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.

MARC N. SIEGEL, M.D.
Resident in Anesthesiology

NIKOLAUS GRAVENSTEIN, M.D.
Assistant Professor of Anesthesiology and Neurosurgery

Departments of Anesthesiology and Neurosurgery
University of Florida College of Medicine
Gainesville, Florida 32610-0254

REFERENCES

1. Sessler DI, Olofsson CI, Rubinstein EH, Beebe JJ: The thermoregulatory threshold in humans during halothane anesthesia. ANESTHESIOLOGY 68:836–842, 1988
2. Whitby JD, Dunkin LJ: Temperature differences in the oesophagus. Preliminary study. Br J Anaesth 40:991–995, 1968
3. Kaufman RD: Relationship between esophageal temperature gradient and heart and lung sounds heard by esophageal stethoscope. Anesth Analg 66:1046–1048, 1987
4. Deal EC Jr, McFadden ER Jr, Ingram RH Jr: Esophageal temperature during exercise in asthmatic and nonasthmatic subjects. J Appl Physiol 46:484–490, 1979
5. Cork RC, Vaughan RW, Humphrey LS: Precision and accuracy of intraoperative temperature monitoring. Anesth Analg 62: 211–214, 1983

(Accepted for publication November 8, 1988.)

Anesthesiology
70:371–372, 1989

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 ($34.43 \pm 0.24°$ C) to those we reported previously using skin-surface gradients ($34.44 \pm 0.23°$ 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

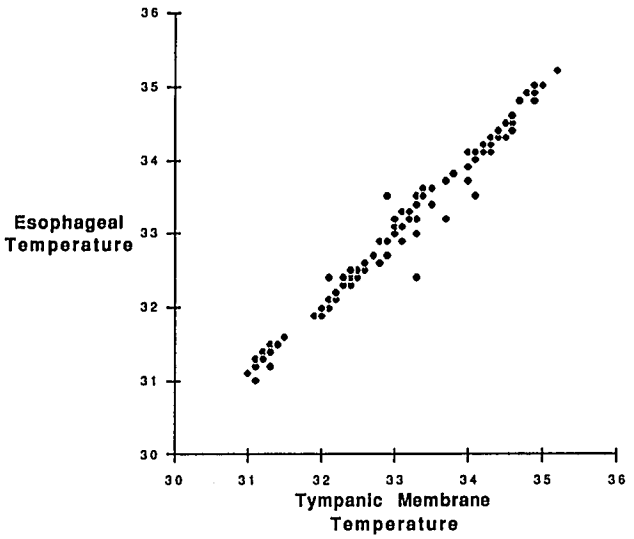

FIG. 1. The correlation between distal esophageal and tympanic membrane temperatures in five patients electively donating a kidney to a relative. The regression equation is: Tympanic = 0.95* (Esophageal) + 1.56, r^2 = 0.98. These data indicate that a thermometer carefully placed in the distal esophagus is a valid measure of central body temperature.

is similar to that in cold-exposed volunteers.[5] These data indicate that thermoregulatory vasoconstriction decreases both capillary and shunt flow and is easily detected using either technique. However, the laser Doppler monitor is expensive and fragile; it also is very sensitive to patient movement, thus poorly suited for postoperative use. Our other studies, in progress and in press, further characterize centrally mediated thermoregulatory vasoconstriction using different measures of cutaneous perfusion. Specifically, using skin-surface gradients to identify centrally mediated thermoregulatory vasoconstriction, we are now quantifying the extent to which regional changes in skin circulation decreases heat loss to the environment.

Dr. Siegel *et al.* are concerned that esophageal temperature may not adequately reflect central body temperature. Because several studies document temperature gradients in the esophagus,[11] we carefully positioned the combined esophageal stethoscope/thermocouples. In each patient, the range of maximal heart sounds was identified (by the same investigator) and the probe placed at the far end of this range. This technique assured that the thermocouple was in the distal quarter of the esophagus, a site that most authors agree is an excellent indicator of central body temperature.[11,12] To validate this technique in our patients, we compared distal esophageal and tympanic membrane temperatures in five kidney donors. The excellent correlation (r = 99) between these measurement sites is shown in figure 1.

