Surg Endosc (1995) 9: 791–796

Surgical Endoscopy

Hypothermia induced by laparoscopic insufflation

A randomized study in a pig model

J. R. Bessell,[1] A. Karatassas,[2] J. R. Patterson,[3] G. G. Jamieson,[4] G. J. Maddern[5]

[1] Department of Surgery, University of Adelaide, The Queen Elizabeth Hospital, Woodville 5011, South Australia, Australia
[2] The Queen Elizabeth Hospital, Woodville 5011, South Australia, Australia
[3] Department of Mathematical Physics, University of Adelaide, Adelaide 5005, South Australia, Australia
[4] Department of Surgery, University of Adelaide, Royal Adelaide Hospital, Adelaide 5000, South Australia, Australia
[5] University of Adelaide Department of Surgery, The Queen Elizabeth Hospital, Woodville 5011, South Australia, Australia

Received: 6 December 1994/Accepted: 14 February 1995

Abstract. Hypothermia is a common postsurgical problem, yet information documenting the impact of laparoscopy on perioperative heat balance is scarce. This paper quantifies the changes in core temperature over a 3-h period of high-flow CO_2 insufflation in a randomized, controlled trial of six pigs. Each animal was anesthetized and studied on three occasions under standardized conditions, acting as its own control via insufflation with no gas compared with insufflation by cold gas and warmed gas.

Insufflation of CO_2 gas at high-flow rates over a prolonged period of time results in a significant fall in core temperature. The provision of warmed rather than cold insufflated gas confers no protection against changes in core temperature during laparoscopic surgery due to the small amount of heat required to warm the gas to body temperature. A much greater effect is the latent heat required to saturate the insufflated gas. Most of the hypothermic effect is due to this, and could be minimized by humidifying the flow.

Key words: Hypothermia — Laparoscopy

Clinical hypothermia has been defined as core temperature below 36°C [16]. Hypothermia and its sequelae are common postsurgical problems, more marked in patients at the extremes of age. Maintenance of perianesthetic normothermia is of great importance to counteract the numerous deleterious consequences of hypothermia [4, 5, 8, 9, 11, 13, 25, 28].

Following the advent of laparoscopic cholecystectomy in 1989, an alternative mode of access to the abdominal cavity became available for a wide variety of gastrointestinal surgical procedures. Dispensing with the need for an "open" laparotomy, the same procedure is now performed via laparoscopic "minimally invasive" access, resulting in less postoperative pain and morbidity from the incision, a shorter hospital stay, and a quicker return to work [18]. This revolutionary development has been rapidly accepted worldwide and currently accounts for a large proportion of intraabdominal procedures, yet controlled studies detailing the influence of laparoscopic surgery on perioperative hypothermia are scarce.

The aim of this study was to assess changes in core temperature during laparoscopy. In particular, it was hypothesized that insufflation of CO_2 gas contributes to hypothermia during laparoscopic surgery if high-flow rates are required over a prolonged period of time. It was also hypothesized that insufflation of warmed rather than cold CO_2 gas minimizes the extent of hypothermia during laparoscopic surgery if high-flow rates are required over a prolonged period of time.

Materials and methods

Ethical approval for this project was granted by the animal ethics committees of Queen Elizabeth Hospital and the University of Adelaide. Six pigs weighing approximately 30 kg were studied. Each pig was anesthetized and studied on three occasions, acting as its own control. The order of these studies was randomized. Studies were performed at 1-week intervals to allow complete recovery of the animal from the anesthetic.

