HEAT LOSS DURING ANAESTHESIA

A. HOLDcroft AND G. M. HALL

SUMMARY
The superficial and deep body temperatures of 23 patients were studied during prolonged anaesthesia for microscopic surgery of the fallopian tubes. The patients were divided randomly into three groups, receiving either halothane 0.5%, halothane 1% or low-dose fentanyl as a supplement to nitrous oxide, oxygen and myoneural block. Other variables were kept constant, and the theatre temperature was maintained at 24 °C. Temperature changes were unrelated to both the type of anaesthesia and the percentage of subcutaneous fat to body weight. Halothane 1% decreased the rate of heat loss in the 3rd hour. Large heat losses occurred on transfer to the recovery room, where the total heat produced increased rapidly and was unrelated to shivering.

Heat loss during anaesthesia occurs not only because of low environmental temperatures and humidity but also because of the infusion of cold fluids, ventilation with cold gases, the exposure of body cavities, the absence of muscle movement and subcutaneous vasodilation. Heat loss may be accentuated by age (Goldberg and Roe, 1966). Morris (1971) showed that theatre temperatures less than 21 °C may result in an oesophageal temperature of less than 36 °C (which he defined as hypothermia) in patients undergoing anaesthesia, and that patients remained normothermic only in temperatures of 24 °C and above. Vale (1973) suggested that operating room temperatures less than 21 °C with a low relative humidity were used often because of the preferences of theatre personnel, and that this could be detrimental to the patient.

Hypothermia in the period after operation may be responsible for shivering, which increases oxygen consumption and contributes to arterial hypoxaemia (Roe, 1966; Bay, Nunn and Prys-Roberts, 1968). Halothane has been shown to increase shivering after operation, which is probably secondary to vasodilation and the consequent heat loss caused by inhibition of the sympathetic nerves (Muldoon and Vanhoutte, 1975). During anaesthesia, however, there appears to be no difference between halothane and other anaesthetic agents in their effect upon body temperature (Morris, 1971), despite their varied effects on the peripheral circulation. Most studies of body temperature during prolonged anaesthesia have involved a variety of operations. This investigation was designed to assess the effect of three anaesthetic techniques on body temperature during a standard surgical procedure.

METHODS
Twenty-three young healthy women were studied after giving informed consent. They were divided into three groups using random numbers. Eight patients received halothane 0.5%, nitrous oxide and oxygen, seven received halothane 1% and the remainder received i.v. fentanyl as a supplementary agent, in a total dose of 0.8–1.5 mg.

Before the induction of anaesthesia the operating room temperature was set at 24 °C, and a slow i.v. infusion of Hartmann’s solution at room temperature was commenced. Skin probes, on the lateral aspect of the mid calf, the ventral surface of the mid thigh, the nipple and the lateral aspect of the upper arm, and the aural temperature probe were connected to the patient and the temperature at these sites was recorded.

Sleep was induced with a small dose of thiopentone 2.5%; following the administration of pancuronium bromide the trachea was intubated and the lungs were ventilated with nitrous oxide and oxygen. Halothane or fentanyl was given to supplement these agents. In those patients who received halothane 1% an infusion of 0.9% sodium chloride 1 litre at room temperature was given over 15 min before surgery to prevent arterial hypotension. The skin and aural temperature probe were connected to the patient and the temperature at these sites was recorded.

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In the operating theatre, the patient was ventilated with an inspired oxygen concentration of 40% (Servomex DCL oxygen analyser) using a Cape-Waine ventilator and a semi-closed circuit with soda-lime. The end-tidal carbon dioxide concentration was...
maintained between 4 and 5% with reference to an infra-red analyser (Elliott Medical Automation Ltd, Rochester, Kent), which had been calibrated against a similar mixture of nitrous oxide in oxygen.

Skin preparation was with chlorhexidine 0.5% in spirit and drappings of double thickness material were spread over the patient, except for the abdomen, before surgery. During the operation, the surgeon irrigated the peritoneal cavity with sodium chloride 0.9% at 40 °C.

