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Skin Circulation and the Thermoregulatory Threshold

To the Editor:—Sessler *et al.*[1] recently published their results of temperature measurements during general anesthesia with halothane, in support of a hypothermic threshold for thermoregulation. Their study was thought-provoking and serves to highlight some of the problems associated with skin circulatory physiology.

Skin temperature is a useful indirect measure of serial changes in skin blood flow (SBF). However, it is a relatively insensitive modality being affected by various factors, such as equipment design, skin texture, contact of thermocouple with skin, and ambient temperature. In Sessler's study, a gradient of 4° C between forearm and fingertip skin temperature during hypothermia was proposed as evidence of cutaneous vasoconstriction and "thermostatic" hypothalamic temperature regulation in 12 patients, corroborated by Laser Doppler and hand venous P_{O_2} measurements in two additional patients. The designation of the 4° C skin temperature gradient was determined by the authors in preliminary studies.[2]

The skin circulation is noted for its regional variations of anatomy and physiology. Total SBF consists of nutrient or capillary flow and arteriovenous (a-v) anastomosis flow. During body cooling, vasoconstriction of a-v anastomoses occurs,[3] so that a-v shunt flow decreases significantly more than nutrient flow.[4] Anastomoses are numerous in the digits, particularly the fingertips, and virtually absent in the forearm skin.[3] Vasomotion of cutaneous precapillery vascular beds, accounting for changes in SBF velocity, occurs less frequently in the digits, compared to more proximal skin areas.[5] One researcher concluded that "observations in one skin area cannot necessarily be applied to others at the periphery."[6]

This study raises a number of interesting issues related to the demonstration and interpretation of cutaneous vasoconstriction. Since the characteristics of SBF to the fingertip and forearm differ considerably, hypothermia would be expected to produce a larger temperature drop at the fingertip, compared to forearm skin, since a-v shunt flow would be predominately affected. Therefore, the observed skin temperature gradients in hypothermic patients could be explained on the basis of local blood flow phenomena instead of "thermostatic" hypothalamic mechanisms. It is not known whether halothane modifies this response, compared to other pharmacologic agents. The Laser Doppler used in this study can measure cutaneous blood flow velocity in any skin area of the body, even if conditions are changing. Photoplethysmography of the fingertip could provide information about the cutaneous pulsatile microcirculation. Thermography could be used to demonstrate skin temperature changes over a wider area compared to a single thermocouple. A combination of these monitors from various sites would provide more information about regional changes in skin circulation, and further characterize central thermoregulatory mechanisms.

I commend Sessler and colleagues for an interesting study. The review of thermoregulation was most informative.

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More on Thermoregulatory Thresholds with Halothane

To the Editor:—We applaud the elucidation by Sessler *et al.* of mechanisms of human thermoregulation during anesthesia;[1] also, we would like to comment on the interpretations of esophageal temperatures measured during the study.

First, when an esophageal stethoscope is positioned at the site of "maximum heart tones," a subjectively determined location, its tip, with temperature sensor, may still be lying in a retrotracheal position. A previous study by Whitby and Dunkin elegantly demonstrated the nonuniform distribution of temperatures along the esophagus, both longitudinally and laterally. These investigators noted that the lower fourth of the esophagus was both the warmest and the most thermally stable segment.[2] Of note was the observation that temperature varied almost 1° C with as little as a 2-cm change in depth of esophagus. In a more recently published study that confirms a temperature gradient along the esophagus, a shift of as little as 2 cm had "very little effect on the sounds," the highest temperatures being recorded at a site 12–16 cm deeper than where best heart and breath sounds occurred (38–42 cm from the incisors).[3]

Second, esophageal temperature can be influenced by active warming of airway gases with a heated humidifier. In a study of how esophageal temperature is influenced by airway temperature, we demonstrated that even passive warming of inspired gases with a heat and moisture

exchanger (Engstrom Edith 1000®) significantly increased mean esophageal temperature by 0.4°, (range 0.2°–1.2° C) behind the trachea (*i.e.*, site of best heart and breath sounds).* In another study, esophageal temperature measured distally was not affected by even extreme changes in airway temperature unlike that measured more proximally.[4]

Temperature in some patients reported by Sessler *et al.* could have, in spite of their positioning the probe at the site of best heart sounds, therefore, still been influenced both by position of the temperature probe, which could result in artifactually cooler temperatures in the control group, and by warming of the esophagus by heated airway gases, which could result in artifactually warmer temperature in the temperature-controlled group. If these influences were operating, esophageal temperatures in these two groups would be skewed in opposite directions.

