BODY TEMPERATURE AND ANAESTHESIA

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"The most effective means (of cooling a man) is to give an anaesthetic"

Pickering (1958)

The body temperature of an anaesthetized patient is measured for two main reasons. First, hypothermia (< 35 °C) commonly occurs in neonates and during prolonged anaesthesia in adults, particularly in those operations involving the body cavities. Secondly, body temperature is used to detect the onset of malignant hyperthermia (MH). It is essential for the anaesthetist to understand the principles of the physiology of temperature regulation if he is to interpret correctly the changes observed.

PHYSIOLOGY OF TEMPERATURE REGULATION

Man is a homeothermic animal and maintains his body temperature within narrow limits, either by increasing heat production or by heat dissipation.

Mechanisms responsible for heat production

Shivering. This is the primary mechanism for heat production and depends on central neuronal coordination (since shivering does not occur below the level of a transection of the spinal cord) and on normal neuromuscular function. Shivering causes a two- to five-fold increase in whole body oxygen consumption.

Non-shivering thermogenesis. The inter-scapular, brown adipose tissue is an important site of heat production in the neonate (Hull, 1966). The metabolism of the brown fat is stimulated by the β-adrenergic effects of noradrenaline. The exact mechanism of the heat production is not known, but it is likely to be either an uncoupling of oxidative phosphorylation of the brown-fat mitochondria in the presence of high concentrations of non-esterified fatty acids, or the stimulation of a lipolysis-lipogenesis cycle with ATP utilization and heat production (Newsholme and Start, 1973).

Vasoconstriction. The response to a cold periphery is vasoconstriction and this acts in support of shivering. However, body conductance (the rate of heat loss/difference between the core and skin temperature) decreases to its lowest value when the core temperature has declined to its normal value. Below this there is no provision for the vasomotor system to decrease heat loss further. It is obvious that some local vasoconstriction can occur, particularly in the periphery, but if this is severe and prolonged it is followed by "cold vasodilatation".

Piloerection. This acts to conserve heat by trapping a still-layer of air close to the surface of the body.

Behavioural thermoregulation. The unpleasant sensation of shivering and its metabolic consequences of an increased oxygen consumption are avoided, if possible, in conscious animals by behavioural responses. The simple response in man is to use warm clothing and move from the cold environment.

Mechanisms responsible for heat dissipation

Sweating. The sweat mechanism is the principal method of heat loss in terms of effectiveness, as the evaporative heat loss may reach eight times the basal heat production. The sweat glands are innervated by cholinergic sympathetic fibres.

Vasodilatation. The importance of vasodilatation is the transfer of heat from the core to the periphery, thus supplying the sweat mechanism with heat for evaporation. The sweating and vasodilator responses move synchronously with an increase in core temperature (Benzinger, 1969). An increased heat loss by convection and radiation is of secondary importance. Vasodilatation involves inhibition of the normal sympathetic vasoconstrictor tone together with active vasodilatation which is cholinergically mediated. It is possible that the active vasodilatation may be secondary to the stimulation of the sweat glands, which is accompanied by the local release of the vasodilator, bradykinin.

Panting. In many animals panting is an important method of evaporative heat loss.

Central temperature regulation

The most common theory of the central control of body temperature assumes that there is a fixed...
“set-point” of body temperature and that any change in core temperature away from this “set-point” initiates either heat producing or heat dissipating mechanisms. Furthermore, it is postulated that this “set-point” may change, for example in exercise or in response to pyrogens (Benzinger, 1969). Hammel and colleagues (1963) have introduced a “variable set-point theory” that permits changes in the core temperature at which thermoregulatory responses are absent.

**Hypothalamic centres.** There are two centres in the hypothalamus concerned with temperature regulation, an anterior, temperature-sensitive area (Aronsohn–Sachs centre) and a posterior, temperature-insensitive area (Krehl–Isenschmidt centre). Cold impulses from the periphery meet in the posterior centre and this area is able to initiate increased heat production by shivering, etc. However, if the core temperature is normal or increased, this is detected by the temperature-sensitive neurones of the anterior centre, which inhibit the posterior centre from stimulating heat production. Thus, the response to a cold stimulus is described as “peripheral cold-stimulation + central warm-inhibition”. The cold receptors in the skin commence firing at a temperature of 33 °C and the frequency increases to a maximum at a skin temperature of 20 °C. It must be emphasized that, under this theory of temperature control, there will be no thermogenesis until the core temperature is below normal, regardless of the skin temperature. Downey, Chiodi and Darling (1967) showed that there was a very weak, central cold reception mechanism by observing a small increase in metabolic rate in paraplegic patients cooled below 35.5 °C.

