Chapter

Techniques to Reduce the Magnitude and Duration of Redistribution Hypothermia in Adults

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Abstract

While much effort has been devoted to correcting intraoperative hypothermia and documenting the adverse outcomes associated with hypothermia, less attention has been directed to preventing redistribution hypothermia in the first place. Methods currently exist that can reduce the magnitude of redistribution hypothermia, but are not widely practiced. This chapter focuses on the pathophysiology of redistribution hypothermia and the currently available methods that can be employed to reduce redistribution hypothermia. Additional promising, but currently unproven, methods are discussed. Since hypothermia causes adverse outcomes, it is anticipated that the reduction in redistribution hypothermia will improve patient outcome.

Keywords: redistribution hypothermia, hypothermia, perioperative hypothermia, intraoperative hypothermia, inhalation induction, anesthesia induction

1. Background

Hypothermia has multiple adverse consequences and should be avoided (**Table 1**) [1, 2]. The Anesthesia Patient Safety Foundation has recently reaffirmed that even mild hypothermia is associated with an increase in complications [3]. In studies assessing whether patients were hypothermic, typically the end-of-case temperature has been used for this determination and its association with complications. With the exception of one study where there was increased blood loss at 36.5°C [4], an increase in complications occurs when the end-of-case temperature is <36.0°C. However, there is increasing recognition that intraoperative temperature matters. The American College of Surgeons consider intraoperative hypothermia to be a modifiable risk factor for surgical site infections; they recommend the maintenance of intraoperative normothermia and the use of prewarming [5]. The 2017 Centers for Disease Control and Prevention (CDC) guidelines recommend maintenance of perioperative normothermia [6].

While much effort has been devoted to documenting adverse outcomes and correcting intraoperative hypothermia, relatively little attention has been directed to preventing intraoperative hypothermia in the first place. "Despite Active Warming, Hypothermia Is Routine in the First Hour of Anesthesia" was written on the cover of the February 2015 issue of Anesthesiology. In a

• Morbid cardiac events (ischemia, infarctions, arrhythmias, sympathetic activation)
• Surgical wound infection
• Coagulopathy, increased blood loss, increased transfusion requirements
• Patient discomfort, postoperative shivering
• More likely to require postoperative ventilation
• Adverse respiratory events in PACU
• Delayed wake-up
• Prolonged PACU stays
• Increased hospital length of stay
• Negative nitrogen balance
• Delayed wound healing
• Increased financial cost of care of hypothermia complications
• Failure to meet MACRA standard

Table 1.
Complications of hypothermia.

retrospective review, Sun et al. found 64% of 58,814 adult patients had a temperature measurement under 36.0°C after 45 min [7]. Some hypothermia complications occur intraoperatively (e.g., coagulopathy and increased transfusion requirements), some postoperatively (e.g., shivering and delayed emergence) and some likely both (e.g., infection risk). The contribution of intraoperative hypothermia to postoperative complications may often be unrecognized. For example, patients may have decreased immunologic defense against infection at the time of incision, that is, during the vulnerable period when infections can become established. It is plausible that, if redistribution hypothermia can be reduced, one may be able to reduce the intraoperative and postoperative complications associated with hypothermia, particularly in situations where patients are at increased risk of developing a greater degree of hypothermia or may have increased risk of hypothermia-associated complications (**Table 2**). End-of-case hypothermia implies intraoperative hypothermia. End-of-case normothermia does not imply intraoperative normothermia. A patient may have been hypothermic intraoperatively, having suffered the consequences of intraoperative hypothermia, achieving normothermia only at the end of the case.

