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Experimental Determination of Heat Flow Parameters during Induction of General Anesthesia

Daniel I. Sessler, M.D.,* Andrew M. Sessler, Ph.D.†

Background: **Alterations in body temperature result from changes in tissue heat content. Heat flow is a complex function of vasomotor status and core, peripheral, and ambient temperatures. Consequently it is difficult to quantify specific mechanisms responsible for observed changes in body heat distribution. Therefore the authors developed two mathematical models that independently express regional tissue heat production and the motion of heat through tissues in terms of measurable quantities.**

Methods: **The equilibrium model expresses the effective regional heat transfer coefficient in terms of cutaneous heat flux, skin temperature, and temperature at the center of the extremity. It applies at steady states and provides a ratio of the heat transfer coefficients before and after an intervention. In contrast, the heat flow model provides a time-dependent estimate of the heat transfer coefficient in terms of ambient temperature, skin temperature, and temperature at the center of the extremity.**

Results: **Each model was applied to data acquired in a previous evaluation of heat balance during anesthesia induction. The relation between the ratio of steady state regional heat transfer coefficients calculated using each model was linear. The effective heat transfer coefficient for the forehead (a core site) decreased approximately 20% after induction of anesthesia. In contrast, heat transfer coefficients in the six tested extremity sites more than doubled.**

Conclusions: **Effective heat transfer coefficients can be used to evaluate the thermal effects of various clinical interventions, such as induction of regional anesthesia or administration of vasodilating drugs. The heat transfer coefficient for the forehead presumably decreased because general anesthesia reduces brain perfusion. In contrast, increased heat transfer coefficients in the extremity sites indicate that thermoregulatory and anesthetic-induced vasodilation more than doubles the core-to-peripheral flow of heat. This flow of heat causes redistribution hypothermia, which is usually the major cause of core hypothermia during anesthesia. (Key words: Mathematical modeling; temperature; thermoregulation.)**

* Professor, Department of Anesthesia, University of California, San Francisco; Professor, Ludwig Boltzmann Institute for Clinical Anesthesia and Intensive Care; Director, Outcomes Research™; and Vice-Chair, Department of Anesthesia and General Intensive Care, University of Vienna.

† Distinguished Senior Scientist, Lawrence Berkeley National Laboratory.

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Address correspondence to Dr. Sessler: Department of Anesthesia, 374 Parnassus Avenue, Third floor, University of California, San Francisco, California 94143-0648. Address electronic mail to: sessler@vaxine.ucsf.edu; on the World Wide Web: www.outcomes.ucsf.edu

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THE three most important autonomic thermoregulatory responses in humans are sweating, vasomotion, and shivering.[1] Active vasoconstriction is largely restricted to the arteriovenous shunts in the fingers and toes.[2,3] Active vasodilation, in contrast, occurs in precapillary arterioles throughout the skin surface.[4,5] However, only arteriovenous shunt vasoconstriction is routinely active at rest in moderate environments.

Vasomotor alterations are induced by many centrally and peripherally acting medications. General anesthetics, for example, produce major alterations in vasomotor tone both by inhibiting tonic thermoregulatory arteriovenous shunt vasoconstriction[6,7] and by inducing direct peripheral vasodilation.[8] As might be expected from these dual actions, anesthetic-induced vasodilation markedly alters regional tissue temperatures and body heat distribution by inducing a core-to-peripheral redistribution of body heat. The result is that redistribution is usually the most important cause of perioperative hypothermia, accounting for 81% of the 1.6°C-hypothermia observed in the first hour after induction of general anesthesia. Even after 3 h, redistribution accounts for 65% of the observed 2.8°C-core hypothermia.[9] Redistri-

bution is also an important cause of hypothermia during neuraxial anesthesia.[10]

Alterations in body temperature result from changes in tissue heat content. Therefore it is of considerable interest to evaluate the flow of heat within the body and the major factors that influence regional heat content. The two major factors are (1) radial conduction of heat and (2) longitudinal convection of heat and local metabolic heat production. In previous investigations, we evaluated regional tissue heat content and temperature by fitting skin and muscle temperatures to parabolic regressions and integrating over volume.[9,10] The flow of heat, however, is a complex function of core temperature, peripheral tissue temperature, vasomotor status, tissue characteristics, and ambient temperature. Consequently we could not quantify the specific mechanism responsible for the observed changes in body heat distribution. Further, it would be difficult to predict from tissue heat content alone how induction of anesthesia (or another vasomotor perturbation) would alter the flow of heat through the body during different circumstances. Quantifying tissue heat flow, however, is critical because alteration in heat flow is the primary factor that influences perioperative core temperature. These phenomena are characterized by an effective heat transfer coefficient and an effective heating rate.

