Temperature monitoring: the consequences and prevention of mild perioperative hypothermia

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Introduction

Homeothermic species require a nearly constant internal body temperature. Significant deviations from "normal" internal temperature cause the metabolic function to deteriorate. Usually, the human thermoregulatory system maintains a core body temperature within 0.2°C of normal, near 37°C. Hypothermia results from exposure to cold, or exposure combined with drugs or illness that decrease thermoregulatory efficacy. Exposure to a cold operating room environment during anaesthesia and surgery commonly combines with anaesthetic-induced inhibition of thermoregulation to produce hypothermia. The prevention and management of temperature-related complications is expedited by an understanding of both normal and drug-influenced thermoregulation.

Normal thermoregulation

Thermoregulation, like many physiological control systems, relies on multiple levels of positive and negative feedback to minimise perturbations from the normal status. Temperature is regulated by signals derived from nearly every type of tissue, including the hypothalamus, spinal cord, deep core tissue and skin surface. Cold signals travel to the hypothalamus and other central structures primarily via Aδ nerve fibres, while warm information travels by unmyelinated C fibres. Most ascending thermal information traverses the spinothalamic tract in the anterior spinal cord. The processing of thermoregulatory information occurs in three phases, namely afferent thermal sensing, central regulation and efferent response.

The hypothalamus regulates temperature by comparing integrated thermal input from the skin surface, neuraxis and deep tissue with threshold temperatures for heat and cold. When integrated input exceeds a threshold, the appropriate response is initiated to maintain adequate body temperature. The slope of response intensity versus core temperature regression is the gain of that response. The difference between the lowest warm and highest cold thresholds indicates the thermal sensitivity of the system. Typically, the interthreshold range, i.e. the temperature range over which no regulatory responses occur, is only a few tenths of a degree Celsius. Because each thermoregulatory response has its own threshold and gain, there is an orderly progression of responses and response intensities are in proportion to need.

How the body determines absolute threshold temperatures is unknown, but the thresholds vary daily in both sexes by ≈ 1°C (circadian rhythm), and monthly in women by ≈ 0.5°C. Exercise, food intake, infection, hypo- and hyperthyroidism, anaesthetic and other drugs (including alcohol, sedatives and and nicotine), and cold and warm adaptation alter threshold temperatures. Central regulation is intact from infancy, but may be impaired in elderly or extremely ill patients.

The hypothalamus responds to temperatures exceeding the appropriate thresholds via response mechanisms that increase metabolic heat production or alter environmental heat loss. These responses allow normal individuals to maintain a core temperature near 37°C, despite widely varying environmental temperatures. As thermoregulatory responses are inhibited (by drugs), the range of environmental temperatures over which normal core temperature can be maintained decreases. For example, when shivering is prevented following the administration of muscle relaxants, hypothermia will develop in an environment that was well tolerated previously. When all thermoregulatory responses

are prevented, the core temperature will remain normal only in a thermoneutral environment.

Quantitatively, behavioural regulation (e.g. dressing appropriately, modifying environmental temperature and voluntary movement) is the most important effector mechanism.

Cutaneous vasoconstriction decreases heat loss via convection and radiation from the skin surface. Total digital skin blood flow is divided into capillary and thermoregulatory arteriovenous shunt components. The arteriovenous shunts are largely limited anatomically to the fingers, toes and nose. They are functionally distinct from capillaries supplying the remainder of the skin. Shunts are open and capillary flow is nearly minimal in a thermoneutral environment. Shunt vasoconstriction is mediated primarily by the release of norepinephrine from presynaptic adrenergic nerve terminals. Vasoconstriction only minimally decreases capillary blood flow.

Nonshivering thermogenesis increases metabolic heat production without producing mechanical work via brown fat oxidation. Nonshivering thermogenesis increases heat production ≈ 100% in infants, but only slightly in adults.

Vigorous shivering roughly doubles metabolic heat production, although this level of intensity cannot be sustained for long. The net efficiency of shivering thermogenesis is somewhat lower than might be expected because muscle metabolism increases blood flow to peripheral tissue, and consequently, heat loss to the environment. Thus, it is used only as a last defense against hypothermia, i.e. its activation threshold is a full degree Celsius below that with vasoconstriction and nonshivering thermogenesis.