The body contains three thermal zones: the core (abdomen, thorax, and brain), the periphery (the extremities), and the skin. At rest, the core temperature is 37.0°C (36.5–37.5°C) and the periphery is 2–4°C cooler. The skin temperature can approach ambient temperature. At rest, most of the basal heat production occurs in the core. Heat travels from the core to the periphery to the skin and out to the environment. In the steady state, the rate of heat loss equals the rate of heat production, and the heat content of the body remains the same. Since temperature is just a measurement that reflects heat content, the temperature remains the same. The body normally maintains core temperature within a narrow range. Within limits, the periphery can act as a temperature buffer as it can add or lose heat, changing its temperature, while keeping the core temperature within a narrow range. The core temperature is the temperature that is physiologically most important [8].

There are behavioral (e.g., seeking an environment of a different temperature and changing clothing) and physiologic defenses to thermal challenges. Under anesthesia only the physiologic defenses are available. As one becomes too warm, the first physiologic defense is to vasodilate. If the temperature increases further,

Risk posed by postoperative hyperdynamic/tachycardic response to hypothermia
Coronary artery disease
Stenotic valvular heart disease
Dynamic obstructive cardiomyopathies
Increased risk or consequence of infection
Immunocompromised
Colon surgery
Foreign body placement (e.g., artificial joints)
Potential for large blood loss increased by hypothermia- induced coagulopathy
Spine surgery
Liver surgery
Prostate resection
Large exposure of tissues that have a propensity to bleed
Hypercarbia exacerbating hypothermia-induced coagulopathy
Increased risk of hypothermia due to patient characteristics
Elderly
Frail
Inability or delay in warming patient or environment
Lateral or prone positioning
Other prolonged positioning
Robotic surgery
Axillary-bifemoral artery bypass
Large surface area burn
Remote location with inability to adjust ambient temperature
Warming devices not available
Risk from hypothermia- induced vasoconstriction
Vascular surgery
Raynaud's disease or syndrome
Free flap with arterial vascular anastomosis

Table 2.
Examples of situations where patients are at increased risk of developing a greater degree of hypothermia or may have increased risk of hypothermia-associated complications.

the patient perspires. As one cools, the first defense is to vasoconstrict. If the temperature decreases further, the patient shivers [9]. These physiologic defenses are impaired during anesthesia.

There is a large vascular supply to the periphery and skin, but at rest these vessels are relatively vasoconstricted and there is relatively little blood flow. The blood flow to the periphery and skin can increase if these blood vessels vasodilate because of the administration of a vasodilator (or there is an increased metabolic need such as what occurs during physical activity). If pharmacologic-induced vasodilation occurs, the increased blood flow to the periphery transfers more heat from the core to the periphery and skin. As a result, the core's temperature decreases while that of the periphery will increase. This process is called redistribution hypothermia. Since heat only travels from higher to lower temperature (second law of

thermodynamics), the heat in the periphery cannot be transferred back to the core. However, warming the periphery decreases the temperature gradient between the core and periphery. A smaller temperature gradient reduces the rate of heat transfer from the core [10]. Thus, more of the heat produced in the core will remain in the core, thus contributing to increasing the core temperature or decreasing the rate of core temperature decrease. If the periphery is warmed to a temperature greater than the core, heat can be transferred from the periphery to the core.

Propofol administration causes vasodilation and thus redistribution hypothermia. Propofol inductions typically result in a decrease in core temperature of about 1.5°C [11–13]. While there is also heat loss to the environment (via conduction, convection, radiation, evaporation, and airway losses), redistribution hypothermia is the major reason for the core temperature decrease in the first 15–60 min of an anesthetic. Although not specified in Sun's results, because propofol is the most common method of anesthetic induction in developed nations, it is likely most of these patients were induced with intravenous propofol and can explain the 64% incidence hypothermia (core temperature < 36.0°C) found in his review [7].

With this understanding, the following physiologic strategies have been studied to reduce redistribution hypothermia: (1) reduce the increased blood flow to the periphery and skin, (2) prewarm the periphery and skin, (3) increase metabolic activity, and (4) warm the environment. This chapter will discuss actual and potential methods available to reduce the magnitude and duration of redistribution hypothermia in adults.