Heat balance and distribution measurements also fail to identify the independent effects of thermoregulatory vasomotion on tissue heat flow in other circumstances. For example, nitroprusside administration facilitates transfer of heat delivered from a cardiopulmonary bypass pump to peripheral tissues. This decreases the core-to-peripheral temperature gradient, and, therefore, the afterdrop.[11] The dose-dependent effects of other vasodilators, nitroglycerin for example, could be estimated using heat transfer coefficients, which would be far easier than performing full heat-balance studies at various doses. After an appropriate dose was established, its exact heat-transfer characteristics could then be determined using formal measurements, as previously described.[9]

Another example is prewarming, which restricts anesthetic-induced redistribution hypothermia[12,13] by reducing the core-to-peripheral tissue temperature gradient.[14] A superficial conclusion might be that anesthetic-induced vasodilation in a prewarmed patient minimally alters the heat diffusion coefficient. This is quite unlikely because studies of peripheral-to-core heat transfer suggest that both thermoregulatory vasomotion and the peripheral effects of anesthetics on blood vessels each contribute to heat diffusion.[15] In circumstances such as these in which heat flow is minimal (because the Second Law of Thermodynamics necessitates that flow be driven by a temperature gradient), the effects of interventions on the diffusion coefficient could still be determined using appropriate mathematical models.

Mathematical models are also likely to help investigators evaluate the effectiveness of thermoregulatory vasomotion during various clinical circumstances. The threshold (triggering core temperature) of vasoconstriction, for example, is reduced approximately 1°C in elderly persons.[16] A reduced threshold surely contributes to the excessive hypothermia in elderly persons.[17] However, the effects of aging on the gain of vasoconstriction (incremental response intensity with further core temperature perturbation[18]) and maximum response intensity remain unknown. Furthermore, the extent to which reduced arteriovenous shunt gain alters tissue heat flow remains unknown. Appropriate models of tissue heat flow would help to determine the extent to which reduced vasoconstriction efficacy (lower gain and maximum intensity) contributes to hypothermia in elderly persons.

Peripheral anesthetic-induced[8] and central thermoregulatory[6,7] factors significantly alter the effective gain and maximum intensity of vasoconstriction and the flow of heat through human tissues. Consequently, both factors markedly alter perioperative core temperature and the efficacy of cutaneous warming.[15,19–21] However, it has proved difficult to evaluate independently the contributions of each. This is, thus, another circumstance in which mathematical models might provide relevant information that has otherwise proved difficult to obtain in clinical protocols.

Heat transfer through living tissues is fully characterized by the bioheat equation.[22,23] However, this equation is impractical in typical experimental and clinical circumstances because it is complicated and necessiates measurement of many difficult-to-obtain values. We developed two mathematical models that independently express regional tissue heat production and the motion of heat through tissues in terms of measurable quantities. We validated these models by applying them to data acquired in our previous evaluation of heat balance during induction of anesthesia and by comparing the results.[9] Using these models, investigators will be able to evaluate tissue heat transfer during the circumstances already described and during other clinically important thermal perturbations.

Analysis

In any object in which there is heat generation and transfer by conduction, the flow of heat is governed by the bioheat equation.[22,23] The bioheat equation, in various forms, fully describes the generation and flow of heat within tissues. However, this equation is impossible to solve in terms of measurable quantities. Therefore, we wanted to develop a model that can be used to reduce the bioheat equation to two functions whose values can be confirmed during at least certain clinical conditions. (Details of this analysis are given in the appendix.)

To that end, we take the bioheat equation (shown in appendix equation A1) and omit factors that are unlikely to contribute substantially in typically perioperative circumstances. Specifically, we neglect the radial convection of heat, that is, we assume that radial flow of heat is conductive. We characterize this flow by an "effective heat transfer coefficient," which we call D. The term D includes true conduction and microconvection by blood, without distinguishing their relative contributions. Longitudinal (convective) blood flow is important because it locally deposits (or absorbs) heat. However, heat also is locally generated by metabolism. Therefore, we define an "effective heating rate," which we call H. This term includes heat deposition by longitudinal blood-borne convection and local metabolic heat generation, again without distinguishing their relative contributions.

