and most peripheral tissues of the trunk and head are not technically part of the core; however,

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little accuracy is lost by ignoring this mass. The core compartment therefore comprises 50–60% of the body mass.⁹

Tissues in which temperature is nonhomogeneous and variable over time define the peripheral thermal compartment. Physically, this compartment consists of the arms and legs. Temperature of the peripheral compartment is usual 2–4°C less than the core temperature in moderate environments.¹ However, this difference can become large during extreme thermal¹⁰ or physiologic¹¹ circumstances. Lower core-to-peripheral temperature gradients result when the environment is warm or when thermoregulatory vasodilation allows facile flow of metabolic heat (which is largely generated in the core) to the periphery. In contrast, vasoconstriction constrains metabolic heat to the core, increasing the core-to-peripheral temperature gradient.

There are typically substantial longitudinal tissue temperature gradients within the extremities, with distal tissues being several degrees cooler than those positioned more proximally. There are also often substantial radial tissue temperature gradients, which become especially large in extreme thermal environments.^{10,12} A cardinal feature of the peripheral compartment is that heat content and distribution change substantially over time and as a function of environmental exposure.¹³ This is in distinct contrast to the core for which temperature is usually precisely regulated.

In a warm environment, especially in vasodilated subjects, tissue temperature in much of the peripheral compartment can equal core temperature. Some authors refer to this situation as an expansion of the core compartment. However, temperature of these tissues will decrease and again become nonuniform in a cooler environment. It is thus easier to consider physical size of the compartments to be fixed and instead allow temperature of the peripheral compartment to vary. It is not wrong to permit flexibility in both the size and the temperature of the compartments; however, doing so adds unnecessary complexity to the discussion.

Heat Flow and Generation

In contrast to the rapid distribution of heat within core tissues, heat flows relatively slowly to peripheral tissues. Core-to-peripheral flow of heat is mediated by blood-borne convection of heat and conduction of heat into adjacent tissues.¹⁴ The convective component can very roughly be modeled as a large and relatively rapid longitudinal flow of heat within the big axial vessels of the extremities. The major factors that influence convective

distribution of heat are peripheral blood flow, counter-current heat exchange between adjacent arteries and veins, and the core-to-peripheral temperature gradient.

The conductive component, in contrast, is a slower radial flow of heat from relatively warm tissue at the central axis to cooler tissues near the skin. Conductive flow is largely determined by the diffusion coefficient, which is to a great extent a function of tissue characteristics. For example, fat insulates nearly three times as well as muscle¹⁵ and provides substantial insulation.^{16,17} Hence conductive heat transfer depends mostly on intrinsic tissue characteristics rather than on thermoregulatory factors, such as vasomotion.

Deposition of heat into peripheral tissues is augmented by heat that is produced by local tissue metabolism and diminished by regional cutaneous heat loss to the environment. Regional heat transfer and local production is fully described by the bioheat equation.¹⁸ However, a precise solution to this equation requires numerous input values that cannot normally be measured. Consequently, a number of equations have been developed to quantify various aspects of tissue heat transfer and local heat production in terms of measurable quantities.^{19,20}

Modifications of the bioheat equation have been used to construct numerous models of tissue heat distribution.^{20,21} Even simple models perform reasonably well within a restricted set of circumstances, such as cold-water immersion. However, they usually prove less accurate during other conditions, prompting refinements in subsequent generations of the models. As a result, some models have become quite complicated.²²

Nonetheless, most models of tissue heat flow have been validated only to a limited extent, and many do not include a sophisticated thermoregulatory component. Ignoring thermoregulatory compensations is acceptable in a situation of extreme thermal stress in which it is reasonable to assume that warm or cold defenses are maximally activated. However, failure to include thermoregulatory defenses can be a substantial limitation in more typical environments in which active control—rather than environmental exposure—is the major determinant of core temperature. Heat balance models therefore usually have proven to be less helpful than initially hoped, leaving investigators largely dependent on experimental data. Fortunately, detailed measurements are becoming available in a variety of circumstances.^{9,12,13,20,23–26}

All metabolic heat must eventually be dissipated to the environment to maintain thermal steady state. Approximately 95% of this heat traverses the skin surface, with

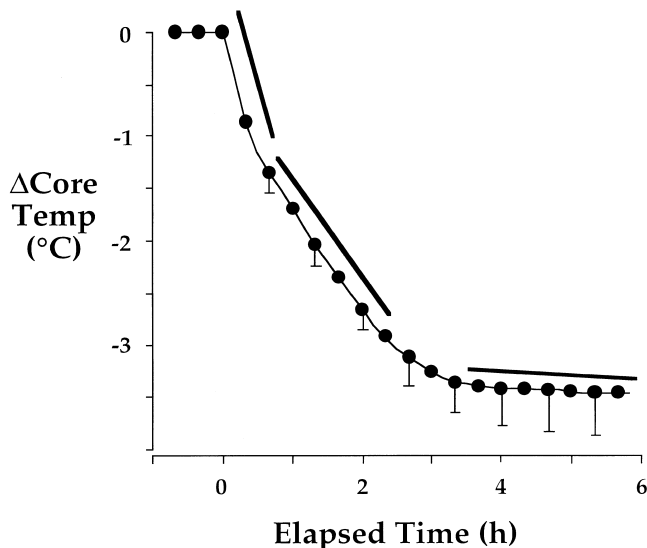


Fig. 1. Typical pattern of hypothermia during general anesthesia. Hypothermia during anesthesia develops with a characteristic pattern. During the first hour, core temperature usually decreases 1–1.5°C. This is followed by a slower, nearly linear decrease in core temperature. Finally, core temperature reaches a plateau and does not decrease further. Each segment of this hypothermia curve has a different cause. Reprinted with permission from Kurz *et al.*⁸⁰

the remaining small amount being lost *via* the respiratory track.^{27,28} In the absence of sweating, only about 10% of cutaneous heat loss is evaporative in adults (although the fraction can be considerable in infants, especially premature infants).²⁹ Sweating, however, is remarkably effective and can dissipate 10 times the basal metabolic rate in a dry, convective environment.^{30,31} The upper chest and face are most sensitive to temperature. However, the common belief that half of body heat is lost through the head only applies when the rest of the body is covered by an arctic survival suit.³² Instead, with comparable levels of insulation, heat loss is roughly proportional to surface area over the entire body and is not much changed by thermoregulatory vasomotion.^{33,34}

General Anesthesia

Nearly all patients administered general anesthesia become hypothermic, typically by 1–3°C,³⁵ depending on the type and dose of anesthesia, amount of surgical exposure,^{36,37} and ambient temperature.^{36–38} Hypothermia develops with a characteristic pattern (fig. 1).¹ Core temperature decreases 1–1.5°C during the first hour.² This initial hypothermia is followed by 2 or 3 h of a

slower, linear, decrease in core temperature.³ Finally, patients enter a plateau phase during which core temperature remains constant. Each segment of this typical hypothermia curve has a different cause.

Redistribution

The core thermal compartment consists of well-perfused tissues of the trunk and head that are maintained at a relatively high temperature. Core temperature, though, poorly represents mean body temperature because peripheral tissues are typically 2–4°C cooler than the trunk and the head.^{9,26} This normal core-to-peripheral tissue temperature gradient is maintained by tonic thermoregulatory vasoconstriction of arteriovenous shunts in the fingers and toes.^{39–42}

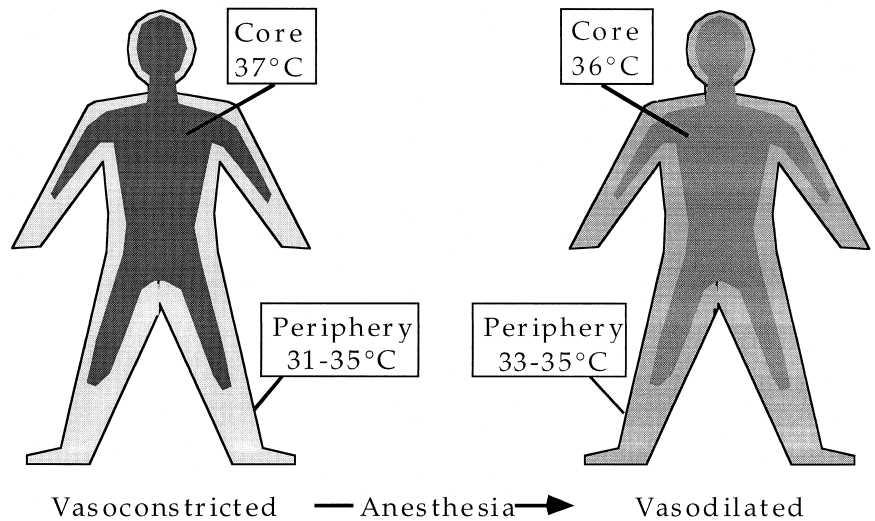
Mechanism. Induction of general anesthesia promotes vasodilation *via* two mechanisms: (1) General anesthesia reduces the vasoconstriction threshold to well below core temperature,^{43–48} thus inhibiting centrally mediated thermoregulatory constriction. (2) Most anesthetics cause direct (peripheral) vasodilation.⁴⁹ Vasodilation allows core heat, which is no longer constrained to the central thermal compartment, to flow down the temperature gradient into peripheral tissues (fig. 2).⁵⁰ This internal redistribution of body heat decreases core temperature and proportionately increases temperature of peripheral tissues; it does not, however, represent any net exchange of heat to the environment, and body heat content remains constant. Of course any systemic cooling that occurs simultaneously will augment core hypothermia.

