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Unintentional hypothermia in the operating room

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Hypothermia, defined as a core temperature below 36° C, is an every day occurrence in most operating rooms. Accidental hypothermia is so common that it is treated as an inevitable consequence of surgery and anaesthesia. Its potentially harmful effects tend to be taken lightly by surgeon and anaesthetist alike. However, as the number of high-risk elderly patients is increasing, and longer procedures are being undertaken, this aspect of patient management requires attention. This article will review thermoregulation, physiologic effects of mild to moderate hypothermia (core temperature 30–36° C), temperature monitoring sites, and methods of preventing perioperative hypothermia in the *adult* patient. Discussion will be confined to incidental hypothermia rather than deliberate cooling, as in cardiac surgery.

It has been said that "the best way to cool a man is to give him an anaesthetic."[1] Why is this so? The temperature of the body core is determined by the balance between heat loss and heat gain. Under conditions of anaesthesia and surgery, this balance is heavily tipped in favour of heat loss and all the normal mechanisms of generating and conserving heat are inhibited.

Mechanisms of heat loss

Heat loss occurs in one of four ways: radiation, convection, evaporation and conduction. In order to understand why certain therapeutic measures are more effective than others in maintaining normothermia, it is important to review these mechanisms of heat loss.

Radiation, the transfer of energy between objects via electromagnetic waves, accounts for 40–50 per cent of the body's total heat loss in the operating room.[2] Radiant heat loss is proportional to the temperature difference between the patient and the environment. Peripheral vasodilation induced by anaesthetic agents raises the skin temperature, encouraging a high flow of heat from the body surface to cool OR surfaces.

Convection refers to the direct transfer of energy by collisions between body surface molecules and moving air molecules. Ambient temperature, air velocity, and surface area are the main determinants of convective heat loss. It is markedly reduced by trapping a layer of stagnant air between the skin and the atmosphere, which is the principle underlying layering of clothing for outdoor winter sports. In naked patients lying in air-conditioned operating rooms, with complete air exchange occurring every five minutes or less, 25–35 per cent of the total heat loss occurs via convection.[3]

Evaporative heat loss occurs because the vaporization of water (or volatile skin preparation solutions) demands heat. To vaporize one gram of water requires 0.58 Kcal, which is drawn from the body as the nearest heat source. Evaporation occurs rapidly from prepped areas of the skin, exposed pleura and peritoneum, and from the respiratory tract, which must humidify dry anaesthetic gases.

Conduction, the transfer of heat by direct contact between objects plays a minor role in intraoperative cooling. It becomes critically important, however, in cold water immersion where thermal conductivity is 32 times that of air. In surgical patients, the use of cool intravenous and irrigating solutions and underlying wet sheets, are examples of conductive losses.

Thermoregulation

Temperature homeostasis in mammals, despite intensive research, is not fully understood. Britt recently reviewed this subject in depth.[4] The