On one occasion the animal was anesthetized for 3 h and temperature was measured without pneumoperitoneum being established. On another occasion, the animal had cold CO_2 insufflated at approximately 25°C (Table 1) for 3 h. The abdominal pressure was maintained at 10 mmHg. The CO_2 was delivered through a modified

Correspondence to: J. R. Bessell

Table 1. Insufflating gas temperature at a flow rate of 10 l/min (manufacturer information)

Time (min)	Temperature (C) with heater turned off	Temperature (C) with heater turned on
0	24.0	25.9
1	25.0	28
2	25.2	29
3	25.4	29.6
4	25.4	30
5	25.5	30.1
6	25.6	30.2
7	25.5	30
8	25.5	29.8
9	25.5	29.9
10	25.3	29.8

LINS-1000 insufflator (Cook Medical Technology, QLD, Australia) via a 10-mm port (Ethicon, Australia) inserted into the peritoneal cavity through the umbilicus. A supraumbilical second 10-mm port allowed a standardized "leak" of CO_2 at 10 l/min from the peritoneal cavity by fully opening the three-way stopcock. This was performed to simulate the repeated gas losses that are experienced clinically during some advanced laparoscopic procedures.

On the remaining occasion, the animal had warmed CO_2 insufflated at approximately 30°C (Table 1) for 3 h; otherwise, experimental parameters remained as for the second occasion.

Pigs were sedated using subcutaneous azaperone (Stresnil, Janssen Pharmaceutica) or ketamine and anesthetized with intravenous pentobarbitone sodium (Nembutal, Boehringer Ingelheim) prior to endotracheal intubation. Anesthesia was maintained by self-ventilation of a 1–1.5% halothane/O_2 mixture at 1,000 ml/min through an open circuit. Core temperature was measured at 15-min intervals by an esophageal thermoresistor (series 700, Yellow Springs Instrument Co., Yellow Springs, OH) displayed on a Tele-Thermometer (model 46, Yellow Springs Instrument Co., Yellow Springs, OH) and by a similar second thermoresistor placed into the peritoneal cavity through the umbilical 10-mm port to monitor intraperitoneal temperature.

Ambient temperature was also recorded at 15-min intervals, and the temperature of the theater environment was maintained close to 24°C. A metallic reflective blanket was wrapped around the animal to reduce thermal loss from cutaneous exposure.

All procedures were performed under aseptic conditions, and animals received a single perioperative dose of intramuscular penicillin/streptomycin. The animals were anesthetized before the placement of and during removal of laparoscopic ports. As only two 10-mm-diameter ports were required, postoperative analgesia was not required. As no intraabdominal procedure was carried out, postoperative ileus did not occur, and no alteration to the animals' eating pattern was observed. The 1-week interval between studies allowed complete recovery from the effects of anesthesia. Before reversal of anesthesia all ports were removed, and fascial and skin defects were closed with sutures. At the completion of the final anesthetic, the animals were killed by an intravenous overdose of pentobarbitone.

The statistical method utilized was repeated-measures analysis of variance, with grouping factors of treatment (no gas, cold gas, warmed gas) and a within factor of time. This method of analysis was the most appropriate for the described experimental situation [12]. Analysis was performed using 5V, BMDP statistical software UCLA (1991), with significance analyses performed at a probability level of 0.05.

Results

Using repeated-measures analysis of variance, regression lines representing the predicted temperature effect over time were fitted to each of the three treatment groups. The mean room temperature over all experiments was 23.7°C. Both intraperitoneal and esophageal temperature were significantly affected by the duration of the experiment and whether or not the animal received gas insufflation. It was found that the regression lines summarizing changes over time for the cold-gas and warm-gas treatment groups were statistically indistinguishable. Consequently, there was no significant temperature difference between animals that received cold or warmed gas over a 3-h period, and these two groups can be considered to behave as one.

The intraperitoneal temperature at the commencement of anesthesia for control animals that received no gas insufflation was 37.7°C; at 3 h a significant rise in temperature to 38°C was observed. The intraperitoneal temperature at the commencement of anesthesia for animals that had cold or warmed gas insufflated was again 37.7°C, but fell to 36°C at 3 h, a statistically significant difference of 1.7°C ($P < 0.001$). The regression line that summarized temperatures recorded by control animals (no gas) was significantly different from the pooled estimate of animals undergoing gas insufflation ($P < 0.001$), reaching almost 2°C after 3 h (Fig. 1).

