Perioperative complications of hypothermia

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Perioperative hypothermia is a common and serious complication of anesthesia and surgery and is associated with many adverse perioperative outcomes. It prolongs the duration of action of inhaled and intravenous anesthetics as well as the duration of action of neuromuscular drugs. Mild core hypothermia increases thermal discomfort, and is associated with delayed post anaesthetic recovery. Mild hypothermia significantly increases perioperative blood loss and augments allogeneic transfusion requirement. Only 1.9°C core hypothermia triples the incidence of surgical wound infection following colon resection and increases the duration of hospitalization by 20%. Hypothermia adversely affects antibody- and cell-mediated immune defences, as well as the oxygen availability in the peripheral wound tissues. Furthermore mild hypothermia triples the incidence of postoperative adverse myocardial events. Thus, even mild hypothermia contributes significantly to patient care costs and needs to be avoided.

Keywords: anaesthesia; hypothermia; complication; perioperative; temperature; thermoregulation.

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The combination of anaesthetic-induced impairment of thermoregulatory control and exposure to a cool operating room environment makes most surgical patients hypothermic. Several prospective, randomized trials have demonstrated a number of hypothermia-induced complications (Table 1). Methods are now available for preventing inadvertent perioperative hypothermia. Consequently, it is now routine to maintain intraoperative normothermia unless therapeutic hypothermia is specifically indicated.

There is no widely accepted definition for the term “mild hypothermia.” Furthermore, the term is not even used consistently within the literature. For the purpose of this review, mild hypothermia will refer to core temperatures between 34 and 36 °C.

**IMPAIRED PHARMACODYNAMICS**

Most enzymes commonly used in the body are highly temperature sensitive; this is also the case for select enzymes which regulate drug metabolism are highly temperature sensitive. The effects of temperature on various muscle relaxants, volatile and intravenous drugs have been investigated and have been shown to have a large effect on their actions.
<table>
<thead>
<tr>
<th>Consequence</th>
<th>Author</th>
<th>N</th>
<th>VTcore (°C)</th>
<th>Normothermic</th>
<th>Hypothermic</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Morbid cardiac events</td>
<td>Frank, et al.</td>
<td>300</td>
<td>1.3</td>
<td>1.4%</td>
<td>6.3%</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Postoperative ventricular tachycardia</td>
<td>Frank, et al.</td>
<td>74</td>
<td>1.5</td>
<td>330 ± 30 pg/ml</td>
<td>480 ± 70 pg/ml</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Adrenergic activation</td>
<td>Kurz et al.</td>
<td>200</td>
<td>1.9</td>
<td>6%</td>
<td>19%</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Surgical wound infection</td>
<td>Kurz et al.</td>
<td>200</td>
<td>1.9</td>
<td>12.1 ± 4.4 days</td>
<td>14.7 ± 6.5 days</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Duration of hospitalization</td>
<td>Beilin, et al.</td>
<td>60</td>
<td>1.0</td>
<td>4800 CPM</td>
<td>2750 CPM</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Lymphocyte proliferation at 24 h postanaesthesia</td>
<td>Schmied, et al.</td>
<td>60</td>
<td>1.6</td>
<td>1 unit</td>
<td>8 units</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Allogeneic transfusion requirement</td>
<td>Schmied, et al.</td>
<td>60</td>
<td>1.6</td>
<td>1.7 ± 0.3 L</td>
<td>2.2 ± 0.5 L</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Intraoperative blood loss</td>
<td>Winkler, et al.</td>
<td>150</td>
<td>0.4</td>
<td>488 ml</td>
<td>618 ml</td>
<td>&lt;0.005</td>
</tr>
<tr>
<td>Intraoperative blood loss</td>
<td>Widman, et al.</td>
<td>46</td>
<td>0.5</td>
<td>516 ± 272 ml</td>
<td>702 ± 344 ml</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Intraoperative blood loss</td>
<td>Johansson, et al.</td>
<td>50</td>
<td>0.8</td>
<td>665 ± 292 ml</td>
<td>698 ± 314 ml</td>
<td>NS</td>
</tr>
<tr>
<td>Urinary excretion of nitrogen</td>
<td>Carli, et al.</td>
<td>12</td>
<td>1.5</td>
<td>982 mmol/day</td>
<td>1798 mmol/day</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Trauma mortality at 24 h</td>
<td>Gentilello, et al.</td>
<td>57</td>
<td>1.0–2.0</td>
<td>7%</td>
<td>43%</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Duration of vecuronium</td>
<td>Heier, et al.</td>
<td>20</td>
<td>2.0</td>
<td>28 ± 4 min</td>
<td>62 ± 8 min</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Keo for vecuronium</td>
<td>Caldwell, et al.</td>
<td>12</td>
<td>2.0</td>
<td>0.20 min⁻¹</td>
<td>0.15 min⁻¹</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Duration of atracurium</td>
<td>Leslie, et al.</td>
<td>6</td>
<td>3.0</td>
<td>44 ± 4 min</td>
<td>68 ± 7 min</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Postoperative shivering</td>
<td>Just, et al.</td>
<td>14</td>
<td>2.3</td>
<td>141 ± 9 ml min⁻¹ m⁻²</td>
<td>269 ± 60 ml min⁻¹ m⁻²</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Duration of postanaesthetic recovery</td>
<td>Lenhardt, et al.</td>
<td>150</td>
<td>1.9</td>
<td>53 ± 36 min</td>
<td>94 ± 65 min</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Thermal discomfort</td>
<td>Kurz, et al.</td>
<td>74</td>
<td>2.6</td>
<td>50 ± 10 mmVAS 18 ± 9 mmVAS</td>
<td>&lt;0.001</td>
<td></td>
</tr>
</tbody>
</table>