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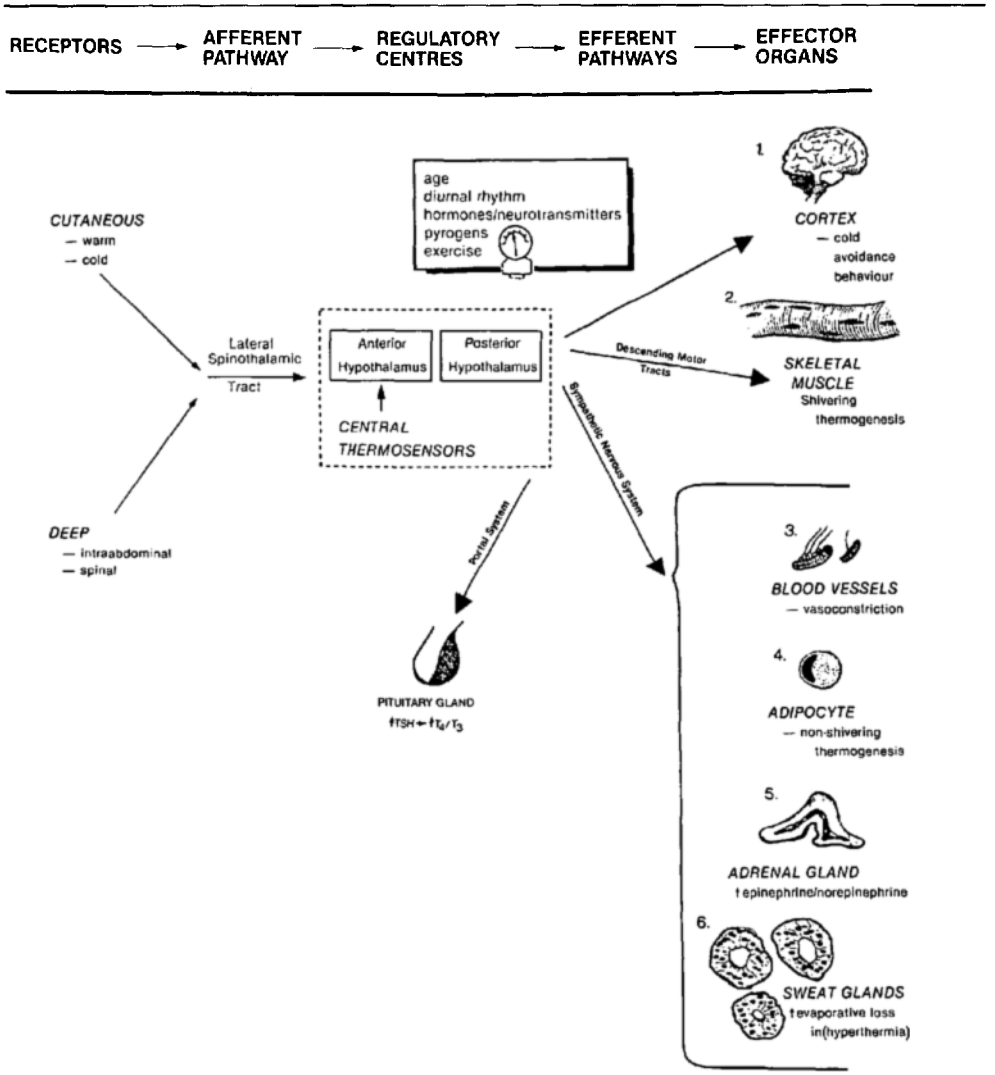

FIGURE Simplified schema of temperature regulation.

following outline, though superficial from a physiologist's point of view, highlights what is relevant to the clinical anaesthetist.

Human thermoregulation is a complex highly sensitive feedback system based on sensing afferents, central integration and set-point, and reflex efferents. The Figure presents a simplified scheme of thermoregulation. Anaesthesia, though it primarily acts on the efferent pathways, can affect all components.

Cutaneous thermoreceptors, most plentiful on exposed areas of the body, are the first to sense an environmental temperature drop. There are two types of receptors, warm-sensitive and cold-sensitive. In response to a temperature above 44° C, the warm sensors increase their firing rate, whereas the

cold sensors are stimulated by temperatures below 24° C. In animals, and probably in man, there are deep temperature sensors in the spinal cord and intra-abdominal viscera. Information from peripheral receptors is relayed via the lateral spinothalmic tract to the anterior hypothalamus. Warm and cold receptors which can respond directly to the blood temperature bathing them are located within the anterior hypothalamus itself.

Whether central or peripheral input plays the dominant role is a matter of continuing debate. Downey *et al.* induced hypothermia in paraplegics with a cooling blanket placed below the level of their lesion, while maintaining warm skin temperatures above the lesion.[5] They found that shivering and an increase in metabolic rate began at a core temperature of 35.5° C despite no sensation of cold from skin receptors. But when the skin above the lesion was cooled, shivering began at a higher core temperature. Numerous experiments have corroborated the interrelationship between superficial and deep receptors.

Together the anterior and posterior hypothalamus make up the temperature-regulating centre. The centre sets a reference temperature which is influenced by diurnal rhythm, age, exercise, pyrogens, hormones, anaesthetic drugs and neurotransmitters. The posterior hypothalamus initiates the appropriate thermoregulatory response when a difference occurs between actual temperature and set-point. The effector responses, controlled either neurally or hormonally, act both to conserve and to generate heat.

Higher cortical centres, acting on information from the hypothalamus can alter behaviour to conserve heat, i.e., seeking out a warm environment, putting on more clothes or increasing voluntary activity. Vasoconstriction, another very effective method of heat conservation, occurs within seconds, mediated by the autonomic nervous system.