The esophageal temperature at the commencement of anesthesia for control animals that received no gas insufflation was 36.9°C; however, after 3 h a significant rise to 37.2°C was recorded. The esophageal temperature at the commencement of anesthesia for animals that had cold or warmed gas insufflated was again 36.9°C, but fell to 36.1°C at 3 h, a statistically significant difference of 0.8°C ($P < 0.001$). There was also a significant difference between the temperatures recorded by control animals and those undergoing gas insufflation ($P < 0.001$), a variation of 1.17°C after 3 h (Fig. 2).

Discussion

Perioperative hypothermia results from the effects of anesthesia, augmented by certain characteristics of the individual patient. General anesthesia influences the development of intraoperative hypothermia by disturbing thermal regulatory mechanisms. This occurs in three phases [22]. In the first instance anesthesia reduces the thermoregulatory threshold for vasoconstriction by 2.5°C, resulting in a core-to-peripheral redistribution of body heat [22]. A second decrease in body temperature is a result of heat loss exceeding metabolic heat production.

Heat production decreases only minimally during anesthesia [27], and respiratory heat loss is relatively small [3]; therefore, the predominant site of heat loss is cutaneous. For example, exposure of the unclad, immobile patient to the cool theater environment [6, 15–17], evaporative water losses from surgical incisions [16], evaporation of surgical skin preparation solution, and use of cold intravenous infusions or irrigating fluids [10, 24] can all cause heat loss through the skin.

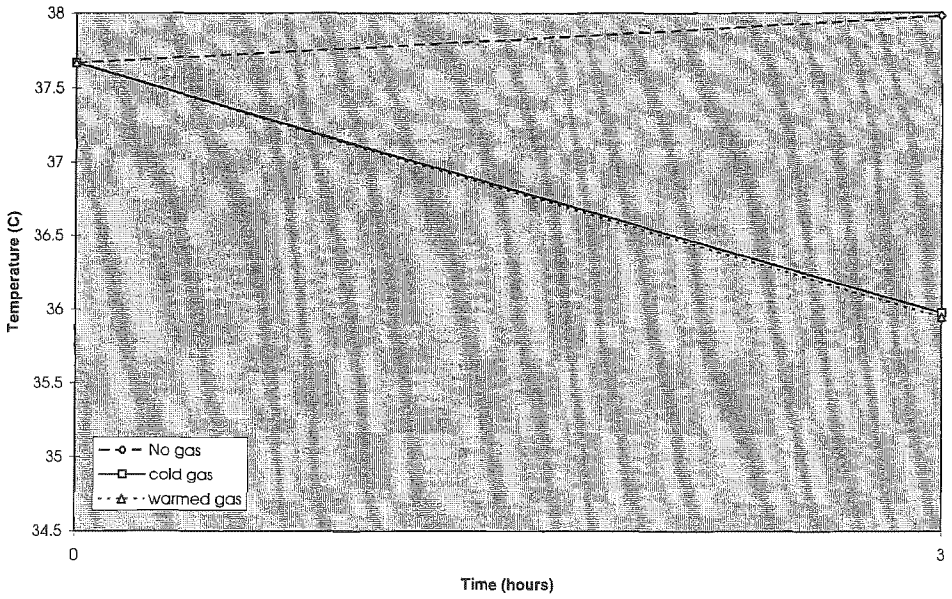

Fig. 1. Intraperitoneal temperature.

