

Clinical complications, monitoring and management of perioperative mild hypothermia: anesthesiological features

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Abstract. *Background and aim of the work:* Perioperative hypothermia is a frequent occurrence and can lead to several complications, which adversely affect the patient's outcome, especially in high risk patient. Nonetheless, central temperature is not frequently monitored in the clinical routine. The aim of this work is to make the point on complications, monitoring techniques, prevention and treatment of mild perioperative hypothermia. *Methods:* We reviewed literature on cardiovascular, haemorrhagic, infectious, and other clinical consequences of mild intraoperative hypothermia, epidemiology and techniques, of temperature monitoring and efficacy of different approaches for the prevention and treatment of mild hypothermia. *Results:* Cardiovascular, haemorrhagic and infectious complications are significantly more frequent in hypothermic than in normothermic patients. Elderly and high risk patients are more prone to develop perioperative hypothermia, and are more liable to hypothermia-related complications. The ideal monitoring site has to be chosen considering both the patients characteristics and surgical procedure. Once identified, hypothermia has to be treated and the most effective systems are represented by active forced-air skin warming system. Active pre-warming during the preoperative period has been also demonstrated to be efficient in reducing the development of intraoperative hypothermia. Humidification and warming of inspired gases, and warming of intravenous fluids are useful techniques when used in a multimodal approach with active skin warming to maintain perioperative normothermia. *Conclusions:* All the patient undergoing surgery for more than 30 minutes should receive an accurate temperature monitoring and a correct management for the maintenance of normothermia. Reducing the incidence and severity of perioperative hypothermia has the potential for drastically reducing complication-related costs. (www.actabiomedica.it)

Key words: Homeostasis, body temperature regulation, anesthesia - general, surgery procedures - operative, perioperative complications

Introduction

Perioperative hypothermia is frequently found and is associated with several complications, which can adversely affect the patient's outcome, especially in high risk patients (1).

Both general and regional anesthesia markedly affect thermal homeostasis by influencing central thermoregulation mechanisms, reducing the sympathetic tone with inhibition of peripheral vasoconstriction

and consequent redistribution of body heat from core to the peripheral compartment.

During general anesthesia normal regulatory responses are inhibited until 2-3°C below physiological temperature (2), while during regional anesthesia the regulatory responses are effective only in unblocked areas of the body (3).

The association of the two techniques further emphasizes these negative effects, resulting in an even more severe degree of central hypothermia (4).

In spite of this, a survey conducted in Europe by the TEMMP (Thermoregulation in Europe, Monitoring and Managing Patient Temperature) group in 2004 showed that central temperature, although very important for a correct management of a surgical patient, is a parameter that is rarely monitored: in a total of 8083 surgical procedures only 19.4% received temperature monitoring. Body temperature was registered in 25% of patients undergoing general anesthesia, and only in 6% of patients who received regional anesthesia (5).

The aim of this review is to make an up-to-date on complications, monitoring techniques, prevention, and treatment of mild perioperative hypothermia, and their effects on the outcome of surgical patients.

Clinical consequences of mild hypothermia

Cardiovascular complications

In a randomized controlled trial in 1997 Frank et al (6) reported a higher prevalence of myocardial ischaemia and ventricular tachycardia in hypothermic patients, who were three times as likely to develop adverse myocardial outcomes in the presence of a 1.3°C hypothermia under physiological values.

These results are in agreement with the findings reported in other previous non-randomized trials, which demonstrated that even mild hypothermia favours the occurrence of tachycardia, hypertension, systemic vasoconstriction and an imbalance between myocardial oxygen supply and demand due to increased levels of circulating catecholamines (7, 8).

On the other hand it is unlikely that shivering, as a cause of increased oxygen consumption, can provoke myocardial ischaemia, also because this regulatory response is unusual in patients at high risk for cardiac complications such as elderly or defedated patients.

Haemorrhagic complications

Hypothermia increases blood losses during surgical procedures impairing platelet function and clotting factor enzyme function. It is still unclear if fibrinolytic activity is also involved, but preliminary data suggest that it is not.