Thermogenesis has two components – shivering and non-shivering. Shivering is a centrally-mediated neural response consisting of involuntary rhythmic contractions of skeletal muscle. Since no mechanical work is done during shivering, all the energy released by contractions appears as heat. Shivering is not a function of the sympathetic nervous system; a totally sympathectomized animal is still able to shiver, although catecholamines facilitate shivering. Shivering increases convective and radiative heat loss by increased muscle movement and blood flow and thus is an inefficient long-term response. Horvath showed that in healthy cold-stressed males, shivering contributed only 11 per cent of the heat production required for thermal balance.[6]

Non-shivering thermogenesis refers to an increase in combustion of fatty acids and glucose, both exothermic reactions. In adult man, the most active metabolic areas are liver and skeletal muscle; in the neonate the highly vascular and richly innervated brown fat plays the major role in thermogenesis. On acute cold exposure, norepinephrine is secreted from peripheral nerve terminals to stimulate calorigenesis in fat and muscle cells.

Ephinephrine and thyroxine are important in long-term cold adaptation to enhance the efficiency of norepinephine-induced thermogenesis, but play a minor role in the initial response to cold stress. Non-shivering thermogenesis occurs in thyroidectomized rats but the metabolic increase is smaller.[7]

Effect of anaesthetic drugs on thermoregulation

Anaesthetic drugs can interfere with thermoregulatory control in a number of ways: by blocking afferent input, by lowering the set-point, or by preventing efferent responses either centrally or peripherally. There are profound interspecies differences in thermal responses to drugs, making extension of results to humans questionable. A drug may act at multiple sites, making it difficult to clarify its action. One experimental method of distinguishing between central and peripheral sites of action of a drug is to administer intraventricular or intrahypothalamic injections in amounts too small to have any systemic effect. Narcotics and barbiturates have been the most widely studied anaesthetic agents using this technique.

Intracerebral injection of morphine has been shown to produce hypothermia in the rat primarily by depressing metabolic heat production.[8] This action can be blocked by micro-injection of a narcotic antagonist directly into the hypothalamus. All opiates produce hypothermia at high doses; the lower the environmental temperature the greater the reduction of body temperature.[9]

Barbiturates produce hypothermia by a somewhat different mechanism than morphine. Microinjections of pentobarbitone into the hypothalamus

do not affect core temperature, but systemic administration leads to a rapid fall. Lomax determined that the rate of temperature fall in rats after pentobarbital injection was chiefly governed by the increase in skin blood flow and therefore by radiative loss.[10] Decreased heat production also occurred but was not the major cause of temperature decline. The combination of barbiturates and narcotics produces a greater depression of body temperature than individually.

Halothane, in common with other inhalational agents, promotes surface blood flow by ganglionic blockade, and presumably depresses central thermoregulatory structures non-specifically, although there is no firm evidence for this.

Muscle relaxants abolish shivering. Curarized patients cool more rapidly intraoperatively than non-curarized patients, attesting to the importance of even subclinical shivering in maintaining body temperature.[11]

Epidural or spinal anaesthesia produce a greater temperature drop than general anaesthetics, mainly due to vasodilatation. Peripheral thermal sensibility is blocked by regional techniques, the warm receptors more rapidly than the cold, but a drop in core temperature is still detected appropriately by the anterior hypothalamus. Shivering and vasoconstriction are abolished below the block. A rise in metabolic heat production is indirectly prevented by a reduction in circulating catecholamine output.[12]

In summary, general and regional anaesthetics impair the response to cooling by preventing vasoconstrriction, abolishing shivering, depressing metabolic rate and prohibiting appropriate behavioural responses.

Intraoperative consequences

What are the clinically important physiological effects of the hypothermia that may develop during surgery? *Intraoperatively*, in the paralyzed, ventilated patient, temperatures down to 33° C can be well tolerated with no obvious detrimental effect. As the temperature slips below 33° C, transition to a "danger zone" occurs.

Cardiovascular system

Most critical are the effects on the cardiovascular system. Cardiac output starts to decline at 32° C;[13] this is despite a shift of blood to the central compartment. At 30° C, cardiac output has decreased by 30–40 per cent. Conduction abnormalities may start to appear at 31° C, ventricular arrhythmias at 30° C.[14] The temperature usually cited for the onset of ventricular fibrillation is 28° C, but fibrillation can occur earlier in the presence of diseased or ischaemic myocardium or with stimuli such as CVP line or endotracheal tube insertion.[15] J-waves, pathognomonic of hypothermia, appear on the electrocardiogram at about 30° C, and may herald ventricular arrhythmias.

