This work was supported by internal funds only. The Department of Outcomes Research is supported by numerous companies with interests in temperature monitoring and thermal management. For example, the unpublished result presented in Figure 4 is based on a study supported by Arizant Healthcare (Eden Prairie, Minnesota). However, Dr. Sessler has no personal financial interest related to this review. He is an ASA representative to the Physician Consortium for Performance Improvement.

General anesthesia profoundly impairs normal tight control of core-body temperature; to a lesser extent, neuraxial anesthesia does as well. Consequently, unwarmed surgical patients nearly all become 1-2°C hypothermic. Randomized trials show that even mild hypothermia increases the risk of morbid myocardial outcomes, bleeding and transfusion requirement, and surgical wound infection. Hypothermia also slows drug metabolism, prolongs recovery, and provokes shivering and thermal discomfort. Various warming methods are available and provide differing combinations of safety, efficacy, cost and ease of use; forced-air remains by far the most common approach.

Normal Thermoregulation

Body temperature is normally tightly regulated to near 37°C. Temperature is sensed at the skin surface and throughout the body; very roughly, the skin, deep tissues, spinal cord, hypothalamus, and remainder of the brain each contribute about 20 percent to central control. Control of body temperature is hierarchical, with the hypothalamus being the dominant center in mammals (it is the spinal cord in birds). Thermal receptors are phenomenally sensitive; for example, humans can detect localized increases in skin temperature of only 3 thousandths of a °C. Precision of thermoregulatory control is thus not limited by receptor sensitivity.

Regulation can be divided into behavioral and autonomic responses. Behavioral defenses are triggered by individuals' perceptions of their thermal environment and include dressing warmly, adjusting a room thermostat and building shelter. Generally, behavior is by far the more powerful thermoregulatory defense and is the reasons humans can live in such diverse environments. The difficulty, of course, is that anesthetized patients have little or no access to behavioral responses – leaving them dependent on less powerful autonomic defenses.

The major autonomic defenses against heat in humans are active pre-capillary vasodilation and sweating. Sweating can dissipate a remarkable amount of heat in a dry, convective environment. One investigator, for example, spent a full hour in an industrial oven at 250°F without adverse effects. The major autonomic defenses against cold are arterio-venous shunt vasoconstriction and shivering. Instead of shivering, all infant mammals (except pigs) use non-shivering thermogenesis to directly convert chemical energy into heat in brown fat and muscle. For example, human infants – even somewhat premature ones – will double metabolic heat production in a cold environment, which is roughly comparable to heat production during sustained shivering. Small mammals generally maintain their preference for non-shivering thermogenesis as adults.

Various species use a remarkable range of thermoregulatory strategies and defenses. For example, penguins cannot sweat and have limited ability to dissipate heat (which they presumably rarely require); but if necessary, they will urinate on their feet and take advantage of the subsequent evaporative cooling. Flamingos stand on one foot to reduce heat loss into the water they stand in and use counter-current mechanisms to further reduce heat loss. Butterflies vary the thickness of wing scales to alter absorption of radiant heat. Camels and just a few other desert mammals take a different approach; instead of sweating, which would waste precious water, they let their core temperature vary up to 10°C during the circadian cycle. Interestingly, most animals regulate core temperature to approximately 37°C. But there are distinct exceptions, such as worms that live near deep-sea thermal vents; they tolerate temperatures as high as 80°C and can have gradients exceeding 60°C across their three-inch-long bodies.

Poikilothermia is defined by lack of autonomic thermoregulatory defenses. But poikilothermic species very much use behavioral thermoregulation. Given warm and cold
environmental options, for example, nearly all poikilothermic species will choose a location that gives them a core temperature near 37°C. Goldfish – which have no intrinsic way to modify their temperature – can be trained to press a button to adjust the temperature of water in their tank. Even bacteria will line up on a thermal gradient at about 37°C. And finally, not all poikilotherms are completely at the mercy of their thermal environments: sharks, for example, heat their eyes to (slightly) improve their vision.