The theatre environment was monitored closely. A newly installed ventilation system gave an estimated 20 air changes per hour. The temperature and relative humidity of the theatre were measured by a wet and dry bulb thermometer, and tables were used to calculate the relative humidity. Temperature recordings from the patient were made every 15 min from the onset of anaesthesia. No warming blanket was used.

Immediately before the tracheal tube was removed the temperature of the inspired and expired gases was measured, and the oesophageal and muscle probes were removed. Before transferring the patients to the recovery room each was covered with a sheet and a blanket.

In the recovery room, skin, aural and rectal temperature recordings were continued for 20–30 min until the patient was ready to return to the ward. The time and duration of any shivering were noted. Three days following the operation the patient’s skinfold thickness was measured using calipers (John Bull, British Indicators Ltd, St Albans, Herts), according to the method of Durnin and Rahaman (1967), and the percentage of fat to body weight was calculated from their table.

All the probes and the recording galvanometer were manufactured by Electrolabatoriet, Copenhagen, and were calibrated previously against a N.P.L. mercury-in-glass total immersion thermometer in a stirred water bath. They were accurate to ±0.1 °C over a range of 15–45 °C. Mean skin temperature was calculated from four probes situated on the lateral aspect of the mid-calf, the ventral surface of the mid-thigh, the nipple and the lateral aspect of the upper arm, using the formula of Ramanathan (1964):

\[
\text{Mean skin temperature} = 0.3 \times (\text{nipple} + \text{arm}) + 0.2 \times (\text{thigh} + \text{calf})
\]

Mean body temperature was determined from aural and mean skin temperature using the following equation, which has been verified recently for neutral thermal environments (Colin et al., 1971):

\[
T_{\text{body}} = (0.66 \times T_{\text{aural}}) + (0.34 \times T_{\text{skin}})
\]

and from this the heat loss or gain can be calculated in kilojoules (kJ) (Burton, 1935):

\[
\text{Total body heat} = \text{mean body temperature} \times \text{specific heat of body} \times \text{mass}
= T_{\text{body}}(°C) \times 0.83 \times 4.18 \times \text{weight (kg)}
\]

The results were compared using Student’s t test, Chi-squared test and linear regression analysis (Armitage, 1971).

RESULTS

Comparison of physical characteristics of groups

The mean ages (± SEM) of the halothane 0.5%, halothane 1% and fentanyl groups were 30 ± 1, 29 ± 2 and 29 ± 1 yr respectively. As the surgeon selected patients who were not overweight their mean weights were 56 ± 3, 57 ± 4 and 56 ± 2 kg respectively. The heights of the patients were similar: 158 ± 2 cm in the halothane 0.5% group, 163 ± 4 cm in the halothane 1% group and 163 ± 2 cm in the fentanyl group.

Environment

The theatre temperature was maintained constant at 24 °C during the study, and the relative humidity was low, ranging from 41 to 55% (see table I). The

<table>
<thead>
<tr>
<th>Relative humidity</th>
<th>Halothane 0.5%</th>
<th>1%</th>
<th>Fentanyl</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean (SEM)</td>
<td>49 (1)</td>
<td>47 (2)</td>
<td>50 (1)</td>
</tr>
</tbody>
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<table>
<thead>
<tr>
<th>Duration of operation (min)</th>
<th>Halothane 0.5%</th>
<th>1%</th>
<th>Fentanyl</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean (SEM)</td>
<td>170 (12)</td>
<td>180 (12)</td>
<td>175 (20)</td>
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</tbody>
</table>