Only prospective, randomized human trials are included; subjective responses were evaluated by observers blinded to treatment group and core temperature. “N” = total number of subjects. VTcore = difference in core temperature between the treatment groups. Different outcomes of the studies by Tanaka, et al. (Tanaka, 2001 #4336), Kurz, et al., and Schmied, et al. are shown on separate rows. Baroflex sensitivity is defined as the change in the R-R interval of the ECG (ms, milliseconds) per 1 mmHg change in systolic blood pressure (Tanaka, 2001 #4336). CPM stands for “counts per minute” and measures radioactivity (after addition of titrated thymidine and cell activation, the amount of radioactivity is proportional to the number of dividing cells). VAS is a 100-mm-long visual analogue scale (0 mm = intense cold, 100 mm = intense heat).
Intravenous anaesthetics

Propofol, a commonly used anaesthetic is paradoxically affected by temperature; a 3 °C decrease in core temperature results in an approximate 30% increase in plasma concentration of propofol. Interestingly, mild hypothermia does not alter hepatic blood flow, or propofol requirement during craniotomy surgery. It is postulated that the increase in propofol plasma concentration is due to a reduced inter-compartmental clearances between the central and shallow compartments. Fentanyl has also been shown to have a relationship with hypothermia; a 5%/°C increase in steady-state plasma concentration of fentanyl can be observed.

Muscle relaxants

Skeletal muscle displays a slight temperature sensitivity while muscle relaxants are markedly affected by temperature. In patients with mild hypothermia (−2 °C) the duration of action of vecuronium was more than doubled. The keo decreased (0.023 min⁻¹ per °C) with lower temperatures, suggesting slightly delayed equilibration of drug between the circulation and the neuromuscular junction during hypothermia. The onset of vecuronium will be significantly delayed if the movement of the drug between the circulation and the neuromuscular junction and its recovery may be also prolonged. Interestingly, when neostigmine is used and as antagonist of vecuronium it does not appear to be altered by mild hypothermia.

In contrast to vecuronium, atracurium does not appear to be as sensitive to temperature. Although the duration of muscle relaxation increases by approximately 60% when core temperature decreases by 3 °C. The recovery index (time for 25–75 percent twitch recovery) for both atracurium and vecuronium is similar during normothermia and hypothermia. As expected the duration of action of rocuronium is prolonged during hypothermic bypass.

Volatile anaesthetics

Anaesthesia potency is driven by the steady-state plasma partial pressure opposed to the actual anesthetic concentration in the cells. Hypothermia increases the solubility of volatile anesthetics but does not appear to alter the potency. An unsupported theory is that hypothermic patients may take longer to recovery from anesthesia because of larger amounts of anesthetic that need to be exhaled.

In the rat, hypothermia does affect the minimum alveolar concentration (MAC) of two anesthetics: halothane and isoflurane. Where MAC is the partial pressure of anesthesia needed to prevent a reaction due to surgical stimulus, the MAC of halothane and isoflurane decreases by approximately 5%/°C as core temperature decreases in the rat. In children it has been demonstrated that a 5.1% decrease in isoflurane MAC is observed for every 1 °C reduction in core temperature.

