BODY TEMPERATURE AND ANAESTHESIA

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The processes of anaesthesia and surgery attenuate normal homeostatic thermoregulation and impose large thermal stresses. The resulting changes in body temperature may be detrimental and it is important, therefore, that anaesthetists understand normal thermoregulation and the ways in which this process may be affected by anaesthesia.

PHYSIOLOGY OF TEMPERATURE REGULATION

Why thermoregulate?
The velocity of enzyme catalysed reactions increases with temperature to a maximum beyond which enzyme activity decreases because of protein denaturation. Many biological reactions have a 10 °C temperature coefficient (Q10) of 2; that is, their rate of reaction doubles for a 10 °C temperature increase [62]. In mammals, protein denaturation begins at about 42 °C and internal temperatures at or above this value can be tolerated only briefly. At the other end of the scale, ice crystals form in mammalian cells at −1 °C and physical disruption of the cells occurs [82]. In intact animals, vital organ function becomes critically impaired at internal temperatures much higher than freezing; for example, consciousness is lost in man at 30 °C and death often occurs at temperatures less than 25 °C as a result of ventricular fibrillation. Accurate maintenance of the internal temperature at a value close to the optimum for enzymatic activity bestows many benefits, including a constant high rate of metabolism, rapid nervous conduction and muscular contraction, decreased viscosity of blood and, perhaps most importantly, freedom from the constraints of environmental conditions.

Homeothermy

Homeothermy is defined by The Thermal Physiology Commission of the International Union of Physiological Sciences as "a pattern of temperature regulation in which the cyclic variation in core temperature, either nychthermally or seasonally, is maintained within arbitrary limits of ±2 °C despite much larger variations in ambient temperature" [25]. Only mammals and birds conform to this definition; however, all other vertebrates exhibit some degree of thermal regulation, mediated usually by behavioural responses [20].

Homeothermy involves sensing body temperature and appropriately driving the mechanisms controlling heat loss and gain so as to maintain a normal value for temperature. Thus thermoregulation is a closed-loop system.

Thermal balance

Homeothermy is achieved by balancing heat gains with heat losses. Within a range of ambient temperatures this may be achieved without metabolic expenditure, by control of peripheral vasculature. Beyond the limits of this "thermonutral zone" (approximately 20–35 °C in resting adults), energy must be expended in order to maintain thermal balance.

Heat gains. These can be considered as obligatory or facultative. Obligatory heat gains are those occurring independently of thermoregulation and imposing demands upon it. This includes the heat derived from basal metabolism (167 kJ m⁻² h⁻¹ (40 Cal m⁻² h⁻¹)), feeding (8% of ingested energy), storage and growth. However, the major source of metabolic heat is muscular exercise; light exercise (walking) results in a three- to five-fold increase in metabolic rate above basal and...
severe exercise can produce up to 20-fold increases.

Facultative heat gains are those available to thermoregulation as a means of restoring thermal balance and may be classified as shivering and non-shivering. Shivering may produce four- to six-fold increases in heat production [31]; 80% of the heat produced is retained by the body, compared with 50% for voluntary exercise. Non-shivering thermogenesis is any source of facultative heat production other than shivering.

Neonates have large quantities of brown adipose tissue over the neck, upper part of the back and around thoracic and abdominal viscera. Activation of brown adipose tissue in human neonates may cause two- to three-fold increases in metabolic rate. Brown fat is controlled by the hypothalamus via sympathetic nerves and circulating catecholamines. In humans older than 1 yr, brown fat is probably unimportant as a source of heat production [26].

Heat losses. Heat is lost from the body only at points of contact with the environment, that is, the skin and respiratory tract. At rest, 75% of basal heat production is lost by convection, conduction and radiation from the body surface. Of these, convection is the largest component and conduction is usually minimal. Losses by insensible perspiration and through the respiratory tract account for the remaining 25% in approximately equal amounts.