Sweating is mediated by cholinergic, post-ganglionic sympathetic nerves. Trained athletes can sweat up to 2 l/hour and lose as much as 10 times their resting metabolic rates. Sweating is the only mechanism by which humans can lose heat in an environment that exceeds core body temperature. Active vasodilation is mediated by the release of a yet-to-be fully-characterised mediator (possibly nitric oxide) from the sweat glands. Heat stress can increase capillary blood flow up to 7.5 l/minute (Table I).

Table I: Thermoregulatory system in humans

Afferent sensing	Skin surface
	Deep tissues
	Spinal cord
	Brain, especially the hypothalamus
Central control	Spinal cord
	Hypothalamus
Efferent defenses	Behavioural
	Sweating and active pre-capillary vasodilation
	Arterioshunt vasoconstriction
	Shivering

Intraoperative temperature monitoring

Input to the central thermoregulatory system is derived largely from deep abdominal and thoracic tissues, the spinal cord and the brain. Thus, no single tissue temperature can be considered to be a "gold standard." Core temperature can be determined by measuring a single temperature that is adjacent to the tympanic membrane, or in the nasopharynx, pulmonary artery or distal oesophagus. Carefully obtained oral, axillary and bladder temperatures approximate core temperatures sufficiently for clinical use, except during cardiopulmonary bypass.

Skin temperatures are approximately 2°C less than core temperature, but the difference between forehead skin and core temperature varies among individuals and within individuals over time. Forehead skin temperature, with a 2°-C compensation is thus only a rough estimate of core temperature. Infrared "tympanic" thermometers and "temporal artery" thermometers are insufficiently accurate for clinical use. Rectal temperature often seriously lags true core temperature, and should thus be avoided.

Usually, body temperature measurements are not necessary during monitored anaesthesia care or minor procedures that are performed under regional anaesthesia. Major surgery, accomplished by epidural or spinal anaesthesia, can be associated with considerable hypothermia. Core temperature monitoring is generally appropriate in such cases. Core temperature alterations immediately following induction of general anaesthesia are hard to predict,

Table II: Temperature monitoring

True core	Pulmonary artery
	Nasopharynx
	Distal oesophagus
	Tympanic membrane, with a thermocouple probe
Generally adequate	Oral
	Axillary
	Bladder
	Zero heat flux
Often inaccurate	Skin surface, including the forehead
	Infrared aural canal "tympanic"
	Infrared temporal artery
	Rectal

and are influenced by a variety of factors. Consequently, temperature monitoring is often not helpful during the first 30 minutes of anaesthesia, and is not required when surgery is completed within this period. By contrast, core temperature should be monitored at no less than 15-minute intervals during general anaesthetics that last longer than 60 minutes.

An oesophageal probe is usually the easiest and most reliable method of reliably measuring core temperature in intubated patients. Axillary, oral or forehead skin surface temperatures can be substituted for oesophageal or nasopharyngeal temperatures during regional anaesthesia, or when patients are mask ventilated. Of these, oral temperature is the most reliable and is also the most suitable for postoperative use (Table II).

Thermoregulation during anaesthesia

Typical doses of general anaesthesia decrease the activation thresholds for responses to hypothermia by ≈ 3°C and increase those defending against hyperthermia by < 1°C. Widening the interthreshold range produces a broad temperature range over which active thermoregulatory responses are absent. Patients are poikilothermic within this range and body temperature changes determined passively by redistribution of heat within the body and the difference between metabolic heat production and heat loss to the environment.