BLOOD LOSS AND TRANSFUSION

The effect of hypothermia on coagulation

A clinical bleeding diathesis is associated with both deliberate and inadvertent hypothermia. Surgeons have long suspected that hypothermia produces a coagulopathy
and increases perioperative blood loss. Three general mechanisms contribute to temperature-related coagulation disorders: platelet function, clotting factor enzyme function, and fibrinolytic activity.

Platelets

Platelet count remains normal during mild hypothermia. Nevertheless, hypothermia has been shown to induce morphologic changes in the platelet structure suggestive of activation. Recently, Faraday, et al. found that moderate and profound degrees of in vitro hypothermia enhanced the binding of platelets to fibrinogen through activation of GPIIb-IIa receptors. These results are in direct contrast to the conclusions based on non-specific measures of platelet function (e.g., bleeding time and shed blood thromboxane release), but are in agreement with other findings describing an activating effect of hypothermia on platelets. These findings suggest that inhibition of intrinsic platelet function is not the cause of this coagulopathy. In addition, these observations support the hypothesis that hypothermia results in coagulopathy by reducing the availability of platelet activators. This hypothesis is supported by the following observations: (a) The generation of thrombin, a potent platelet agonist, decreases under hypothermic conditions, and (b) hypothermia results in the release of a circulating anticoagulant with heparin-like effects. Kestin, et al. similarly concluded that prolongation of the bleeding time in patients after cardiopulmonary bypass is likely the result of a lack of available platelet activators rather than an intrinsic platelet defect. The suggestion that hypothermia might promote pathologic platelet aggregation in the presence of platelet activators may have clinical relevance under conditions in which hypothermic blood is exposed to platelet activators such as extracorporeal circuits or atherosclerotic plaque.

Clotting factors

An insidious feature of hypothermic coagulopathy is that standard coagulation tests, including the prothrombin time and the partial thromboplastin times remain normal. The reason is that the tests are normally performed at 37 °C — no matter what the patient’s temperature might be. These same tests, though, are prolonged by hypothermia when they are performed at the patient’s actual core temperature. However, the clinical importance of this prolongation remains debatable. Hypothermia has been shown to prolong activated partial thromboplastin time by 10% compared with test temperatures at 37 °C and both hypo- and hyperthermia increased the response to heparin in vitro. Recently, Shimokawa, et al. have demonstrated a hypothermia-induced delay in the coagulation cascade and reduced platelet function in vivo, whereas the conventional coagulation tests performed at normothermia failed to detect these impairments in haemostasis performance. The isolated effect of hypothermia on hemostasis has been also investigated in healthy humans: reduction of body core temperature to 32 °C was associated with a small decrease in the prothrombin time and platelet count, whereas coagulation variables at 34 °C did not differ from normothermia.

Fibrinolysis

The fibrinolytic system normally regulates the balance between formation of haemostatic plugs and restoration of blood flow after clot formation. Fibrin is a major
structural element in formed clots, but is subject to degradation by plasmin. Plasmin is the activated enzymatic form of plasminogen. The conversion of plasminogen to plasmin is, therefore, the core of the fibrinolytic mechanism. This reaction is enhanced by two types of plasminogen activators, but tissue-type plasminogen activator is the most important. Inadequate fibrinolysis predisposes patients to thrombosis, while excessive fibrinolysis predisposes them to haemorrhage. Multivariate analysis of 232 adult trauma patients demonstrated a significant hypercoagulability associated with enzyme activity slowing and decreased platelet function at core temperatures \( \leq 34 \, ^\circ\text{C} \), whereas fibrinolysis was not significantly affected at any of the measured temperatures.\(^{20}\) Preliminary data suggests that fibrinolysis remain normal during mild hypothermia, but is significantly increased during hyperthermia, suggesting that hypothermia-induced coagulopathy does not result from excessive clot lysis. The corresponding effects of thermal disturbances on plasminogen activator have yet to be determined, but thromboelastogram data suggests that hypothermia impairs clot formation, rather than facilitating clot degeneration.\(^{21}\) Recently, Swawn, et al. reported that the thrombolytic action of tissue plasminogen activator (tPA) is also reduced as a function of temperature, with \( \sim 1\% \) loss of the clot mass for each \( 1 \, ^\circ\text{C} \) increase at a temperature range of \( 30–40 \, ^\circ\text{C} \).