Sweating is the major source of heat loss under thermoregulatory control. The maximum rate of sweating is of the order of 2 litre h⁻¹; this gives a calculated heat loss of about 2.7 MJ m⁻² h⁻¹ (650 Cal m⁻² h⁻¹) or about 15 times basal heat production. Sweating is exhaustible and maximum daily sweat production is about 5 litre.

Heat storage

The body is not in a continuous state of heat balance; net gains or losses result in changes in the overall heat content of the body, normally with little or no change in the core body temperature. This is brought about by changes in the temperature of the "body shell" (those parts of the body in contact with the environment and hence usually at a lower temperature than the body core).

Temperature receptors

There is growing evidence that temperature receptors exist in many body tissues. They can be classified into two main groups: central, which sense the temperature of the body core, and peripheral, which sense the temperature of the body shell and environment.

Central thermoreceptors. It is well recognized that the anterior hypothalamus is temperature sensitive and it was thought that hypothalamic temperature contributed more than 60% of the total thermoregulatory input [3,4]. More recently, it has become apparent that many body tissues contain temperature receptors, including the spinal cord, the midbrain, the lower brain stem, abdominal organs and skeletal muscle, and the concept of the hypothalamus as the sole sensor of core temperature has given way to one of multiple thermal inputs with the hypothalamus contributing perhaps as little as 20% of the total [76].

It is not known whether central thermoreception is accomplished by specific single cells or if it is a result of temperature dependent changes within neural networks.

Peripheral thermoreceptors. Recordings from afferent cutaneous nerves show clearly that specific warm and cold receptors exist in the skin. The histological nature of these receptors is unknown, as are the mechanisms responsible for the transduction of temperature into nervous activity. It has been suggested that cold sensitivity results from the activity of a ouabain-sensitive, sodium-potassium ATPase, while warm sensitivity is attributable to the different temperature coefficients of sodium and potassium membrane permeabilities [9]. Cold impulses travel mainly in A-delta fibres. Discharge in these fibres begins at skin temperatures of about 40 °C and is maximal (8-14 impulses per second) at 25-30 °C. Below 25 °C the mean frequency of discharge remains the same, but burst activity becomes more common. This burst activity may be the means of encoding thermal information at low temperatures. Above 45 °C, cold receptors show a paradoxical increase in discharge frequency. Warm impulses are transmitted in C fibres. Discharge begins at about 30-35 °C and is maximal at 40-45 °C. Warm receptors do not exhibit burst discharge.

In addition to steady rates of discharge for a given temperature, peripheral thermoreceptors also demonstrate accommodation (a declining response to a steady thermal stimulus) and patterns of discharge related to the rate of change of temperature [14, 55]. Studies of human thermal sensibility indicate that patterns of discharge in
single warm or cold fibres are insufficient to account for the observed level of subjective thermal discrimination and some degree of central processing must occur [14].

Input-output coupling

In mammals, integration of thermal input and control of effector organs is performed primarily by the hypothalamus, although a certain degree of processing of thermal input occurs in the spinal cord, and some limited thermoregulatory responses are possible at spinal segmental level. It appears that thermoregulation utilizes “loose” input-output coupling such that a given stimulus may provoke one of several different effector responses. For example, in animal experiments, cold stress may result in the behavioural response of selection of a warm environment or the autonomic response of shivering [76]. The role of the hypothalamus in thermoregulation is often likened to a thermostat; however, the biological correlate of the thermostatic set point remains unknown. There is no evidence for a neural reference signal with which the integrated thermal input is compared, and it has been suggested that a balance of warm and cold thermal inputs creates a dynamic set point [76].

ANAESTHESIA AND TEMPERATURE REGULATION

During anaesthesia and surgery, several factors combine to interfere with normal thermoregulation. These include abolition of behavioural responses, attenuated hypothalamic function, reduced metabolic rate, reduced effector responses and abnormally large thermal stresses.

General anaesthesia totally abolishes the behavioural responses which form such an important part of normal thermoregulation. Local anaesthesia also impairs behavioural responses although, if unsedated, the patient can at least complain if he/she feels too cold or too hot.