A quantitative study of systemic heat balance and regional body heat distribution in volunteers indicated that after 1 h of anesthesia, core temperature decreased 1.6°C, with redistribution contributing 81% to the decrease. During the subsequent 2 h of anesthesia, core temperature decreased an additional 1.1°C, with redistribution contributing only 43%. Redistribution therefore contributed 65% to the total decrease in core temperature during the first 3 h of anesthesia (fig. 3).⁹ Core-to-peripheral redistribution was therefore the primary cause of hypothermia during the initial phase of anesthesia—and in this study remained the dominant cause even after 3 h.

Major Factors Influencing Magnitude. The extent to which induction of general anesthesia induces redistribution hypothermia in individual patients depends on a number of factors. Among the most important is the patient's initial body heat content. Core temperature, which is precisely regulated, remains essentially normal

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Fig. 2. Cartoon showing redistribution hypothermia after induction of general anesthesia. Body heat content is not normally distributed evenly. Instead, tonic thermoregulatory vasoconstriction maintains a core-to-peripheral temperature gradient. Induction of general anesthesia inhibits vasoconstriction, allowing a core-to-peripheral redistribution of body heat. Reprinted with permission from Sessler DI: Temperature monitoring, *Anesthesia*, 4th edition. Edited by Miller RD. New York, Churchill Livingstone, 1994, pp 1363–82.



even in warm environments.^{51,52} However, body heat content increases as peripheral tissues absorb heat; after a number of hours in a sufficiently warm environment, peripheral tissue temperature approaches core temperature.⁵³ Because flow of heat needs a temperature gradient, redistribution magnitude is limited when peripheral and core temperatures are similar.^{54,55}

An additional important factor is body morphology. Obese patients redistribute considerably less than those of normal weight; conversely, very thin patients redistribute more (fig. 4).⁵⁶ Reduced redistribution hypothermia in obese patients apparently results because the major thermoregulatory problem in these well-insulated patients is dissipation of metabolic heat. Consequently, they spend much of their time in vasodilation, which differs from the constricted state most patients maintain in a hospital environment. The result is that their peripheral tissue temperature is higher than normal, which reduces core-to-peripheral flow of heat after induction of anesthesia.

The fractional contribution of redistribution to core hypothermia naturally depends on the amount of redistribution. However, it also depends on the contribution of systemic heat loss, which in turn depends on numerous factors (discussed in Linear Phase section). Factors that increase systemic heat imbalance, such as a cool environment³⁶⁻³⁸ or large surgical incision,⁵⁷ will increase the total amount of core hypothermia but decrease the fraction that results from redistribution.

Linear Phase

The second portion of the hypothermia curve is a relatively slow, linear decrease in core temperature. It

results simply from heat loss exceeding metabolic heat production. Metabolic rate is reduced by 15–40% by during general anesthesia.^{9,58,59} The exact causes for this reduction are not well-established, but decreased brain metabolism contributes, as does mechanical ventilation, which spares the diaphragm and chest wall muscles. To some extent, the process can be reversed by administration of amino acids^{59,60} or caffeine.⁶¹⁻⁶³

Cutaneous heat loss is mediated by the same four fundamental mechanisms that modulate heat transfer between any two substances: radiation, conduction, convection, and evaporation (fig. 5).⁶⁴ Among the four heat loss pathways, only conductive loss depends linearly on the temperature difference between patients and their environments. Nonetheless, surprisingly little accuracy is lost by considering total heat loss from all four mechanisms to be a linear function of the skin-to-ambient temperature difference over the typical range of operating room temperatures.

It is during the linear phase the factors altering heat loss are most apparent. This includes relatively great heat loss in infants and children, more hypothermia during large than small operations, and reduced hypothermia when surgery is performed in a warm environment.³⁶⁻³⁸ This is also the period in which passive insulation and active intraoperative heating is most effective.⁶⁵⁻⁶⁸

Radiation. Among the four potential heat-loss mechanisms, radiation usually contributes most.⁶⁹ Radiation is the transfer of heat from one surface to another *via* photons and therefore does not depend at all on the temperature of the intervening air. The amount of heat lost by radiation is a function of the emissivity of the two surfaces and the difference between the fourth power of

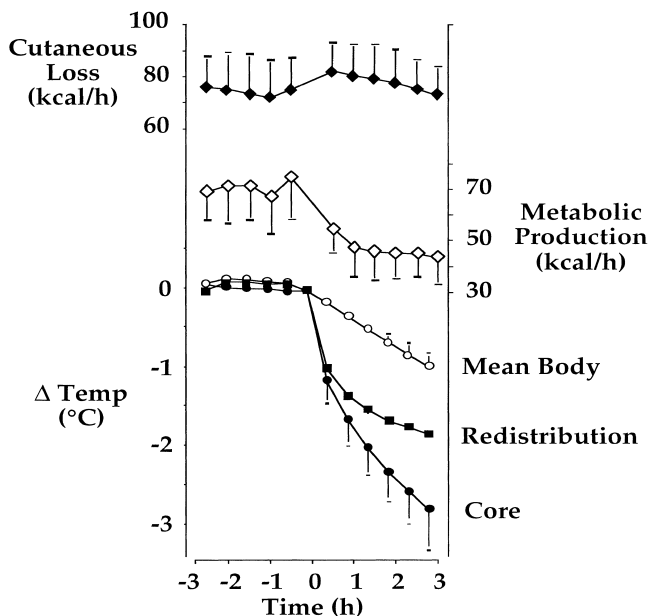


Fig. 3. Quantification of redistribution hypothermia. Overall heat balance was calculated from the difference between metabolic heat production and heat loss. To separate the contributions of decreased overall heat balance and internal redistribution of body heat to the decrease in core temperature, the change in overall heat balance was multiplied by body weight and the specific heat of humans. The resulting change in mean body temperature (mean body) was subtracted from the change in core temperature (core), leaving the core hypothermia specifically resulting from redistribution (redistribution). After 1 h of anesthesia, core temperature decreased $1.6 \pm 0.3^\circ\text{C}$, with redistribution contributing 81% to the decrease. During the subsequent 2 h of anesthesia, core temperature decreased an additional $1.1 \pm 0.3^\circ\text{C}$, with redistribution contributing only 43%. Redistribution contributed 65% to the entire $2.8 \pm 0.5^\circ\text{C}$ decrease in core temperature during the 3 h of anesthesia. Induction of general anesthesia is identified as elapsed time zero. Reprinted with permission from Matsukawa *et al.*⁹

their temperatures in degrees Kelvin. *Emissivity* defines an object's ability to absorb and emit heat; these two effects are always identical, otherwise objects would continue to absorb or lose heat indefinitely. Objects that absorb and emit heat perfectly are termed "black bodies" and have an emissivity of one. In contrast, perfect mirrors have an emissivity of zero. Human skin (of all colors) acts very much like a black body and has an emissivity near 0.95 for the infrared light.

Conduction and Convection. Convection is often considered "facilitated (or forced) conduction" because the two types of heat loss share a fundamental mechanism. Conduction is the direct transfer of heat from one surface to a second adjacent surface. Heat transfer, in this case, is proportional to the difference in the surface temperatures and any insulation between them. This

insulation might be a real barrier (such as a sheet inserted between a patient and a circulating-water mattress) or effective insulation when the surfaces themselves conduct heat poorly.

Air movement reduces the buildup of heat near the skin surface by displacing warmed air with cooler air. This convection markedly increases flow of heat compared with still air, with the improvement being proportional to the square root of the air velocity. (Convection is the basis of the popular "wind-chill factor.") Even in the relatively still air of an operating room (with a typical velocity near 20 cm/s), convection remains the second most important source of heat loss during anesthesia and surgery. It becomes the dominant source of heat loss in highly convective environments, such as laminar flow units.