The core temperature that triggers each defense defines its threshold. Human body temperature is normally maintained between the sweating and the vasoconstriction thresholds – defined as the interthreshold range – which usually spans only a few tenths of a °C. The shivering threshold is typically a full °C below the vasoconstriction threshold. People are thus already fairly hypothermic by the time they start shivering.

**Anesthetic-Induced Thermoregulatory Impairment**

General anesthetics only slightly increase the sweating threshold and thus minimally impair thermoregulatory defenses against heat. In contrast, all general anesthetics profoundly reduce the thresholds for vasoconstriction and shivering. Intravenous anesthetics – such as propofol, opioids and central alpha-2 agonists – reduce the vasoconstriction and shivering thresholds as a linear function of plasma concentration. The volatile anesthetics differ in disproportionately reducing the vasoconstriction and shivering thresholds at higher doses.

The consequence of anesthetic-induced thermoregulatory impairment is a 10-to-20-fold increase in the interthreshold, usually to between 2 and 4°C. Since, by definition, no autonomic regulation occurs within the interthreshold range, anesthetized patients are effectively poikilothermic over a broad range of temperatures extending from slightly above normal to well below normal (Figure 1).

With rare exceptions, such as meperidine, anesthetics synchronously reduce the vasoconstriction and shivering thresholds; so while both decrease, the normal 1°C difference between the two thresholds is maintained at any dose. The vasoconstriction threshold during anesthesia is similar in infants, children and adults. However, it is reduced by about a °C in the elderly. Neuraxial anesthesia also increases the interthreshold range, although the magnitude of the effect is considerably smaller than with general anesthesia.

**Heat Balance**

Heat distribution in humans can roughly be divided into peripheral and core thermal compartments. Deep tissues of trunk and head constitute the core and represent about half the adult body mass. Temperature of these well-perfused tissues is nearly homogenous and is thus well represented by any single core temperature.

The arms, legs and skin constitute peripheral tissues. Peripheral tissue temperatures are distinctly inhomogeneous and there are usually substantial differences within this mass. Many temperatures must therefore be integrated to accurately characterize peripheral compartment temperature. Peripheral tissues serve as a thermal buffer for the core. Thus by allowing peripheral tissue temperature to vary considerably, the thermoregulatory system can maintain core temperature while rarely invoking metabolically expensive defenses such as sweating or shivering.

![Figure 1: The major autonomic thermoregulatory response thresholds in volunteers given desflurane, alfentanil, isoflurane or propofol. All the anesthetics slightly increase the sweating threshold (triggering core temperature), while markedly and synchronously decreasing the vasoconstriction and shivering thresholds. Used with permission.](image-url)

Continued on page 36
The Second Law of Thermodynamics specifies that heat can only flow down a thermal gradient. On average, over time, there must thus be a core-to-peripheral tissue temperature gradient to allow heat generated in the core to dissipate into the environment. The magnitude of the core-to-peripheral temperature gradient is determined by vasomotor tone and by past and present thermal environments, but is usually 2-4°C in hospital environments.7

In a typical (cool) operating suite, patients vasoconstrict to constrain metabolic heat to the core and maintain core temperature. But as noted above, general anesthesia profoundly impairs thermoregulatory control. After induction of anesthesia, the vasoconstriction threshold thus decreases to well below body temperature and thermoregulatory arteri-venous shunts open. The result is a large flow of heat from the core to peripheral thermal compartment and consequent rapid 1-1.5°C reduction in core temperature.7 This internal redistribution of body heat – rather than net loss of heat to the environment – is the primary cause of hypothermia during the initial hour after induction of anesthesia (Figure 2). Redistribution is also the primary initial cause of hypothermia during neuraxial anesthesia, although vasodilation results largely from block-induced sympathectomy rather than impairment of central thermoregulatory control.8