General anaesthesia

The loss of consciousness during general anaesthesia abolishes thermal sensation, but it is generally held that the sensitivity of thermoreceptors is not impaired [21]. The effect of anaesthesia on the various effector systems has never been studied directly. With the exception of ketamine, all general anaesthetic agents impair thermoregulation, presumably by attenuation of hypothalamic function. The effect of this is to widen the range of core temperatures over which no thermoregulatory responses occur. That thermoregulatory responses are still possible during anaesthesia has been known since the first attempts at surface hypothermia, when reductions in core temperature were limited in spontaneously breathing patients by severe shivering [42]. Recently, the phenomenon of thermoregulation during anaesthesia has been rediscovered by observation of vasoconstriction in hypothermic, but not in normothermic, patients [65, 66]. It is not known if these responses represent “normal” thermoregulation at a lower set point. Studies comparing different anaesthetic techniques have failed to demonstrate significant differences in heat losses [27, 28, 52, 67], with the exception of ketamine, which seems sparing of thermoregulation [15, 30], and ether anaesthesia, during which increases in body temperature were observed in children undergoing facial surgery [51].

Other agents given during anaesthesia also have major effects on thermal balance. Neuromuscular blocking drugs abolish shivering and it has been observed that paralysed patients cool more rapidly than non-paralysed patients, although this may relate more to the type of surgical procedures performed than to the use of myoneural block [18]. Vasodilators redistribute body heat to the peripheral tissues and increase the potential for loss of heat to the environment. Opioids, barbiturates, phenothiazines and butyrophenones have central and peripheral actions tending to decrease body temperature.

The pattern of temperature change during general anaesthesia is well documented and consists of an initial large decrease in core body temperature on induction of anaesthesia and for the first 1 h, with a slower reduction in core temperature thereafter. The initial change is caused partly by redistribution of heat to the periphery and is variable in size; the subsequent reduction is caused entirely by loss of heat to the surroundings and in several studies has been measured at approximately 0.3 °C h⁻¹ [18, 28, 33, 48, 57, 71, 81]. Estimates of net heat loss during anaesthesia have been remarkably consistent at 42–67 kJ h⁻¹ (10–16 Cal h⁻¹) [18, 27, 69, 71, 72]. Higher rates of heat loss have been observed in the elderly [18].

Local anaesthesia

Hypothalamic thermoregulation remains intact during regional anaesthesia, but abnormal heat
losses produced by vasodilatation, combined with impairment of shivering in the area of the block, and rapid i.v. infusion of cold fluids, may result in core hypothermia. If the patient is not sedated he or she complains of feeling cold; vasoconstriction and shivering occur above the level of the block.

There were no differences in the changes of aural temperature or total body heat when extradural analgesia was compared with four different general anaesthetic techniques during pelvic surgery. However, rewarming during the recovery period was prolonged, presumably because of decreased ability to shiver and continuing increased heat losses [28]. The shivering seen commonly during obstetric extradural anaesthesia may not be thermoregulatory in nature. There is often no sensation of cold in these patients as would be expected during normal thermoregulation. It has been suggested that stimulation of spinal cord thermoreceptors causes the shivering, but in volunteers 80 ml of ice-cold saline injected extradurally produced no effect [56]. It has been proposed also that differential block of warm and cold nerve fibres may be the cause; however, one would again expect a sensation of cold to occur if this were the case. It has been observed recently that women having extradural analgesia in labour have higher vaginal and oral temperatures than those receiving pethidine, and the authors suggested that this may be caused either by decreased sweating in the area of the block or by some effect of the extradural analgesia in increasing heat production [17].

**Thermal stresses**

Cold operating theatres, skin preparation lotions, irrigating fluids, i.v. fluids and dry anaesthetic gases combine with exposure of body viscera to impose large thermal stresses on patients.

**Recovery**

Mild hypothermia (core temperature > 33 °C) during anaesthesia is of little consequence perioperatively [44], but any heat debt incurred must be repaid during the recovery period when thermoregulatory function is restored. Unless active measures are taken to prevent shivering in the recovery period, large increases in oxygen consumption occur and can lead to hypoxaemia [1, 10, 58, 63].