Hypothermia that results from administration of cool intravenous fluids does not easily fit into one of the major heat-loss categories. However, it is best considered a type of conduction because the administered fluids are warmed to body temperature by conduction from blood and tissues. Warming administered fluids therefore requires transfer of heat from body tissues to the cool fluid (even if the fluid is now well-distributed

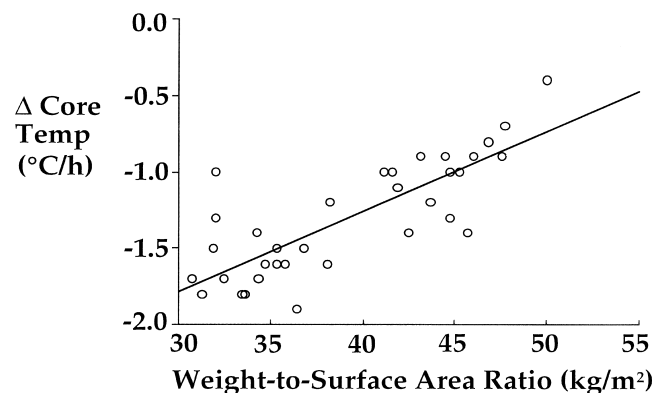


Fig. 4. The influence of body morphology on redistribution hypothermia. The amount of redistribution hypothermia (reduction in core temperature during the first hour of anesthesia) was inversely proportional to the weight-to-surface area ratio: $\Delta \text{core temp} = 0.0524 \cdot \text{Wt/SA} - 3.35$; $R^2 = 0.66$. The 95% confidence interval for the slope was $0.040-0.065^\circ\text{C} \cdot \text{h}^{-1} \cdot \text{kg}^{-1} \cdot \text{m}^2$. Reduced redistribution hypothermia in obese patients apparently results because the major thermoregulatory problem in these well-insulated patients is dissipation of metabolic heat. Consequently, they spend much of their time vasodilated, which differs from the constricted state most patients maintain in a hospital environment. The result is that peripheral tissue temperature in obese patients is higher than normal, which reduces core-to-peripheral redistribution of heat after induction of anesthesia. Reprinted with permission from Kurz *et al.*⁵⁶

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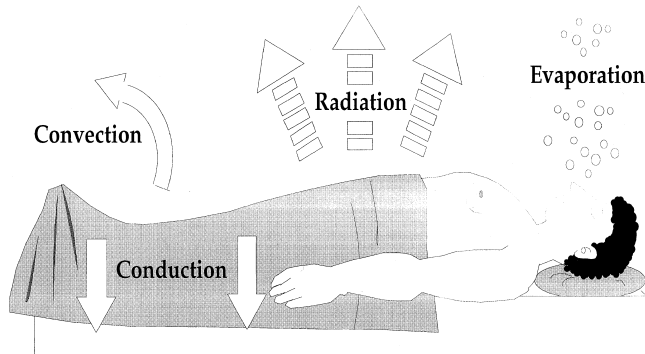


Fig. 5. The linear second phase of the hypothermia curve. The second phase of the hypothermia curve results from heat loss exceeding metabolic heat production. Typical heat production is approximately $1 \text{ kcal} \cdot \text{kg}^{-1} \cdot \text{h}^{-1}$. At steady state, heat loss must equal this amount. During anesthesia and surgery, however, heat loss is often far greater. The major cause of loss is radiation, accounting for roughly 60% of the total. Radiative loss is proportional to the difference of the fourth powers of room wall (*i.e.*, ambient) and skin temperatures. The remaining loss is largely convective, which is proportional to the difference between skin and air temperatures multiplied by the square root of air speed. Respiratory evaporative loss contributes only approximately 10% to the total and cutaneous evaporative loss remains relatively small except during sweating. However, evaporative loss from within surgical incisions can be substantial. Conductive loss is unimportant during anesthesia.

within the body). Reducing heat content of the remaining body tissues naturally reduces their temperature.

Evaporation. Evaporative heat loss is derived from the heat of vaporization of water, which is a substantial 0.58 kcal/g . To put this in perspective, evaporation of 1 g water at 100°C needs nearly six times as much energy as heating it from 0 to 100°C . Fortunately, adults normally lose little water through intact skin. Only about 5% of the basal metabolic rate is thus lost from evaporation of transcutaneous water during normal (nonsweating) circumstances. Respiratory evaporative losses are also small, usually also less than 10% of the basal metabolic rate.^{27,70}

There is substantial evaporative loss from within surgical incisions. For example, it is well-established that core hypothermia is more pronounced during large³⁷ than small³⁶ operations, with most of the difference presumably resulting from evaporative loss. Furthermore, Roe³⁷ demonstrated that approximately half the total heat loss is evaporative in rabbits with large abdominal incisions. Some caution is necessary when extrapolating this result to humans because humans have a relatively smaller abdomen than rabbits. Even more importantly, rabbit skin is well-insulated by fur; therefore, a smaller proportion of total heat loss will occur *via* their

skin. The contribution of evaporation from within surgical incisions remains to be determined in humans because of technical difficulties. An additional difficulty is that surgical incisions presumably also substantially increase loss by radiation.

Perioperative heat loss also results from evaporation of skin-preparation solutions. Heat loss resulting from skin preparation has been measured and mathematically modeled.⁷¹ Heat loss is significantly less with water- than alcohol-based solutions. Heating the solutions and radiant warming somewhat decrease heat loss, but also unacceptably increase the risk of chemical skin irritation. The decrease in mean body temperature in a 70-kg patient ranges from -0.2 to -0.7°C/m^2 depending on the solution and condition of application. The smallest decrease occurs during radiant warming and washing with water-based solutions, whereas the largest decreases result when the skin is washed with warm or cold alcohol-based solutions. These losses are not trivial, but, in most patients, will be small compared with other causes of core hypothermia.

Core Temperature Plateau

The final phase of the typical intraoperative hypothermia curve is a core temperature plateau that usually develops after 2–4 h of anesthesia and surgery. It is characterized by a core temperature that remains constant, even during prolonged surgery. The core temperature plateau is sometimes passive, and sometimes actively maintained.

Passive Plateau. A passive plateau results when metabolic heat production equals heat loss, without activating thermoregulatory defenses. Mammals must maintain a thermal steady state over the long-term to keep core temperature constant. However, several factors complicate the situation during anesthesia and surgery: (1) Anesthesia significantly decreases metabolic heat production.^{9,58} (2) Heat loss may be abnormally high because of a relatively cool operating room environment,^{36–38} administration of cool intravenous and irrigating fluids,^{72,73} and evaporative and radiative losses from within surgical incisions.⁵⁷ (3) Behavioral compensations are not available to unconscious patients, and autonomic responses are impaired,^{43–45} at least until patients become quite hypothermic.

The combination of anesthetic-induced reduction in heat production and surgical factors that abnormally increase heat loss means that in normothermic surgical patients a passive plateau rarely develops. However, each degree reduction in core temperature (at constant

ambient temperature) reduces heat loss roughly by 10%. Metabolic heat production also decreases passively, but at a somewhat slower rate (*i.e.*, 6%/°C).⁴ Patients becoming sufficiently hypothermic will therefore eventually reach a passive core temperature plateau when heat loss finally decreases to the point that it equals heat production.

A passive core temperature plateau is most common during relatively small operations in patients who are well-covered with effective insulators.^{74,75} The amount and effectiveness of insulation is a key issue here because ambient temperature and insulation are primary factors that influence heat loss. A special case of passive insulation is observed when active cutaneous warming markedly reduces heat loss. In such situations, heat loss may equal production even during very large operations in a cold environment.

Active Plateau. Patients who become sufficiently hypothermic trigger thermoregulatory vasoconstriction and develop an actively maintained plateau. The critical difference between a passive plateau and one that is actively maintained is that an active plateau depends on thermoregulatory vasoconstriction to decrease heat loss and, especially, to alter distribution of heat within the body. In this regard, core temperature is maintained much as it is normally.

A core temperature between 34 and 35°C is necessary to trigger thermoregulatory vasoconstriction with typical concentrations of most anesthetics.^{43,44,48} Once triggered, vasoconstriction is effective, although largely *via* an unexpected mechanism. Vasoconstriction, surprisingly, only slightly reduces cutaneous heat loss.^{33,76} The reason appears to be that constriction is largely restricted to arteriovenous shunts in the fingers and toes.⁴⁰ In contrast to its modest effect on systemic heat balance, shunt vasoconstriction has an important influence on the distribution of body heat.