2. Studied methods to reduce redistribution hypothermia

2.1 Reducing the increased blood flow to the periphery and skin

2.1.1 Etomidate

Compared to propofol, etomidate inductions result in a lesser initial temperature drop (1.4 vs. 0.5°C) [12]. Because of the adrenal axis suppression resulting from etomidate [14], the author does not recommend using etomidate just for thermal stability. However, if etomidate is used for other indications, one would expect a thermal benefit.

2.1.2 Ketamine

Compared to propofol, ketamine inductions result in a lesser initial temperature drop (1.5 vs. 0.9°C) [13]. Because of the risk of emergence reactions and hallucinations from an anesthetic dose of ketamine [15], the author does not recommend using ketamine just for thermal stability. However, if an anesthetic dose of ketamine is used for other indications, one would expect a thermal benefit.

2.1.3 Phenylephrine infusion

Ikeda et al. have demonstrated that a phenylephrine infusion of 0.5 mcg/kg/min starting immediately before induction with 2.5 mg/kg propofol results in an initial lower temperature decrease compared to propofol after the first hour (1.2 vs. 0.5°C decrease after 1 h) [16]. Presumably the vasoconstriction from phenylephrine opposes the vasodilation resulting from propofol administration. In addition, the patients who received the phenylephrine infusion maintained a higher mean arterial

blood pressure (83 ± 9 vs. 72 ± 8 mm Hg, mean ± SD). (It seems plausible that any technique discussed in this section that reduces vasodilation has the potential to accrue an additional benefit of reducing induction-associated hypotension. This hypothesis requires investigation.)

2.1.4 Phenylephrine bolus

A 160 mcg bolus of phenylephrine immediately prior to 2.2 mg/kg propofol reduces the mean decrease in core temperature by about 0.43°C in the first hour than those who did not receive the phenylephrine bolus [17, 18]. While redistribution hypothermia can continue for up to 3 h, a large part of the temperature decrease occurs within the first 15 min. The vasoconstricting effect of a bolus of phenylephrine lasts sufficiently long to oppose much of the maximal vasodilation resulting from propofol induction. While most patients decrease their blood pressure after propofol administration, the bolus phenylephrine reduced the incidence of propofol-induced hypotension from 98 to 58% [17, 18]. While generally effective, the 160 mcg dose was used on all patients in this study but may not be optimal. Some patients still became hypotensive (systolic BP < 85 mm Hg), and 1 patient in this group of 50 patients increased the systolic blood pressure to >180 mm Hg [17, 18]. It remains to be determined if a weight-based dose could be found that further reduces the incidence of hypotension, avoids dangerous hypertension, and still maintains the thermal benefit.

2.1.5 Inhalation inductions

Ikeda et al. demonstrated less core hypothermia when anesthesia is induced with inhaled sevoflurane than with intravenous propofol (1.5 vs. 0.8 °C decrease after 1 h) [11]. This study of 10 patients in each group was done at a time when the concept of redistribution hypothermia was still in development and the harmful effects of even mild hypothermia were not as well appreciated as they are today. A recent study (50 patients in each of six groups) replicated and strengthened these findings [17, 18]. Inhalation inductions of 8% sevoflurane in either 100% oxygen or 50% oxygen/50% nitrous oxide resulted in a higher mean temperature by about 0.5°C than those who received 2.2 mg/kg propofol in patients aged 18–55 years [17, 18]. Inhalation inductions were also found effective in reducing redistribution hypothermia in older (56–88 years, mean 67.2 years) patients. Elderly patients have an increased risk for hypothermia [19–21] for reasons that include decreased metabolic activity, decreased muscle mass, an impaired vasoconstriction response, and an impaired shivering response. A previous study also concluded that inhalation induction is more hemodynamically stable than IV propofol inductions [22]. In contrast to propofol inductions where significant hypotension can occur immediately, an inhalation induction typically causes a more gradual decrease in blood pressure which can be treated before severe hypotension develops.