The best method of preventing shivering in the recovery period is to avoid peroperative hypothermia [32]. It has been suggested, on the basis of different electromyographic patterns, that tremor occurring during emergence from anaesthesia is unrelated to thermoregulatory shivering [64]. These authors viewed emergence tremor as the result of disinhibited spinal reflexes. In animals, however, recovery from anaesthesia is associated with a period of hypersensitivity of the hypothalamus to local cooling, so that exaggerated thermoregulatory responses may offer an alternative explanation [21].

The simplest and most effective method of treating post anaesthetic shivering is skin warming with a radiant heater [74]. Methylphenidate and opioids are only partially successful in inhibiting shivering and periods of postoperative paralysis and ventilation are used commonly to allow time for passive rewarming following major surgery and in patients with limited cardiorespiratory function [60, 61].

**MEASUREMENT OF BODY TEMPERATURE**

**Thermometry**

The probes used most commonly are thermocouples or thermistors with an accuracy of ±0.1–0.2 °C, which is more than adequate for clinical use. Liquid-in-glass and liquid crystal thermometers, while sufficiently accurate, are not available in forms which allow core body temperatures to be measured and are of little use in anaesthesia.

Deep body thermometers have recently been described which heat the skin to produce an area of zero thermal flow between skin and subcutaneous tissues and hence obtain an estimate of deep body temperature [16, 77, 79]. Static temperatures measured with deep body thermometers correlate well with other measures of core temperature, but a slow response to thermal transients makes them unsuitable for clinical anaesthesia.

**Sites of measurement**

Body temperature is monitored during anaesthesia to ensure that thermal balance is maintained and to detect the onset of malignant hyperthermia. To document thermal balance adequately, it is necessary to record both core and peripheral temperatures, in order to estimate whole body heat (see below). This is done rarely,
if ever, except in a research setting; usually one of the following measures of core body temperature is used.

**Oesophageal.** The lower 25% of the oesophagus gives a reliable approximation of blood and cerebral temperature, provided that the thoracic cavity is not open. Readings from elsewhere in the oesophagus are influenced by the temperature of the inspired gases. A distance of 24 cm beyond the corniculate cartilages has been recommended as the minimum in adults, and a formula has been derived for use in children [84-86]. Alternatively, positioning at the site of loudest heart sounds, as heard through an oesophageal stethoscope, has been used. This site is not suitable for use in the awake patient.

**Nasopharyngeal.** A temperature probe positioned behind the soft palate gives a less reliable measure of cerebral temperature than a correctly positioned oesophageal probe [87]. Accurate positioning and fixation are difficult and leakage of gases around a tracheal tube may influence the measurements [23].

**Rectal.** Despite having been shown to be inaccurate and misleading, the rectum remains one of the most commonly used sites for measuring core body temperature. Rectal temperature is influenced by heat-producing gut flora, blood returning from the lower limbs and insulation of the probe by faeces. It is commonly 0.5-1 °C higher, and responds to changes more slowly, than other measures of core temperature [2, 12]. Rectal temperature is not suitable for use as a clinical monitor of core body temperature during anaesthesia in adults, especially when early detection of malignant hyperthermia is the primary objective.

**Bladder.** Temperature probes inserted in indwelling urinary catheters have been used to measure core body temperature and, while more accurate than rectal measurements, they are expensive, invasive and are subject to the same slow responsiveness if urine flow is less than 270 ml h⁻¹ [6, 12, 29, 73].

**Tympanic membrane.** The tympanic membrane and aural canal temperatures provide a rapidly responsive and accurate estimate of hypothalamic temperature and correlate well with oesophageal temperature [2, 12]. Damage to the tympanic membrane has been reported following the use of tympanic membrane probes, so the aural canal is now the preferred site. The probes are tolerated well by patients and so provide a means of measuring core temperature before induction of anaesthesia and in the recovery period.

An infra-red tympanic membrane thermometer has been described which eliminates the possibility of tympanic membrane damage and would seem ideal for use when intermittent measurement of core temperature is all that is required [75].