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<thead>
<tr>
<th>Inspired gas temperature (°C)</th>
<th>Halothane 0.5%</th>
<th>1%</th>
<th>Fentanyl</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean (SEM)</td>
<td>29 (0.5)</td>
<td>28 (0.3)</td>
<td>28 (0.6)</td>
</tr>
</tbody>
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<thead>
<tr>
<th>Expired gas temperature (°C)</th>
<th>Halothane 0.5%</th>
<th>1%</th>
<th>Fentanyl</th>
</tr>
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<tbody>
<tr>
<td>Mean (SEM)</td>
<td>32 (0.2)</td>
<td>31 (0.2)</td>
<td>32 (0.6)</td>
</tr>
</tbody>
</table>

inspired gas temperature varied from 27 to 29 °C and the expired gas temperature was in the range of 30–33 °C. There was no significant difference
between the groups with regard to the patient’s environment, both in temperature, humidity and operative technique. Blood losses were small (100–200 ml) and the area of peritoneal irrigation and exposure to air was standardized as far as possible. The duration of operation varied from 1½ h to 4½ h; the mean time was 3 h (table I).

Correlation of deep body temperatures

Correlations were made between oesophageal, rectal, tympanic and muscle temperatures. The oesophageal and rectal temperatures did not show a close correlation. The regression equation was \[ y = 0.7x + 10.7 \] (for 143 pairs of numbers), and the regression coefficient was 0.68. However, there was a very close correlation of aural with oesophageal temperatures \( (r = 0.93) \) compared with rectal and aural temperatures \( (r = 0.75) \) (fig. 1). The aural temperature was chosen, therefore, to represent deep body temperature in the calculation of heat loss and gain.

Muscle temperature was consistently less than oesophageal or rectal temperature. The correlation coefficient for oesophageal compared with muscle temperature was 0.49 and for rectal and muscle temperatures, 0.36 (fig. 2).

Temperature changes during anaesthesia

During induction the mean skin temperature change varied \((-0.53\) to \(0.95^\circ C)\), but the average value was a small increase of \(0.16^\circ C\). The mean decrease in aural temperature was \(0.06^\circ C\) (range \(-0.3\) to \(0.3^\circ C)\). Total body heat increased slightly by a mean of \(10 \text{ kJ}\) during induction.

The mean skin temperature changes for all three groups are shown in figure 3. There were wide variations in skin temperature changes in the 1st hour of anaesthesia, but after this period the temperature was more stable. The average change in mean skin temperature per hour was a decrease of \(0.1-0.3^\circ C\). Only in the temperature changes during the 3rd hour was there a significant difference between the groups. Those who received halothane \(1\%\) showed a smaller temperature change than those who received halothane \(0.5\%\) \((P<0.01)\) or fentanyl \((P<0.05)\).

The aural temperature (fig. 4) showed trends similar to the mean skin temperature, but there was no significant difference between the three groups \((P>0.05)\). The rate of heat loss during anaesthesia is shown in table II. There was no significant difference between the amount of heat loss in the three groups \((P>0.1)\), although the loss of heat during the administration of fentanyl was less than when halothane was used.
TABLE II. The heat loss per hour of the anaesthetized patients in the halothane 0.5%, halothane 1% and fentanyl groups

<table>
<thead>
<tr>
<th></th>
<th>Halothane 0.5%</th>
<th>Halothane 1%</th>
<th>Fentanyl</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total heat loss during anaesthesia (kJ h⁻¹)</td>
<td>Mean 54</td>
<td>64</td>
<td>18</td>
</tr>
<tr>
<td></td>
<td>SD 32</td>
<td>49</td>
<td>76</td>
</tr>
<tr>
<td></td>
<td>SEM 11</td>
<td>19</td>
<td>27</td>
</tr>
<tr>
<td>Heat loss related to surface area (kJ m⁻² h⁻¹)</td>
<td>Mean 34</td>
<td>39</td>
<td>12</td>
</tr>
<tr>
<td></td>
<td>SD 20</td>
<td>29</td>
<td>48</td>
</tr>
<tr>
<td></td>
<td>SEM 7</td>
<td>11</td>
<td>17</td>
</tr>
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**FIG. 4.** The aural temperature changes during each hour of anaesthesia and the average change per hour in patients anaesthetized with halothane 0.5%, halothane 1% or fentanyl.

