Body Temperature During Surgery and Anesthesia*

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The recognition of fulminant hyperthermia and accidental hypothermia during anesthesia and surgery has given a new impetus to clinical thermometry in the operating room. It is suggested that in order to recognize and avoid these conditions in the anesthetized patient, a routine of continuous monitoring of body temperature be added to our armamentarium for patient care.

One of the problems that has to be solved is the standardization and selection of a physiologically meaningful site for the monitoring of body temperature. In order to select such a proper site, a brief discussion of functional anatomy and physiology of body temperature is here presented, together with a discussion of the advantages and disadvantages of the traditional sites, placing emphasis on a new site—namely the tympanic membrane.

The relative constancy of body temperature was recognized by Claude Bernard as a part of the regulation of the “milieu intérieur” required for the health and proper functioning of the cellular elements of warm-blooded animals. The healthy man does not exist with internal body temperature much outside the normal range of 36°-38°C, although during hard work and in febrile diseases he may tolerate for short periods of time temperature as high as 40°-41°C. Denaturation of vital cellular proteins occur above 44°C. Man may tolerate hypothermic states for short periods of time during which his body temperature is as low as 27°-29°C, but these temperatures are incompatible with life except under the most careful medical supervision and even then not for periods longer than a few days.

The center for regulation of body temperature is in the hypothalamus (fig. 1). In this area two regions, anterior and posterior hypothalamus, have been recognized. The anterior region, also known as the “heat disposal” region in the event of total body heat gain, is involved in the initiation of thermoregulatory reflexes. These reflexes will activate the cardiovascular and respiratory system, cutaneous blood vessels, and sweat glands through the autonomic nervous system to help balance body heat through heat loss.

Cold stimulates the central nervous system in the posterior hypothalamus and the endocrine system through the pituitary gland. Its primary action on the periphery is to stimulate sensations of cold and possibly other cutaneous nerve endings which send thermoregulatory signals to the “temperature maintenance region.” The posterior region of the hypothalamus, on receiving “cold signals” through the “shivering center,” will activate the shivering reflex which is one of the most powerful physiological actions of the body in the production of heat.

Hormonal action of cold takes place through the hypothalamus. By lowering the temperature of blood going to the brain, cold stimulates the hypothalamus, which in turn affects the pituitary and the release of thyroid stimulating hormone (TSH) and adrenocorticotropic hormone (ACTH). These two hormones promptly act on their target organs to release thyrotropic and adrenal hormones which serve to increase heat production in the body tissue (fig. 1 and fig. 2). These hormones also serve to potentiate the direct effect of cold in producing extra body heat by shivering. There are interactions of the various endocrine glands on each other, and thus it is not easy to obtain a clear evaluation of the part played by a single glandular component (fig. 2).

Monoamines in the hypothalamus act as mediators of temperature response. Local infusion of 5-hydroxytryptamine have been shown to raise tem-
TEMPERATURE REGULATION

TEMPERATURE MAINTENANCE REGION
HEAT DISPOSAL REGION
NEUROHORMONAL MECHANISM
IN ANTERIOR PITUITARY
METABOLISM ELEVATES

THYROID GLAND
ACTIVITY INCREASED

SKELETAL MUSCLE
SHIVERING

PERSPIRATION

CUTANEOUS BLOOD VESSELS
CONSTRUCTION (DILATION?)

SYMPATHETIC GANGLION

Fig. 1—Functional anatomy of physiologic temperature regulation.

perature in cats. It is suggested that the release of monoamines and their relative local concentrations act on the temperature cells of the anterior hypothalamus. Norepinephrine and epinephrine are concerned with regulating heat loss, and 5-hydroxytryptamine is involved with heat production.

In fever, in response to leukocyte pyrogens, the pyrogens act directly on the cells of the anterior hypothalamus. The magnitude of skin temperature appears of little importance in the regulation of body temperature. Logically, it makes the most sense to have the sensor for warmth inside the core of the body where the heat-generating metabolic activity originates, and the sensor for cold on the outside where the cold environment is located.

“Human Thermostat.” Theodor H. Benzinger has proposed the concept of a “human thermostat” or a “temperature eye” being located in the hypothalamus. The temperature eye will sense the body temperature, as the retina of the eye is capable of sensing light. The set point, 37°C, of the human thermostat is located in the hypothalamus area.

The shifting of the set point from 37°C will cause the activation of thermoregulatory reflexes and, in effect, create the constancy of body temperature. Anesthetic agents, by depression of the hypothalamus, can cause lowering of the set point. The same effect has been attributed to aspirin, while it has been postulated that pyrogen will raise the set point of the human thermostat. Considering the functional anatomy and central regulation of body temperature, it becomes apparent that the logical site to monitor the central temperature is located in the cranium.

Rectal Temperature. The most widely used rectal temperature has been highly criticized for not being a true representative of core temperature. In monitoring of rectal temperature, one has to realize that this area does not have any thermal significance of its own. There are no thermoreceptive elements in the rectum. It is far away from the central nervous system and does not have direct relationship with the crossroad of circulation, that is, heart and great vessels. Sir George Pickering has observed “no clear relationship between changes in rectal temperature and changes in vasomotor tone.”