In our model, heat then only flows radially, and in cylindrical coordinates is described by the following:

$$\frac{\partial T}{\partial t} = \frac{D}{r}\frac{\partial}{\partial r}\left(r\frac{\partial T}{\partial r}\right) + H \qquad (1)$$

where T is temperature in °C, t is time in seconds, D is the effective heat transfer coefficient (cm^2/s), and H is the heat production and deposition (°C/s). These quantities can be expressed in the following way:

$$D = \frac{k}{s\rho}, \qquad (2)$$

$$H = \frac{P}{s\rho}, \qquad (3)$$

where the heat conduction coefficient k is measured in units of $cal \cdot cm^{-1} \cdot s^{-1} \cdot °C^{-1}$ and the heat production rate P is measured in units of $cal \cdot cm^{-3} \cdot s^{-1}$. The density of material is ρ (g/cm^3) and the specific heat is s ($cal \cdot g^{-1} \cdot °C^{-1}$).

We have taken D and H to be a function of t but not of position. The experimental observation of a parabolic variation of temperature in steady state is consistent with these assumptions because a parabolic temperature distribution is the solution of equation 1 in the steady state when $\partial T/\partial r = 0$. An example of this radial distribution is shown in figure 5 of Belani *et al.*[24]

Next we developed two different models.

The Equilibrium Model

In equilibrium, long before and long after changes in vasomotor status or heat generation rate, we have $\partial T/\partial t = 0$. We designate the equilibrium period before induction of anesthesia with a superscript "−". We similarly designate the equilibrium period after induction of anesthesia with a superscript "+". Measurements have shown that, in equilibrium, T is a parabolic function of radius (r).[9,24]

$$T(r) = T_c^{\pm} - \frac{r^2}{r_s^2}(T_c^{\pm} - T_s^{\pm}), \qquad (4)$$

where T_c is temperature at the center of the cylinder, T_s is temperature at the surface, and r_s is the radius at the surface. With a parabolic temperature distribution and no further assumptions, we can derive from equation 1 formulas for the ratio of parameters after those before vasomotor tone is altered. The results are

$$\frac{H^+}{H^-} = \frac{Q^+}{Q^-}. \qquad (5)$$

$$\frac{D^+}{D^-} = \frac{Q^+}{Q^-}\left(\frac{T_c^- - T_s^-}{T_c^+ - T_s^+}\right). \qquad (6)$$

where $Q^{\pm}$ is the heat flow from the surface. The ratio of the equilibrium effective heating rates before and after induction of anesthesia is simply H^+/H^-; D^+/D^- is similarly the ratio of the equilibrium effective heat transfer coefficients before and after induction of anesthesia.

Heat Flow Model

Previously we showed that heat flow from the skin surface into air per unit of area, Q, is proportional to $T_s - T_a$, where T_s is the skin temperature and T_a is the ambient temperature in °C.[25] That is,

$$Q = \frac{k_a}{L_a}(T_s - T_a), \qquad (7)$$

where k_a is the effective heat flow parameter in the air and L_a is a characteristic distance over which the tem-

perature decreases from T_s to T_a. Convection, radiation, and conduction are all included in equation 7. The flow of heat out of a limb is given by

$$Q = \frac{2k}{r_s}(T_c - T_s). \quad (8)$$

Most of these quantities are time dependent, although that dependence has not been explicitly indicated. We define a new quantity $\lambda(t)$ whose time dependence we will study in detail

$$\lambda(t) = \frac{2L_a}{r_s}\frac{k}{k_a}. \quad (9)$$

We show in the appendix that

$$\lambda(t) = \frac{T_s - T_a}{T_c - T_s}. \quad (10)$$

Forming the ratio of lambda for times long before a change in vasomotor tone, λ^-, and long after, λ^+, we see that the ratio is given by

$$\frac{\lambda^+}{\lambda^-} = \left[\frac{T_s^+ - T_a^+}{T_c^+ - T_s^+}\right]\left[\frac{T_s^- - T_a^-}{T_c^- - T_s^-}\right]^{-1} \quad (11)$$

It is clear that the lambda ratio is exactly the same as the D ratio (equation 6) because both depend only on the ratio k^+/k^-. Thus, we have two different formulas for the changes in effective heat transfer coefficient, and, most importantly, both formulas only involve quantities that can be measured.