Body heat is largely generated by metabolically active organs in the core thermal compartment. Tonic thermoregulatory vasoconstriction normally constrains a portion of that heat in the core compartment, producing the normal 3 or 4°C core-to-peripheral temperature gradient. As discussed previously, induction of anesthesia inhibits constriction, which allows heat to flow from core to peripheral tissues. Once in the periphery, heat cannot return to the core because heat traveling up a temperature gradient would violate the Second Law of Thermodynamics. Reemergence of vasoconstriction therefore cannot recover heat already lost to peripheral tissues. It

does, however, restrict further flow of heat from the core to the peripheral tissues.

The major consequence of thermoregulatory vasoconstriction is therefore that the core remains relatively warmer than might be expected based on systemic heat balance. Typically, this produces a core temperature plateau.⁷⁷ Depending on the environmental temperature and size of the operation, it may be manifested as a slowing in the core cooling rate or even an increase in core temperature.^{78,79}

Peripheral tissues, however, do not fare so well after vasoconstriction. Not only does cutaneous heat loss continue almost unabated,^{33,76} but less heat flows peripherally from the core. The result is that peripheral tissues gradually cool. Eventually, this reduction in tissue temperature decreases cutaneous heat loss. However, the core temperature plateau appears well before a thermal steady state, suggesting that constraint of centrally generated metabolic heat to the thermal core, rather than a reduction in cutaneous loss, is the dominant factor. The major clinical implication of this mechanism is that an actively maintained plateau is typically not a thermal steady state. Instead, body heat content and mean body temperature continue to decrease.

Constraint of metabolic heat has been quantified during general anesthesia.⁸⁰ Vasoconstriction was triggered in volunteers who were in the linear phase of their hypothermia curve. Core temperature, which was decreasing at a rate of approximately 0.6°C before vasoconstriction, remained virtually constant during the subsequent 3 h. The core temperature plateaus resulted in part from a reduction in cutaneous heat loss, but also from constraint of 20 kcal to the core thermal compartment (fig. 6).

Limb Tourniquets. Limb tourniquets constitute a special case of the core temperature plateau. The reason is that tourniquets can be considered a vasoconstriction so intense that no blood or heat is exchanged between the isolated extremity and the remainder of the body. Metabolic heat from the core therefore cannot escape to the isolated region, and from there to the environment. The clinical consequence is that heat is constrained to the core thermal compartment, which then remains relatively warm. The ability of limb isolation to slow core cooling has been shown in volunteers immersed in cold water.⁸¹

In adult patients, a limb tourniquet slows the rate at which core hypothermia develops, or induces, a plateau. Pediatric patients, however, are usually treated in an environment sufficiently warm to maintain normother-

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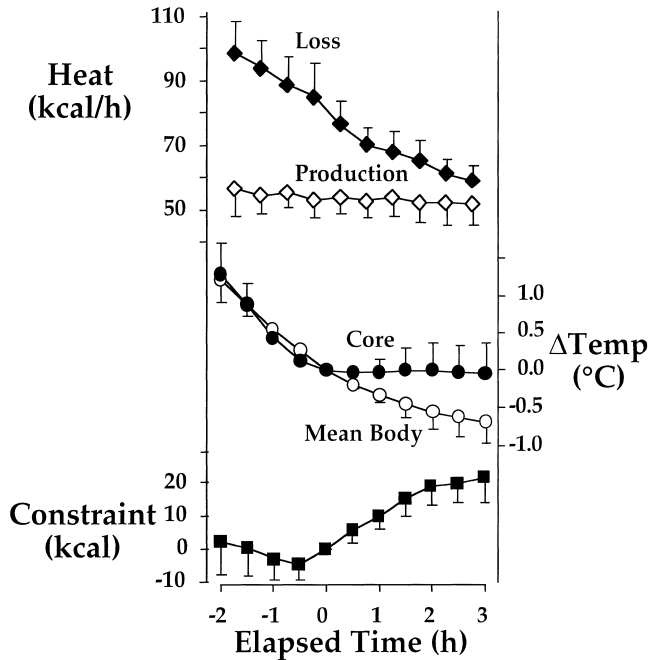


Fig. 6. The core temperature plateau. The third phase of the hypothermia curve coincides with reemergence of thermoregulatory vasoconstriction. Vasoconstriction decreased cutaneous heat loss (adjusted for evaporative and respiratory loss) approximately 25 kcal/h. However, heat loss always exceeded heat production. Consequently, mean body temperature, which decreased at a rate of approximately 0.6°C/h before vasoconstriction, subsequently decreased at a rate of approximately 0.2°C/h. Core temperature also decreased at a rate of approximately 0.6°C before vasoconstriction, but remained virtually constant during the subsequent 2 h. Because mean body temperature and body heat content continued to decrease, constraint of metabolic heat to the core thermal compartment contributed to the core temperature plateau; that is, vasoconstriction reestablished the normal core-to-peripheral temperature gradient by preventing metabolic heat (which is largely generated in the core) from escaping to peripheral tissues. Constrained heat is presented cumulatively, referenced to vasoconstriction at elapsed time zero. Data are presented as the mean \pm SD. Reprinted with permission from Kurz *et al.*⁸⁰

mia without necessitating activation of thermoregulatory vasoconstriction. As a result, application of a limb tourniquet increases core temperature relative to patients not similarly treated.⁸² Core temperature increases yet further when two tourniquets are applied. For example, pediatric patients without tourniquets treated in a warm environment remained normothermic. In contrast core temperature increased $1.0 \pm 0.6^\circ\text{C}$ in 90 min in those with one tourniquet and $1.7 \pm 0.6^\circ\text{C}$ in those with bilateral tourniquets (fig. 7).⁷⁹

A natural consequence of the complete thermal isolation provided by tourniquets is that distal tissues become unusually cold. Not surprisingly then, eventual release of

the tourniquet is associated with redistribution of heat from the core to the previously isolated extremity and a substantial core temperature “afterdrop” (the rapid decrease in core temperature associated with an internal reduction of body heat).^{83,84} Thus, tourniquets help to prevent hypothermia (or even augment core temperature) while inflated; however, they also cause precipitous hypothermia at release.

Pediatric Patients

Infants and children tend to have relatively globular bodies with larger fractions of their mass in the torso than in adults. Therefore, redistribution apparently contributes somewhat less to the initial postinduction hypothermia in infants than in older children and adults.⁷⁸ Presumably, infants redistribute less because their extremities are small compared with their trunk and head and therefore cannot absorb much heat from the core.

The head constitutes a far larger fraction of the total surface area in infants than adults.⁸⁵ Cutaneous heat loss from the head in adults is no greater a fraction of the total than would be expected from its surface area. In infants, however, heat loss from the head may be a proportionately larger fraction of the total, perhaps because the skull and scalp are thin, allowing loss of heat delivered to the brain. Heat loss from the scalp can even be used to detect distressed fetuses.⁸⁶⁻⁸⁸

Cutaneous heat loss is roughly proportional to surface

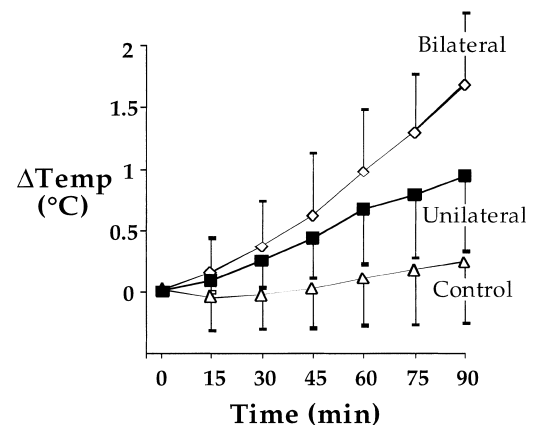


Fig. 7. The influence of one and two leg tourniquets on core temperature in pediatric patients. The change in nasopharyngeal temperature in patients given unilateral leg tourniquets, bilateral leg tourniquets, or no tourniquets (control) during 90 elapsed min. Time zero indicates induction of anesthesia. Patients in the control group remained nearly normothermic. In contrast, central temperatures increased $1.0 \pm 0.6^\circ\text{C}$ in 90 min in patients with unilateral tourniquets and $1.7 \pm 0.6^\circ\text{C}$ in those with bilateral tourniquets. Reprinted with permission from Bloch *et al.*⁷⁹

area, whereas metabolic heat production is largely a function of mass. Consequently, it is relatively easy for infants and children to lose large amounts of heat *via* the skin surface.^{34,89} Intraoperative heat loss in infants can therefore easily exceed metabolic heat production.⁹⁰ The linear hypothermia phase is therefore usually rapid in infants and children because of a high surface-area-to-weight ratio. In contrast, respiratory losses are similar to that in adults.