During the next few hours of anesthesia, changes in core body temperature are mostly determined by systemic heat balance; that is, the difference between metabolic heat production and heat loss to the environment. Eventually, patients may reach a thermal equilibrium where heat production and loss are equal. But patients who become sufficiently hypothermic will trigger arteri-venous shunt constriction, which constrains metabolic heat to the thermal core and prevents further core hypothermia.9 As discussed above, the vasoconstriction threshold during anesthesia depends on the type of anesthesia and its dose; but typically, vasoconstriction during general anesthesia re-emerges at about 34.5°C. Perioperative heat balance has been reviewed in detail.10

Temperature Monitoring

There are four consistently reliable core-temperature monitoring sites: pulmonary artery, distal esophagus, nasopharynx and tympanic membrane (as measured with a thermocouple). Except during the most extreme thermal perturbations – for example, cardiopulmonary bypass – these sites rarely differ by more than a few tenths of a °C and can be used interchangeably. During general endotracheal anesthesia, the esophagus is the most obvious place to measure core temperature since esophageal measurements are easy to obtain and resistant to artifact. The difficulty is that in many patients, none of the core-temperature sites may be readily available or convenient. Core temperature can nonetheless be reasonably estimated from other sites, including the bladder, axilla, rectum and mouth. Reliability will be enhanced by thoughtful selection of the measurement site in a given patient and use of good technique. Postoperatively, electronic oral temperatures are generally reliable; in contrast, infrared aural canal (“tympanic”) monitors perform poorly.11

Skin temperature is well below core temperature; furthermore, the core-to-skin gradient varies among patients and over

Figure 2: To separate the contributions of decreased overall heat balance and internal redistribution of body heat to the decrease in core temperature, we multiplied the change in overall heat balance by body weight and the specific heat of humans. The resulting change in mean body temperature (“heat balance”) was subtracted from the change in core temperature (“measured”), leaving the core hypothermia specifically resulting from redistribution (“redistribution”). After one hour of anesthesia, core temperature had decreased 1.6 ± 0.3°C, with redistribution contributing 81 percent to the decrease. During the subsequent two hours of anesthesia, core temperature decreased an additional 1.1 ± 0.3°C, with redistribution contributing only 43 percent. Redistribution thus contributed 65 percent to the entire 2.8 ± 0.5°C decrease in core temperature during the three hours of anesthesia. Induction of general anesthesia is identified as elapsed time zero; all values after elapsed time zero differ significantly from those preceding induction of anesthesia. Adapted with permission.7
time within patients. Infrared aural canal (“tympanic”) and forehead thermometers are not sufficiently accurate for clinical use. Anesthetic-induced thermoregulatory impairment and perioperative temperature monitoring have been reviewed in detail.\textsuperscript{12}

**Consequences of Hypothermia**

Randomized trials have consistently demonstrated that mild hypothermia (i.e., 1.5-2°C) causes substantial morbidity (Table 1, page 39). The three most serious complications caused by hypothermia are morbid myocardial events, wound infections and coagulopathy.

The only randomized trial specific to cardiovascular events was conducted in 300 vascular surgery patients whose core temperatures differed by 1.3°C at the end of surgery. The primary outcome, a composite of serious cardiac complications, was reduced by a factor-of-three in patients assigned to normothermia.\textsuperscript{13} Adverse cardiac events most likely result from autonomic stimulation, which increases circulating catecholamines and blood pressure.

All surgical wounds become contaminated; whether contamination progresses to clinical infection is largely determined by adequacy of host defense, especially oxidative killing by neutrophils. Hypothermia may increase wound infection risk by provoking vasoconstriction, which reduces delivery of oxygen and immune cells to surgical incisions, by directly impairing function of macrophages and other immune cells, and by reducing scar formation and wound healing.