Comparison and Assumptions

In the analytic work we assumed that D and H are independent of radius (r) and, therefore, only a function of time (t). The mathematical models depend on the assumption of a parabolic distribution of temperature. Although previous measurements have shown this to be the case,[25] the measurements are gross and the models demand this behavior in detail. In the equilibrium model (equations 5 and 6), the parabolic assumption is used less critically than in the heat flow model (equation 11), in which the heat flux out of the body (essential to the argument) depends critically on the temperature distribution just under the skin.

The heat flow model also assumes that the air takes up heat proportional to $(T_s - T_a)$, and this is only approximately true. For example, immediately after vasodilation, the heat transfer coefficient presumably has changed already, but there is no change in the input to the formula for $\lambda(t)$. Presumably, vasodilation initiates a wave of tissue temperature change that invalidates the parabolic assumption and a burst of heat into the air (because the heat conduction coefficient increases), which warms air near the skin and invalidates the $(T_s - T_a)$ assumption. However, if we make the heat flow approximation for air (see appendix equation A12 for the formula) and insert this in equation 6, then we immediately obtain equation A11 (*i.e.*, under the heat flow approximation, the equilibrium model is the same as the heat flow model). The heat flow model, providing a function of time $[\lambda(t)]$, is more general than the equilibrium model, which only produces one number D^+/D^-. We will show experimentally that the two models agree well, but we do not show for what range of time $\lambda(t)$ is significant. The argument given here suggests that it is not correct at the onset of vasodilation, but it may be valid not far from that time.

Clinical Protocol

The physiologic data we present were obtained from our previous evaluation of regional heat distribution during induction of general anesthesia.[9] Briefly, six minimally clothed male volunteers in an ≈22°C environment were evaluated for 1 h before induction of fentanyl–propofol anesthesia and for 3 h subsequently.

Core temperature was measured at the tympanic membrane. At four sites, right leg muscle temperatures were recorded using disposable, 8-mm, 18-mm, and 38-mm, 21-gauge needle thermocouples inserted perpendicular to the skin surface. Skin-surface temperatures were recorded immediately adjacent to each set of needles. Cutaneous thermal flux[26,27] was measured from single transducers on the thigh and calf, each positioned midway between the needles.

Tissue and skin-surface temperature and thermal flux were similarly measured from the upper and lower arms. Skin-surface temperature and heat flux also were measured on the forehead. Because the head was covered with a plastic canopy (to measure oxygen consumption) during the preinduction period, initial values at this site were obtained just before induction of anesthesia.

Tissue temperatures, as a function of radial distance from the center of the extremity, were calculated using skin-surface temperatures and the muscle temperatures (8 mm, 18 mm, and 38 mm below the surface) using

Table 1. Ratio of Effective Heating Rates and Heat Transfer Coefficients 1 h before and 3 h after Induction of General Anesthesia

	H^+/H^-	D^+/D^-	λ^+/λ^-
Upper calf	0.9 ± 0.2	1.4 ± 0.2	1.4 ± 0.7
Lower calf	0.9 ± 0.2	2.0 ± 0.7	2.9 ± 1.6
Upper thigh	0.8 ± 0.3	1.4 ± 0.6	1.4 ± 0.7
Lower thigh	0.8 ± 0.3	2.1 ± 2.3	2.0 ± 1.4
Upper arm	1.0 ± 0.3	1.6 ± 0.7	1.4 ± 0.5
Lower arm	1.3 ± 0.5	2.2 ± 1.3	1.9 ± 0.7
Forehead	0.9 ± 0.1	0.8 ± 0.8	0.8 ± 0.1
Average	1.0 ± 0.2	1.6 ± 0.5	1.7 ± 0.7

The ratio of effective heating rate, determined using Eq. 5, is shown in the first column. A single thermal flux transducer was used for the upper and lower thigh; a single transducer was similarly used for the upper and lower calf. Consequently, H^+/H^- are identical for both segments of the thigh and calf. The effective heat transfer coefficients, as determined using Eq. 6, are presented in the second column, and as determined using Eq. 11, are presented in the third column. Data are mean ± SD.

parabolic regression.‡ The temperature at the center of the upper thigh was set to core temperature, providing a fifth temperature for the regression in this location. In contrast, temperatures at the center of the lower leg and arm segments were estimated from the regression equation with no similar assumption. Results of the parabolic regression were expressed by the equation

$$T(r) = T_c + a_2 r^2, \qquad (12)$$

where $T(r)$ is the temperature in °C at radius r (centimeters), T_c (°C) is the temperature at the center of the leg segment, and a_2 (°C/cm^2) is a regression constant.