The threshold for intraoperative vasoconstriction is similar in adults and pediatric patients.^{78,91} Once constricted, an effective core temperature plateau develops in infants and children.

Neuraxial Anesthesia

It is not widely recognized that neuraxial anesthesia produces thermal perturbations that may be as large or larger than those observed during general anesthesia. The mechanisms are in some ways similar, but also differ in numerous important respects.

Redistribution

Just as with general anesthesia, redistribution of body heat is the major initial cause of hypothermia in patients administered spinal or epidural anesthesia. Neuraxial anesthesia inhibits thermoregulatory control centrally,⁹²⁻⁹⁴ but a far more important effect of major conduction anesthesia is blocking of peripheral sympathetic and motor nerves, which prevents thermoregulatory vasoconstriction and shivering.⁹⁵⁻⁹⁷

Redistribution during epidural anesthesia has been quantified in volunteers.²⁶ Core temperature decreased $0.8 \pm 0.3^\circ\text{C}$ in the first hour of anesthesia. Redistribution contributed 89% to this initial decrease and needed a net transfer of 20 kcal from the trunk to the extremities. During the subsequent 2 h of anesthesia, core temperature decreased an additional $0.4 \pm 0.3^\circ\text{C}$, with redistribution contributing 62%. Redistribution, therefore, contributed 80% to the entire $1.2 \pm 0.3^\circ\text{C}$ decrease in core temperature during the 3 h of anesthesia (fig. 8).

Redistribution during neuraxial anesthesia is typically restricted to the legs. Although the mass of the legs far exceeds that of the arms, they contribute comparably to redistribution. Consequently, redistribution decreases core temperature about half as much during major conduction anesthesia²⁶ as during general anesthesia.⁹ In both cases, however, redistribution remains the most important cause of core hypothermia during the first

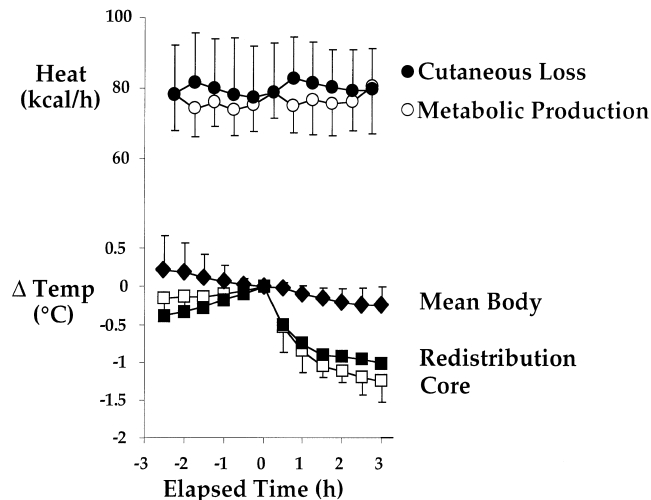


Fig. 8. Heat balance during epidural anesthesia. Overall heat balance was only slightly negative (loss exceeding production) before induction of anesthesia and subsequently changed little. To separate the contributions of decreased overall heat balance and internal redistribution of body heat to the decrease in core temperature, the change in overall heat balance was divided by body weight and the specific heat of humans. The resulting change in mean body temperature (mean body) was subtracted from the change in core temperature (core), leaving the core hypothermia specifically resulting from redistribution (redistribution). After 1 h of anesthesia, core temperature decreased $0.8 \pm 0.3^\circ\text{C}$, with redistribution contributing 89% to the decrease. During the subsequent 2 h of anesthesia, core temperature decreased an additional $0.4 \pm 0.3^\circ\text{C}$, with redistribution contributing 62%. Redistribution contributed 80% to the entire $1.2 \pm 0.3^\circ\text{C}$ decrease in core temperature during the 3 h of anesthesia. The increase in the redistribution curve before induction of anesthesia indicates that thermoregulatory vasoconstriction was constraining metabolic heat to the core thermal compartment. Such constraint is, of course, the only way in which core temperature could increase while body heat content decreased. Induction of epidural anesthesia is identified as elapsed time zero. Results are presented as the mean \pm SD. Reprinted with permission from Matsukawa *et al.*²⁶

hour of anesthesia. The magnitude of this redistribution depends largely on the patient's initial thermal state and can be ameliorated by cutaneous warming before induction of anesthesia.⁹⁸ As during general anesthesia, core temperature subsequently decreases linearly at a rate determined by the inequality between heat loss and production.

Lack of an Active Core Temperature Plateau

The linear hypothermia phase during general anesthesia is discontinued by reemergence of thermoregulatory vasoconstriction.⁸⁰ Neuraxial anesthesia differs in that peripheral nerve block, rather than central inhibition of thermoregulatory control, is the primary cause of hypothermia.²⁶ Sufficient core hypothermia will trigger vaso-

constriction and shivering in unblocked regions (*i.e.*, the arms) if thermoregulation is not excessively impaired by old age⁹⁹ or sedative medications.^{44,46} However, upper body shivering is relatively ineffective and often insufficient to prevent further hypothermia. Furthermore, shivering *per se* is often treated pharmacologically,^{45,47,100,101} which negates its thermogenic function.

The nerve block associated with neuraxial anesthesia continues throughout anesthesia. Patients therefore have little ability to vasoconstrict or shiver in the lower body during regional anesthesia, no matter what their central thermoregulatory status.¹⁰² Progressive hypothermia therefore is not limited by development of an active core temperature plateau the way it is during general anesthesia. Inability to vasoconstrict and shiver in the lower body is usually of little consequence if the surgical procedure is small because a passive plateau usually develops in well-insulated patients undergoing minor procedures. In contrast, patients undergoing large operations with neuraxial anesthesia have the potential for serious hypothermia to develop.¹⁰³

Hypothermia during neuraxial anesthesia is far more common than generally appreciated. Several studies indicate that hypothermia during neuraxial anesthesia for large operations is nearly as common and severe as during general anesthesia.^{104,105} An insidious aspect of this hypothermia is that neuraxial anesthesia not only inhibits autonomic thermoregulatory control, but also inhibits behavioral control.¹⁰⁶ Patients therefore do not necessarily feel cold and consequently do not complain to their anesthesiologist. Because temperature monitoring remains rare during major conduction anesthesia,¹⁰⁷ it is common that neither patients nor their anesthesiologists appreciate the extent of hypothermia.

Neuraxial and General Anesthesia

Most studies that evaluate hypothermia during regional and general anesthesia conclude that core temperature decreases more with general than with regional anesthesia.^{103,104} The mechanisms by which hypothermia develop provide some indication of what might be expected during various clinical circumstances. In this analysis, we will assume operations of comparable size and administration of similar amounts of unwarmed intravenous fluid.

Redistribution of body heat is the primary cause of hypothermia during the first hour of neuraxial²⁶ or general⁹ anesthesia. However, redistribution decreases core temperature about twice as much during general as during regional anesthesia. During relatively short pro-

cedures, hypothermia is likely to be greater in patients administered general anesthesia. Patients administered regional anesthesia will generally start the linear hypothermia phase at a relatively high temperature because they initially lost less to redistribution. Furthermore, hypothermia may develop at a reduced rate during neuraxial anesthesia because metabolic heat production remains near normal.

After long and large operations, the opposite pattern is likely to prevail because in patients administered general anesthesia a core temperature plateau will develop, whereas those undergoing neuraxial anesthesia often continue to become hypothermic. Whether core hypothermia is worse with general or regional anesthesia is thus likely to depend largely on the duration and magnitude of surgery.

Combined Neuraxial and General Anesthesia. The patients at greatest risk for inadvertent perioperative hypothermia are those in whom regional and general anesthesia are combined. The reason is that these patients will initially become rapidly hypothermic because of redistribution to all four extremities. During the linear phase, they will continue to cool, but at the relatively higher rate associated with general anesthesia.

Three additional factors contribute to hypothermia during combined regional-general anesthesia. The first is that neuraxial anesthesia *per se* reduces the vasoconstriction threshold. General anesthesia does also, but the epidural effect is superimposed on the general anesthetic effect. As a result, vasoconstriction occurs later and at a lower core temperature in patients administered both epidural and general anesthesia.⁷⁷ The second factor is that general anesthesia inhibits the shivering that might otherwise increase heat production during neuraxial anesthesia. The third and most important factor is that peripheral nerve block prevents vasoconstriction in the legs.^{96,108,109} As a result, vasoconstriction—once initiated centrally—is relatively ineffective, and core temperature continues to decrease (fig. 9).