The reliance on rectal temperature for the last hundred years has been responsible for the failure of clinicians to recognize and use the concept of central thermoreception in clinical thermometry. Our measurements at the Medical College of Virginia and the measurements of others in the anesthetized patient have shown that rectal temperature deviates widely from tympanic (central) measurements. Be-

Fig. 2—Flow diagram of hormonal response to cold. (Reprinted with permission from Masson et Cie [eds.] Les Concepts de Claude Bernard Sur Le Milieu Intérieur. Paris: Libraires de L’Académie de Médecine, 1967, and from T. H. Benzinger.)
cause of these anatomical and physiological aspects of the rectum, such deviations make it nonrepresentative of core body temperature.

**Esophageal Temperature.** As will be discussed further, esophageal temperature in the anesthetized patient who does not experience sudden heat loss or heat gain somewhat represents core temperature in reference to central temperature. However, one has to realize, as J. D. Whitby and L. S. Dunkin have shown, that the esophageal temperature recorded in the anesthetized and intubated patient depends on the site of the esophagus at which it is taken. The longitudinal variation is greater than the lateral and can be by as much as 6°C. The lowest temperatures are found in the upper and middle third of the esophagus. Both longitudinal and lateral variations level out in the lower third. The lower fourth of the esophagus is both the warmest and the most stable. To reach this area, thermocouple leads should be inserted 45 cm from the nostril. This area is situated in the lower mediastinum below the pulmonary vein and between the heart and the descending part of the aorta. Carlsten and Crimley have shown that the esophageal temperature closely follows the temperature of intracardiac blood.

**Tympanic Temperature.** The tympanic membrane temperature, because of its proximity and similar blood supply, is very close to the central temperature. By placing a thermocouple in juxtaposition to the tympanic membrane, T. H. Benzinger has shown that its temperature accurately reflects the temperature of blood coursing through the brain (fig. 3). With the use of gradient calorimetry, he has demonstrated that the changes in tympanic temperature are concordant with one of the thermoregulatory reflexes, namely the sweat rate, whereas skin temperature changes do not reflect sweat rate and are paradoxical and discordant.

Figure 4a, from Benzinger’s study shows that when some subjects swallowed ice, cranial temperature decreased. At the same time, the sweat rate showed a parallel heat loss which was repeatedly constant.

Figure 4b shows the same subjects. When they swallowed ice, their skin temperature increased while at the same time their sweat rate, or heat loss, decreased (discordant relationship).

**Clinical Thermometry in the Operating Room.** In a group of about 30 patients (mostly adult) undergoing general anesthesia and surgery, continuous body temperature measurement was made from a thermocouple placed against the tympanic membrane. This source was compared with continuous and simultaneous measurements from the esophagus and rectum. The period of comparison ranged from 1.5 hours to 12 hours, depending on the length of the operation, and the time the patient spent in the recovery room. For the rectum and the esophagus, a soft plastic probe without cotton cover was used. For the tympanic membrane, a probe with a special “Q-tip” was used.

Each thermocouple was connected to a monitoring recorder, and three temperatures being monitored were transcribed directly to a Leeds and Northrup Company, Speedomax continuous writer.¹

![Fig. 3—Tympanic thermometer consisting of disposable thermocouple introduced into external auditory conduit. (Reprinted with permission from Bild Der Wissenschaft, Volume Three, July, 1964, and from T. H. Benzinger.]

¹ We would like to thank Leeds and Northrup Company for providing the monitoring equipment, and also acknowledge the support of Dr. John L. Patterson, Jr., Director, Cardiopulmonary Labs and Research, and Dr. C. Paul Boyan, Chairman, Department of Anesthesiology.
Figure 5 shows simultaneous monitoring of rectal, esophageal, and tympanic temperature in a patient during open heart surgery. Immediately after the bypass there is a profound fall in body temperature. (From a tympanic temperature of $37^\circ C$ to $32^\circ C$). This has prompted some clinicians to advocate this technique in patients with fulminant hyperthermia to lower the body temperature. Figure 5 also shows the deviations of rectal temperature from tympanic and esophageal under the conditions of extracor-

![Figure 4a](image1)

**Fig. 4a**—Concordant patterns of sweating rate and cranial (tympanic) temperatures observed during repeated oral ingestion of ice. (Reprinted with permission from Masson et Cie [eds.] *Les Concepts de Claude Bernard Sur Le Milieu Intérieur*, Paris: Libraires de L'Académie de Médecine, 1967, and from T. H. Benzinger.)

![Figure 4b](image2)

**Fig. 4b**—Discordant patterns of sweating and skin temperatures observed during repeated oral ingestion of ice. (Reprinted with permission from Masson et Cie [eds.] *Les Concepts de Claude Bernard Sur Le Milieu Intérieur*, Paris: Libraires de L'Académie de Médecine, 1967, and from T. H. Benzinger.)
difficulty in recovery of the heart during open heart surgery.