**Clinical outcome**

As demonstrated in a study by Schmied, et al. mild hypothermia increases blood loss. In this study, patients were randomly assigned to normothermia or mild hypothermia during elective primary hip arthroplasty. A reduction of just \( 1.6 \, ^\circ\text{C} \) in core hypothermia temperature increased blood loss by \( 500 \, \text{ml} \) (30\%) and significantly augmented allogeneic transfusion requirement. The same group subsequently confirmed the haemostatic benefits of maintaining intraoperative normothermia in a retrospective analysis. In contrast, another study of blood loss during hip arthroplasty failed to identify a temperature-dependence to blood loss.\(^{22}\) Why the results should differ so much in similar — and apparently well-conducted — studies remains unclear. Possibilities include differences in surgical technique or even in the populations that were evaluated. Recently, two more randomized controlled trials have confirmed that only \( \approx 0.5 \, ^\circ\text{C} \) core hypothermia increases blood loss by \( 200–300 \, \text{ml} \) in patients undergoing hip arthroplasty under spinal anaesthesia.\(^{23}\)

Recently, evidence indicates that blood transfusions may be more toxic than previously believed. Numerous studies have found a relationship between hypothermia, blood loss and transfusion requirements. In 2008, Rajagopalan et al. conducted a comprehensive meta-analysis of randomized, controlled trials that compared normothermic patients with those who experienced mild hypothermia (\( 34–36 \, ^\circ\text{C} \)). A total of 14 studies were used in Ranjagopalan’s analysis of blood loss, and 10 studies were included in the analysis of transfusion requirements. In this study, it was argued that mild hypothermia increases blood loss by approximately 16\%.\(^{24}\) Although there is not a linear relationship between blood loss and transfusion requirements, increased blood loss surely increases transfusion requirements. In the meta-analysis by Rajagopalan it was found that mild hypothermia significantly increases the relative risk for transfusions by approximately 22\%.\(^{24}\) As recent evidence\(^ {25} \) has shown, blood transfusions more toxic than previously assumed, thus, a decreased risk of transfusion is very clinically relevant.
ADVERSE MYOCARDIAL EVENTS

Myocardial infarction is one of the leading causes of unanticipated perioperative morbidity and mortality. In a landmark study using prospective randomized data, Frank, et al. demonstrated that patients assigned to only 1.4°C core hypothermia were three times as likely to experience adverse myocardial outcomes.26

The mechanism by which mild hypothermia triggers myocardial events remains unclear, although shivering is clearly not the primary mechanism. Hypothermia causes hypertension and tachycardia in elderly patients and those at high risk for cardiac complication. In this regard, they differ from young, healthy subjects who demonstrate virtually no hemodynamic response to mild hypothermia.27 Cold-induced hypertension in the elderly is associated with a three-fold increase in plasma norepinephrine concentrations28, which is at least likely to augment cardiac irritability and facilitate development of ventricular arrhythmias.

It has long been suggested that patients with cardiopulmonary disease may not tolerate the increased metabolic demands associated with post-operative shivering and may have adverse outcomes. However, post-operatively, oxygen consumption (i.e., metabolic rate) rarely increases even by a factor of three, and then only during extreme circumstances.29–31 Because advanced age32 and opioid administration33 are associated with reduced shivering, it is likely that the average elderly patient who is undergoing surgery and receives adequate analgesia experiences a relatively small increase in metabolism due to post-operative shivering.

A temperature threshold of approximately 1°C below normothermia activates sympatheural (noradrenaline), as well as adrenomedullary (adrenaline) responses in awake healthy volunteers during cold exposure.34 The cardiovascular physiology associated with these reactions involves an increase in the various indices of cardiac work.34 In addition, studies demonstrate that this β-adrenoceptor-mediated increase in the myocardial work in normal, mildly hypothermic volunteers is linked to an increase in myocardial perfusion.35 Thus, mild core hypothermia does not evoke coronary vasoconstriction in healthy human subjects, and actually increases myocardial tissue perfusion, in a manner that matches the increase in the heart rate-systolic pressure product, an index of myocardial oxygen consumption.35 However, even in the absence of vasoconstriction, increased myocardial metabolic requirements in the presence of flow-limiting coronary lesions may predispose patients to myocardial ischemia.