Cardiopulmonary Bypass

Hypothermia is frequently used during cardiac surgery because it provides substantial protection against cardiac and cerebral ischemia.¹¹⁰⁻¹¹³ For the same reason, it is also used during certain neurosurgical procedures, especially repair of aneurysms. Procedures performed with cardiopulmonary bypass differ from other types of surgery in that heat exchange to the pump dwarfs all other

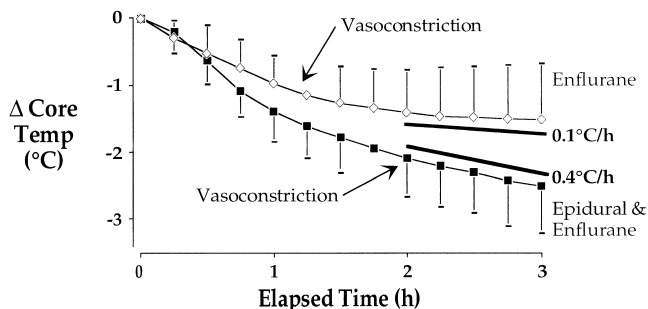


Fig. 9. Pronounced hypothermia during combined epidural-general anesthesia. Patients undergoing colorectal surgery were randomly assigned to combined epidural-enflurane anesthesia or enflurane alone. In appropriate patients, epidural anesthesia was maintained by an infusion of bupivacaine. Arteriovenous shunt vasoconstriction was observed nearly 1 h later in the patients administered combined epidural-general anesthesia, which is consistent with the established centrally mediated thermoregulatory impairment produced by neuraxial anesthesia. There was a distinct core temperature plateau after 2 h in the patients administered general anesthesia alone; in contrast, core temperature continued to decrease at a rate of 0.4°C/h in the patients who were also administered epidural anesthesia. Reprinted with permission from Joris *et al.*⁷⁷

sources of heat loss and gain. Furthermore, the thermal perturbations are of much greater magnitude and induced considerably faster than during routine surgery. Consequently, cardiac surgery usually involves major alterations in tissue heat content and in the regional distribution of heat.

Tissue Heat Distribution

Even during routine surgery, there are substantial alterations in tissue heat distribution and these differences are often the major cause of observed core temperature variation. During cardiopulmonary bypass, however, the rate at which heat is added to or removed from the core is extraordinary. For example, it is not unusual for net heat exchange with the bypass pump to exceed 400 kcal, even during bypass at a moderate temperature, with the bulk of this heat being transferred within 30 min.¹¹⁴ This rate of net heat transfer corresponds to the entire hourly basal metabolic rate every 10 min.

Distribution within the Core Thermal Compartment. The extraordinary thermal stress imposed by cardiopulmonary bypass cannot be fully distributed even within the core thermal compartment during the rapid cooling and rewarming phases of bypass. Consequently, there are often transient temperature differences among various core monitoring sites. These differences are not measurement artifacts; instead, they reflect true variation in tissue temperature within the trunk and head. Fortu-

nately, though, these differences are short-lived and typically resolve within minutes of completing cooling or rewarming.^{115,116}

During cardiopulmonary bypass, a two-compartment model of body heat distribution may fail. Consequently, it is sometimes helpful to define a third "intermediate" compartment. This term is used to designate tissues such as the rectum and bladder. These tissues are normally considered to be part of the core, but are not perfused well enough to remain homogeneous during the enormous and rapid heat fluxes associated with cardiopulmonary bypass. Consequently, it is common for temperatures in such sites to lag several degrees behind core sites.^{114,117} Temperatures in these intermediate sites nonetheless remain much closer to core temperature than do more peripheral tissues.

Core-to-peripheral Tissue Temperature Gradient.

The major thermal difficulty with cardiopulmonary bypass is that heat is transferred to the core compartment much faster than it can be dissipated to peripheral tissues. As aforementioned, flow of heat to peripheral tissues is limited by blood-borne convection of heat and conduction of heat into adjacent tissues. The division between longitudinal convection and radial conduction is, of course, somewhat artificial. There is some radial blood flow and, therefore, some radial convection. Similarly, there is a degree of longitudinal conduction, although this mechanism is unlikely to contribute much. It is therefore useful to consider the two mechanisms independently. Finally, local tissue metabolism produces some heat locally that depends on neither convection nor conduction.

Neither convection nor conduction is nearly sufficient, even in vasodilated subjects, to compensate for the rapid changes in core heat content during cardiopulmonary bypass. Figure 10 illustrates the changes in core and peripheral compartment heat content during cardiopulmonary bypass at 31 or 27°C.²⁵ Cooling decreased core heat content far more than peripheral tissue heat content in both cases. Core heat content subsequently increased rapidly, and in each case, exceeded prebypass values by the end of the rewarming phase of bypass. In patients cooled to 27°C, for example, total body heat content decreased 419 ± 49 kcal during cooling. During pump rewarming, core heat content increased to 66 ± 23 kcal more than precooling values, whereas peripheral heat content remained 70 ± 42 kcal less than precooling values. Body heat content at the end of rewarming was thus 4 ± 52 kcal less than at the onset of cooling.

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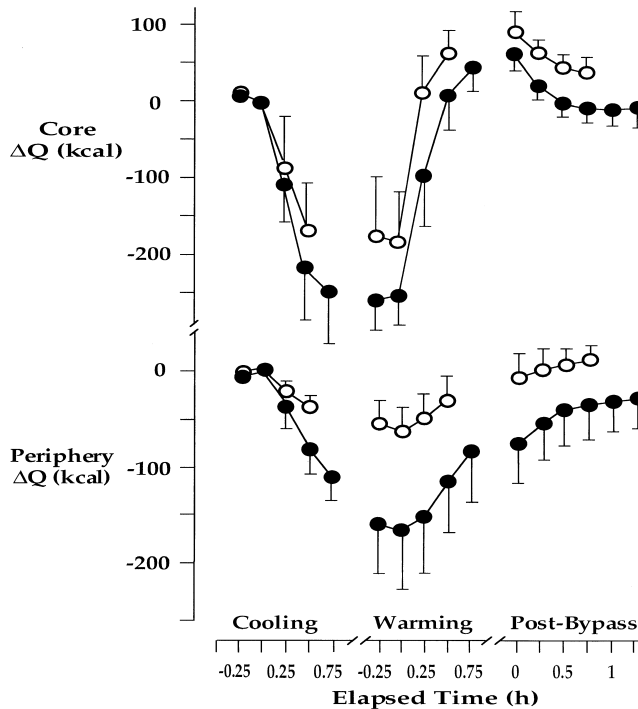


Fig. 10. Changes in core and peripheral tissue heat content during and after cardiopulmonary bypass at 31 and 27°C. The beginning of bypass cooling was designated as elapsed time zero for cooling. Because the duration of cooling differed among patients—and especially between the groups—a second zero elapsed time was defined as the beginning of rewarming. Similarly, the end of rewarming identified elapsed time zero for the postbypass period. Fewer data points are shown for the 31°C group (open circles) than for the 27°C group (filled circles) because both the cooling and the rewarming periods were shorter. In the 31°C group, total body heat content decreased 231 ± 93 kcal. During pump rewarming, peripheral heat content increased to 7 ± 27 kcal less than precooling values, whereas core heat content increased to 94 ± 33 kcal more than precooling values. Body heat content at the end of rewarming was 87 ± 42 kcal more than at the onset of cooling. In the 27°C group, total body heat content decreased 419 ± 49 kcal. During pump rewarming, core heat content increased to 66 ± 23 kcal more than precooling values, whereas peripheral heat content remained 70 ± 42 kcal less than precooling values. Body heat content at the end of rewarming was 4 ± 52 kcal less than at the onset of cooling. Results are presented as the mean \pm SDs. Reprinted with permission from Rajek *et al.*²⁵

However, the distribution of heat was quite abnormal, with more than usual being in the core compartment.

The result of this abnormal distribution of heat was a substantial core-to-peripheral tissue temperature gradient, especially during the rapid cooling and rewarming phases of bypass. Average peripheral tissue temperature in the 27°C patients decreased to a minimum of $29.8 \pm 1.7^\circ\text{C}$, and subsequently increased to $32.8 \pm 2.1^\circ\text{C}$ at the end of bypass. The core-to-peripheral tissue temperature

gradient was thus $4.6 \pm 1.9^\circ\text{C}$ at the end of rewarming, which was about twice the initial difference.