When a heat load is imposed on the body, the temperature-sensitive neurones of the anterior hypothalamus initiate the sweating and vasodilator mechanisms. These mechanisms are so sensitive that an increase in core temperature of 0.5 °C produces a seven-fold increase in sweat rate and body conductivity (Benzinger, 1969). The sweat rate is directly proportional to the excess temperature above the set-point and shows the characteristics of a proportional control system. A cold periphery can exert some inhibition on the sweat mechanism, although the site of neuronal interaction is not known. The reason for this cold-inhibition is thought to be the prevention of sweating in a cold environment in which evaporative heat loss would be reduced. This anti-homeostatic phenomenon has been used by Hammel and his colleagues (Hammel, 1963; Hammel et al., 1968) to support the “variable set-point hypothesis”. The response to a heat stimulus may be summarized as “central warm stimulation + some peripheral cold-inhibition”.

**Neurotransmitters.** The discovery by Feldberg and Myers (1964) of the ability of 5-hydroxytryptamine (5HT) and noradrenaline (NA) injected into the third ventricle of cats to increase and decrease body temperature, respectively, raised the possibility of producing a temperature-control theory based on individual neurotransmitters. Unfortunately the actions of 5HT and NA were found to show a wide species difference (Feldberg, Hellon and Lotti, 1967) and the presence of cholinergic pathways further complicated the picture (Hall and Myers, 1972). The following simplified explanation is derived from the work of Myers and Yaksh (1969). The temperature-sensitive neurones in the anterior hypothalamus are stimulated by cold to release 5HT, which activates a cholinergic pathway to the posterior heat-production centre. When NA-containing cells are activated by warming, the 5HT–cholinergic pathway is inhibited. This inhibition enables a second cholinergic pathway to activate an efferent heat dissipation system.

Bacterial pyrogens have been used for many years in the study of temperature control. In 1973, Feldberg and Gupta showed that fever caused by bacterial pyrogen was associated with the release of prostaglandin E1 (PGE1) into the cerebro-spinal fluid. The direct injection of PGE1 into the third ventricle (Milton and Wendland, 1971) produced a hyperthermic response, but at a far lower concentration than NA or 5HT. It is conceivable that the role of the catecholamines is one of control of prostaglandin activity rather than a direct effect on the hypothalamic neurones. The inhibition of prostaglandin synthesis by aspirin and paracetamol provides an explanation of their antipyretic action (Vane, 1971).

**SITES FOR TEMPERATURE MEASUREMENT**

Several sites have been advocated for the measurement of body temperature during anaesthesia.

**Oesophagus.** It is essential that the temperature probe is placed in the lower quarter of the oesophagus to avoid the cooling effects of the inspired anaesthetic gases (Whitby and Dunkin, 1969, 1970). Although the lower oesophagus is perhaps the site of choice in the anaesthetized patient, it is not suitable for use in the periods before or after operation.

**Nasopharynx.** Whitby and Dunkin (1971) showed that the nasopharyngeal temperature was less reliable than the lower oesophageal in estimating cerebral temperature. The nasopharyngeal probe was subject
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to inadequate exclusion from the outside air, a leakage of gases from around the endotracheal tube and accidental displacement.

Rectum. Benzinger (1969) observed that the rectal temperature sometimes deviated markedly and paradoxically from cranial temperature when a subject was exposed to a thermal stimulus, and suggested that the use of rectal temperature as an indication of core temperature was a major reason for the lack of progress in the understanding of thermo-regulatory physiology. The measurement of rectal temperature during anaesthesia is of little value and should be undertaken only when access to other sites is not possible.

Tympanic membrane. Tympanic thermometry was widely used by Benzinger (1959) and Benzinger and Taylor (1963) as an indication of cerebral temperature in their studies elucidating the control of body temperature. The validity of this assertion has been questioned (McCook, Peiss and Randall, 1961; Randall et al., 1963), and Nadel and Horvarth (1970) concluded that the temperature of the tympanic membrane was related to the ambient temperature. Webb (1973) found a good correlation between oesophageal and tympanic temperatures during cardiopulmonary bypass, but occasional bleeding from the ear was seen in the heparinized patient. Because of the risk of damage to the tympanic membrane, Keatinge and Sloan (1975) used the anterior wall of the aural canal with a servo-controlled heating device applied to the outer ear to prevent any local cooling. Holdcroft and Hall (1978) found that the aural canal temperature showed a good correlation with oesophageal temperature in the periods before, during and after operation, and was well tolerated by the patients.