**Blood.** The thermistors of pulmonary artery catheters enable continuous measurements of blood temperature to be made and provide the best estimate available of core body temperature.

**Muscle.** In animal experiments, muscle temperature was found to indicate the onset of malignant hyperthermia before any other core temperature measurement [41]. It is obviously not practical to monitor muscle temperatures in every patient undergoing anaesthesia, and end-tidal carbon dioxide partial pressure may provide a more reliable and earlier warning of the onset of malignant hyperthermia [19].

**Skin.** Measuring the temperature of the skin at a single site gives no information other than the temperature of that area of skin. More specifically, neither mean skin nor body core temperature can be derived from a single measurement of skin temperature. The use of multiple skin sites enables a weighted mean value for skin temperature to be derived and hence a value for total body heat [59].

**CALCULATIONS AND ASSUMPTIONS**

**Mean skin temperature**

The several different schemes for calculating mean skin temperature from individual measurements have been assessed during anaesthesia and in the recovery period [27, 70]. Accurate estimations of mean skin temperature require measurement at 10 or more sites. However, when compared with a 15-site method, the four-site method of Ramanathan was found to give 89% agreement within 1 °C [70] and was later shown to have 95% confidence limits of 0.2 °C at 33 °C [27]. Therefore, if access to measurement sites is limited, the formula of Ramanathan offers an acceptable alternative. There has been no assessment of these formulae during the use of active warming techniques when large variations in regional skin temperatures might be expected, for example, with warming mattresses or radiant heaters.
Mean body temperature
This is generally calculated as:
\[ T_{\text{body}} = (0.66 \times T_{\text{core}}) + (0.34 \times T_{\text{skin}}) \]
where \( T_{\text{body}} \) = mean body temperature; \( T_{\text{core}} \) = core temperature; \( T_{\text{skin}} \) = mean skin temperature. This has been verified for use in hot and neutral, but not in cold environments [11].

Total body heat
The heat content of a body is:
mean temperature \times weight \times specific heat
The specific heat of the human body is taken as 3.475 \( \text{kJ} \cdot \text{°C}^{-1} \) (0.83 \( \text{Cal} \cdot \text{°C}^{-1} \)) and the formula becomes [8]:
\[ \text{Total body heat} = T_{\text{body}} \times \text{weight (kg)} \times 3.475 \text{kJ} \]

Methods of control of body temperature during anaesthesia

Ambient temperature
Morris [45-48] demonstrated that, if the operating theatre temperature was maintained greater than 24 °C, all patients remained normothermic during anaesthesia (oesophageal temperature > 36 °C). At ambient temperatures less than 21 °C all patients became hypothermic while, between 21 and 24 °C, 70% of patients remained normothermic. Several other workers have failed to corroborate these findings [19, 57, 78] and other factors such as humidity and airflow are probably also important.

Humidifiers
The latent heat of vaporization of water is 2.45 \( \text{kJ} \cdot \text{ml}^{-1} \) (585 \( \text{Cal} \cdot \text{ml}^{-1} \)). The administration of dry anaesthetic gases which must be humidified in the lungs results in loss of heat from the body which may be calculated as 6.45 \( \text{kJ} \cdot \text{h}^{-1} \)-litre of minute ventilation (1.54 \( \text{Cal} \cdot \text{h}^{-1} \)-litre of minute ventilation). This loss of heat can be totally prevented by adequate humidification of the inspired gases. Experimentally, use of heated humidifiers to supply inspired gases fully saturated at 37 °C results in a reduction in net heat losses of 58.6 \( \text{kJ} \cdot \text{h}^{-1} \) (14 Cal h\(^{-1}\)) in dogs [68] and 44.38–66.98 \( \text{kJ} \cdot \text{h}^{-1} \) (10.6–16 Cal h\(^{-1}\)) in man [33, 69, 71, 72]. It can be seen from the above that these heat savings are almost sufficient to restore thermal balance during anaesthesia [5, 54]. The most efficient of the heat and moisture exchangers are capable of delivering gases at 27 °C and 85% relative humidity with non-re-breathing systems, and 29 °C and 99% relative humidity with a circle system [83]. This results in savings of 3.22–4.19 \( \text{kJ} \cdot \text{h}^{-1} \)-litre of minute ventilation (0.77–1.0 Cal h\(^{-1}\)-litre of minute ventilation). Circle systems alone deliver inspired gases at about 25 °C and 40% relative humidity and reduce heat loss by about 1.26 \( \text{kJ} \cdot \text{h}^{-1} \)-litre of minute ventilation (0.3 Cal h\(^{-1}\)-litre of minute ventilation). Adequate heated humidification of the inspired gases therefore offers a simple and effective way of restoring thermal balance during anaesthesia.