**Temperature changes during recovery**

The mean skin and aural temperature changes from the end of the operation to the time when the patient left the recovery room are shown in figure 5. During the time of transfer from the theatre to the recovery room, the average mean skin temperature decreased by 0.3 °C in the halothane 0.5% group and by 0.7 °C in the fentanyl group. Changes in the aural temperature were smaller, but total heat loss was 22 kJ m⁻² in the halothane 0.5% group and 54 kJ m⁻² in the fentanyl group.

After transfer to the recovery room the rate of rewarming was of the order of 1 °C per hour, with a total heat gain of more than 100 kJ m⁻² h⁻¹. The type of anaesthesia made no significant difference to these temperature changes (P > 0.1).

**Shivering**

Ten of the patients who received halothane shivered, compared with only three patients in the fentanyl group (P > 0.1). When all the patients were divided into two groups—shivering and non-shivering—no significant difference was found between them in respect of mean skin, tympanic or body temperature at the beginning of recovery (P > 0.1), the temperature gradient (mean skin minus aural temperature) (P > 0.05), or the previous heat loss during anaesthesia (P > 0.1). Shivering was unrelated to the actual temperature of the patient (P > 0.1), that is, a mean skin temperature of less than 33 °C or an aural temperature less than 35 °C.

During the recovery period there were no significant differences between the two groups in respect of mean skin, mean body or aural temperature, and heat gained (P > 0.1).

**Relationship of temperature changes to physical characteristics of patients**

The rate of temperature changes during anaesthesia was unrelated to age, weight, surface area and percentage fat. The best correlation was the mean skin temperature to % fat (y = 0.03x - 1.0; r = 0.26).

There was no significant correlation between % fat and the presence of shivering (P > 0.1).

**DISCUSSION**

Previous studies of the change of temperature during anaesthesia have used a variety of surgical procedures, anaesthetic agents and operating and recovery room environments. This study attempted to standardize the operative procedure, the type of patient and the environment, in order to demonstrate the effect of
halothane and fentanyl on body temperature. The patients were similar, being a group of fit, fairly lean, young women, undergoing a standard operation using microsurgical techniques. The average time of operation was 3 h, which ensured an adequate number of recordings. The anaesthetic technique was varied only according to the supplementary agent used, and the semi-closed circuit allowed warmed gases to be delivered to the patient. Thus, an ideal context for the assessment of heat loss during anaesthesia was available.

Body temperature is regulated by the basal metabolic rate, shivering, cutaneous vaso-activity and sweating (Hemingway and Price, 1968). Superficial body temperature can be measured by skin probes placed over various parts of the body, and deep body temperature by oesophageal, rectal and tympanic thermometers. The oesophageal temperature is most stable in the lower quarter of the oesophagus (Whitby and Dunkin, 1968) and gives an indication of cardiac temperature. Rectal temperature may be affected by faeces and pelvic irrigations, and changes in rectal temperature tend to lag behind oesophageal. Tympanic temperature is altered by local cooling, as is the temperature of the external aural canal (Greenleaf and Castle, 1972). However, when draughts and cold air are excluded, aural temperature parallels oesophageal during rapid changes in body temperature, whereas rectal temperature does not (Keatinge and Sloan, 1975).

The results showed that monitoring of aural temperature was an accurate clinical method of measuring deep body temperature and was well tolerated by both awake and anaesthetized patients. It was not found necessary to position the probe accurately on the tympanic membrane but only to secure the lead firmly to the external auditory meatus. No complications occurred throughout the use of this method, although the oesophageal probe produced occasional trauma to the pharynx during insertion.

Muscle temperature monitoring was not continued into the recovery period because of the effects of trauma, but it might have proved useful in determining the increase in heat production by striated muscle. The deltoid muscle was used for ease of insertion, and there were no problems, after operation, associated with its use.