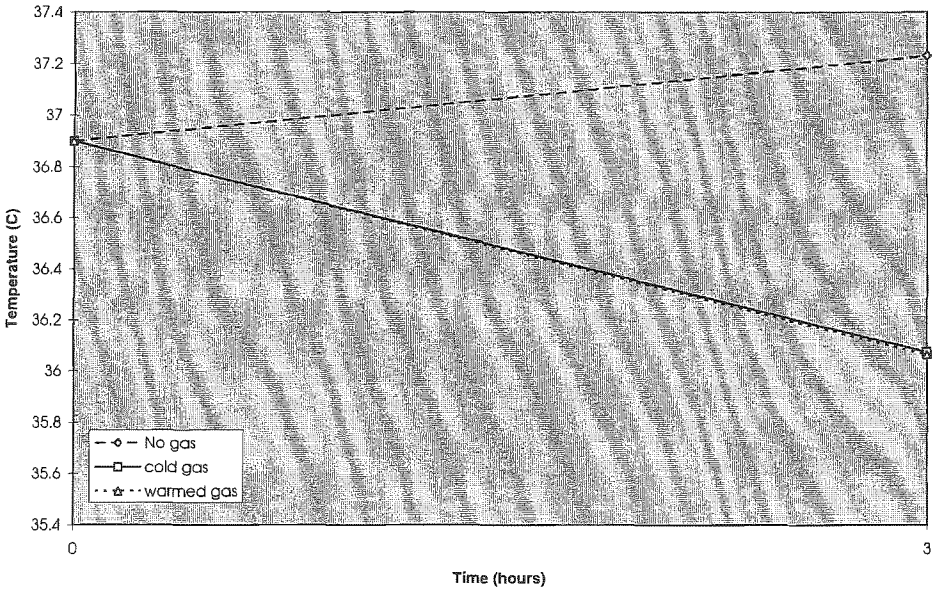

Fig. 2. Esophageal temperature.

Certain types of surgery, such as laparotomy, contribute to loss of heat from surgical incisions by increasing the surface area of the patient available for heat exchange [21, 28]. After 3–4 h, core temperature finally reaches a plateau. Patients that are kept relatively warm require no active thermoregulation at this stage. If not kept warm, thermoregulatory vasoconstriction decreases cutaneous heat loss [23], and sequesters metabolic heat to the core [2].

Patient characteristics such as age, size, and associated medical conditions augment both the degree of hypothermia and the resultant effects. For example, in elderly patients with limited cardiopulmonary reserves, marked postoperative thermogenic shivering can dramatically increase oxygen consumption, risking cardiac arrythmias, failure, or myocardial infarction [9, 28].

The importance of perioperative hypothermia becomes apparent when the numerous deleterious effects it may cause are considered. Conditions such as increased susceptibility to dermal infection [25], induction of a hypokalemic state [4, 11], impaired myocardial function [13], respiratory depression, negative nitrogen balance [5], thrombocytopenia, and depletion of clotting factors [8] have been reported. The net effect of these complications is reflected in the mortality rate of patients thus affected. One study reported a 24% mortality in postoperative patients who remained hypothermic after 2 h compared with 4% of their normothermic counterparts [26]. There is a financial penalty as well, because hypothermic patients are reported to spend up to 1 h longer in the recovery ward [7].

In the past it had been assumed that the impact of

laparoscopy would be to decrease the risk of heat loss by comparison with the corresponding "open" procedure. This assumption was based on knowledge that the predominant thermal loss during surgery is from exposed surfaces. During laparotomy the open abdomen exposes a greater surface area, whereas during laparoscopy with the abdomen sealed there is less potential heat loss from convection as well as conduction and radiation. Despite the peritoneal cavity not being in contact with the ambient theatre environment, thermal loss is at least comparable during open and laparoscopic cholecystectomy [29], and some factors unique to laparoscopy may actually increase the risk of heat loss during this type of surgery [14, 20].

Laparoscopic procedures frequently take longer to perform than their open counterparts, predisposing the patient to greater heat losses from prolonged exposure in the anesthetized state. In addition, during laparoscopy heat loss also occurs due to the use of CO_2 gas which is insufflated into the peritoneal cavity to provide a surgical access. Laparoscopic insufflators use high-pressure bottles as the source of CO_2. In delivering gas from a bottle source to the patient, the gas pressure must be brought from a pressure in the range 1,350 mmHg (180 kPa) to 37,600 mmHg (5,000 kPa) down to a pressure of 15 mmHg (2 kPa). Associated with the change of pressure at the regulator is gas expansion. As the gas expands, it cools, and being in contact with the regulator, this cools as well. The degree of cooling is dependent on the flow rate; at high gas-flow rates the cooling will be more pronounced. In this study the temperature of the CO_2 as it exited the insufflator at a flow rate of 10 l/min was approximately 25°C (Table 1).