In adults, anesthetic inductions are achieved most commonly by intravenous, not inhalation, inductions for reasons that include inhalation inductions take extra time, room contamination with anesthetic gases, and possible patient dissatisfaction. An inhalation induction takes 1–2 min longer than an intravenous induction [17, 18] and that lost time may be recovered by a quicker wake-up because of the patient being warmer. However, Muzi et al. demonstrated that the speed of inhalation induction approached that of an intravenous induction using a primed circuit [23]. Although many anesthesia practitioners may assume patients would not want the inhalation technique, when offered a choice, 50% chose an inhalation induction, 33% chose IV induction, and 17% were undecided [24].

Inhalation inductions are not for everyone. Medical contraindications would include concern of increased intracranial pressure, indication for hypothermia, contraindication to hyperthermia (e.g., multiple sclerosis), increased aspiration risk, unfavorable airway anatomy, and patient fear of face masks. Since patients may lighten more rapidly when the face mask is removed for endotracheal intubation than with propofol, it may be prudent to avoid inhalation inductions when intubation may be a more prolonged process as there may potentially be an increased risk of awareness than a propofol induction. Examples would include inserting double-lumen tubes or training novice laryngoscopists.

However, there are additional potential benefits to preforming inhalation inductions. First, there will be no pain on propofol injection. Second, trainees will get more practice with airway management. In current practice, most patients after IV induction immediately receive a laryngeal mask airway (LMA) or endotracheal intubation. Third, future propofol shortages can be mitigated by employing inhalation inductions. Fourth, LMAs may be easier to insert while patients are breathing spontaneously as the airway tends to open during inspiration and there is less of an obstruction to proper LMA positioning than a totally collapsed airway one typically gets after IV propofol inductions. Fifth, there will be less second-hand exposure to propofol, currently a candidate factor in propofol addiction. Sixth, inhalation inductions may be a superior alternative over other induction agents to patients with allergies to propofol. Seventh, with intravenous inductions, atelectasis develops very quickly. One would expect that with spontaneous ventilation, there may be less atelectasis, but this will need to be studied. In patients breathing spontaneously via an LMA after IV propofol induction, one does not have to manage a patient who becomes apneic, thus eliminating extra tasks and saving time while starting a case. Lastly, propofol supports bacterial growth [25]. There is an increased number of colony-forming units in the stopcocks of patients who received propofol (10× at 24 h and >100× at 48 h) compared to those who did not [26]. While it is not established that this is a cause of increased infections, the avoidance of propofol would eliminate this as a concern. Removing the stopcocks could also address this concern but that adds cost and likely would not be universally done.

2.1.6 Nitrous oxide

Previous work suggests an ongoing thermal benefit to using nitrous oxide. Ozaki et al. found nitrous oxide impairs thermoregulation less than sevoflurane or isoflurane [27]. The threshold for vasoconstriction was 35.8 ± 0.3°C (mean ± SD) in the patients given 50% nitrous oxide combined with 0.5 MAC sevoflurane, which was statistically significantly greater than that in those given 1.0 MAC sevoflurane: 35.1 ± 0.4°C. Similarly, the threshold for vasoconstriction was 35.9 ± 0.3°C in the patients given 60% nitrous oxide combined with 0.5 MAC isoflurane, which was statistically significantly greater than that in those given 1.0 MAC isoflurane: 35.0 ± 0.5°C. The use of nitrous oxide allows for the thermal defense of vasoconstriction to activate before the patient becomes more hypothermic.