Respiratory gas humidification and/or heating does slightly increase (*i.e.*, 0.4° C) *proximal* esophageal temperature. However, as noted by Dr. Siegel and colleagues, *distal* esophageal temperatures are unaffected by inspired gas conditioning in adults.[15] Because our measurements were made in the distal esophagus, they are certainly valid reflections of central temperature. In any case, overestimating central temperature by several tenths of a degree in the patients given additional warming would not account for the 2° C difference between the groups. Nor would it influence interpretation of the study.

Dr. Siegel and colleagues suggest that, in studies of thermoregulation, the "best and least controversial" temperature should be recorded. In doing so, they imply that tympanic membrane (*e.g.*, hypothalamic) temperature is the "gold standard." There is little physiological support for this approach, because it is now known that integrated input from the entire body determines thermoregulatory responses. Evidence in animals and birds indicates that multiple, redundant thermal inputs from nearly every part of the body are integrated by both the spinal cord and hypothalamus.[14] It is likely that thermal sensing is similar in humans.

Thus, the most useful approach to temperature monitoring is to recognize that temperatures of different tissues are "real," but that the physiological and practical significance of each varies. Fortunately, "central" (*e.g.*, tympanic membrane, distal esophageal, and rectal) temperatures are similar in general surgical patients, so measurements at any one of these sites reflects the bulk of the thermal input to the central regulatory system.

DANIEL I. SESSLER, M.D.
Assistant Professor of Anesthesiology
University of California, San Francisco
San Francisco, California 94143

EDUARDO H. RUBINSTEIN, M.D., PH.D.
Professor of Anesthesiology and Physiology
University of California, Los Angeles
Los Angeles, California 90024

REFERENCES

1. Morris RH: Operating room temperature and the anesthetized, paralyzed patient. Surgery 102:9597, 1971
2. Ingram DL, Smith REL: Brain temperature and cutaneous blood flow in the anesthetized pig. J Appl Physiol 29:698–704, 1970
3. Grant RT, Bland EF: Observations on arteriovenous anastomoses in human skin and in bird's foot with special reference to the reaction to cold. Heart 15:385–407, 1931
4. Coffman JD, Cohen AS: Total and capillary fingertip blood flow in Raynaud's phenomenon. N Engl J Med 285:259–263, 1971
5. Sessler DI, Olofsson CI, Rubinstein EH: The thermoregulatory threshold in humans during nitrous oxide-fentanyl anesthesia. ANESTHESIOLOGY 69:357–364, 1988
6. Sessler DI, Olofsson CI, Rubinstein EH, Beebe JJ: The thermoregulatory threshold in humans during halothane anesthesia. ANESTHESIOLOGY 68:836–842, 1988
7. Goetz RH: The rate and control of the blood flow through the skin of the lower extremities. Am Heart J 31:146–182, 1946
8. Hertzman AB, Randall WC: Regional differences in the basal and maximal rates of blood flow in the skin. J Appl Physiol 1:234–241, 1948
9. Holloway GA Jr, Watkins DW: Laser Doppler measurement of cutaneous blood flow. J Invest Dermatol 69:306–309, 1977
10. Fagrell B: Dynamics of skin microcirculation in humans. J Cardiovasc Pharmacol 7(Suppl 3):S53–S58, 1985
11. Whitby JD, Dunkin LJ: Temperature differences in the oesophagus: Preliminary study. Br J Anaesth 40:991–995, 1968
12. Kaufman RD: Relationship between esophageal temperature gradient and heart and lung sounds heard by esophageal stethoscope. Anesth Analg 66:1046–1048, 1987
13. Mercer JB, Jessen C: Central thermosensitivity in conscious goats: Hypothalamus and spinal cord versus residual inner body. Pflugers Arch 374:179–186, 1978
14. Simon E: Temperature regulation: The spinal cord as a site of extrahypothalamic thermoregulatory functions. Rev Physiol Biochem Pharmacol 71:1–76, 1974

(Accepted for publication November 8, 1988.)