This higher haemorrhagic risk was well demonstrated for patients undergoing colo-rectal (9) and hip replacement (10) surgery, where hypothermic patients required allogenic blood transfusion more frequently than normothermic patients.

In 1999 Johansson et al. (11) excluded correlations between mild hypothermia and haemorrhagic complications; however, a more recent trial showed that a reduction in central temperature as little as 0.5°C may be enough to increase surgery-related blood losses (12).

Infectious complications

In contrast with usual surgeons' belief, hypothermia facilitates surgical wound infection by two different mechanisms: firstly it induces a regulatory peripheral vasoconstriction with a significant reduction of subcutaneous oxygen tension (13), which is strictly correlated with an increased incidence of surgical wound infection (14, 15). Secondly, hypothermia directly impairs the immune function inhibiting T-cell mediated antibody production (16, 17), and the non-specific oxidative bacterial killing of neutrophils (18), whose activity also depends on oxygen supply (19, 20).

In agreement with those findings, Kurs et al. (9) reported more frequently infections of the surgical wound in patients developing mild perioperative hypothermia when compared to those actively maintained as normothermic, and this was associated with a delayed wound healing and longer hospital stay.

Other complications

In a randomized, controlled trial Lenhardt et al. (21) demonstrated that mild perioperative hypothermia delayed the discharge from the post anesthesia care unit (PACU) even when core normothermia was not included as a discharge criterion.

Moreover, central temperature can markedly affect the pharmacokinetic and pharmacodynamic properties of several drugs, including muscle relaxants. The duration of the effect of vecurnium is more than doubled in patients with a 2°C reduction in core temperature (22); while the effect of atracurium on nicotinic receptors is prolonged by 60% by a reduction in core temperature of 3°C (23).

Postoperative shivering is an expression of oxygen debt developed during the intraoperative period and paid at awakening. As stated above, the increased oxygen consumption produced by shivering is unlikely to provoke cardiovascular complications by itself, but it can act as a co-factor in patients with borderline myocardial perfusion. Furthermore, thermal discomfort associated with shivering in the awakening period can be considered as a complication by itself, being often described by patients as the worse and most stressful experience of the whole hospital stay, and can result in a relevant increase in circulating levels of catecholamine, resulting in a higher incidence of tachycardia and hypertension (7).

Other consequences of mild hypothermia are hypokalemia and hypomagnesemia (24), whose clinical effects still need to be clarified, and an increased cardiotoxicity of local anesthetics (25).

It has been also hypothesized that hypothermic patients may be more prone to deep venous thrombosis due to peripheral vasoconstriction, venous stasis, and hypoxia; such an effect still needs to be demonstrated, but may potentially interact with the already existing platelet dysfunction and coagulopathy.

Temperature monitoring

In 2004 the TEMMP (Thermoregulation in Europe, Monitoring and Managing Patient Temperature) group conducted a multicenter survey collecting information in a single day on the clinical practice of temperature monitoring and management from a representative sample of all hospitals in 17 European countries (5). The questions addressed the number of surgical procedures performed, type of anesthesia provided, type and site of temperature monitoring and method of patient warming. The survey collected information from a total of 8083 surgical procedures. Patient temperature was monitored in 19.4% of the cases, while active patient warming was applied in 38.5%. Interestingly, body temperature was monitored in 25% of patients receiving general anesthesia, but only 6% of cases during regional anesthesia ($P=0.0005$). The nasopharynx was the monitoring site mostly used in patients with general anesthesia,

while the tympanum was preferred with regional anaesthesia. Fourty three% of patients with general anaesthesia were warmed compared to 28% with regional anaesthesia ($P=0.0005$). For active warming, forced-air warming was mostly used irrespectively of the anaesthesia technique.

These findings demonstrated that intraoperative temperature monitoring is still widely uncommon in Europe, while active patient warming is not a standard of care. This is even more relevant if we consider that there is already sufficient evidence demonstrating that, if not correctly treated, patients become hypothermic during anesthesia; all patients should receive an accurate perioperative management aimed at preventing the development of even mild hypothermia.