Data Analysis

The ratio of the effective heat transfer coefficients (equilibrium model), D^+/D^-, was determined using equation 6. The lambda ratio (heat flow model), λ^+/λ^-, was determined using equation 11. The two methods of estimating these ratios were compared using linear regression. Data are presented as mean ± SDs.

Results

Regional thermal flux was comparable before (t = −1 h) and after (t = 3 h) induction of anesthesia. Consequently, the ratio H^+/H^- was 1 ± 0.2. In contrast, the ratio of the effective heat transfer coefficients, D^+/D^-, as determined using equation 6 (equilibrium model) was 1.6 ± 0.5. This ratio was similar, 1.7 ± 0.7, when calculated using equation 11 (heat flow model; table 1).

There was a linear relation between the two methods of determining the ratio of effective heat transfer coefficients before and long after induction of general anesthesia: $\frac{\lambda^+}{\lambda^-} = 1.0 \cdot \left(\frac{D^+}{D^-}\right) + 0.0$, $r^2 = 0.62$. The highest λ^+/λ^- value was from the lower calf, whereas the lowest value was the forehead (fig. 1).

The quantity $\lambda(t)$, as given by equation 10, increased most in the lower calf, lower arm, and lower thigh. $\lambda(t)$ in the proximal extremity segments also increased, but somewhat less. In contrast, the ratio decreased ≈20% in the forehead, which was the only core site we evaluated (fig. 2). When the forehead was excluded, $\lambda(t)$ more than doubled after induction of general anesthesia (fig. 3).

Discussion

Regional body heat distribution and core-to-peripheral flow of heat can be quantified using a sufficient number of muscle and skin temperatures.[9] These measurements,

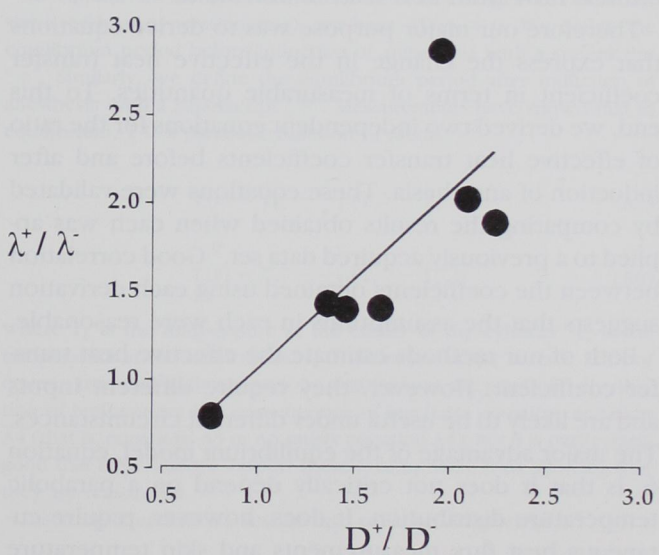

Fig. 1. Regression comparing two methods to determine the ratio of effective heat transfer coefficients before (t = −1 h) and long after induction of general anesthesia (t = 3 h). The effective heat transfer coefficients, D^+/D^-, were determined using equation 6. The effective heat transfer coefficients, λ^+/λ^-, were determined using equation 11. $\frac{\lambda^+}{\lambda^-} = 1.0 \cdot \left(\frac{D^+}{D^-}\right) + 0.0$, $r^2 = 0.62$. The highest λ^+/λ^- value is from the lower calf, whereas the lowest value is the forehead.

‡ Regression constants were determined by reflecting temperatures around the centerline and fitting the values to a second-order polynomial least-squares regression.