Nitrous oxide has been under challenge for several decades. Two of the reasons why nitrous oxide has been out of favor with many practitioners have been the concern of major cardiovascular morbidity and mortality and an increased risk of surgical site infections (SSI). In combination with another retrospective study of 49,016 patients where nitrous oxide use was associated with decreased 30-day mortality and decreased in-hospital mortality/morbidity, the results of the ENIGMA II have essentially eliminated these concerns [28–31]. ENIGMA II concludes "Our findings support the safety profile of nitrous oxide use in major non-cardiac surgery. Nitrous oxide did not increase the risk of death and cardiovascular complications or surgical site infection, the emetogenic effect of nitrous oxide can

be controlled with antiemetic prophylaxis, and a desired effect of reduced volatile agent use was shown." [4] The other major reason for not using nitrous oxide has been the concern for postoperative nausea and vomiting (PONV). If a patient has been administered an antiemetic, there is a small nonsignificant increased risk of severe PONV. ENIGMA II concludes "Nitrous oxide increases the risk of severe PONV by only a small percentage, and the increased risk is essentially eliminated by antiemetic drug prophylaxis. Concern about severe PONV thus does not appear to be a valid reason to avoid nitrous oxide."

Except for potential environmental concerns, there is little reason not to use nitrous oxide in cases that are not of long duration (>4–6 h) unless there are physical contraindications (e.g., gas space expansion). Besides from its potential thermal benefit, nitrous oxide has been shown to reduce chronic pain in specific populations (Asians and other patients with variants in the methylenetetrahydrofolate reductase gene) [32]. The United States is in the midst of an opioid epidemic. The majority of heroin users got their start from medically prescribed opioids [33]. Nitrous oxide also has analgesic efficacy and may reduce intraoperative opioid use. Further research is needed, but the possibility of reducing chronic pain and intraoperative opioid use may have benefit in combatting the opioid epidemic [34].

2.2 Prewarm the periphery and skin

Prewarming is the active warming of the body surface, often via forced-air warming, prior to induction of general or central neuraxial anesthesia. It is currently the most effective method of reducing redistribution hypothermia. It has been extensively studied, and, in addition to demonstrating warmer core temperatures, improved outcomes (decreased blood loss, transfusion requirement, and infection rate) have been demonstrated. (A recent chapter reviews much of the relevant detail and will not be repeated here [10]. A small representative sample of studies are listed [35–39].) Prewarming is fundamentally different from all other techniques in that it's the only technique that exogenously adds heat content to the patient. However, the technique is not universally used [40]. Obstacles to its use include (1) requirement of space, equipment, supplies, and personnel time, (2) change in the pattern of patient flow, (3) patient refusal or intolerance, (4) requirement of cleaning if reusable equipment is utilized, (5) insufficient availability of a power supply, (6) requirement to train personnel, (7) bypass of the holding area, (8) additional equipment maintenance requirement, and (9) inadequate knowledge of the value of prewarming [10].

Prewarming works by adding heat content to the periphery. This decreases the temperature gradient between core and periphery and thus decreases the heat transfer and redistribution hypothermia. Any method that can increase the peripheral temperature will reduce redistribution hypothermia. Any event that decreases peripheral temperature will increase redistribution. Thus, all reasonable efforts should be made to keep the periphery warm before induction of anesthesia. After application of forced-air warming, it will take time (usually 30 min) until an increase in core temperature occurs [41, 42]. This delay occurs because the periphery needs to be warmed before there is a significant effect on core temperature.

The efficacy of prewarming can be limited by sweating, thermal discomfort, and efficacy of the warming device. Sessler et al. found that 30 min of prewarming increased peripheral tissue heat content by more than the amount normally distributed during the first hour of anesthesia [43]. Since there are other and ongoing mechanisms of heat loss, prewarming more than 30 min will likely benefit many patients. However, if it is difficult to arrange for 30+ min of prewarming or the patient does not tolerate the longer durations, even 10–20 min of prewarming is effective in reducing hypothermia and shivering [44].