Most general anaesthetics produce a similar pattern and magnitude of thermoregulatory impairment. This pattern is illustrated in Figure 1 using propofol. Opioids inhibit thermoregulatory control similarly. By contrast, even very high plasma concentrations of midazolam have little thermoregulatory effect. The vasoconstriction threshold in infants who are anaesthetised with halothane or isoflurane

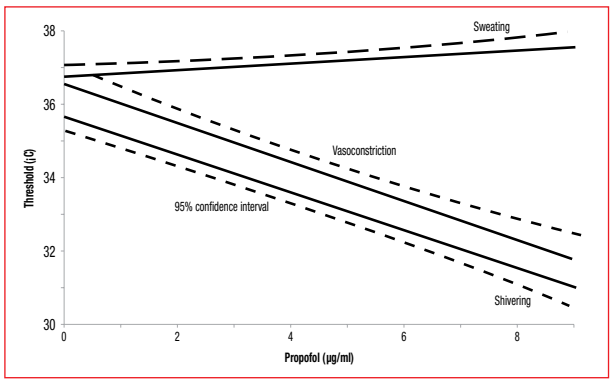

Figure 1: Linear reduction of vasoconstriction and shivering thresholds with propofol
Solid lines show concentration-response regressions. Dotted lines indicate 95% confidence intervals for the thresholds. Propofol administration linearly, but slightly, increased the sweating threshold. Conversely, propofol markedly decreased the vasoconstriction and shivering thresholds. Thermoregulatory responses are similar in magnitude during administration of other general anaesthetics, although volatile anaesthetics non-linearly decrease cold-response thresholds, with a disproportionately greater effect at high concentrations. [Matsukawa T, Kurz A, Sessler DI, et al. Propofol linearly reduces the vasoconstriction and shivering thresholds. Anesthesiology. 1995;82(5):1169-1180]

differs little to that in adults. Intraoperative vasoconstriction thresholds are increased by painful stimulation, but decreased by advanced age.

Perioperative hypothermia

The typical pattern of intraoperative hypothermia is illustrated in Figure 2. Core temperature decreases precipitously for one hour (redistribution hypothermia), then decreases slowly for 23 hours (a linear decrease), and finally becomes constant (plateau phase).

Hypothermia during the first hour of anaesthesia results largely from the core-to-peripheral redistribution of body heat. Subsequently, core and mean body temperatures decrease when heat loss exceeds heat production. And finally, a core temperature plateau results when the re-emergence of thermoregulatory vasoconstriction decreases cutaneous heat loss and constrains metabolic heat to the core thermal compartment

Numerous factors contribute to core hypothermia during the first hour of surgery:

- Patients are undressed in a cool environment.
- Their skin is prepared for surgery with a cold solution which is allowed to evaporate.
- The induction of anaesthesia decreases metabolic heat production and produces cutaneous vasodilation.
- Cold intravenous fluids directly decrease the core temperature.
- The patients' lungs are ventilated with dry gases which increase respiratory heat loss.

Nonetheless, unanaesthetised individuals easily tolerate typical operating room temperatures without becoming hypothermic, and the evaporation of skin preparation fluids cannot explain the observed hypothermia. Furthermore, general anaesthesia decreases metabolic heat production by only 15-30%, and minimally increases cutaneous heat loss.