Some laparoscopic procedures require only modest CO_2 flow rates; however, other advanced laparoscopic procedures such as colorectal and esophageal operations frequently result in large gas leaks due to the use of multiple large ports up to 33 mm in diameter, insertion and removal of laparoscopic instruments, extraction of electrocautery smoke which may obscure vision, aspiration of gas by the sucker as intraperitoneal fluid is removed, and inadvertent removal of ports not fixed securely to the abdominal wall. Insufflation may therefore be required at high-flow rates to maintain adequate pneumoperitoneum over a sustained period. These factors of prolonged surgery, flow rate greater than 3 l/min, and frequent gas extraction have previously been confirmed to result in thermal losses [20].

To counteract the cooling effect of CO_2 some companies have provided insufflators with built-in heating elements, despite the absence of controlled evidence that laparoscopy contributes to perioperative hypothermia or that gas-warming devices are protective against it. The only study to address this issue reported that postoperative temperatures in 20 patients receiving warmed CO_2 (35–35.5°C) were within 0.1°C of pre- and intraoperative findings [19]. This contrasted to a control group receiving unwarmed CO_2 (21°C) where a thermal loss of 0.3°C per 50 l of consumed CO_2 was reported. Unfortunately, this valuable study was not randomized and different operations were performed both between and within groups. Noncommercial warming devices were used, and similar but not identical flow rates and volumes of CO_2 were used. These methodological factors could have introduced errors and the conclusion that the use of physiologic temperature CO_2 helps diminish thermal loss remains to be proven.

The commercial insufflator used in this study was provided with a 100-W heating element in contact with the gas flow regulator. Application of heat at the regulator prevents cooling of the CO_2 delivered to the patient. With the heater element activated, the regulator could be held at 45°C, and the temperature of the CO_2 as it exited the insufflator at a flow rate of 10 l/min was approximately 30°C (Table 1).

The methodology of this experiment controlled for the predominant confounding factors which are known to cause intraoperative hypothermia; cutaneous heat losses from exposure to an ambient theater environment of less than 24°C, evaporative water losses from surgical incisions, and use of cold intravenous infusions or irrigating fluids. Although inhaled gases were not warmed, the control arm of this experiment (no gas insufflation) indicates that respiratory heat losses were not a contributing factor, and this correlates with previous reports that respiratory heat losses are minimal [3]. Therefore the only factor acting to alter body temperature between the three arms of the experiment was the insufflation of cold or warmed gas.

Insufflation of CO_2 gas at a flow rate of 10 l/min over a 3-hour period resulted in a statistically significant decrease in body temperature. The magnitude of this hypothermic effect (up to 2°C) due solely to laparoscopy would exert a clinically significant impact, especially when added to the numerous other factors tending to reduce body temperature during general anesthesia. The changes we observed concur with two previous uncontrolled studies addressing the matter of laparoscopic hypothermia, in which it was reported that changes in core temperature as a result of laparoscopy can be expected to fall by only 0.3°C for each 50 l of CO_2 delivered [19, 20]. Although it is true that an average leak of 10 l/min is unlikely to be tolerated for prolonged periods in most laparoscopic operations, this exaggerated "worse-case" scenario was chosen as the model to unmask any effect which may be potentially disguised by a more clinically modest situation. It is also perhaps trivial that the difference between the cold and warmed gas was only 5°C, but this represents the limit of the capabilities of commercially available insufflators. The design of the study was intended to reflect the reality faced by surgeons in current clinical operating conditions, therefore precluding the study of a potentially more useful warmed-gas temperature of 40°C, for example. Yet, if laparoscopic gas warmers were to heat gas to higher temperatures, that might introduce a deleterious drying effect on intraabdominal membranes, and therefore the gas would also have to be humidified. Unfortunately, this would cause condensation inside the insufflator and present

an electrical safety hazard. For the time being, the provision of humidified, physiological-temperature gas is a technological problem.