Respiratory system

There are no clinically significant changes in respiratory mechanics in the temperature range of 30–36° C, in the ventilated patient. Severinghaus showed that dead space increased by bronchodilatation in dogs, but Nunn concluded that in humans this does not happen.[16] Respiratory drive does not cease until 24° C.

Hypoxic pulmonary vasoconstriction is impaired by even mild hypothermia. Benumof and Wahrenbrock determined in dogs that the hypoxic response at 31° C was less than one half that at 40° C.[17]

Inhalational agents become more soluble with decreasing temperature, and also more potent. The increase in solubility slows the rate of rise of anaesthetic partial pressure in the alveoli, slowing induction. Opposing this is the fact that hypothermia reduces MAC. The reduction per degree change in body temperature varies from agent to agent. For example, halothane requirement falls by five per cent with each degree centigrade, cyclopropane by only two per cent per degree.[18] The net effect of these opposing actions is that MAC is achieved at roughly the same rate at all temperatures.[19]

Similarly, carbon dioxide and oxygen increase in solubility with hypothermia, lowering PCO_2 and PO_2 and raising pH. Blood gas tensions and pH value are measured in the laboratory at a temperature of 37° C and traditionally corrected with nomograms to determine PCO_2 and pH at the patient's temperature. This assumes that the ideal pH at all temperatures is 7.42. However, Rahn *et al.* hypothesized from both laboratory work and observations on hibernating animals, that the rising pH and dropping PCO_2 which occur as body temperature is lowered maintains a constant ionic charge on proteins.[20] This is the critical parameter which must be defended to optimize enzyme function, not pH.

Ream *et al.* argue that the best method of managing the acid–base status of hypothermic patients is to use uncorrected values.[21] For example, an arterial blood specimen drawn at 32° C, which at that temperature has a pH of 7.47 and a PCO_2 of 4.3 kPa (32 mmHg) will, with rewarming, yield a pH of 7.42 and a PCO_2 of 5.3 kPa (40 mmHg). Thus no ventilatory adjustment would be required. Minute ventilation may have to be reduced to compensate for the fall in CO_2 production occurring in hypothermia.

The considerations for oxygen administration during hypothermia are affected by the haemoglobin dissociation curve. The curve shifts to the left with cooling, primarily due to an increased affinity of haemoglobin for oxygen. However, haemoglobin saturation remains constant with decreasing temperature, despite the change in oxygen solubility which occurs. As blood cools the oxygen solubility increases, dropping its partial pressure. The correction factor for PO_2 depends on the level of haemoglobin saturation. If saturation is below 90 per cent, PO_2 falls by 7.2 per cent with each degree centigrade drop, but if PO_2 is above 500 mmHg the reduction is only 1.3 per cent per °C. In the practical range of saturation (90–100 per cent) the correction factor is complex and must be derived from a nomogram or equation.[22] For calculation of alveolar–arterial gradients, temperature-corrected blood gas values must be used.

Central nervous system

In the central nervous system there is a seven per cent reduction in cerebral blood flow per degree Centigrade drop in temperature. This is due to a combination of factors – decreased cardiac ouput, increased viscosity and increased cerebrovascular resistance. There is a corresponding reduction in cerebral metabolic rate so that demand does not outstrip supply.

During experimental hypoxia, low-grade hypothermia (34° C) seems to exert a cerebral protective effect. At this temperature, the accumulation of lactic acid, a suspected mediator of hypoxic neuronal damage is reduced compared to normothermia.[23] Below 34° C, however, the potential cardiovascular complications outweigh the possible protective benefit to patients with marginal cerebral oxygenation.