Two randomized trials evaluated surgical site infection in 200 patients undergoing colon resection\textsuperscript{14} and in 421 general surgical patients.\textsuperscript{15} Both found a three-fold reduction in wound infection risk. The temperature difference between the randomized groups was 1.9°C in Kurz et al.\textsuperscript{14} and – amazingly – was not reported in Melling et al.\textsuperscript{15} The findings are notable in that hypothermia was maintained only intraoperatively, and returned to normal within a couple of postoperative hours, whereas wound infections are typically identified one to two weeks after surgery. That hypothermia augments the risk of wound infection was possibly the first demonstration that intraoperative anesthetic management has long-term consequences – a general topic that remains under active investigation.\textsuperscript{16}

Local tissue temperature – including temperature inside surgical incisions – is in equilibrium between core and ambient temperature and decreases roughly in proportion to core hypothermia. Platelet function (via release of thromboxane A\textsubscript{2}) is a function of local tissue temperature. It is thus unsurprising that mild hypothermia increases blood loss and, consequently, transfusion requirement. The effect of hypothermia on each has been reviewed in a recent meta-analysis.\textsuperscript{17}

Hypothermia also provokes a host of lesser complications, including slowed drug metabolism, prolonged recovery, thermal discomfort and shivering.

**Maintaining Normothermia**

The major initial cause of core hypothermia in most patients is core-to-peripheral redistribution of body heat. The amount of heat that redistributes is a strong function of the core-to-peripheral tissue-temperature gradient. Interventions that reduce the gradient thus reduce redistribution hypothermia. The easiest way to reduce the gradient is simply to warm patients before induction of anesthesia. Warming must be intense enough to provoke thermoregulatory vasodilation and sufficiently prolonged to transfer 50 or more kcal into the body (i.e., 30 minutes of forced-air warming\textsuperscript{18}). Pre-warming has little effect on core temperature, which remains regulated, but does increase peripheral tissue temperature and body heat content. Subsequent induction of general or regional anesthesia thus provokes little redistribution hypothermia because core and peripheral tissue temperatures are already similar (Figure 3). At least five randomized trials have demonstrated the efficacy of pre-warming\textsuperscript{19,20} and the strategy probably deserves more use than it currently gets.

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{fig3.png}
\caption{During the preinduction period (–60 to 0 min), volunteers were either actively warmed or passively cooled (no warming). At induction of anesthesia (0 min), active warming was discontinued and volunteers were exposed to the ambient environment. Initial tympanic membrane temperatures were similar before each preinduction treatment. During the 60 minutes following induction of anesthesia, core temperature decreased less when volunteers had been prewarmed (\textit{Delta T} = -1.1±0.3°C) than when the same volunteers had not been prewarmed (\textit{Delta T} = -1.9±0.3°C). Data are presented as means ± standard deviations. Adapted with permission.\textsuperscript{19} Continued on page 38}
\end{figure}
Forced-air is by far the most common intraoperative warming approach because it is inexpensive, easy to use, effective and remarkably safe. Recently, some investigators have proposed that forced-air warming might disperse bacteria within operating rooms. This is a curious assertion since six studies demonstrate that properly used forced-air systems do not increase bacterial counts.21,22 Furthermore, activation of forced-air warming does not reduce operating room air quality, even during laminar-flow ventilation (Figure 4). Finally, according to the Centers for Disease Control and Prevention (CDC), “... for most surgical site infections, the source of pathogens is the endogenous flora of the patient’s skin, mucous membranes, or hollow viscera ...”23 Forced-air is thus not only a perfectly appropriate way to keep surgical patients normothermic, but remains the only perioperative warming system that has been shown in randomized trials to significantly reduce surgical site infection risk.15,24

Only a tiny amount of heat is lost via ventilation, even with high fresh-gas flows; airway heating and humidification is thus an ineffective approach to maintaining perioperative normothermia. Similarly, only a small amount of heat is lost via conduction from the posterior surface. It is thus unsurprising that circulating-water mattresses are only marginally effective; they also occasionally cause burns. Nonetheless, newer systems that combine posterior heating with pressure relief materials (i.e., PerfecTemp, Medline) appear to maintain normothermia as well as forced-air, even in patients having major open abdominal surgery. Electric blankets also appear to maintain normothermia as well as forced-air. At least some circulating-water garments and anterior-surface pads transfer even more heat than forced-air. And finally, a novel system that combines a very small vacuum with circulating water, restricted to the hand and forearm (vitalHEAT vH2, DynaTherm Medical), also seems to be as effective as forced-air.