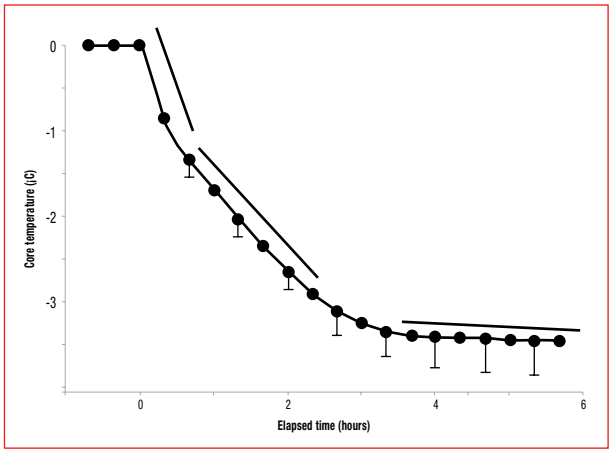

Figure 2: Typical intraoperative hypothermia pattern

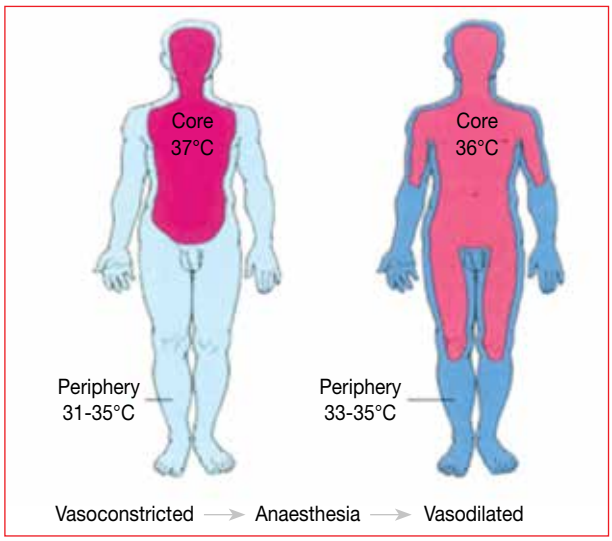

Figure 3: Peripheral warming at the expense of core temperature

Since decreased heat production and increased heat loss are insufficient to explain the initial core hypothermia following the induction of general anaesthesia, another mechanism must be invoked. Core temperature poorly represents mean body temperature and body heat content because the temperature in peripheral tissue (roughly half the body mass) changes considerably depending on thermoregulatory status, environmental temperature and duration in a particular environment. Typically, peripheral tissue temperature (and heat content) is considerably lower than core temperature. Although anaesthetic-induced vasodilation only minimally increases cutaneous heat loss to the environment, it allows the mixing of heat in the core and peripheral thermal compartments. The result is peripheral warming at the expense of core temperature (Figure 3). Redistribution hypothermia contributes ≈ 80% to observed hypothermia during the first hour of anaesthesia, and often remains the major cause of core hypothermia, even after three hours of anaesthesia. The similar internal redistribution of heat causes the initial hypothermia during epidural anaesthesia.

The initial hypothermia which develops following the induction of anaesthesia results when vasodilation allows heat in warm core tissue to mix with cooler peripheral tissue. This warms the periphery at the expense of the core temperature. Although the core temperature decreases precipitously, body heat content (and mean body temperature) remains nearly constant

It is likely that the slow, linear decrease in core temperature that is typically observed during the second and third hours of anaesthesia simply results from heat loss that exceeds metabolic heat production. Heat loss presumably also exceeds production during the first hour of anaesthesia, but usually contributes much less to core hypothermia than redistribution.

When patients are in a relatively warm environment and undergo small operations, the core temperature plateau that develops after the second to fourth hour of anaesthesia may be passive, i.e. without thermoregulation. By contrast, when patients are sufficiently hypothermic, this plateau is often accompanied by active thermoregulatory vasoconstriction. Peripheral vasoconstriction decreases cutaneous heat loss and constrains metabolic heat (which is mostly generated centrally) to the relatively small core thermal compartment, thereby preventing further hypothermia, and sometimes even increasing core temperature. Heat loss from peripheral tissue not only continues, but this tissue receives less metabolic heat. The core temperature plateau does not represent a true thermal steady state (heat production equalling heat loss) in these circumstances because body heat content continues to decrease (Table III).

Table III: Intraoperative hypothermia

Initial hour	Mostly core-to-peripheral redistribution of body heat
Subsequent 2-4 hours	Heat loss exceeding heat production: • Radiation • Convection
Core temperature plateau	• A passive equilibrium in a warm environment • *The re-emergence of thermoregulatory vasoconstriction:* Typically at a core temperature near 34.5°C

The consequences of hypothermia

Temperatures only 23°C below normal provide significant protection against cerebral ischaemia and hypoxaemia in animals. Thus, mild hypothermia may be useful during procedures that are likely to cause ischaemia, such as carotid endarterectomy and neurosurgery. However, it is important to recognise that mild perioperative hypothermia has yet to be shown to improve outcome, despite large studies which have evaluated cerebral aneurism surgery, brain trauma and acute myocardial infarction. Currently, the only well documented benefits of therapeutic mild hypothermia (≈ 33-34°C) is improved neurological function after out-of-hospital cardiac arrest and in asphyxiated newborns. And at least in susceptible swine, mild hypothermia both slows triggering of malignant hyperthermia and reduces the severity of the syndrome once triggered. By contrast, mild perioperative hypothermia (i.e. 2°C) causes many complications, including morbid myocardial events, coagulapathy, surgical site infection, reduced drug metabolism, thermal discomfort and shivering.