Strict thermoregulation attenuates myocardial injury during coronary artery graft surgery as reflected by reduced levels of cardiac specific troponin.36

WOUND INFECTION AND HEALING

Wound infections are common and serious complications of anesthesia and surgery. For example, the wound infection risk in patients undergoing colon surgery is approximately 10%.37,38 Surgical wound infections prolong hospitalization by 5 to 20 days per infection, and substantially increase costs.37

Hypothermia may facilitate perioperative wound infections in two ways. First, sufficient intraoperative hypothermia triggers thermoregulatory vasoconstriction.39 Thermoregulatory vasoconstriction significantly decreases subcutaneous oxygen tension in humans40 and the incidence of wound infections correlates with subcutaneous oxygen tension.38
Second, considerable evidence indicates that mild core hypothermia directly impairs immune function including T-cell-mediated antibody production and “non-specific” oxidative bacterial killing by neutrophils. Thus, hypothermia may directly impair neutrophil function, or impair it indirectly by triggering subcutaneous vasoconstriction and thereby producing tissue hypoxia.

Vasoconstriction-induced tissue hypoxia may also impair wound healing. Scar formation requires hydroxylation of abundant proline and lysine residues, allowing cross-linking within and between collagen strands to provide tensile strength. The hydroxylases catalyzing this reaction depend on the substrate oxygen. Hypothermic vasoconstriction decreases oxygen supply to tissues and thus results in decreased collagen deposition.

The first few hours following bacterial contamination constitute a decisive period during which infection is established. The effects of antibiotic administration and of hypoperfusion are especially important during this period. Similarly, wound hypoperfusion (achieved by epinephrine infiltration or “dehydration shock”) aggravates test infections when induced up to 2.5 hours after the inoculation, but has no effect when induced later.

Only 1.9 °C core hypothermia triples the incidence of surgical wound infection following colon resection. This is clinically important as indicated by the fact that patients with wound infections were hospitalized one week longer on average than patients without infection. In addition, hypothermia increases the duration of hospitalization by 20% even when infected patients were excluded from the analysis—apparently because healing per se was significantly impaired.

This result is consistent with studies by Carli, et al. showing that mild hypothermia aggravates post-operative protein wasting. In addition, disorders of hemostasis are characterized not only by prolonged bleeding, but also by slow wound healing. Hemostatic reactions, particularly platelet plug formation, play a primary role in initiating the first and perhaps the second stage of wound healing. Activated platelets initiate wound healing and angiogenesis by the release of growth and chemotactic factors. It is thus likely that hypothermia-induced coagulopathy because of low availability of platelet activators also contributes to impaired wound healing.

Cancer recurrence

Removing the primary tumor is important for eliminating the major pool of metastasizing cells, but the surgical procedure itself might promote metastases. This is attributed to several mechanisms acting in synergy, including mechanical release of tumor cells, enhanced angiogenesis and immunosuppression. Especially the immunsystem is affected by perioperative hypothermia. Inadvertant hypothermia might thus increase cancer recurrences in patients undergoing cancer surgery.

THERMAL DISCOMFORT AND POSTOPERATIVE RECOVERY

Increased solubility of volatile anesthetics and reduced metabolism of intravenous drugs suggests that hypothermia might prolong emergence and recovery from general anesthesia. The issue has been addressed several times. However, most available studies suffer major methodological flaws that preclude accurate interpretation of their results. Typical problems include: 1) patients not randomly assigned to normothermia or hypothermia; 2) temperatures measured at inadequate sites (e.g., axilla,
mouth); 3) fitness for discharge evaluated by an observer not blinded to intraoperative thermal management and postoperative temperatures; and 4) core temperature per se being among the discharge criteria.

Recently, a prospective, randomized trial demonstrated that mild hypothermia significantly delayed discharge of adult patients from the post-anesthesia care unit. Recovery duration was prolonged even when core normothermia was not a discharge criteria (Figure 1). Interestingly, similar prolongation of recovery duration was not observed in infants and children. A limitation of that study, though, is that patients were not randomly assigned to specific intraoperative thermal management.

Even mild hypothermia produces marked postoperative thermal discomfort. Patients often identify feeling cold in the immediate postoperative period the worst part of their hospitalization — sometimes rating it worse than surgical pain. Given the appropriate efforts to treat surgical pain, it would similarly seem appropriate to treat thermal discomfort. It is also likely that thermal discomfort is physiologically stressful, and contributes to observed increases in postoperative blood pressure, heart rate, and plasma catecholamine concentrations.

**SHIVERING**

Shivering is an important complication of hypothermia. However, there is increasing evidence that shivering-like tremor is a complicated response that includes at least three different patterns of muscular activity, some of which may not even be thermoregulatory.