It is of interest that control pigs anesthetized for 3 h at a mean temperature of 23.7°C recorded a rise in body temperature. A positive correlation between the rate of body temperature rise and ambient room temperature has been reported in adult surgical patients under general anesthesia [6], and this phenomenon has also been shown to occur in dogs [1].

Our results have left one question unanswered: Why was warmed gas no better, although it exited the insufflator 5° warmer than the cold gas? A simple thermodynamic calculation indicates that the heat required to raise the temperature of the CO_2 gas flowing at 10 l/min from 25 to 37°C is 0.9 W, and the heat required to raise the temperature of the CO_2 gas from 30 to 37°C is 0.48 W.

Both of these are miniscule in comparison to the basal metabolic rate of 80 W, and would reduce body temperature by less than 0.1° over 3 h. This confirms that the observed core temperature differences cannot be explained by the difference in gas temperature used in this study. So where did the heat go? A further thermodynamic calculation shows that the latent heat required to evaporate body water in the pig to saturate the initially dry CO_2 stream of 10 l/min at 37°C is 18 W. This indicates that the evaporation of body water to saturate the CO_2 is a much greater source of heat requirement and the corresponding predicted temperature drop after 3 h is 1.6°C for a 30-kg animal. Within the errors of measurement, this would account for most of the observed intraperitoneal temperature drop of 1.7°C.

In addition, a subsequent experiment prompted by these findings has shown that the temperature of the gas, which was measured at the patient outlet of the insufflator, rather than as it entered the abdomen, actually falls exponentially along the insufflator tubing until it reaches room temperature. It was observed that the gas temperature will fall roughly 63% for every 1.5-m length of insufflator tubing. Because standard insufflator tubing is 3 m long, the temperature of gas entering the abdomen in the "warm" and "cold" cases actually differed by only 0.7°C.

Our results indicate that laparoscopic gas insufflation causes a significant fall in core temperature, and the provision of warmed rather than cold gas using currently available insufflators will confer no protection against a fall in perioperative core temperature heat loss. However, it is suggested that humidification of the insufflated CO_2 would largely resolve the problem of laparoscopy-induced hypothermia, but in addition, insufflator tubing should be equipped with an insulated heating wire to prevent warmed-gas from equilibrating with room temperature as it flows to the patient. However, the provision of humidified, heated gas to minimize perioperative hypothermia is a problem which remains to be overcome. Controlled studies will be required to validate the clinical utility of future generations of insufflator apparatus, with monitoring of gas saturation and temperature as it exits the device and as it enters the patient's abdomen.

Acknowledgement. The authors would like to express their appreciation to Cook Medical Australia, Ethicon Endosurgery Australia, Ken Porter, Adrian Hines, Olympia Kapaniris, Sue Millard, and Phil Leppard for their assistance with this project.