It is well known that the initial reduction in core temperature due to heat redistribution from the core to the shell is almost 1°C in the first 40 minutes after anesthesia induction (1, 2, 4).

Accordingly, it can be reasonable to monitor the patient's temperature in all patients undergoing surgical procedures longer than 30 minutes.

When deciding for the site of temperature monitoring, we can differentiate between central and peripheral compartments (26).

The central compartment includes all highly perfused tissues, mostly consisting in head and trunk, whose temperature remains almost stable. Accordingly, central compartment temperature can be monitored in either the pulmonary artery, the nasofarinx, the tympanic membrane, or in the distal third of the oesophagus (28, 27).

The best monitoring site should be chosen based on the characteristics and site of the surgical procedure the patient is undergoing.

Nonetheless, clinicians frequently use other more peripheral monitoring sites. When comparing peripheral sites of temperature monitoring, Cattaneo et al. (29) clearly demonstrated that intermediate monitoring sites, such as bladder and rectal monitoring, represent the most accurate estimate of true core temperature. Their main pitfall is that they need longer equilibration times if compared to tympanic temperature monitoring, and can be markedly affected by surgery in patients undergoing abdominal or urologic procedures.

Prevention and treatment of mild hypothermia

As already mentioned, in the initial period after induction of anesthesia hypothermia is mostly caused by the redistribution of heat from the core to the shell.

The amount of re-distributed heat is a function of the gradient between the two compartments, which depends on operating room temperature, vasomotorial status of the patient, and his adiposity, and, finally, on the effect of the anesthetic drugs.

A very simple and effective, though underused, strategy to prevent perioperative hypothermia can be based on the reduction of this gradient by a pre-warming the patient's skin surface before surgery.

It has been demonstrated that pre-warming patients with active forced-air warming systems before they arrive in the operating room can prevent or at least minimize re-distributive hypothermia during both general (30) and regional (31) anesthesia, allowing good results with minimal economic and logistic cost, and also improving subjective patient comfort.

Another strategy of prevention the initial re-distribution hypothermia is based on the reduction of the heat gradient between the core and the shell by increasing the heat content in the peripheral compartment. This effect can be obtained by administering vasodilators.

Oral nifedipine (20 mg 12 hours before surgery followed by other 10 mg 1 hour before inducing anesthesia) has been reported to significantly reduce re-distributive hypothermia in surgical patients (32).

During the intraoperative period, heat is mainly lost by radiation and convection from the skin surface.

The easiest system to reduce heat loss would be maintaining a sufficiently high temperature in the operating room; however, this is usually not possible because of the thermal discomfort complained by operating room team when room temperature exceeds 23°C.

Other strategies for maintenance of perioperative normothermia include passive insulation and active warming. Several studies indicate that active methods are more efficient than passive insulation in maintaining perioperative normothermia (5, 9, 33-35.)

Passive Insulation

Several devices are available to minimize heat dispersion and insulate the patient from the environment, including *surgical draping*, *cotton blankets*, and *metallized plastic covers*. All these systems of passive insulation have the same efficiency in minimizing thermal dispersion, which is reduced by about 30% (36).

The mechanism of action of passive insulation is based on insulation of the air layer between insulating covers and patient's skin surface, and efficiency of this system is directly proportional to the covered surface area.

Increasing the number of insulating layers, as well as actively warming insulating blankets do not increase the efficiency this system of passive insulation because of this very heat capacity (37).

Active cutaneous warming systems

The forced-air warming devices are the most used and efficient active warming systems (33, 35, 38, 40). They consist in an electrically powered heater-blower unit and a plastic, or paper patient cover, and provide heat by convection also reducing the loss due to radiation.

These devices are able to maintain normothermia even in patients undergoing large surgical procedures (40), and if employed in the intraoperative period, they increase central temperature by almost 0.75°C/hour.

Resistive heating blankets are as efficient and cheaper than forced-air system because they do not require a disposable cover (38). They are particularly useful for accidental hypothermia treatment (39) because of their efficacy in transferring a large fraction of the heat generated by the device to the patient.