Muscle. The primary site of increased heat production in malignant hyperthermia is striated muscle, and in the porcine syndrome the measurement of muscle temperature was found to indicate the onset of the syndrome before any other estimate of core temperature (Lucke, Hall and Lister, 1976).

Skin. Shanks (1975c) examined the many methods available for deriving a mean skin temperature during anaesthesia and concluded that measurements of skin temperature at 10 or more sites was required. If access to a large number of sites was not possible, the four-point method of Ramanathan (1964) was a good approximation.

The measurement of the temperature of the great toe has been advocated as a guide to the degree of peripheral vasodilatation, particularly after cardiac surgery (Matthews, Meade and Evans, 1974a, b; Brock, 1975). It must be emphasized that the temperature of the great toe does not reflect the mean skin temperature, and to suggest that there is an absolute toe temperature which should be achieved by a given time in the period after cardiac surgery is ingenuous (Matthews, Meade and Evans, 1974a).

HEAT LOSS DURING ANAESTHESIA

The possibility of a profound decrease in body temperature during anaesthesia is now widely recognized. The following factors influence the rate at which heat is lost:

Age of patient. The newborn infant is particularly likely to develop hypothermia because of its low basal metabolism, imperfect sweat mechanism and large surface area to body weight ratio (Hey, 1972). Dilworth (1973) has discussed the methods available for the maintenance of body temperature in the neonate.

At the other extreme of age, Goldberg and Roe (1966) observed large decreases in core temperature in the elderly and suggested that this was as a result of decreased heat production.

Theatre environment. Vale (1973) has pointed out that the modern theatre environment with a temperature of 21 °C and a low humidity is for the comfort of the theatre personnel and not in the best physiological interests of the patient. Morris (1971a) concluded that the ambient temperature was the major factor determining body temperature during anaesthesia, and that all patients remained normothermic (>36 °C) at a theatre temperature of 24–26 °C, regardless of the patient's age, type of surgery or anaesthetic agent used. In the study of Holdcroft and Hall (1978), core temperatures of 35 °C were recorded during prolonged pelvic surgery despite the theatre temperature of 24 °C.

Type of operation. Operations involving the pleural cavity are often associated with large heat losses (Dyde and Lunn, 1970). Morris and Wilkie (1970) and Morris (1971b) found no difference between intra-abdominal surgery and surgery not involving body cavities, when assessing the influence of ambient temperature on oesophageal temperature. Catastrophic decreases in body temperature to values <32 °C have been recorded during prolonged vascular surgery (Newman, 1971; Searle, 1971) but Shanks (1975b) concluded that the temperature response in this type of surgery was normal. Vale
high environmental temperature and humidity was not confirmed by the study of Magbagbieola (1973). He examined the effect of premedication with atropine on the rectal temperature of 200 children living in the tropics and observed a mean increase of 0.12 °C (range -0.5 to +1.4 °C).

Anaesthetic agents may interfere with the normal thermoregulatory mechanisms at several sites, both central and peripheral. The most important are the vasodilatation produced by extradural and spinal analgesia and inhalation agents such as halothane, and the abolition of shivering by the use of neuromuscular blocking drugs. There have been few studies comparing the effects of individual anaesthetic agents on body temperature, probably because of the difficulty of standardizing the operating conditions. Engelman and Lockhart (1972) found that, in children, a halothane in nitrous oxide in oxygen anaesthetic produced a greater decrease in rectal temperature than ketamine, although the ambient temperature was not well controlled. Naito and others (1974) showed an increase in rectal temperature in infants and children anaesthetized with diethylether but no significant change in those anaesthetized with halothane for the repair of a hare-lip or cleft palate. Holdcroft and Hall (1978) failed to show any significant difference in heat loss between patients ventilated with nitrous oxide in oxygen and supplemented with 0.5% halothane, or 1% halothane or fentanyl for up to 3 h of anaesthesia.

Infusion of cold i.v. fluids. The administration of i.v. fluids at room temperature causes a reduction in total body heat as such fluids are warmed to body temperature. The infusion of large quantities of cold blood produces a very rapid decrease in oesophageal temperature, and values less than 30 °C have been reported (Boyan and Howland, 1961). When the blood was warmed, however, Boyan and Howland (1963) reported a significant decrease in the incidence of cardiac arrest during massive blood transfusion. The theoretical and practical aspects of blood warmers were reviewed by Russell (1974).