At 33° C, impaired cerebration begins to develop in the conscious patient. At 30° C, loss of consciousness and pupillary dilatation supervene.[24]

Metabolic/endocrine system

Metabolic changes include a decline in basal metabolic rate of the order of five to seven per cent per °C. At 32° C, hyperglycaemia can occur.[25] This is thought to be due to decreased insulin release and impaired peripheral utilization of glucose. Administered insulin has little effect and, in any case, the condition reverts on rewarming. In the awake human the initial sympathetic response to surface cooling is elevation of plasma catecholamines. However, in deeply anaesthetized animals, this catecholamine response is minimal.[27]

Musculoskeletal system

Prolonged hypothermia augments neuromuscular blockade by two mechanisms. During a rapid drop in temperature, the neuromuscular junction may be initially resistant to non-depolarizing relaxants because of an increase in the amount of acetylcholine released in response to a nerve impulse. However, hypothermia reduces acetylcholine mobilization into readily available stores so that after 20 minutes they become depleted, augmenting neuromuscular blockade. Miller and Roderick found that the serum clearance rate of curare and pancuronium decreases with decreasing temperature.[28] This is probably the major reason that hypothermia enhances the potency of these drugs. The breakdown of atracurium is temperature-dependent, such that a reduction in temperature from 37° C to 26° C increases the half-life of atracurium two-fold.[29]

Renal haematologic/gastrointestinal systems

Glomerular filtration rate progressively declines with cooling to reach 50 per cent of normal at 30° C. However, urine flow is not reduced until about 20° C because of early depression of tubular reabsorption, and may even be higher than normal. The increase in blood viscosity is usually not clinically important unless temperature falls below 30° C. Platelet count falls at temperatures below 32° C, possibly due to platelet sequestration in the portal circulation, but normalizes with rewarming.[30] Intestinal motility decreases below 34° C, with ileus occurring frequently.

Postoperative consequences

It is in the postoperative period that the consequences of even a mild heat debt become most apparent. There are three major complications to be aware of (1) shivering, (2) peripheral vasoconstriction and, (3) delayed drug clearance.

Shivering can produce up to a 400–500 per cent increase in metabolic rate within skeletal muscles.[31] The increased oxygen consumption and carbon dioxide production raise ventilatory requirements at a time when gas exchange may be impaired by residual drugs, partial upper airway obstruction, pain or intrinsic lung disease. Blood flow to the muscles involved increases dramatically, demanding a higher cardiac output. In the elderly patient, or one with coronary artery disease, attempts to increase cardiac output to the required level may result in myocardial ischemia. Failure of the cardiorespiratory system to meet the metabolic demands of shivering has been shown to lead to reduced mixed venous saturation and, in the presence of shunting, arterial hypoxaemia.[32] There may be a shift to anaerobic metabolism with lactic acidosis resulting.

Another outcome of intraoperative hypothermia, which only becomes a problem postoperatively, is peripheral vasoconstriction. Vasoconstriction may be a cause of unexplained hypertension in the recovery room. It can also mask hypovolemia so that as the patient gradually warms and vasodilates there is a sudden drop in blood pressure.

Delayed drug clearance can have particular significance in the elderly who already suffer from inefficient clearing mechanisms. The maximum rate of renal excretion of a drug can decline by 10 per cent/0.6° C fall in body temperature.[33] Hepatic detoxifying processes are significantly delayed with even a 3–4° C drop, which particularly affects narcotics[34] and barbiturates.[35] The effect of hypothermia on actions of non-depolarizing relaxants has been discussed above.

Temperature monitoring sites

The temperature effects cited above all refer to true core temperatures. The body has been described as a "thermal onion" with layers of temperature gradients surrounding a thermal core. Therefore, the anaesthetist's choice of temperature monitoring site is important. A skin temperature of 32° C is of little concern, an oesophageal temperature of 32° C is a major concern.

Axillary

The most commonly used axillary probe normally reads 0.5° C below oral, or 1° C below rectal temperature. It is the best monitor of muscle temperature and therefore the first to rise with malignant hyperthermia, but it is a poor measure of core temperature. It is useful for signalling trends, carries no risk, and at $0.17/patient is a bargain.

Nasopharyngeal

A temperature probe in the nasopharynx positioned posterior to the soft palate provides an estimate of hypothalamic temperaature. It can be affected by leakage of air around the endotracheal tube cuff.[36] There is a possibility of epistaxis, even with careful insertion and a theoretical risk of cros-contamination unless the probes are properly sterilized.

Oesophageal

An oesophageal temperature probe gives a good approximation of myocardial temperature. Whitby and Dunkin found that it must be accurately positioned in the lower one-quarter of the oesophagus to avoid being influenced by the temperature of inspired gases.[37] An even more dependable site for myocardial temperature is the pulmonary artery, if a Swan-Ganz catheter is in place.