Circulating water mattresses, used for decades for the intraoperative warming, have the disadvantage of being placed under the patient. The back is a relatively small fraction of the total surface, that is already well insulated by the foam of covers placed on the operating table; on the other hand, most of the heat is lost from the anterior surface of the body (40), which is not sheltered by the device.

Finally, it must be considered that blood flow of the patient's back is reduced by compression produced

by the body weight, and this further reduces the ratio between transferred and dissipated heat. Accordingly, these systems are less effective than other systems, especially in the adult patient. On the contrary, these systems maintain an acceptable efficiency in pediatric patients, due to the more favorable proportion of warmed skin surface, and the reduced effects of body weight on the back's blood flow.

Radiant warmers generate infrared radiation and have the advantage of avoiding direct contact between the device and the patient's skin. This is especially useful for neonatal intensive care units (ICU) and pediatric surgery, facilitating the access to the patient. The efficiency of radiant warmers depends on the distance between the device and the skin of the patient, as well as its direction.

For the same reasons this approach can be particularly useful also in ICU and trauma patients. Trauma patients are always hypothermic and have a high haemorrhagic risk; accordingly, radiant warmers can represent a very useful tool, allowing to effectively warm the patient without affecting the diagnostic and therapeutic process.

The main limit of this method is that it is not able to stop heat losses by convection; and we know that most of the heat is lost with convective rather than with radiant mechanism (40, 41). This limit makes the method not usable in most clinical situations, with the exception of neonatal intensive care units and pediatric surgery.

The *Negative-Pressure Warming* is a recently introduced method involving the creation of a vacuum applied to the hand and forearm. The aim is to allow heat shift from peripheral to central compartment by preventing the hypothermia-related vasoconstriction (42), which provokes the postoperative insulation between central and peripheral compartment.

The theoretical basis of this system seems trivial both because the central compartment represents about half of total body mass (43, 44), and because thermoregulation mechanisms usually maintain the peripheral to central temperature gradient between 2 and 4°C (45, 46).

Two initial studies showed a great efficacy of this device in increasing central compartment heating, almost 10°C/hour (47, 48); however, other investigations

failed to confirm true benefits in using this technique (49, 50).

Internal Warming Systems

Intravenous Fluid Warming reduces heat loss due to the infusion of room temperature solutions.

One litre of crystalloid solution or a unit of refrigerated blood decrease body temperature approximately 0.25°C (51).

Nevertheless, although fluid warming is useful in preventing the reduction of core temperature when large volumes of fluids are administered (38), it is not able to maintain normothermia by itself, and should not be considered as an alternative method to actively warm the patient.

Less than 10% of heat production is lost in the airways (52, 53) to warm and humidify inspiratory gases (54).

Although several studies indicate that *active airway heating and humidification* slightly contribute to the maintenance of central normothermia (40, 54), these techniques are useful to preserve ciliary function and prevent bronchospasm (55), and are easily and economically obtained by using heat and moisture exchanging filters ("artificial noses") (56).

Other techniques of active warming have been described, such as *peritoneal dialysis*, *arteriovenous shunt*, and *cardiopulmonary bypass*, which is the most effective system to actively warm a patient (57); however, it is clear that these systems cannot be routinely applied to prevent and treat mild perioperative hypothermia.

Amino Acid Intravenous Infusion has also been demonstrated to increase the metabolic heat production in patients under general anesthesia. Sellden et al. (58) reported that patients receiving an intravenous infusion of an amino acid solution during surgery maintained a core temperature almost 0.5°C higher than those receiving crystalloid.

Conclusions

Perioperative hypothermia is a frequent finding in surgical patients, increasing the incidence of cardiovascular, hemorrhagic and infectious complications,

which can adversely affect final outcome of surgical patients. In spite of this central temperature seldomly monitored in the clinical practice.

Based of whether surgical patients receive general or regional anesthesia, they all develop perioperative hypothermia when surgical procedure lasts more than 30 minutes. Accordingly, physicians in charge of the patient should monitor core temperature and actively prevent and/or minimize its reduction using a multimodal approach, which can include active pre-warming before surgery, as well as passive insulation, fluid warming, and forced-air skin warming during the surgical procedure.