References

1. Allen DP (1986) Effect of anaesthesia upon temperature and blood pressure. Trans Am Soc Anesthesiol 14: 367
2. Belani KG, Sessler DI, Larson MD, Lopez M, Washington DE, Ozaki M, McGuire J, Merrifield B, Hynson J, Schroeder M (1993) The pupillary light reflex: effects of anesthetics and hyperthermia. Anesthesiology 79: 23–27
3. Bickler P, Sessler DI (1990) Efficiency of airway heat and moisture exchangers in anesthetized humans. Anesth Analg 71: 415–418
4. Boelhouwer R, Bruining H, Ong G (1987) Correlations of serum potassium fluctuations with body temperature after major surgery. Crit Care Med 15: 310–312
5. Carli F, Emergy PW, Freemantle CAJ (1989) Effect of perioperative normothermia on postoperative protein metabolism in elderly patients undergoing hip arthroplasty. Br J Anaesth 63: 276–282
6. Clark RE, Orkin LR, Rovenstine EA (1954) Body temperature studies in anesthetized man—effect of environmental temperature, humidity and anesthesia system. JAMA 154: 311–319
7. Conahan TJ (1982) Heating reduces recovery time (cost) in outpatients. Anesthesiology 67: 128–130
8. Ellis P, Kleinsaser L, Speer R (1957) Changes in coagulation occurring in dogs during hypothermia and cardiac surgery. Surgery 41: 198–210
9. Heymann AD (1977) The effect of incidental hypothermia on elderly surgical patients. J Gerontol 32: 146–148
10. Imrie MM, Hall GM (1991) Body temperature and anaesthesia. Br J Anaesth 64: 346–354
11. Laszlo A, Sprung J, Polic S, Kampine JP, Bosnjak ZJ (1990) Effects of hypothermia and potassium variations on maximum diastolic potential. Anesthesiology 73: 3A
12. Ludbrook J (1994) Repeated measurements and multiple comparisons in cardiovascular research. Cardiovasc Res 28: 303–311
13. Mattheussen MD, Boutros A, Van Aken H, Flameng W (1990) Effect of the volatile anesthetics on the hypothermic myocardium. Anesthesiology 73: A574
14. Monagle J, Bradfield S, Nottle P (1993) Carbon dioxide, temperature and laparoscopic cholecystectomy. Aust N Z J Surg 63: 186–189
15. Morris RH (1971) Influence of ambient temperature on patient temperature during intraabdominal surgery. Ann Surg 173: 230–233
16. Morris RH, Kumar A (1972) The effect of warming blankets on maintenance of body temperature of the anesthetized, paralysed adult patient. Anesthesiology 36: 408–411.
17. Morris RH, Wilkey B (1970) The effects of ambient temperature on patient temperature during surgery not involving body cavities. Anesthesiology 32: 102–107
18. Neugebauer E, Troidl H, Spangenberger W, Dietrich A, Lefering R, The Cholecystectomy Study Group (1991) Conventional versus laparoscopic cholecystectomy and the randomized controlled trial. Br J Surg 78: 150–154
19. Ott DE (1991) Correction of laparoscopic insufflation hypothermia. J Laparoendosc Surg 1: 183–186
20. Ott DE (1991) Laparoscopic hypothermia. J Laparoendosc Surg 1: 127–131
21. Roe CF (1971) Effect of bowel exposure on body temperature during surgical operations. Am J Surg 122: 13–15

22. Sessler DI (1993) Perianesthetic thermoregulation and heat balance in humans. FASEB J 7: 638–644
23. Sessler DI, Hynson J, McGuire J, Moayeri A, Heier T (1992) Thermoregulatory vasoconstriction during isofluorane anaesthesia minimally decreases heat loss. Anesthesiology 76: 670–675
24. Shanks CA, Ronai AK, Schafer MF (1988) The effects of airway heat conservation and skin surface insulation on thermal balance during spinal surgery. Anesthesiology 69: 956–958
25. Sheffield CW, Sessler DI, Hunt TK (1994) Mild hypothermia during isofluorane anesthesia decreases resistance to E. coli dermal infection in guinea pigs. Anesth Analg 38: 201–205
26. Slotman GJ, Jed EH, Burchard KW (1985) Adverse effects of hypothermia in postoperative patients. Am J Surg 149: 495–501.
27. Stevens WC, Cromwell TH, Halsey MJ, Eger EI, Shakespeare TF, Bahlman SH (1971) The cardiovascular effects of a new inhalational anesthetic, Forane, in human volunteers at constant aretrial carbon dioxide tension. Anesthesiology 35: 8–16
28. Tollofsrud S, Gunderson Y, Anderson R (1984) Perioperative hypothermia. Acta Anaesthesiol Scand 28: 511–515
29. Wallasvaara MP (1992) Ventilation and body temperatures during laparoscopic vs open cholecystectomy [Abstr]. Anesth Analg 74: S340