Postoperative myocardial infarction is perhaps the most common serious perioperative complication that occurs in approximately 8% of moderate- to high-risk patients. Three quarters of postoperative myocardial infarctions are

clinically silent. However, both silent and clinically apparent postoperative infarctions have the same mortality of roughly 10%. Approximately 80% occur within two postoperative days. Mild perioperative hypothermia increases the risk of morbid myocardial events, especially infarction. The increase appears to be mediated by autonomical activation, with consequent tachycardia and hypertension.

Just 2°-C core hypothermia significantly increases the incidence of myocardial ischaemia in high-risk patients undergoing peripheral vascular surgery. Myocardial ischaemia probably results less from post-anaesthetic shivering than from sympathetic activation, hypertension and tachycardia.

Mild hypothermia reduces platelet function and decreases activation of the coagulation cascade. Consistent with these in vitro data, hypothermia significantly increases blood loss and allogeneic transfusion requirements during elective primary hip arthroplasty. Blood loss increases roughly 0.5 units for each degree Celsius decrease in core temperature.

Mild hypothermia contributes to a common and serious complication of anaesthesia and surgery and wound infection by directly impairing immune function, especially oxidative killing by neutrophils, and decreasing cutaneous blood flow which reduces tissue oxygen delivery. Together, these effects of mild hypothermia triple the clinical incidence of surgical wound infections, and substantially increase the duration of hospitalisation in patients undergoing elective colon resection. Hypothermia-induced protein wasting and decreased collagen synthesis is also likely to impair wound healing.

Mild hypothermia decreases the metabolism of most drugs. For example, the duration of action of vecuronium is more than doubled at 34.5°C, compared with 36.5°C. The effect of hypothermia on metabolism of atracurium and propofol is somewhat less, but is still clinically important. Consistent with decreased drug metabolism and exaggerated drug effect, mild hypothermia significantly prolongs the duration of postoperative recovery, even when temperature is not a discharge criterion.

Postoperative thermal discomfort *per se* is not life-threatening. Nonetheless, patients who become hypothermic often remember the worst aspect of surgery as feeling cold postoperatively. Efforts to prevent and treat such unpleasant sensations deserve the same attention as those that are currently given to pain management.

Shivering-like tremor occurs in ≈ 40% of unwarmed patients recovering from general anaesthesia, but is now rare. Shivering is usually preceded by core hypothermia and peripheral vasoconstriction, indicating that it is thermoregulatory. Perianaesthetic tremor increases oxygen consumption

Table IV: The conseqeunces of perioperative hypothermia

Major	Morbid myocardial events
	Coagulopathy and increased transfusion requirement
	Surgical site infection
Minor	Reduced drug metabolism
	Prolonged recovery
	Shivering, tachycardia and hypertension
	Thermal discomfort

≈ 200% and may exacerbate postoperative pain. Although most of the tremor has electromyographic patterns that characterise normal shivering, some resemble pathological clonus. The tremor can be treated by skin-surface warming, clonidine (75 µg intravenously), or meperidine administration (25 mg intravenously). Meperidine is more effective than equipotent doses of other opioids, possibly because the drug is a central alpha-receptor agonist, in addition to its primary action at µ-opioid receptors (Table IV).

Tremor during epidural anaesthesia is normal thermoregulatory shivering triggered by core hypothermia, and preceded by peripheral vasoconstriction above the level of sympathetic blockade. It can be treated by skin surface warming above the level of blockade, intravenous or epidural meperidine (25 mg) administration, or epidural sufentanil administration (50 µg).

The prevention and treatment of hypothermia

Initial rapid core hypothermia during epidural or general anaesthesia is difficult to treat because it results largely from the internal redistribution of heat. However, it can be largely prevented by warming the skin and peripheral tissue before induction to decrease the core peripheral temperature gradient. Given the large heat capacity of the peripheral thermal compartment, generally 30-60 minutes of moderate warming is required.