This simple and easy therapy has been demonstrated to be associated with a significant reduction of surgery-related costs by reducing the incidence of complications and accelerating hospital discharge.

References

1. Leslie K, Sessler DI. Perioperative hypothermia in the high-risk surgical patient. *Best Practice & Research Clin Anaesth* 2003; 17,4: 485-98.
2. Sessler DI. Perioperative heat balance. *Anesthesiology* 2000; 92: 578-96.
3. Emerick TH, Ozaki M, Sessler DI, et al. Epidural anesthesia increases apparent leg temperature and decreases the shivering threshold. *Anesthesiology* 1994; 81: 491-9.
4. Joris H, Ozaki M, Sessler DI, et al. Epidural anesthesia impairs both central and peripheral thermoregulatory control during general anesthesia. *Anesthesiology* 1994; 80: 268-77.
5. The thermoregulation in Europe, Monitoring and Managing Patient Temperature (TEMMP) study group. Survey on intraoperative temperature management in Europe. *Eur J Anaesthesiol* 2007; in press.
6. Frank SM, Fleisher LA, Breslow MJ, et al. Perioperative maintenance of normothermia reduces the incidence of morbid cardiac events: a randomized clinical trial. *JAMA* 1997; 277: 1127-34.
7. Frank SM, Beattie C, Christopherson R, et al. Unintentional hypothermia is associated with postoperative myocardial ischemia. *Anesthesiology* 1993; 78: 468-76.
8. Frank SM, Higgins MS, Breslow MJ, et al. The catecholamine, cortisol, and hemodynamic responses to mild perioperative hypothermia. *Anesthesiology* 1995; 82: 83-93.
9. Kurz A, Sessler DI, Lenhardt RA & Study of wound infections and temperature group. Perioperative normothermia to reduce the incidence of surgical-wound infection and shorten hospitalization. *New England J of Med* 1996; 334: 1209-15.
10. Schmied H, Kurz A, Sessler DI, et al. Mild intraoperative hypothermia increases blood loss and allogenic transfusion requirements during total hip arthroplasty. *Lancet* 1996; 347: 289-92.
11. Johansson T, Lisander B, Ivarsson I. Mild hypothermia does not increase blood loss during total hip arthroplasty. *Acta Anaesthesiol* 1999; 43: 1005-1010.
12. Winkler M, Akca O, Birkenberg B, et al. Aggressive warming reduces blood loss during hip arthroplasty. *Anesth and Analg* 2000; 91: 978-84.
13. Sheffield CW, Sessler DI, Hopf HD, et al. Centrally and locally mediated thermoregulatory responses alter subcutaneous oxygen tension. *Wound Rep Reg* 1997; 4: 339-45.
14. Greif R, Akca O, Horn EP, Kurz A, Sessler DI, Outcomes Research Group. Supplemental perioperative oxygen to reduce the incidence of surgical wound infections. *N Engl J Med* 2000; 342: 161-7.
15. Hopf HW, Hunt TK, West JM. Wound tissue oxygen tension predicts the risk of wound infection in surgical patients. *Arch Surg* 1997; 132: 997-1005.
16. Farkas LG, Bannantyne RM, Janes JS, Umamaheswaran B. Effects of two different climates on severely burned rats infected with *Pseudomonas aeruginosa*. *Eur Surg Res* 1974; 6: 295-300.
17. Saririan K, Nickerson DA. Enhancement of murine in vitro antibody formation by hyperthermia. *Cell Immunol* 1982; 74: 306-12.
18. Van Oss CJ, Absolam DR, Moore LL, Park BH, Humbert JR. Effect of temperature on the chemotaxis, phagocytic engulfment, digestion and O₂ consumption of human polymorphonuclear leukocytes. *J Reticuloendotel Soc* 1980; 27: 561-5.
19. Hohn DC, MacKay RD, Halliday B, Hunt TK. The effect of oxygen tension on the microbicidal function of leukocyte in wound and in vitro. *Surg Forum* 1976; 27: 18-20.
20. Mader JT. Phagocytic killing and hyperbaric oxygen: Antibacterial mechanism. *HBO Rev* 1982; 2: 37-49.
21. Lenhardt R, Marker E, Goll V, et al. Mild intraoperative hypothermia prolongs postoperative recovery. *Anesthesiology* 1997; 87: 1318-23.
22. Heier T, Caldwell JE, Sessler DI, Miller RD. Mild intraoperative hypothermia increases duration of action and spontaneous recovery of vecuronium blockade during nitrous oxide-isoflurane anesthesia in humans. *Anesthesiology* 1991; 74: 815-9.
23. Leslie K, Sessler DI, Bjorksten AR, Moayeri A. Mild hypothermia alters propofol pharmacokinetics and increases the duration of action of atracurium. *Anesth Analg* 1995; 80: 1007-4.
24. Polderman KH, Peerdeman SM, Girbes AR. Hypophosphatemia and hypomagnesemia induced by cooling in patients with severe head injury. *Journal of Clin Anesth* 1995; 7: 359-66.
25. Freisz M, Timour Q, Mazze RI, et al. Potentiation by mild hypothermia of ventricular conduction disturbances and reentrant arrhythmias induced by bupivacaine in dogs. *Anesthesiology* 1989; 70: 799-804.
26. Sessler DI. Perioperative thermoregulation and heat balance. *Proceedings of the NY Academy of Sciences* 1997; 813: 757-77.
27. Webb GE. Comparison of oesophageal and tympanic tem-