As might be imagined, core-to-peripheral temperature gradients are even more extreme during cardiopulmonary bypass at 17°C (fig. 11). In this study,¹² core temperature decreased to a minimum of $16.8 \pm 1.1^\circ\text{C}$ during bypass, and then increased to $36.5 \pm 0.3^\circ\text{C}$. The average peripheral tissue temperature at the onset of bypass was $33.7 \pm 0.7^\circ\text{C}$; tissue temperature decreased to a minimum of $22.6 \pm 2.1^\circ\text{C}$ during bypass and subsequently increased to $31.8 \pm 1.5^\circ\text{C}$ during 104 ± 18 min of rewarming. The core-to-peripheral tissue temperature gradient was $1.6 \pm 0.8^\circ\text{C}$ at the onset of bypass, $-5.9 \pm 0.9^\circ\text{C}$ at the end of cooling, and $4.7 \pm 1.5^\circ\text{C}$ at the end of rewarming. The core-to-peripheral tissue temperature gradient was thus increased almost by a factor of three

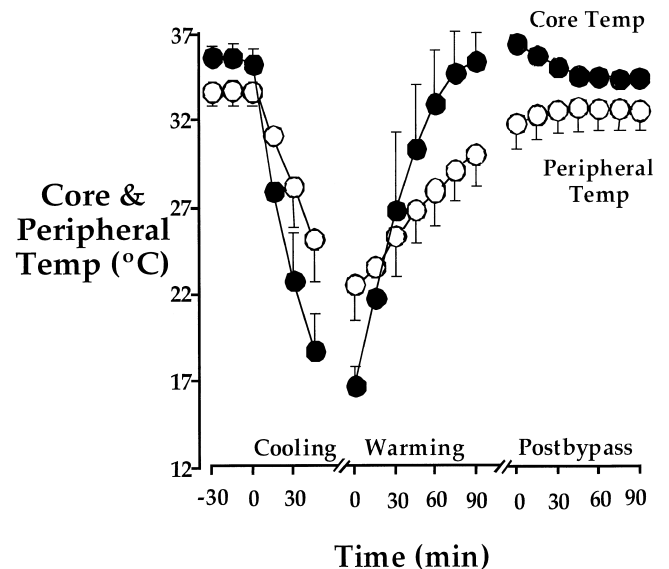


Fig. 11. Core and peripheral temperatures during and after deep hypothermic cardiopulmonary bypass. The beginning of bypass cooling was designated as elapsed time zero for cooling. Because the duration of cooling differed among patients, a second zero elapsed time was defined as the beginning of rewarming. Similarly, the end of rewarming identified elapsed time zero for the postbypass period. Core temperature (solid circles) decreased to a minimum of $16.8 \pm 1.1^\circ\text{C}$ during bypass, and then increased to $36.5 \pm 0.3^\circ\text{C}$. The average peripheral tissue temperature at the onset of bypass was $33.7 \pm 0.7^\circ\text{C}$; tissue temperature decreased to a minimum of $22.6 \pm 2.1^\circ\text{C}$ during bypass, and subsequently increased to $31.8 \pm 1.5^\circ\text{C}$ during 104 ± 18 min of rewarming. The core-to-peripheral tissue temperature gradient was $1.6 \pm 0.8^\circ\text{C}$ at onset of bypass, $-5.9 \pm 0.9^\circ\text{C}$ at the end of cooling, and $4.7 \pm 1.5^\circ\text{C}$ at the end of rewarming. The core-to-peripheral tissue temperature gradient was increased almost by a factor of three during bypass, despite an essentially normal postbypass core temperature. Results are presented as the mean \pm SD. Reprinted with permission from Rajek *et al.*¹²

during bypass, despite an essentially normal postbypass core temperature.

Radial and Longitudinal Tissue Temperature Distributions. Abnormal tissue temperature distribution during cardiopulmonary bypass is not restricted to the core-to-peripheral axis. There are also substantial radial and longitudinal inequalities within the peripheral compartment. This is a natural consequence of the two major pathways by which heat flows peripherally from the core: longitudinal convection and radial conduction. Blood-borne convection of heat is faster than radial diffusion; however, the distances involved are proportionately much greater. That is, the length of the extremities far exceeds their width. Consequently, convection and conduction both remain clinically important impediments to peripheral heat transfer.

The center of the thigh can be considered part of the core because temperature in this region normally equals core temperature, even during rapid thermal perturbations.¹² Calf and foot tissues, however, are normally considerably cooler than the core; furthermore, their temperatures change slowly in response to core perturbations. The disparity between core and regional temperature increases progressively in more distal aspects of the extremities.

During one study of bypass at 17°C, for example, temperature in the central axis of the leg at the end of cooling was $6.0 \pm 4.3^\circ\text{C}$ greater than core temperature at the upper calf, $7.1 \pm 4.2^\circ\text{C}$ greater at the lower calf, and $6.3 \pm 4.1^\circ\text{C}$ greater in the foot. At the end of rewarming, the situation was reversed, with the upper calf being 2.9°C less than the core temperatures. At that time, temperature at the center of the lower calf exceeded core temperature by $4.9 \pm 2.0^\circ\text{C}$, whereas the foot exceeded core temperature by $6.7 \pm 3.4^\circ\text{C}$. These data indicate that longitudinal convection of heat along extremities is insufficient to equilibrate peripheral and core tissue temperatures during the rapid thermal perturbations associated with hypothermic bypass.

The second factor limiting transfer of heat from the core to peripheral tissues is the slow rate at which heat is conducted radially. This is manifested as a radial tissue temperature gradient. Radial inequalities are far more important in the proximal than in the distal extremities because the tissue temperature gradients between the center axis and skin are greater.

Figure 12 shows that at the end of cooling during bypass to 17°C, temperatures at the center of the upper and lower thigh were $8.0 \pm 5.2^\circ\text{C}$ and $7.3 \pm 4.2^\circ\text{C}$, respectively, cooler than skin temperature. There was

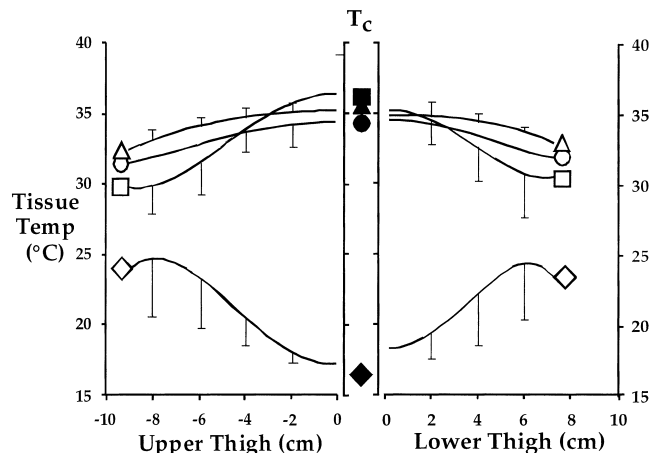


Fig. 12. Radial tissue temperature gradients in the thigh during and after cardiopulmonary bypass at 17°C. Average radial upper and lower thigh temperatures at the onset of cooling (open triangles), at the start of rewarming (open diamonds), at the end of rewarming (open rectangles), and 1 h after the end of rewarming (open circles). Temperatures were estimated using fourth-order regression coefficients calculated from individual thigh skin and muscle temperatures. Distance zero indicates the center of each extremity segment. Solid symbols indicate the corresponding core temperatures at the each time. Results are presented as the mean \pm SD. Reprinted with permission from Rajek *et al.*¹²

therefore a substantial (and nonlinear) radial temperature gradient through the thigh. At completion of rewarming, there was again a substantial temperature gradient, but now with tissue at the center of the upper and lower thigh being $7.0 \pm 2.2^\circ\text{C}$ and $6.4 \pm 2.3^\circ\text{C}$ warmer than skin temperature. In contrast, there was little radial temperature gradient in the calf.

Afterdrop

The term *afterdrop* refers to the rapid decrease in core temperature after discontinuation of cardiopulmonary bypass.^{118,119} It results from insufficient rewarming of peripheral tissues that leaves a large core-to-peripheral temperature gradient at the time bypass is discontinued. Hypothermia then results from redistribution of heat from the core to peripheral tissues. It is thus analogous to the redistribution hypothermia that accompanies induction of general anesthesia.