Less than 10% of metabolic heat is lost via respiration, even when patients are ventilated with dry, cool gas. Passive airway humidification (heat and moisture exchangers) can prevent most of this loss, and active gas heating and humidification prevents it all. However, simple thermodynamic calculations indicate that airway heating and humidification in adults cannot produce clinically significant alterations in body heat content. Technically adequate clinical studies find that inspired gas conditioning fails to prevent hypothermia in adults. (It is likely that the clinical impression that airway heating is effective results from the artifactual heating of proximal oesophageal thermometers.) Consequently, active airway heating and humidification is rarely indicated. Similarly, heat and moisture-exchanging filters contribute little to the maintenance of normothermia.

Since most heat is lost through the skin; although evaporation from large surgical incisions may contribute significantly, preventing cutaneous heat loss minimises the slow, linear decline in core temperature during surgery. Cutaneous heat loss can be passively decreased by covering the skin with a cloth or paper surgical drapes, blankets or plastic bags. A single layer of each insulator reduces heat loss by ≈ 30%, but the efficacy of different cover types is similar because most insulation is provided by the layer of still air trapped under the cover. Thus, adding additional layers does not proportionately increase the benefit. Combining three layers of warmed cotton blankets only reduces heat loss by 50%. Because cutaneous heat loss is roughly proportional to the surface area, the choice of covering material is far less important than the total surface area covered.

Active warming can prevent most cutaneous heat loss, and can even eliminate the loss of heat across the skin surface. Circulating water blankets are more effective when placed over, rather than under, patients because little heat is lost through the 5 cm of foam that pads most operating room tables. Similarly, newer circulating water wraparound garments are quite effective, although expensive. Water temperature should not exceed 40°C to minimise the risk of pressure or heat necrosis, and should be set at even lower temperatures in patients with arterial vascular insufficiency. Recently developed systems which appear to be effective include over- or under-body resistive heating and circulating water applied to extremities. Forced air is by far the best validated warming approach and offers a good combination of safety, efficacy, ease of use and price (Figure 4).

Surgical Care Improvement Project and Physicians Quality Reporting Initiative

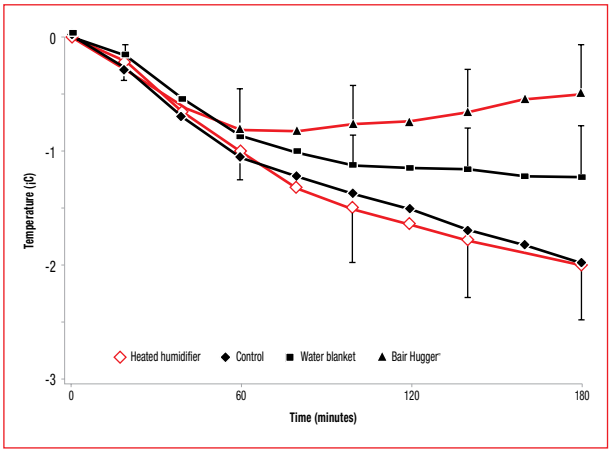

Figure 4: Comparison of three intraoperative warming therapies
Tympanic membrane temperature decreased uniformly during the first hour after induction of general anaesthesia in each group. Temperature then increased over the remaining two hours in patients randomised to forced air warming (Bair Hugger®), but remained nearly constant in those lying on a circulating water blanket set at 40°C. Patients in the control and heated humidifier groups continued to become more hypothermic throughout the operation. Uniform initial hypothermia during the first hour of anaesthesia results from the internal redistribution of heat and illustrates the difficulty in treating this temperature decrease. [Hynson J, Sessler DI. Intraoperative warming therapies: a comparison of three devices. J Clin Anesth. 1992;4(3):194-119]

The Surgical Care Improvement Project (SCIP) and the Physicians Quality Reporting Initiative (PQRI) are separate measures. However, they were designed in concert and are harmonised. Thus, they each have identical requirements. SCIP is a hospital-based reporting requirement. By contrast, PQRI applies to individuals, and is linked to a 2% Medicare bonus for reporting. Presumably, the bonus will soon be provided for meeting requirements, and not just reporting effort. The American Society of Anesthesiologists (ASA) strongly supported the inclusion of thermal management in PQRI because it is the only measure that is specific to operating room anaesthesia. Without it, anaesthesiologists would have been excluded from the 2% Medicare bonus. (I was an ASA delegate to the PQRI committee.)