- perature monitoring during cardiopulmonary bypass. *Analgesia and Anesthesia* 1973; 52: 729-33.
28. Stone JG, Yound WL, Smith CR, et al. Do temperature recorded at standard monitoring sites reflect actual brain temperature during deep hypothermia? *Anesthesiology* 1991; 75: A483.
 29. Cattaneo CG, Frank SM, Hesel TW, EI- Rahmany HK, Kim LJ, Tran KM. The accuracy and precision of body temperature monitoring methods during regional and general anesthesia. *Anesth Analg* 2000; 90: 938-45.
 30. Just B, Trevien V, Delva E, Lienhart A. Prevention of intraoperative hypothermia by preoperative skin-surface warming. *Anesthesiology* 1993; 79: 214-8.
 31. Glosten B, Hynson J, Sessler DI, McGuire J. Preanesthetic skin-surface warming reduces redistribution hypothermia caused by epidural block. *Anesth Analg* 1993; 77: 488-93.
 32. Vassilieff N, Rosencher N, Deriaz H, et al. Effect of premedication by nifedipine on intraoperative hypothermia. *Ann Francaises de Anesth et de Reanim* 1992; 11: 484-7.
 33. Borms SF, Engelen SLE, Himpe DGA, Theunissen WJH. Bair-Hugger forced-air warming maintains normothermia more effectively than Thermo-Lite insulation. *J Clin Anesth* 1994; 6: 303-7.
 34. Krenzischek DA, Frank SM, Kelly S. Forced-air warming versus routine thermal care and core temperature measurements sites. *J Post Anesth Nurs* 1995; 10: 69-78.
 35. Casati A, Fanelli A, Ricci P, et al. Shortening the discharging time after total hip replacement under combined spinal/epidural anesthesia by actively warming the patient during surgery. *Minerva Anesthesiol* 1999; 65: 507-14.
 36. Sessler DI, McGuire J, Sessler AM. Perioperative thermal insulation. *Anesthesiology* 1991; 74: 875-.
 37. Sessler DI, Schroeder M. Heat loss in humans covered with cotton hospital blankets. *Anesth Analg* 1993; 77: 73-7.
 38. Negishi C, Hasegawa K, Mukai S, et al. Resistive heating and forced-air warming are comparably effective. *Anesth Analg* 2003; 96 (6): 1683-7.
 39. Greif R, Rajek A, Laciny S, Bastanmehr H, Sessler DI. Resistive heating is a more effective treatment for accidental hypothermia than metallic-foil insulation. *Ann Emerg Med* 2000; 35: 337-45.
 40. Hynson J, Sessler DI. Intraoperative warming therapies: A comparison of three devices. *J Clin Anesth* 1992; 4: 194-9.
 41. Hardy JD, Milhorat AT, DuBois EF. Basal metabolism and heat loss of young women at temperatures from 22 degrees C to 35 degrees C. *J Nutr* 1941; 21: 383-403.
 42. Plattner O, Ikeda T, Sessler DI, Christensen R, Turakhia M. Postanesthetic vasoconstriction slows postanesthetic peripheral-to-core transfer of cutaneous heat, thereby isolating the core thermal compartment. *Anesth Analg* 1997; 85: 899-906.