Inhalation of cold, dry anaesthetic gases. Shanks (1974) compared the heat loss in a group of patients ventilated with dry anaesthetic gases with those receiving saturated gases at body temperature. The average hourly heat loss was decreased by 10.9 kcal h⁻¹ (45.6 kJ h⁻¹) in the humidified group.

Heat production in the period after operation

The heat loss that occurs during anaesthesia is regained after operation by a combination of shivering and vasoconstriction. Bay, Nunn and Prys-Roberts (1968) measured the oxygen consumption in patients who shivered after surgery and found increases of 135-468% over basal values. These enormous increases in oxygen consumption impose a further burden on a cardiorespiratory system which may already be depressed after prolonged anaesthesia. If sufficient oxygen is not delivered to the muscle to maintain aerobic metabolism, then anaerobic pathways are activated, with the production of lactate. Thus, the maintenance of normothermia during anaesthesia will prevent an undesirable increase in oxygen consumption after surgery.

Prevention of heat loss during operation

Vale (1973) demonstrated a large decrease in heat loss during anaesthesia by the use of a heat-retaining mattress filled with methyl-cellulose gel. Other investigators have achieved more variable results using warming blankets or heat-retaining mattresses (Lewis and MacKenzie, 1972; Morris and Kumar, 1972; Goudsouzian, Morris and Ryan, 1973) and the majority of patients probably require additional measures. Vale (1972) pointed out that water-circulating mattresses and electric blankets may cause skin burns unless careful attention is directed towards ensuring good contact with the surface of the patient. Shanks (1975a, b) showed that the combination of surface insulation, warmed i.v. fluids and humidification of the inspired gases increased body heat in operations involving the body cavities and even in vascular surgery. Vivori and Bush (1977) claimed that humidification and heating of the anaesthetic gases was not required in children, as the other methods were sufficient.

The use of warmed i.v. fluids, humidification of the inspired gases and a warming blanket in a theatre with an ambient temperature greater than 24 °C should prevent heat loss during anaesthesia.

MALIGNANT HYPERTERMIA (MH)

Body temperature may be monitored during anaesthesia to detect the onset of MH. Keaney and Ellis (1971) attempted to define MH as "... a progressive
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rise in body temperature at a rate of at least 2 °C per hour." This definition fails to indicate that the increase in body temperature is but one sign of the uncontrolled (or malignant) increase in muscle metabolism. Furthermore, it is probably unwise to diagnose MH on temperature alone, since rapid changes in body temperature are not uncommon following induction of anaesthesia. For example, a patient anaesthetized for pelvic surgery with thiopentone, pancuronium, nitrous oxide in oxygen and fentanyl developed an increase in core temperature from 36.0 to 38.2 °C over 15 min, shortly after induction of anaesthesia. The patient cooled slightly to 37.9 °C during the next 60 min and rapidly subsided to 36.0 °C after operation (A. Holdcroft and G. M. Hall, unpublished results). MH was excluded in this patient by the absence of any of the metabolic changes associated with the stimulation of muscle metabolism, such as acidosis, hypercarbia and hyperkalaemia. It is the practice in this centre to diagnose MH only when an increase in body temperature is accompanied by the appropriate changes in acid–base status and plasma potassium concentration.

The value of the routine operative measurement of body temperature to detect the onset of MH is superficially attractive but as yet unproven. In the porcine syndrome, Lucke (1975) found that striated muscle was the best site for temperature monitoring and that changes in core temperatures may be considerably delayed. The earliest metabolic change detected in the porcine syndrome was an increase in the end-tidal Pco₂ concentration (Hall, Lucke and Lister, 1975). If a carbon dioxide analyser is available, then it should be used in suspected cases.

The cause of death in MH may not be the hyperthermia per se. Pettigrew and others (1974) used induced hyperthermia to treat advanced carcinomatosis and showed that patients tolerated a body temperature of 41.8 °C for 4 h, if their fluid and electrolyte balance was maintained. Indeed, in this series there was only one death attributable to hyperthermia at a temperature of 43 °C, and this conforms with the general biological principle that the lethal temperature for an animal is approximately 6 °C greater than its normal body temperature. In MH, some patients die with a body temperature less than 42 °C and it seems likely that the metabolic changes, particularly the acidosis and hyperkalaemia, are responsible. A more detailed consideration of MH is undertaken by Britt (1972) and Relton, Britt and Steward (1973).

REFERENCES