Warming mattresses and blankets
The most common variety of warming mattress has water circulated through plastic tubing from a thermostatically controlled heater. The hazards involved in the use of electric mattresses preclude their use during anaesthesia.

Warming mattresses have been demonstrated to be ineffective when used alone to counter heat losses during anaesthesia and surgery [39, 47, 78] and there have been several reports of thermal injury resulting from their use [13]. This has resulted in suggestions that their use be discontinued; however, the combination of a warming mattress with a heated humidifier has been shown to be more efficient in preventing heat loss than each technique alone [80]. A similar benefit has been demonstrated using unheated towelling to increase insulation of non-operated parts [71].

Recently, a hot air mattress has been introduced which claims to produce a warm “micro climate” under surgical drapes and has been reported as effective in reducing heat losses in children [22, 53]. It has not been evaluated in the adult surgical patient.

Metallized plastic blankets appear to be of little value in preventing intraoperative heat loss [7, 57].

Radiant heaters
Radiant heaters have been used extensively in the postoperative period to speed rewarming and to suppress shivering. The heat supply from an overhead radiant heater has been estimated as 74 \( \text{kJ} \cdot \text{h}^{-1} \) (17.7 Cal h\(^{-1}\)) [24] and core temperature increased more rapidly with radiant heat than in control groups after cardiac surgery [34–36]. However, the major benefit from radiant heating
is suppression of shivering [43, 50, 74], which results in decreased oxygen uptake, carbon dioxide production and peripheral vasoconstriction [32]. These devices are therefore of value where hypothermia has been allowed to occur and following cardiopulmonary bypass when the after decrease in core temperature is difficult to avoid. There may also be a role for radiant heaters in the anaesthetic room to prevent loss of body heat during the presurgical period.

**Oesophageal rewarmers**

These devices consist of a double-lumen oesophageal tube through which water is circulated at up to 42 °C. They were described originally for use in core rewarming of hypothermic patients [37, 38, 40], but have been used to prevent hypothermia during anaesthesia [39]. From an *in vitro* study the rate of thermal transfer over the temperature range 19–37 °C was 339 kJ h⁻¹ (81 Cal h⁻¹) [38]. In normothermic individuals, the temperature gradient between the rewarming tube and the patient is much less and rates of thermal transfer lower. In postoperative cardiac surgical patients, the rate of rewarming and the incidence of shivering using the oesophageal device were not significantly different from a control group [authors’ unpublished data]. These devices probably have a role in rewarming from profound hypothermia where surface rewarming is inappropriate, but they are expensive and invasive and cannot be recommended for routine peroperative use.

**Intravenous fluids**

The infusion of 1 litre of fluid at 20 °C results in a thermal loss of 71 kJ (17 Cal) as the body warms the fluid to 37 °C. Similarly, 1 litre of blood at 4 °C requires about 125 kJ (30 Cal) for warming. Such losses are avoided easily and are unnecessary.

**CONCLUSION**

Thermal balance during anaesthesia may be restored by minimizing the thermal stresses imposed on the patient. A warm operating theatre, adequate heating and humidification of the inspired gases and warming of i.v. fluids is all that is required in most cases. Post-anesthetic shivering is potentially dangerous, especially in patients with limited cardiorespiratory reserve, and is best treated by radiant heating of the skin.

**REFERENCES**

BODY TEMPERATURE AND ANAESTHESIA

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