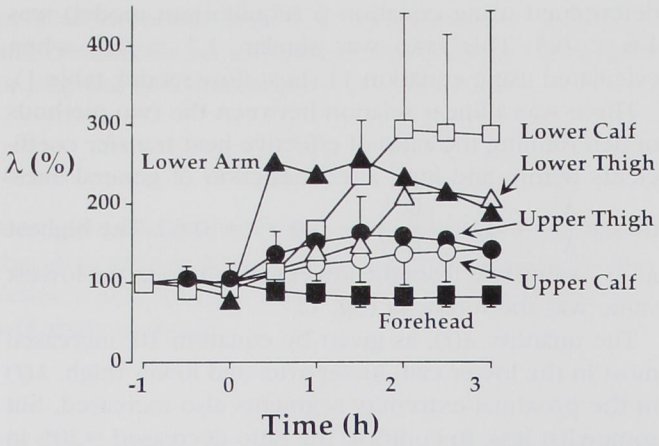

Fig. 2. The quantity $\lambda(t)$, as given by equation 10, as a percentage of the initial values. Elapsed time zero identifies induction of general anesthesia. Values are expressed as means ±SDs. The initial and final values were used to calculate λ^+/λ^- in figure 1. Upper arm values are not shown because they are virtually identical to those for the upper calf. Many error bars are omitted for clarity, but they were generally similar to those displayed for the upper calf.

however, are difficult and invasive, which limits their general applicability. Further, the results apply only to specific experimental circumstances, and it would be difficult to determine the effects of vasomotor changes on heat flow from heat distribution alone.

Therefore our major purpose was to derive equations that express the change in the effective heat transfer coefficient in terms of measurable quantities. To this end, we derived two independent equations for the ratio of effective heat transfer coefficients before and after induction of anesthesia. These equations were validated by comparing the results obtained when each was applied to a previously acquired data set.[9] Good correlation between the coefficients obtained using each derivation suggests that the assumptions in each were reasonable.

Both of our methods estimate the effective heat transfer coefficient. However, they require different inputs and are likely to be useful under different circumstances. The major advantage of the equilibrium model, equation 6, is that it does not critically depend on a parabolic temperature distribution. It does, however, require cutaneous heat flux measurements and skin temperature and temperature at the center of the extremity. Regional thermal flux is easy to measure using special transducers.[26] Furthermore, heat flux was essentially unchanged by general anesthesia, as expected from previous observations.[28] The results with this model thus depended primarily on skin and central extremity temperatures under the circumstances of our study. The ratio D^+/D^- increased because anesthetic-induced vasodilation reduced the gradient between skin and central extremity temperature. This model also applies only during equilibrium and provides only a ratio of the heat transfer coefficients rather than absolute values.

The heat flow model, equation 10, provides absolute and time-dependent estimates of the effective heat transfer coefficient. Furthermore, it is potentially valid in nonequilibrium situations (although not exactly when anesthesia is induced). From figure 2 it is apparent that steady state is reached in about 2 h, which is consistent with the studies of Ducharme *et al.*[29,30] One might expect that the rate at which steady state develops would be a function of segment radius and thus develop faster in the arms than in the legs, and would be faster in the calves than in thighs. However, this theoretical radial dependence was not apparent in the data, suggesting that regional blood flow varies considerably and influences the kinetics of heat exchange in various extremity segments.

The heat flow model depends much more on a parabolic tissue temperature distribution than the equilib-

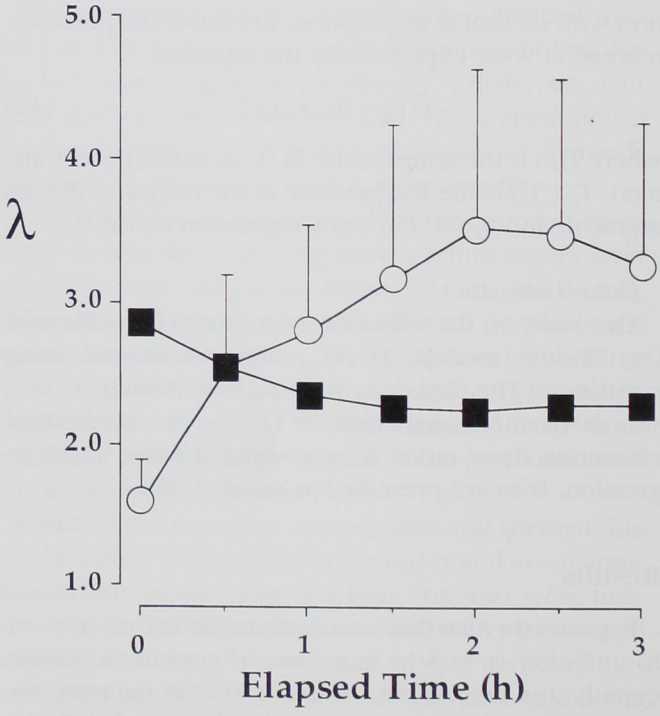

Fig. 3. Absolute values of the quantity $\lambda(t)$, as given by equation 10, after induction of general anesthesia at elapsed time zero. Forehead (core, squares) $\lambda(t)$ was plotted separately from the peripheral sites (circles). Initially, the six peripheral values were averaged in each volunteer; the means ± SDs were then determined from these values.