Because of redistribution hypothermia, ideally, every patient undergoing general or neuraxial anesthesia should be prewarmed [45, 46]. If the patient receives just a peripheral nerve block, there is little risk of hypothermia. Prewarming (and forced-air warming) should not be applied over ischemic limbs. Normally when there is heat transfer to an area of the body, blood circulation removes the heat from that area, thus decreasing the local temperature. If there is impaired blood flow, it is possible that the heat accumulation from prewarming or intraoperative forced-air warming could cause tissue damage. (In therapeutic hyperthermia, temperatures >42.0°C have been associated with tissue damage such as fat necrosis [47].) For similar reasons, forced-air warming over the lower extremities should be turned off during aortic cross-clamping. Also, in theory, there may be more risk of cell death from warming ischemic tissue because of the resulting increase in metabolic oxygen demand in combination with the impaired blood supply. It may be prudent to avoid prewarming when there is a contraindication to hyperthermia (e.g., risk of neurologic ischemia and pregnancy).

There is no data to guide the decision to use prewarming on patients who are hyperthermic preoperatively. Patients are hyperthermic because either (1) their cooling mechanisms have been overwhelmed as that which occurs in heat exhaustion or heatstroke (nonfebrile hyperthermia) or (2) they have an elevated temperature set point as occurs with many infections (febrile hyperthermia). The nonfebrile patients probably should be allowed to have their core temperature normalized and thus probably should not be prewarmed. It has been suggested that the febrile patients should be allowed to remain hyperthermic intraoperatively [48]. There is overwhelming evidence that fever is part of a coordinated defense system [49, 50]. The lines of evidence include evolutionary, correlative, antipyretic, and hyperthermia/hypothermia studies [49]. For example, infectious illnesses in animals are of longer duration, and mortality rates increase if the fever is treated [49]. Some of the enzymes in the immune system have a temperature optima in the febrile range. In addition, if the temperature of these patients decrease to below their elevated temperature set point and the set point does not change during the anesthetic, then these patients will behave postoperatively as though they are hypothermic (e.g., increasing metabolism and cardiac output, shivering), even if their temperature is >37.0°C. Thus, although unproven, there is reason to maintain the febrile hyperthermia intraoperatively. It is an unanswered question as to whether these patients should be prewarmed.

2.3 Increase metabolic activity

2.3.1 Amino acid administration

The preoperative administration of amino acids increases metabolic heat production and leads to the release of insulin and leptin resulting in a mean temperature increase of 0.46°C [51]. These hormones may also affect central thermoregulation. If amino acid infusion is started after hypothermia develops, rewarming is not augmented [52]. It is possible that the amino acid-induced increase in cardiopulmonary demands may be problematic in frail patients and those with reduced cardiopulmonary reserve. Since there is limited evidence, this technique is considered experimental.

2.3.2 Fructose administration

The preoperative administration of fructose increases metabolic heat production and affects central thermoregulation [53]. However, in patients with hereditary fructose intolerance (HFI), the infusion of fructose can lead to liver damage, kidney

damage, convulsions, and death. HFI often goes undiagnosed. The prevalence of HFI is estimated at 1 in 20,000, similar to the incidence of malignant hyperthermia events.

2.4 Warming the environment

As discussed above, anything that is practical and can be done to keep the patient warmer will likely result in the periphery remaining warmer and thus less redistribution. There is often a difference of opinion among various operating room personnel as to what temperature of the operating room should be. A cooler environment will increase the rate of heat loss from the patient. With the resultant decrease in peripheral heat content, the magnitude of redistribution hypothermia will be greater [8].

There are five methods of heat loss (conduction, convection, radiation, evaporation, and loss via the airway). Radiation and convection losses are most important [54]. One of the major determinants of radiative heat loss is the temperature difference between the radiator (i.e., the patient) and the environment. A greater temperature difference will result in a greater heat loss. Another major determinant is the absorption/reflection properties of the environment. The author is unaware of any clinical data regarding these factors.