The denominator for SCIP and PQRI includes patients who are having surgical procedures with general or neuraxial anaesthesia that lasts at least 60 minutes in which the need for intentional hypothermia is not documented. Use of cardiopulmonary bypass is considered to be *de facto* evidence of intentional hypothermia. Thus, patients having monitored anaesthesia care, peripheral nerve blocks alone and short procedures are excluded. The numerator includes both a process and intermediate outcome component. The outcome component is met when patients have a documented body temperature ≥ 36°C within 30 minutes before the end of anaesthesia, and/or within 15 minutes thereafter. There is no requirement to use a specific type of thermometer or to measure temperature at a particular site. However, it is likely that accurate thermometers and a reliable core temperature site should provide higher values. The process component is met by the use of active over-body warming, such as forced air (Table V).

Table V: Surgical Care Improvement Project and Physicians Quality Reporting Initiative

Numerator	• Body temperature ≥ 36°C within last 30 minutes of anaesthesia • Body temperature ≥ 36°C within 15 after anaesthesia • Use of active over-body warming
Denominator	• Surgical procedure with general or neuraxial anaesthesia • Lasting 60 minutes • *Intentional hypothermia not documented:* Cardiopulmonary bypass considered to be intentional

Recommendations

Body temperature is normally tightly regulated. However, general anaesthetics profoundly impair normal thermoregulatory control, with the consequence that unwarmed surgical patients almost inevitably become 1-3°C hypothermic. Large randomised trials have demonstrated that even mild perioperative hypothermia causes numerous severe complications, including morbid myocardial outcomes, increased blood loss and transfusion requirements, prolonged recovery and hospitalization, and surgical wound infections. Because thermal disturbances are associated with severe consequences, now the standard of care is to monitor temperature during general anaesthesia, and to maintain normothermia, unless otherwise specifically indicated. Table VI lists the most important learning points for this review.

Table VI: Major learning points

Thermoregulation	*Afferent sensing:* Skin, deep tissue, the spinal cord and the brain *Central regulation:* Brain, especially the hypothalamus Behavioral and autonomic defenses, sweating, vasoconstriction, and shivering
Temperature monitoring	*Core:* Pulmonary artery, nasopharynx, oesophagus and tympanic *General adequate:* Oral, axillary and bladder
Intraoperative hypothermia	Core-to-peripheral redistribution Heat loss exceeding production Passive or active core temperature plateau
Maintaining normothermia	Forced air is a good combination of safety, efficacy and low cost
SCIP and PQRI	*Numerator:* Normothermia or active over-body warming *Denominator:* Surgery > 60 minutes with general or neuraxial anaesthesia

PQRI: Physicians Quality Reporting Initiative, SCIP: Surgical Care Improvement Project

Bibliography

1. Sessler DI. Temperature monitoring and perioperative thermoregulation. Anesthesiology. 2008;109(2):318-338.
2. Sessler DI. Perioperative heat balance. Anesthesiology. 2000;92(2):578-596.
3. Sessler DI. Complications and treatment of mild hypothermia. Anesthesiology. 2001;95(2):531-543.
4. Frank SM, Fleisher LA, Breslow MJ, et al. Perioperative maintenance of normothermia reduces the incidence of morbid cardiac events: a randomized clinical trial. JAMA. 1997;277(14):1127-1134.
5. Rajagopalan S, Mascha E, Na J, Sessler DI. The effects of mild perioperative hypothermia on blood loss and transfusion requirement: a meta-analysis. Anesthesiology. 2008;108(1):71-77.
6. Kurz A, Sessler DI, Lenhardt RA. Study of wound infections and temperature group: perioperative normothermia to reduce the incidence of surgical-wound infection and shorten hospitalization. N Engl J Med. 1996;334(19):1209-1215.