rium model does. Both models, however, require tissue temperature at the center of the extremity (T_c). We estimated this temperature from skin and tissue temperatures using a parabolic regression. However, core temperature could be substituted for T_c in the thigh with little loss of accuracy. A single 38-mm-long needle thermocouple would probably be sufficient in the arms and most of the leg. The lower calf, however, differed from other extremity sites in that λ^+/λ^- was considerably greater than D^+/D^-. The cause of this difference remains unclear, but it may result from distortion of the parabolic tissue temperature gradient in the distal leg by the relatively large adjacent bone at this site.

The effective heat transfer coefficient for the forehead (a core site) decreased ≈20% after induction of anesthesia. This decrease presumably resulted because propofol anesthesia decreases brain perfusion, approximately in proportion to the reduction in cerebral metabolic rate.[31] In contrast, the diffusion coefficient for the six tested extremity sites more than doubled after induction of anesthesia. This increase indicates that thermoregulatory and anesthetic-induced vasodilation doubles the core-to-peripheral flow of heat. Of course, this flow of heat causes redistribution hypothermia, which is usually the major cause of core hypothermia during anesthesia.[9]

In conclusion, heat transfer coefficients were estimated by applying two independent mathematical models to data acquired in a previous evaluation of heat balance during induction of anesthesia. We found a linear relation between the ratio of steady state regional heat transfer coefficients calculated using each model, which suggests that both are reasonably accurate. The effective heat transfer coefficient for the forehead (a core site) decreased ≈20% after induction of anesthesia. This decrease presumably reflects anesthetic-induced reduction in brain perfusion. In contrast, heat transfer coefficients in the extremities more than doubled after induction of general anesthesia, indicating that thermoregulatory and anesthetic-induced vasodilation doubles the core-to-peripheral flow of heat.

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Appendix: Derivation of Change in Heat Flow Parameters

Generalities

In any object, where there is heat generation and transfer by conduction, the flow of heat is governed by the bioheat equation[22,23]:

$$\rho c \frac{\partial T}{\partial t} = k\nabla^2 T + P \tag{A1}$$

where the heat conduction coefficient, k, is measured in units of cal · $cm^{-1} \cdot s^{-1} \cdot °C^{-1}$ and the heat production rate, P, is measured in units of cal · $cm^{-3} \cdot °s^{-1}$. The density of material is ρ (g/cm^3) and the specific heat is s (cal · $g^{-1} \cdot °C^{-1}$) and where T is temperature in °C and t is time in seconds.

We may define an effective heat transfer coefficient, D (cm^2/s), and an effective heat generation coefficient, H (°C/s), by

$$D = \frac{k}{s\rho}, \tag{A2}$$

$$H = \frac{P}{s\rho}, \tag{A3}$$

In the human body where blood flow is important in locally depositing (or absorbing) heat and in transferring heat, we may still use the same equation, but with D acting as the effective heat transfer coefficient and H as the effective heating rate. Thus D includes conduction and convection, whereas H includes metabolic generation and heat deposition. Because the flow of heat radially is what we want to model, we have

$$\frac{\partial T}{\partial t} = \frac{D}{\partial r}\frac{\partial}{\partial r}\left(r\frac{\partial T}{\partial r}\right) + H \tag{A4}$$

where we have taken D and H as functions of T, but not of position.

The Equilibrium Model

In equilibrium (long before and long after changes in vasomotor status or heat generation rate), we have $\partial T/\partial t = 0$. We define the equilibrium period before induction of anesthesia with a superscript "−". Similarly, we define the equilibrium period after induction of anesthesia with a superscript "+". Measurements have shown that in equilibrium, T is a parabolic function of radius.[9,24]

$$T(r) = T_c^- - \frac{r^2}{r_s^2}(T_c^- - T_s^-); \tag{A5}$$

$$T(r) = T_c^+ - \frac{r^2}{r_s^2}(T_c^+ - T_s^+); \tag{A6}$$

where T_c is the temperature at the center of the cylinder, T_s is the temperature at the surface, and r_s is the radius at the surface. It is, of course, true that with our various assumptions the parabolic distribution of temperature is a consequence of our basic equation, equation A4 (that is, equations A5 or A6 satisfy equation A4), but it is particularly good that experiments support these assumptions, thus showing that they are reasonable.