Prevention. In a general sense, afterdrop results because bypass machines can warm the core much more quickly than heat can be distributed to peripheral tissues. This results in a large core-to-peripheral temperature gradient and a subsequent redistribution of substantial amounts of heat. Not surprisingly, reestablishing normal peripheral tissue temperature is easier when

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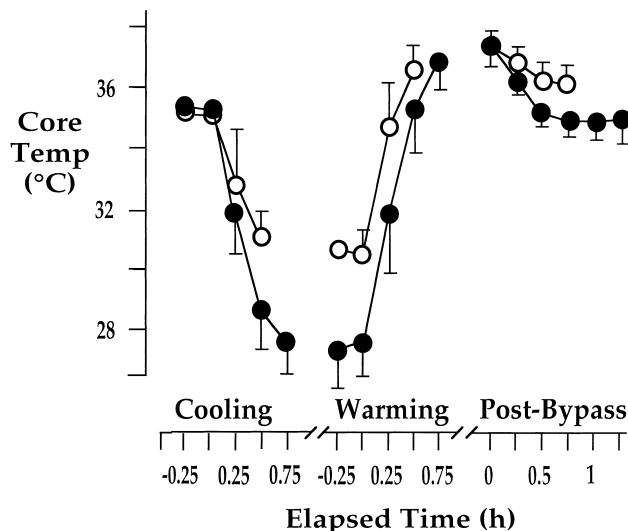


Fig. 13. Change in core temperature during cooling and re-warming during bypass. Designated times are the same as in figure 10. Data are presented as the mean \pm SD. Patients cooled to a target temperature of 31°C are shown with open circles, whereas those cooled to 27°C are shown with filled circles. Reprinted with permission from Rajek *et al.*²⁵

bypass has been conducted at a moderate temperature. Afterdrop is therefore less problematic when bypass is conducted at 32–34°C than at temperatures near 28°C. For example, a recent study found that afterdrop was 2.3°C when bypass temperature was 27°C, but was only 1.5°C at a bypass temperature of 31°C (fig. 13).²⁵

Afterdrop is largely a kinetic problem because sufficiently prolonged bypass heating would eventually warm peripheral tissues. One strategy for minimizing afterdrop is to somewhat prolong re-warming in an effort to reduce the extreme core-to-peripheral temperature gradients that result from very rapid re-warming. The difficulty with this approach is that the thermal benefits of prolonged bypass must be balanced against the well-known complications of bypass, including damage to hematopoietic cells and resulting inflammation and activation of the coagulation system.^{120–123}

An additional strategy is to administer vasodilating drugs in an effort to facilitate core-to-peripheral transfer of heat. Infusion of nitroprusside during the re-warming phase of bypass was reported in 1980 to reduce afterdrop from $2.6 \pm 0.2^\circ\text{C}$ to $1.5 \pm 0.4^\circ\text{C}$.¹²⁴ Bypass temperature was not specified in that manuscript, but was presumably near 28°C. Pump flows were also not specified, making it difficult to assess the extent to which nitroprusside produced vasodilation. The major difficulty with this study, however, was lack of randomized

assignment to nitroprusside treatment. Nonetheless, a recent study of bypass conducted at 28°C confirmed that vasodilation induced by either nitroprusside or isoflurane reduced afterdrop magnitude by a third.¹²⁵

In a more recent study, patients undergoing bypass at 32°C were randomly assigned to a control group or treatment with nitroprusside sufficient to increase pump flow from 2.5 to 3.0 $\text{l} \cdot \text{min}^{-1} \cdot \text{m}^{-2}$. Nitroprusside administration, in this case, had little influence on afterdrop magnitude. These data suggest that nitroprusside administration is not an effective method of reducing afterdrop, at least in patients cooled only to approximately 32°C. This result is consistent with the relatively small afterdrop magnitude observed in these patients.²⁵

Afterdrop during cardiac surgery is the thermal analog to the redistribution hypothermia that accompanies induction of anesthesia. Another amelioration strategy is to use a variant of prewarming discussed previously; namely, active warming of peripheral tissues. The amount of heat provided by clinically available cutaneous warmers is tiny compared to that transferred by bypass pumps. Warming might nonetheless be beneficial because the heat can be directed to the relatively sequestered peripheral tissues. Consistent with this theory, forced-air warming applied to the legs, starting with re-warming and continued through recovery, halved afterdrop from 1.2 to 0.5°C.¹²⁶ The difficulty, however, was that detailed analysis of regional heat balance and tissue temperature gradients did not support the proposed mechanism. Instead, it was apparent that core temperature was preserved because forced-air warming improved systemic heat balance rather than reducing core-to-peripheral redistribution of body heat.

Respiratory heat loss is trivial.²⁸ Consequently, airway heating and humidification has little influence on core temperature even during routine surgery.^{65,127} The thermal fluxes are an order of magnitude larger during cardiac surgery, making the relative respiratory contribution proportionately smaller yet. As might be expected, then, airway heating and humidification has no clinically relevant influence of core temperature during bypass.¹¹⁸ Heating with circulating-water mattresses is also unlikely to be effective because they transfer relatively little heat.⁶⁵

Return to Normothermia

Surgical patients become hypothermic largely because general^{43,44,48} and regional^{77,93,128} anesthesia impairs

thermoregulatory defenses, as do most sedatives.^{45-47,100} Consequently, most surgical patients become hypothermic unless they are actively warmed.^{65,66,129} The amount of heat lost during surgery is substantial. For example, based on the reported specific heat of humans,¹ a 3°C reduction in mean body temperature corresponds to a debt of approximately 175 kcal in a 70-kg patient. This is roughly the basal heat production for 3 h.⁵⁸

Full postoperative recovery of protective thermoregulatory responses may be limited by residual volatile anesthetic¹³⁰ or opioids administered to treat surgical pain.^{45,46} Nonetheless, brain anesthetic concentrations usually decrease rapidly during the initial postoperative period,¹³¹ allowing reemergence of thermoregulatory responses, including vasoconstriction and shivering.^{130,132-135} These responses combine to decrease cutaneous heat loss,³³ constrain metabolic heat to the thermal core,¹³⁶ and increase metabolic heat production.¹³² As a result, core temperatures usually increase toward normal values after anesthesia is discontinued.

Arteriovenous shunt vasoconstriction is universal during postanesthetic recovery in hypothermic patients.¹³³ Similarly, shivering is common. Nonetheless, postoperative core temperatures in hypothermic patients usually increase relatively slowly, often requiring 2-5 h to return to normal values.^{133,137,138} Such slow return to normothermia contrasts markedly with the rapid increases observed in unanesthetized volunteers,¹³⁹ suggesting that prolonged hypothermia does not result simply from the magnitude of the heat debt, but because thermoregulatory compensations are not maximally activated in surgical patients—presumably because of residual volatile anesthetics and opioids administered for treatment of pain.

Summary

Hypothermia during general anesthesia develops with a characteristic three-phase pattern. The initial rapid reduction in core temperature after induction of anesthesia results from an internal redistribution of body heat. Redistribution results because anesthetics inhibit the tonic vasoconstriction that normally maintains a large core-to-peripheral temperature gradient. Core temperature then decreases linearly at a rate determined by the difference between heat loss and production. However, when surgical patients become sufficiently hypothermic, they again trigger thermoregulatory vasoconstriction, which restricts core-to-peripheral flow of heat.

Constraint of metabolic heat, in turn, maintains a core temperature plateau (despite continued systemic heat loss) and eventually reestablishes the normal core-to-peripheral temperature gradient. Together, these mechanisms indicate that alterations in the distribution of body heat contribute more to changes in core temperature than to systemic heat imbalance in most patients.

Just as with general anesthesia, redistribution of body heat is the major initial cause of hypothermia in patients administered spinal or epidural anesthesia. However, redistribution during neuraxial anesthesia is typically restricted to the legs. Consequently, redistribution decreases core temperature about half as much during major conduction anesthesia. As during general anesthesia, core temperature subsequently decreases linearly at a rate determined by the inequality between heat loss and production. The major difference, however, is that the linear hypothermia phase is not discontinued by reemergence of thermoregulatory vasoconstriction because constriction in the legs is blocked peripherally. As a result, in patients undergoing large operations with neuraxial anesthesia, there is the potential of development of serious hypothermia.

Hypothermic cardiopulmonary bypass is associated with enormous changes in body heat content. Furthermore, rapid cooling and rewarming produces large core-to-peripheral, longitudinal, and radial tissue temperature gradients. Inadequate rewarming of peripheral tissues typically produces a considerable core-to-peripheral gradient at the end of bypass. Subsequently, redistribution of heat from the core to the cooler arms and legs produces an afterdrop. Afterdrop magnitude can be reduced by prolonging rewarming, pharmacologic vasodilation, or peripheral warming.

Postoperative return to normothermia occurs when brain anesthetic concentration decreases sufficiently to again trigger normal thermoregulatory defenses. However, residual anesthesia and opioids given for treatment of postoperative pain decreases the effectiveness of these responses. Consequently, return to normothermia often needs 2-5 h, depending on the degree of hypothermia and the age of the patient.

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