 43. Matsukawa T, Sessler DI, Sessler AM, et al. Heat flow and distribution during induction of general anesthesia. *Anesthesiology* 1995; 82: 662-73.
 44. Kurz A, Sessler DI, Christensen R, Dechert M. Heat balance and distribution during the core-temperature plateau in anesthetized humans. *Anesthesiology* 1995; 83: 491-9.
 45. Rajek A, Lenhardt R, Sessler DI, et. Tissue heat content and distribution during and after cardiopulmonary bypass at 17°C. *Anesth Analg* 1999; 88:1 220-5.
 46. Rajek A, Lenhardt R, Sessler DI, et al. Tissue heat content and distribution during and after cardiopulmonary bypass at 31°C and 17°C. *Anesthesiology* 1998; 88: 1511-8.
 47. Grahn D, Brock-Utne JG, Watenpaugh DE, Heller HC. Recovery from mild hypothermia can be accelerated by mechanically distending blood vessels in the hand. *J Appl Physiol* 1998; 85: 1643-8.
 48. Mathur A, Grahn D, Dillingham MF, Brock-Utne JG. Treatment of mild hypothermia using the "thermo-stat™" facilitates earlier discharge from the post anesthesia care unit (abstract). *Anesthesiology* 1999; 91: A1232.
 49. Smith CE, Parand A, Pinchak AC, Hagen JF, Hancock DE. Failure of negative pressure rewarming (thermostat) to accelerate recovery from mild hypothermia in postsurgical patients (abstract). *Anesthesiology* 1999; 91: A1175.
 50. Taguchi A, Arkilic CF, Ahluwalia A, Sessler DI, Kurz A. Negative pressure rewarming vs. forced air warming in hypothermic postanesthetic volunteers. *Anesth Analg* 2001; 92: 261-266.
 51. Sessler DI. Consequences and treatment of perioperative hypothermia. *Anesth Clin North Am* 1994; 12: 425-56.
 52. Sessler DI. Complication and treatment of mild hypothermia. *Anesthesiology* 2001; 95: 531-43.
 53. Lenhardt R. Monitoring and thermal management. *Best Pract & Res Clin Anesth* 2003; 17 (4): 569-81.
 54. Bickler P, Sessler DI. Efficiency of airway heat and moisture exchangers in anaesthetized humans. *Anesth Analg* 1990; 71: 415-8.
 55. Deriaz H, Fiez N, Lienhart A. Influence d'un filtre hygrophobe ou d'un humidificateur-réchauffeur sur l'hypothermie peropératoire. *Ann Fr Anesth Réanim* 1992; 11: 145-9.
 56. Forbes AR. Temperature, humidity and mucus flow in the intubated trachea. *Br J Anesth* 1974; 46: 29-34.
 57. Gentilello LM, Cobean RA, Offner PJ, Soderberg RW, Jurkovich GJ. Continuous arteriovenous rewarming: Rapid reversal of hypothermia in critically ill patients. *J Trauma* 1992; 32: 316-325.
 58. Altura BM, Altura BT, Carella A, Turlapaty PDMV, Weinberg J. Vascular smooth muscle and general anesthetics. *Fed Proc* 1980; 39: 1584-91.
 59. Sellden E, Lindahl SG. Postoperative nitrogen excretion after aminoacid-induced thermogenesis under anesthesia. *Anesth Analg* 1998; 87: 641-6.

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