It is impossible to transfer substantial amounts of heat into patients by warming intravenous fluids because the fluids cannot be heated to much above core temperature. However, it is very much possible to cool patients by giving large amounts of unwarmed crystalloids, colloids or blood. A liter of crystalloid or colloid at ambient temperature reduces mean-body temperature in a 70-kg adult by 0.25°C; a unit of blood reduces mean-body temperature by the same amount (it is twice as cold, but half the volume). It is thus unnecessary to warm fluids when small amounts (i.e., ≤1 liter/hour) are given; but it may be appropriate to warm larger volumes, especially if core temperature is drifting. Fluid warming, though, should never be the first-line defense against hypothermia because it cannot compensate for substantial heat loss from the skin surface and from within surgical incisions.

Consequences of perioperative hypothermia and warming strategies have been reviewed in detail.25

**Figure 4:** Mean particle concentration at a putative surgical site (green) versus the background (baseline) particle load (blue) in operating rooms tested with an Arizant 522 upper-body forced-air blanket (Eden Prairie, Minnesota). Three different test conditions are shown: forced-air warming system set to off (“No Air”), ambient (“Ambient Air”) or high (“Warm Air”). Each of the measurements at the surgical site was highly statistically significantly less than the baseline concentration (P<0.001); however, there were no statistically significant differences among the three surgical site measurements (P=0.39). Error bars are 95 percent confidence intervals. The horizontal red line shows the 2-log reduction in background particles that defines adequate laminar flow performance. Forced-air warming did not worsen the ability of the laminar flow environment to shield wounds from ambient particles.

**PQRS and SCIP**

The Physician Quality Reporting System (PQRS) and Surgical Care Improvement Project (SCIP) are national attempts to improve various aspects of surgical care. Each includes provisions related to perioperative normothermia; fortunately, the provisions are harmonized and thus essentially identical for the two organizations. The incentives for participation are that compliance with SCIP provisions is publically reported and that reports to PQRS are linked to Medicare payments.

The PQRS and SCIP measures combine process and an intermediate outcome. The process component is use of warming techniques deemed effective and the outcome component is core temperature. The measures apply to surgical patients having...
### Table 1: Major in Vivo Consequences of Mild Perioperative Hypothermia in Humans.