Convection refers to heat transfer resulting from the bulk movement of a fluid (i.e., gas or liquid). A patient will transfer heat to warm the air immediately around him or herself. Convective airflow will move this warm air away from the patient and replace it with cooler ambient air. Thus, heat loss will continue to warm the newly adjacent cool air. The cooler the adjacent air, the greater the rate of heat loss from the patient.

Surgeons generally prefer a cooler room because they are working, are under lights that may emit heat, may be under stress, are gowned, may be in physical contact with other personnel, and may also be wearing lead aprons. An uncomfortable surgeon may not work at his/her best and may drip perspiration into the surgical wound. With modern operating rooms where the air is replaced many times an hour, the temperature can be adjusted within minutes. Thus, a reasonable compromise would be to keep the operating room warm until the patient is prepped and draped and then cool the room for the benefit of the surgical team. Once the patient is draped, convective losses are reduced except from the surgical wound.

3. Candidate methods to reduce redistribution hypothermia

Unfortunately, none of the abovementioned techniques fully solves the redistribution hypothermia problem. It is plausible that either reducing propofol dosages or combining techniques may provide additional thermal benefit. The following techniques show promise but require formal investigation:

1. Ketamine in analgesic doses is commonly used as part of a multimodal analgesia strategy. It is plausible that reducing the propofol dose by an analgesic dose of ketamine would reduce the magnitude of redistribution hypothermia. The induction dose of propofol (2.2 mg/kg) is similar in mg to the induction dose of ketamine (2 mg/kg). Reducing the propofol dose by 30 mg and replacing it with 30 mg ketamine seems reasonable.

2. Kazama et al. found that patients can be induced with a reduced total dose of propofol and with less hypotension when diluted propofol was administered

as an infusion [55]. It is plausible that, by using less propofol, there would be a lesser amount of redistribution hypothermia (and less hypotension).

3. A blended propofol-inhalation induction would utilize less propofol and thus potentially reduce redistribution hypothermia.

4. Combining prewarming with any of the other techniques (e.g., prewarming and inhalation induction, prewarming and phenylephrine prior to propofol).

5. Combining prophylactic phenylephrine with inhalation inductions.

4. Summary

At this time, prewarming is the most studied and likely the most effective method of reducing redistribution hypothermia, and improved outcomes have been documented. Unfortunately, it is not universally used. Given the priority of operating room expediency, either inhalation inductions or prophylactic administration of bolus phenylephrine are practical and can be used in virtually every anesthetizing location. Even though these techniques have been demonstrated to reduce redistribution hypothermia, and post-induction temperatures are similar to what one sees after prewarming and a propofol induction, we can only anticipate but not yet infer the same improved outcomes will accrue. Although a strong correlation of adverse outcomes and hypothermia has been documented in numerous studies, an outcome study is needed. Inhalation inductions or prophylactic administration of phenylephrine reduces redistribution hypothermia by reducing vasoconstriction; they do not add heat content. Prewarming reduces redistribution hypothermia by warming the periphery and adds heat content to the patient. Because the periphery needs to get warmed before forced-air warming increases the core temperature, it is likely that prewarmed patients will rewarm more rapidly, which is likely beneficial.

It is important to keep the operating room warm until the patient is prepped and draped. The temperature of a modern operating room can be decreased rapidly for the comfort of the operating room personnel. Putting a warm blanket on a patient as he/she enters a cold operating room does little to rewarm a patient. The skin temperature receptors have a disproportionate influence on the hypothalamus. The warm blanket may make the patient feel warmer, but the patient may still have lost significant heat content to the cool environment.

Besides from thermal benefits, financial benefits may accrue from reducing redistribution hypothermia. In the United States, the new Medicare Access and CHIP Reauthorization Act of 2015 (MACRA) temperature target (35.5°C) may now be easier to achieve [56]. Avoidance of unpleasant side effects (e.g., shivering) may result in less patient dissatisfaction. Reducing hypothermia-associated complications will reduce costs.

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