Figure 6 shows another example of a patient undergoing open heart surgery with multiple "crash cooling" and "rewarming" periods during extracorporeal circulation. A similar deviation between rectal and esophageal, and tympanic temperature is noticed.

Figure 7 shows the rewarming period and accidental overheating of a patient under extracorporeal circulation with multiple "crash cooling" periods. It can be seen that in case of accidental overheating, the deviations between different sites of temperature discussed in figure 5 can exist.

**Hypothermia During Anesthesia and Surgery.**

During clinical thermometry we noticed, as others have, that under modern operating room conditions (humidity of about 50% and temperature between 68°F and 75°F) the majority of patients undergoing different types of surgery had some degrees of fall in body temperature. Depending on the type and length of the operation, some patients could develop profound hypothermia.

Man being a homothermic animal has the ability to maintain his normal body temperature regardless of environmental temperature. An anesthetized man loses this ability. He becomes somewhat
poikiothermic and follows the environmental temperature which, in such circumstance, is that of the operating room.

By depressing the central nervous system, anesthetic agents depress the center of temperature regulation thus decreasing body temperature and contributing to the fall of temperature in the anesthetized patient. Sir George Pickering has emphasized this fact, by saying, "The best way to cool a patient is to anesthetize a patient."

Anesthetic agents cause cellular depression and decreases in metabolism and heat production. The effect of these agents on the "shivering center" and on the periphery in concert with muscle relaxants will block the act of shivering, and as long as the patient is anesthetized and paralyzed, no compensatory mechanism for heat gain exists. The fact that peripheral circulation has lost the capability of constriction will contribute further to the magnitude of heat loss in the anesthetized patient.

Premedication with any of the usual drugs has the effect of relaxing muscle tone, thus making the patient more susceptible to a fall in body temperature when taken into a cold operating room. On recovery from anesthesia, thermostatic reflexes appear, and if the body temperature is lowered, shivering and cutaneous vasoconstriction occur with a concomitant rise in oxygen consumption. The greater the fall in temperature, the greater the oxygen consumption during the recovery period.

In comparison with other anesthetic agents used, halothane can cause more heat loss during anesthesia, more shivering at the termination of anesthesia, and more rise in oxygen consumption.

The importance of increased postoperative oxygen consumption may be considerable, particularly in patients with preexisting respiratory or circulatory disease.

In the event that a postoperative airway obstruction exists, the patient will not be able to tolerate anoxia if, because of increased oxygen consumption due to shivering, the ventilation demand is increased. In patients with abdominal incisions and thoracotomy, increase in ventilatory demand may present postoperative difficulties.

Increase in oxygen requirement will also increase demands on circulation. Patients with cardiovascular problems and unreplace blood loss will be further handicapped in dealing with this. During recovery from hypothermia, the rewarming acidoses can contribute greatly to morbidity of heat loss during anesthesia.

Robert M. Morris' study on the relationship between operating room temperature and the temperature of the anesthetized patient shows that a significant linear correlation exists between the patient's esophageal temperatures and their operating room temperatures. He classified the operating rooms according to their effect on patients' temperature: (1) rooms below 21°C in which all patients became hypothermic; (2) 21°C to 24°C (70°F to 75°F) rooms in which 70% of the patients remained normothermic and 30% became hypothermic; and (3) 24°C to 26°C (75°F to 79°F) rooms in which all patients remained normothermic. He concluded that, 21°C can be classified as the "critical ambient temperature" for lightly anesthetized paralyzed adults.

Boyan and his associates have shown that if, in addition, large amounts of cold blood are transfused, the fall in temperature can be greater. A contributing factor to this is the cold solution used for prepping. Deep anesthesia (with or without muscle relaxants) can also cause great heat loss. Since basal heat production decreases with age, the resultant heat deficit is greater in the elderly.

In addition to heat loss, secondary to vasodilation and decreased heat production, the opening of the abdominal cavity is an important factor with the anesthetized patient. Inside the peritoneal cavity is a large surface from which much heat can evaporate, and irrigation of the peritoneal cavity with cold solutions leads to increased heat loss. At a comfortable operating room temperature (20°C to 22°C),
most patients experience a fall in body temperature.

Figure 8 emphasizes the effects of age (85 years old), the type of operation (intra-abdominal), the length of procedure (5 1/2 hours), and cold ambient temperature (operating room temperature 21.1°C) on the extent of the fall in body temperature. At times during the procedure, by means of a warm blanket, an effort was made to warm the patient. In spite of this effort, the drifting of hypothermia took place, and the patient regained a normal body temperature only after he was taken to the recovery room (temperature 26.6°C).

This is in concert with the observation of Robert M. Morris and his associates that a warm blanket under the paralyzed and anesthetized adult is no substitute for a warm operating room. One can see from this experiment that in the event of slow and progressive heat loss, there are minimal deviations in the monitoring of the different sites of body temperature. But under clinical circumstances when one can expect sudden heat gain and heat loss, we believe that it seems more important to use tympanic or esophageal monitoring of temperature.

REFERENCES