<table>
<thead>
<tr>
<th>Potential Complications</th>
<th>First author</th>
<th>Year</th>
<th>N</th>
<th>ΔT&lt;sub&gt;core&lt;/sub&gt; (°C)</th>
<th>Normothermic</th>
<th>Hypothermic</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Surgical wound infection</td>
<td>Kurz&lt;sup&gt;14&lt;/sup&gt;</td>
<td>1996</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>Melling&lt;sup&gt;15&lt;/sup&gt;</td>
<td>2001</td>
<td>421</td>
<td>?</td>
<td>5%</td>
<td>14%</td>
<td>0.001</td>
</tr>
<tr>
<td>Duration of hospitalization</td>
<td>Kurz&lt;sup&gt;14&lt;/sup&gt;</td>
<td>1996</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>Frank&lt;sup&gt;13&lt;/sup&gt;</td>
<td>1997</td>
<td>300</td>
<td>1.3</td>
<td>8 (5-13, range)</td>
<td>8 (5-11)</td>
<td>N.S.</td>
</tr>
<tr>
<td>Blood loss</td>
<td>Schmied&lt;sup&gt;26&lt;/sup&gt;</td>
<td>1996</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>Blood loss</td>
<td>Winkler&lt;sup&gt;27&lt;/sup&gt;</td>
<td>2000</td>
<td>150</td>
<td>0.4</td>
<td>0.5 L</td>
<td>0.6 L</td>
<td>0.002</td>
</tr>
<tr>
<td>Blood loss</td>
<td>Widman&lt;sup&gt;28&lt;/sup&gt;</td>
<td>2002</td>
<td>46</td>
<td>0.5</td>
<td>0.5 ± 0.3 L</td>
<td>0.7 ± 0.3 L</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Blood loss</td>
<td>Persson&lt;sup&gt;29&lt;/sup&gt;</td>
<td>2001</td>
<td>59</td>
<td>1.0</td>
<td>0.29 ± 0.03 L</td>
<td>0.30 ± 0.05 L</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Blood loss</td>
<td>Hofer&lt;sup&gt;30&lt;/sup&gt;</td>
<td>2005</td>
<td>60</td>
<td>1.8</td>
<td>1.5 ± 0.5 L</td>
<td>2.7 ± 1.0 L</td>
<td>0.01</td>
</tr>
<tr>
<td>Blood loss</td>
<td>Johansson&lt;sup&gt;31&lt;/sup&gt;</td>
<td>1999</td>
<td>50</td>
<td>0.8</td>
<td>1.0 ± 0.4 L</td>
<td>1.0 ± 0.4 L</td>
<td>N.S.</td>
</tr>
<tr>
<td>Blood loss</td>
<td>Vassiliades&lt;sup&gt;32&lt;/sup&gt;</td>
<td>2003</td>
<td>94</td>
<td>0.9</td>
<td>0.6 ± 0.5 L</td>
<td>0.8 ± 0.5 L</td>
<td>0.015</td>
</tr>
<tr>
<td>Blood loss</td>
<td>Wong&lt;sup&gt;33&lt;/sup&gt;</td>
<td>2007</td>
<td>103</td>
<td>?</td>
<td>Median 200 ml</td>
<td>Median 400 ml</td>
<td>0.01</td>
</tr>
<tr>
<td>Transfusion requirement</td>
<td>Nesher&lt;sup&gt;34&lt;/sup&gt;</td>
<td>2003</td>
<td>60</td>
<td>1.0</td>
<td>1.7 units</td>
<td>Median 3.5 units</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Transfusion requirement</td>
<td>Schmied&lt;sup&gt;26&lt;/sup&gt;</td>
<td>1996</td>
<td>60</td>
<td>1.6</td>
<td>10 ± 55 ml</td>
<td>80 ± 154 ml</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Transfusion requirement</td>
<td>Kurz&lt;sup&gt;14&lt;/sup&gt;</td>
<td>1996</td>
<td>200</td>
<td>1.9</td>
<td>0.4 ± 0.4 L</td>
<td>1.1 ± 0.9 L</td>
<td>0.013</td>
</tr>
<tr>
<td>Transfusion requirement</td>
<td>Hofer&lt;sup&gt;30&lt;/sup&gt;</td>
<td>2005</td>
<td>60</td>
<td>1.8</td>
<td>0.4 ± 0.4 L</td>
<td>1.1 ± 0.9 L</td>
<td>0.013</td>
</tr>
<tr>
<td>Postoperative troponin 1</td>
<td>Nesher&lt;sup&gt;34&lt;/sup&gt;</td>
<td>2003</td>
<td>60</td>
<td>1.0</td>
<td>22 ± 9 ng/ml</td>
<td>8 ± 5 ng/ml</td>
<td>0.05</td>
</tr>
<tr>
<td>Morbid cardiac events</td>
<td>Frank&lt;sup&gt;13&lt;/sup&gt;</td>
<td>1997</td>
<td>300</td>
<td>1.3</td>
<td>1%</td>
<td>6%</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Myocardial damage</td>
<td>Nesher&lt;sup&gt;34&lt;/sup&gt;</td>
<td>2003</td>
<td>60</td>
<td>1.0</td>
<td>8 ± 5 ng/ml</td>
<td>22 ± 9 ng/ml</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Urinary excretion of nitrogen</td>
<td>Carli, et al&lt;sup&gt;35&lt;/sup&gt;</td>
<td>1989</td>
<td>12</td>
<td>1.5</td>
<td>982 mmol/day</td>
<td>1,798 mmol/day</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Duration of vecuronium</td>
<td>Heier, et al&lt;sup&gt;36&lt;/sup&gt;</td>
<td>1991</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>Duration of atracurium</td>
<td>Leslie&lt;sup&gt;37&lt;/sup&gt;</td>
<td>1995</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 (oxygen consumption)</td>
<td>Just&lt;sup&gt;38&lt;/sup&gt;</td>
<td>1992</td>
<td>14</td>
<td>2.3</td>
<td>141 ± 9 ml·min&lt;sup&gt;1·m&lt;sup&gt;2&lt;/sup&gt;&lt;/sup&gt;</td>
<td>269 ± 60 ml·min&lt;sup&gt;1·m&lt;sup&gt;2&lt;/sup&gt;&lt;/sup&gt;</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Duration of postanesthetic recovery</td>
<td>Lenhardt&lt;sup&gt;39&lt;/sup&gt;</td>
<td>1997</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>Adrenergic activation</td>
<td>Frank&lt;sup&gt;40&lt;/sup&gt;</td>
<td>1995</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>Thermal discomfort</td>
<td>Kurz&lt;sup&gt;41&lt;/sup&gt;</td>
<td>1995</td>
<td>74</td>
<td>2.6</td>
<td>50 ± 10 mm VAS</td>
<td>18 ± 9 mm VAS</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Composite complications</td>
<td>Wong&lt;sup&gt;33&lt;/sup&gt;</td>
<td>2007</td>
<td>103</td>
<td>?</td>
<td>32%</td>
<td>54%</td>
<td>0.027</td>
</tr>
<tr>
<td>Mortality after major trauma</td>
<td>Gentillo&lt;sup&gt;42&lt;/sup&gt;</td>
<td>1997</td>
<td>57</td>
<td>?</td>
<td>2 / 29 (7%)</td>
<td>12 / 28 (43%)</td>
<td>&lt;0.05</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. ΔT<sub>core</sub> = difference in core temperature between the treatment groups. Different outcomes of some studies are shown in separate rows. Table is restricted to hypothermia-related complications. VAS is a 100 mm-long visual analog scale (0 mm = intense cold, 100 mm = intense heat). Just et al. is but one of dozens of studies showing that hypothermia provokes shivering. Results presented as means ± SDs or median [interquartile range] unless otherwise specified. N.S. = not significant. Adopted from Sessler DI, Kurz A, Anesthesiology News, 2008.
general or neuraxial anesthesia lasting at least 60 minutes who do not have documented intentional hypothermia. They can be met by fulfilling any one of the three following conditions: 1) active intraoperative over-body warming; 2) body temperature $\geq 36^\circ C$ within 30 minutes before the end of anesthesia; or 3) body temperature $\geq 36^\circ C$ within 15 minutes after anesthesia.