In studies of thermoregulation, there is a premium on recording the best and least controversial measurement of core temperature. For this purpose, we suggest either the tympanic membrane or another site with a temperature that correlates as closely with core temperature. As Cork *et al.* have shown, this would include nasopharyngeal temperature.[5]

* Siegel MN, Gravenstein N: Use of heat and moisture exchanger significantly influences esophageal temperature monitoring. Submitted for publication.

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In Reply:—Dr. Loubser's comments provide a welcome opportunity to discuss thermoregulatory vasoconstriction in detail. Metabolic heat is lost primarily *via* convection and radiation from the skin surface.[1] Decreasing cutaneous blood flow reduces environmental heat loss and is the most consistently used thermoregulatory mechanism.[2] In 1931, Grant and Bland observed that total digital skin blood flow is divided into capillary and arteriovenous shunt components, and subsequent studies have confirmed that peripheral skin has a dual blood supply.[3]

Thermoregulatory vasoconstriction is believed to occur primarily in the cutaneous arteriovenous shunts.[4] As Dr. Loubser notes, these shunts are concentrated in the fingers and toes so that blood flow to distal extremities during cold exposure is more affected than flow to more proximal skin (*e.g.*, forearm).[3] It is precisely this distribution that makes finger and toe temperatures useful indices of thermoregulatory vasoconstriction. Forearm temperature is minimally affected by thermoregulatory vasoconstriction (*e.g.*, forearm and core temperatures are well correlated).[5] In contrast, cold exposure decreases shunt flow, which decreases peripheral skin-surface temperature.

The decrease in peripheral flow that we observed is almost completely *centrally mediated* and does not result from changes in local ambient temperature.[2] The pattern of vasoconstriction indicates that peripheral cooling does not result from passive central cooling: if peripheral cutaneous flow decreased passively, the gradients would increase smoothly as central temperature decreased. Instead, the skin temperature gradients remained $\approx -1°$ C until vasoconstriction, and then increased rapidly to values $\geq 4°$ C (see figure 3 in reference 6). In studies using other anesthetics (which inhibit thermoregulation more than halothane), we have observed patients with central temperatures $\approx 32°$ C who maintained negative skin-surface temperature gradients.

Central-finger tip gradients are frequently used to evaluate peripheral vasoconstriction because they correlate well with plethysmography,[7] and thermal and helium dilution.[8] Since forearm temperature is $\approx 3°$ C below core temperature,[6] a central-finger tip gradient of 7° C is comparable to a forearm-finger tip gradient of 4° C. When the thermoregulatory threshold during halothane anesthesia is determined using central-finger tip gradients, the results are similar (34.43 ± 0.24° C) to those we reported previously using skin-surface gradients (34.44 ± 0.23° C).[6] Furthermore, the duration of anesthesia preceding a finding of significant vasoconstriction with each method differed by less than 10 min.

Since thermoregulatory vasoconstriction produces a rapid, 5–10-fold decrease in peripheral flow, it is easily detected by both central-finger tip and forearm-finger tip gradients. Comparing two skin-surface temperatures (rather than central and skin temperatures) has the advantage of eliminating confounding factors caused by differences in equipment design, skin texture, core temperature, and ambient temperature. Adequate and consistent contact between skin and the thermocouples is easily maintained using self-sticking Mon-a-Therm® probes.

Although thermoregulatory vasoconstriction is believed to occur primarily in the cutaneous arteriovenous shunts, capillary flow also decreases.[4] Capillary flow can be determined using the laser Doppler perfusion monitor which correlates well with ^{133}Xe washout,[9] and dynamic capillaroscopy.[10] We have recently demonstrated a good correlation between the laser Doppler perfusion index and skin-surface temperature gradients (Doppler index = $-7.9 * \text{Gradient} + 67$, $r^2 = 0.63$).[5] We also have used multiple peripheral skin-surface monitors to demonstrate that the pattern of vasoconstriction during anesthesia