Rectal

Rectal probes have been traditionally used as a measure of core temperature despite several disadvantages. During cardiac bypass rewarming, rectal temperature has been shown to correlate poorly with tympanic and myocardial temperatures.[38] Rectal temperatures can be affected by heat-producing organisms in the bowel, cool blood returning from the legs, as well as the obvious problem of insulation by faeces. Benzinger states that if the rectal temperature deviates from the oesophageal or tympanic measurement it is the rectal observations that are in error.[39] More accurate, though more expensive, is the use of a thermocouple probe incorporated into a Foley catheter for bladder temperatures.

Tympanic

Tympanic membrane (or aural canal) thermistors,

closely approximate hypothalamic temperatures[40] and are tolerated by conscious patients, making this mode useful for postoperative or intensive care monitoring. However, their readings can be rendered inaccurate by cerumen and the canal or membrane can be traumatized. With the availability of new sensors embedded in cotton, this possibility is slight, but the ear canal should be examined by otosocope before insertion to ensure the patency of the tympanic membrane.

Skin

The use of skin probes, either liquid crystal strips or flat metal discs has drawbacks. Their accuracy is questionable and, even if they are accurate, temperature at only one skin site correlates poorly with core temperature.[41] Monitoring a large number of skin sites to determine mean skin temperature is generally of interest only to the researcher. Of clinical interest, however, is the correlation between cardiac output in patients in shock and great toe temperature observed by Joly and Weil.[42] This simple method of assessing tissue perfusion is an adjunct to more sophisticated measures.

In summary, the best single monitor of core temperature for hypothermic patients is a correctly situated oesophageal probe. Many other sites can be chosen depending on availability and cost, but their limitations should be borne in mind.

Prevention

As anaesthetists it is our responsibility to minimize the temperature drops which accompany surgery. There is no simple solution. Identification of patients at risk is the first priority. The Table lists the surgical and patient conditions which alert the anaesthetist to the possibility of this complication. Patients for major vascular procedures are at high risk not only because of the duration of surgery, blood loss, and exposure of peritoneum but because use of the aortic clamp allows the distal vessel bed to approach thermal equilibrium with the atmosphere. Sickle cell disease, digital reimplantation, and cold agglutinins are mentioned, not because patients with these factors are particularly susceptible to cooling, but because the consequences of even mild hypothermia are significant in these patients. Vasoconstriction and increased viscosity from hypothermia can lead to stasis in peripheral vascular beds, thereby promoting sickling. Vascular reanastomis may be unsuccessful in the presence of vasospasm in a cool digit. Some cold agglutinins are triggered by temperatures of 32° C, a temperature frequently reached in the peripheral tissues of anaesthetized patients.

TABLE Risk factors for intra-operative hypothermia

Surgical factors
Duration > 3 hours
Major body cavities opened
Major blood loss
Major vascular surgery
Patient factors
Age – 65–80 moderate risk
– > 80 high risk
Burns
Paraplegia
Trauma victims post-resuscitation
Ethanol intoxication
Exfoliative dermatits
Myxoedema
Adrenal insufficiency
Paget's Disease
Sickle cell disease
Digital reimplantations
Cold agglutinins

Surprising temperature drops can occur prior to the patient's arrival in the operating theatre, due to their lack of clothing, the vasodilating effects of the pre-medication, and the long wait in the chilly corridor.[43] To avoid this, keep patients well covered during their journey to the operating room and ensure that they are placed in a warm holding area.

Prophylaxis should be started early, because the greatest heat loss takes place in the first hour due to exposure, prepping, and sudden vasodilatation on induction. Morris found that in operating rooms at temperatures less than 21° C, mean body temperature fell within the first hour of anaesthesia by 1.3° C, during the second hour by 0.3° C, and by only 0.1° C in the third hour.[44]

Intraoperative methods of minimizing heat loss will be discussed in decreasing order of effectiveness. Most effective, without doubt, is reducing radiant heat loss by raising ambient temperature.