Limitations of the PQRS and SCIP measures include the fact that “active over-body” warming is deemed acceptable, although some over-body systems may not be especially effective and at least several other warming systems are also effective. This aspect of the measures remains controversial and may change as additional evidence becomes available. It is also possible that the measure may, at some point, become a pure outcome measure that will only be met by a body temperature $\geq 36^\circ C$. “Body temperature” is not currently defined; a variety of systems and measurement sites can thus be used, although many measurement sites are sub-optimal and some temperature monitoring systems are clearly inaccurate. And finally, neither measure includes “hard” outcomes such as cardiovascular complications, blood loss, transfusion requirement or surgical site infection.

Both measures are designed for simplicity and to be rigorous and easily auditable. Consequently, the provisions do not recognize the subtleties of clinical practice. But the basic message, that surgical patients should be kept normothermic, is well supported by many randomized trials. Maintaining normothermia is already the community standard of care, irrespective of PQRS and SCIP specifics. That said, how normothermia is maintained is entirely at the clinician’s discretion. There is absolutely no requirement to use any particular warming approach for any particular patient in any particular environment. Whatever works is perfectly acceptable!

References are available at the back of the online version of this NEWSLETTER at www.asahq.org or by request by e-mailing communications@asahq.org.
Perioperative Thermoregulation and Heat Balance

References:
4. Talke P, Tayefeh F, Sessler DI, Jeffrey R, Noursalehi M, Richardson C. Dexametomidine does not alter the sweating threshold, but comparably and linearly reduces the vasoconstriction and shivering thresholds. Anesthesiology. 1997; 87:835-841.