Inserting equations A5 and A6 into equation A4 gives us

$$-\frac{4D^-}{r_s^2}(T_c^- - T_s^-) + H^- = 0 \tag{A7}$$

$$-\frac{4D^+}{r_s^2}(T_c^+ - T_s^+) + H^+ = 0 \tag{A8}$$

and hence

$$\frac{D^+}{D^-} = \left(\frac{H^+}{H^-}\right)\left(\frac{T_c^- - T_s^-}{T_c^+ - T_s^+}\right). \tag{A9}$$

We can apply this model to an extremity, in which case T_c becomes the temperature in the center of the limb, T_s is the skin temperature, and r_s is the radius at the skin. The heat flow is given by $Q = -k\nabla T$ and at the skin surface

$$Q = -k \left.\frac{\partial T}{\partial r}\right|_{r_s} = \frac{2k}{r_s}(T_c - T_s). \quad \text{(A10)}$$

By combining steady state equations before (−) and after (+) a change in vasomotor status or heat production (t = 0) into a single equation (±), we obtain from equations A7, A8, and A10:

$$\frac{-4D^{\pm}}{r_s^2}\left(\frac{r_s Q^{\pm}}{2k^{\pm}}\right) + H^{\pm} = 0 \quad \text{(A11)}$$

Using equations A2 and A4 we obtain

$$\frac{H^+}{H^-} = \frac{Q^+}{Q^-}. \quad \text{(A12)}$$

Using this result, equation A12, and inserting it in equation A9:

$$\frac{D^+}{D^-} = \frac{Q^+}{Q^-}\left(\frac{T_c^- - T_s^-}{T_c^+ - T_s^+}\right). \quad \text{(A13)}$$

Equations A12 and A13 express the change in effective heat transfer coefficient, D, and in effective heating rate, H, in terms of measurable quantities.

The Heat Flow Model

Previously we showed that heat flow from the skin surface into air per unit area, Q, is proportional to $T_s - T_a$, where T_s is the skin temperature and T_a is the ambient temperature in °C.[25] That is,

$$Q = \frac{k_a}{L_a}(T_s - T_a), \quad \text{(A14)}$$

where k_a is the effective heat flow parameter in the air and L_a is a characteristic distance over which the temperature decreases from T_s to T_a. Convection, radiation, and conduction are all included in equation A14. (We introduce the quantity L_a to keep the dimensions of k_a the same as those of k, but only the combination k_a/L_a comes into our analysis.)

The flow of heat out of a limb is given in equation A10. Equating the two expressions for heat flow (equations A10 and A14) and solving for T_s, we obtain:

$$\lambda(t) = \frac{T_s - T_a}{T_c - T_s}, \quad \text{(A15)}$$

where the new quantity $\lambda(t)$ is defined by

$$\lambda(t) = \frac{2L_a}{r_s}\frac{k}{k_a}. \quad \text{(A16)}$$

We have explicitly indicated the time dependence of $\lambda(t)$, because this analysis does not assume equilibrium. Of course, T_c, T_s, and T_a are also time dependent. Thus in this model we have a formula involving only one parameter, $\lambda(t)$, and there is no need to determine heat flux. Besides the assumption of cylindrical symmetry, this model is especially sensitive to tissue temperature near the skin surface because it depends on the derivative of $T(r)$. We have assumed a parabolic tissue temperature distribution based on previous studies[24]; however, even small deviations from this idealized function might markedly alter estimates of $\lambda(t)$. And, finally, the ratio of the value of $\lambda(t)$ long after a change in vasomotor tone or heat production, λ^+, to the value of $\lambda(t)$ long before a change in vasomotor tone or heat production, λ^-, namely λ^+/λ^-, is just the quantity D^+/D^-. This follows because both ratios only depend on the ratio of k values (compare equations A2 and A16). Explicitly,

$$\frac{\lambda^+}{\lambda^-} = \left[\frac{T_s^+ - T_a^+}{T_c^+ - T_s^+}\right]\left[\frac{T_s^- - T_a^-}{T_c^- - T_s^-}\right]^{-1}. \quad \text{(A17)}$$

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