Ambient temperature

Morris determined that there was a "critical ambient temperature" of 21° C for adults, regardless of whether surgery involved major body cavities or

not.[45] Patients in rooms colder than 21° C invariably become hypothermic, while those in rooms above this critical temperature usually maintained oesophageal temperatures over 36° C after two hours of surgery. Unfortunately, this is not a comfortable thermal environment for surgeons. Since the greatest temperature fall occurs in the first hour, a compromise arrangement whereby the room temperature could be maintained at 22–24° C until the patient is draped seems sensible. This type of management depends on the theatre having an individually controlled and rapidly responsive thermostat. The simple expedient of covering as much body area as possible will diminish convective and radiative heat loss. Mobile overhead radiant warmers would be useful during insertion of invasive monitoring lines and prepping.

Humidification

Humidification of anaesthetic gases will reduce hourly heat loss by about 10–15 per cent or 9–10 Kcal·hr^{-1}.[46] A heat loss of 60–70 Kcal will drop the core temperature of the average size adult by 1° C. Humidification can be accomplished via heat–moisture exchangers or electrically heated humidifiers.

The most efficient heat–moisture exchangers provide an absolute humidity of 28–32 mgH_2O·L^{-1} at 6 L·min^{-1} flow.[47] These light-weight devices are easily inserted into either a Bain circuit or circle system and will virtually eliminate heat loss due to vaporization from the respiratory tract. However, they can collect mucous and will develop increased resistance from moisture-trapping over the course of a long case.

The electrically heated humidifiers can transport heat directly to major bronchi and mediastinal vessels. There is some debate over the significance of this pathway. Theoretical calculations of the amount of heat possible to conduct via the airway are disappointingly low because of the low specific heat of air.[48] Nevertheless, heated humidifiers have been proven, in clinical practice, to be beneficial.[49] Stone *et al.*, by warming and fully humidifying gases to 37° C, maintained nasopharyngeal temperatures in adults undergoing major procedures significantly above those in a control group.[50] Caldwell, Crawford and Sinclair found that hypothermia could be prevented post cardiopulmonary bypass by a heated humidifier in circuit.[51]

Tracheal oedema ("hot pot tracheitis") has been reported from the inadvertent administration of gases at 43° C.[52] Therefore, inspired temperature must be monitored. An essential safety feature is a thermostat which cuts off current when the output vapour temperature exceeds 41° C. Other problems with the use of electrically heated humidifiers are "rain-out" in the circuit, bacterial contamination and possible electrical hazards.

Blood and fluid warmers

Two units of blood infused at 4° C can lower core temperature by 0.5° C. Rapid infusion of ten units has resulted in cardiac arrest from hypothermia. Blood can be warmed prior to transfusion by a hot water bath or by an electro-magnetic oscillatory device. With the latter method, haemolysis can occur in the presence of inadequate mixing, or with unit volumes less than 250 ml.

Russell, in 1974, extensively reviewed blood warmers on the market with respect to both efficacy and safety.[53] He recommended that the ideal warmer should be able to provide blood at a temperature greater than 32° C at flow rates above 150 ml·min^{-1}. At that time, the Fenwal unit was the only one of 11 tested, to meet his criteria. More recently, Cherry *et al.* found the Gorman-Rupp DW 1000 warmer to compare favourably with the Fenwal.[54]

As Russell pointed out, the other advantages of warming blood, besides avoidance of hypothermia, are a two and one half fold reduction in viscosity of the administered blood, and venodilatation. Disadvantages are haemolysis if temperature exceeds 45° C, and risk of bacterial contamination if the unit is allowed to infuse too slowly.

Warming crystalloid solutions can be recommended as risk-free; the calculated heat gain of warming one litre of lactated Ringer's solution from room temperature to 37° C is 16 Kcal. Therefore, the heat loss resulting from the infusion of six to eight litres of crystalloid during a lengthy case could drop the patient's temperature by 2° C.

Warming blankets

Conductive heat loss to the underlying mattress can be reduced by a thermal mattress heated by water circulating around a closed circuit at temperatures of up to 41° C. Morris concluded that a warming blanket was ineffective in preventing hypothermia in

adults anaesthetized in "cold" operating rooms (mean temperature 19.7°C). In procedures lasting up to three hours, there was no difference in oesophageal temperatures between patients on warming blankets and control patients.[55] He suggested the reasons for this were (1) the actual surface area of the body exposed to the mattress was less than one-third of the total (2) sheets were interposed for safety and (3) peripheral vasoconstriction reduced the warming blanket's effectiveness as a heat exchanger. Its maximum temperature of 41°C only provides a small gradient for heat transfer. Since the minimum temperature is 4°C, it is a far more useful means of cooling than warming. Interestingly, the combination of a heated humidifier and a warming mattress is more effective than either blanket or humidifier alone.[56] A plausible explanation is that the skin is less vasoconstricted due to conservation of body heat with humidification and thus more heat exchange with the mattress can occur.