Shivering is an autonomic thermoregulatory response that can be observed in a number of situations, one being during peri-operative care. Due to cold operating

![Figure 1](https://example.com/figure1.png)

**Figure 1.** Mild intraoperative hypothermia prolongs postoperative recovery. One hundred fifty patients were randomly assigned to normothermia or \(\approx 2.5 \, ^\circ\)C core hypothermia. Fitness for discharge was determined using defined criteria by observers blinded to patient temperature and group assignment. The percentage of patients fit for discharge are plotted against time, using survival curve analysis. Hyperthermia significantly delayed discharge by \(\approx 20 \, \text{minutes}\) (left side of figure). When normothermia (core temperature \(\geq 36 \, ^\circ\)C) also was required for discharge, the difference between the groups increased to nearly two hours (right side of figure). These data indicate that mild hypothermia significantly prolongs postanesthetic recovery. Reprinted with permission from Lenhardt R et al.: Mild intraoperative hypothermia prolongs postoperative recovery. *Anesthesiology* 1997; 87:1318–23.
rooms and anesthesia induced vasodilatation, patients often become hypothermic during surgery. The best thermoregulatory response to counteract a decrease in body temperature is through shivering. A 2 fold increase in metabolic heat production can be sustained over increased durations.51

Although shivering may not be regarded as clinically important as infection or overall surgical outcomes, it can have a profound effect on patient recovery and especially comfort. Shivering is notably uncomfortable and patients even report that it is more uncomfortable than pain from surgery.52 Aside from thermal discomfort shivering can exacerbate wound pain,52 increase intracranial53 and intraocular pressure54 and for mothers during labor and delivery, it can cause further discomfort.55

Another potentially complication related to shivering is the increase in metabolic demands. With increased metabolism comes an increase in oxygen consumptions sometimes as high as 2 or 3 fold.29,56 Increased oxygen demands induced through shivering may create problems for patients predisposed with existing intrapulmonary shunts, fixed cardiac output, or limited respiratory reserve an increase in oxygen demands. Shivering however, is rare in elderly due to impaired thermoregulatory control and thus the potential increase in oxygen consumption is generally absent in this population. Even in younger individuals, shivering is seldom associated with clinically relevant hypoxia as hypoxia itself is an inhibitor for the shivering response.

Minor consequences of hypothermia

Hypothermia is associated with mild hypokalemia,57,58 but the clinical significance of this observation appears trivial. The cardio-toxicity of bupivacaine is markedly increased by mild hypothermia.59 Hypothermia has a mild effect on somatosensory evoked potentials, but the changes are unlikely to alter clinical management. Neither hypothermia nor hyperthermia significantly alter electroencephalographic values.60

Pulse oximeter function usually is well maintained even in intensely vasoconstricted patients. (Consequently, it is not prudent to use a pulse oximeter as an indicator of extremity flow.)61) However, sufficient vasoconstriction (usually resulting from the combination of hypothermia and vascular volume depletion) can obliterate the saturation signal. The signal can be restored by local warming or a finger nerve block.62 Interestingly, thermoregulatory vasoconstrictive per se slightly increases SaO2, but the increase is only ≈2% which is not clinically important.63

Cost of complications

In selected patient populations, maintaining normothermia has already been shown to be cost-effective. For example, reduced need for allogeneic transfusion in patients undergoing hip arthroplasty more than offsets the cost of forced-air and fluid warming.64 Similarly, treatment of infections and prolonged hospitalization is far more expensive than active warming during colon resection.63

CONCLUSION SUMMARY

Sympathoneural and adrenomedullary responses to perioperative mild hypothermia may result in a derangement of myocardial energetics, and triples the incidence of adverse myocardial outcomes in high-risk patients. Mild hypothermia significantly increases blood loss and significantly augments allogeneic transfusion requirement.
Only 1.9 °C core hypothermia triples the incidence of surgical wound infection following colon resection and increases the duration of hospitalization by 20%. This outcome may be due to a direct adverse effect of hypothermia on antibody- and cell-mediated immune defences, or an indirect effect, through low oxygen availability in the peripheral wound tissues. Mild perioperative hypothermia changes the kinetics and action of various anaesthetic and paralyzing agents, increases thermal discomfort, and is associated with delayed postanaesthetic recovery. Finally, mild core hypothermia affects pulse oximetry monitoring and various electrophysiologic indices of the central and peripheral nervous system, with questionable clinical significance, as yet.

REFERENCES