It must be appreciated when using this device that third degree thermal burns have been reported, particularly over bony prominences in obese and diabetic patients.[57] To avoid this serious complication, Vale advocated a heat-retaining methylcellulose gel mattress which has the high thermal capacity of water, yet cannot be overheated.[58]

Reflective blankets

Studies of the effectiveness of reflecting blankets (metallized plastic sheeting) have produced conflicting results. Bourke *et al.* found significant, though small, differences in core temperatures between test and control patients when using the aluminized Tyvek 1443 blanket.[3] They recommended using a reflective blanket when more than 60 per cent of the body surface area could be covered and when the procedure would last more than two hours. Radford and Thurlow found the "Space Blanket" manufactured by Thermos was unable to prevent core hypothermia during neurosurgery.[59] Condensing of perspiration over a long case will reduce the infra-red reflectiveness. This may account for the differing conclusions of the two studies, since the Tyvek 1443 has perforations to avoid moisture trapping whereas the Space Blanket does not. Other authors have found that clear plastic sheeting is just as effective as metallized sheeting in providing thermal protection.[60]

Anaesthetic technique

There is little in the literature comparing the effects of individual anaesthetic agents on body temperature, presumably because of the difficulty in standardizing operating conditions. Holdcroft and Hall discovered no difference among three anaesthetic techniques in core temperatures after three hours of anaesthesia, (1) 0.5 per cent halothane (2) one per cent halothane (3) fentanyl with a background of nitrous oxide and pancuronium.[61] Engelman and Lockhart comparing the rectal temperatures in children during halothane or ketamine anaesthesia found that halothane was associated with a greater fall in rectal temperature.[62]

Core rewarming

A theoretical risk of rapid external rewarming is that cold acidotic blood may be shunted centrally, precipitating arrhythmias and further reducing core temperature. Experience with victims of environmental exposure suggests that active core rewarming is safer than external rewarming if core temperature is less than 32°C.[63] Therefore, if the patient's temperature has dropped below this level intraoperatively it would seem prudent to institute some method of core rewarming. Techniques that can be used in the general surgical suite are nasogastric, peritoneal or thoracic instillation of body temperature saline, depending on which site is most accessible.

Postoperative management

The postoperative management of hypothermia patients is dictated by their core temperature and their risk category.

If the temperature is 33–36°C, a healthy patient can be extubated, but consideration should be given to electively ventilating the elderly or those with severe cardiac or respiratory problems. Measures are required in the recovery room both to minimize ongoing heat loss and to provide external heat. Further heat loss can be prevented by covering the patient as completely as possible, changing underlying wet sheets, and warming infusions. An overhead radiant warmer is a very effective and comfortable way of external heating. If this is not available, a warming blanket with a single sheet interposed, placed over top of the patient rather than underneath, is useful.

The three major postoperative consequences of

temperatures of 33–36° C are shivering peripheral vasoconstriction and delayed drug clearance. The shivering patient requires an optimum airway and oxygen administration. The vasoconstrictor response may result in haemodynamic instability as blood volume shifts gradually from the central compartment to the periphery with rewarming. The delayed drug clearance and potentiation of muscle relaxants will have a greater impact on the elderly.

If the temperature is below 33° C arrhythmias, reduction of cardiac output, and central nervous system impairment may be added to the aforementioned problems. The patient should be electively ventilated with warmed humidified gases and have ECG monitoring until the temperature reaches at least 35° C. External rewarming measures as described above should be instituted as well.

Conclusion

In conclusion, three basic recommendations can be made:

1 Core temperature should be monitored, preferably oesophageally, in patients at risk for hypothermia.
2 Conserving and warming measures should be instituted early since the greatest drop in temperature occurs within the first hour of the case.
3 No patient, particularly over age 65, should be discharged from recovery room with a core temperature below 36° C. Increased oxygen consumption will continue either by shivering or non-shivering thermogenesis until the heat debt has been restored.

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