Mean skin temperature is measured accurately by the unweighted mean temperature of 15 selected sites, but this method shows 67% agreement, within 0.2 °C, to the mean based on the weighting system of Ramanathan (1964) using only four skin temperature measurements (Mitchell and Wyndham, 1969), in awake resting men. Shanks (1975) found that this four-site system reaches a 95% agreement to within 1 °C of the true mean skin temperature during anaesthesia, but when changes of temperature were measured he reported a result comparable to that of a 10-site system used by Colin and Houdas (1965).

The most practicable method of measuring mean skin temperature in this study was the four-site system, but this had not been studied in the recovery period and had not been shown to be completely comparable to the multiple probe system. Therefore, a comparison of 15-, 12-, and four-site formulae was made in another group of non-selected surgical patients, before, during and after anaesthesia (see Appendix).

Our results showed that the 15-site system of Mitchell and Wyndham (1969) and the four-site system of Ramanathan (1964) gave results which were not significantly different, both during anaesthesia and recovery.

Morris (1971) measured oesophageal temperature during different types of anaesthesia and found that the largest decrease in temperature occurred during the 1st hour. The average figure for this period was 1.3 °C but by the 3rd hour the temperature change had decreased to 0.1 °C. We did not observe such dramatic changes in temperature in the 1st hour, but the aural temperature showed similar but smaller changes. Cohen (1967) reported an acute decrease in oesophageal temperature of 1 °C on induction of anaesthesia. This finding was not substantiated in this study, and an overall heat gain occurred, despite the use of non-depolarizing muscle relaxants. During anaesthesia, measurements of mean skin temperature indicated that, even in a relatively warm environment, temperatures may decrease by 0.1–0.3 °C per hour. There was a wide variation in temperatures in the 1st hour, and then the patients stabilized. The small temperature change in the 3rd hour in patients receiving halothane 1% may be a result of sympathetic vasoconstriction to maintain the arterial pressure, despite the fact that no patient had an arterial systolic pressure of less than 100 mm Hg.

Vale (1973) noticed that during the recovery period a rapid increase in heat production occurred. We found that, whereas the heat loss during anaesthesia had been at a rate of —15 to —39 kJ m⁻² h⁻¹, during the recovery period heat was gained at more than 100 kJ m⁻² h⁻¹. If this energy is produced by the oxidation of glucose, oxygen will be consumed at about 50% more than basal values. Thus failure to
increase the inspired oxygen concentration or to maintain an adequate cardiac output and oxygen-carrying capacity (mainly haemoglobin) could lead rapidly to anaerobiosis and lactic acidemia. The maintenance of normothermia during anaesthesia is essential to counteract the effects of postoperative hypoxaemia, hypotension and anaemia on oxidative metabolism.

There was a large decrease in skin temperature during transfer to the recovery room, despite the beginning of patient activity and maintenance of the environmental temperature. This can only be accounted for by the removal of the drapes from the patient. This decrease was larger than the hourly loss during anaesthesia and, in view of the detrimental effects of hypothermia, should be prevented by suitable covering.

The cause of shivering after operation has been related to the administration of halothane (Cohen, 1967), and particularly to the central body temperature (Jones and McLaren, 1965). Moir and Doyle (1963) found a lower temperature (more than 0.5 °C less) in shivering compared with non-shivering patients. In our study, neither the anaesthetic used nor the skin or aural temperatures appeared to relate to the occurrence of shivering, and it is interesting that the average total heat gained by patients in the recovery period did not increase following shivering. This suggests microscopic muscle activity, undetectable to the naked eye.

The other factors that were unrelated to the change in temperature were the age of the woman and the percentage of body fat. Body fat has classically been thought of as an insulating layer protecting the central core temperature from the environment. It is also a highly active metabolic tissue and has been described as an "electric blanket" (Renold and Cahill, 1965), or heat producing organ, which is important in the maintenance of body temperature.

In future studies of temperature changes during anaesthesia, adequate standardization of patients, operations and the environment should be achieved to reduce the many variables that affect body temperature. Short studies of less than 2 h will be subject to the large variations in results that we have shown in our investigation. The amount of subcutaneous fat appeared to have little effect on the degree of insulation, but different findings might occur in more obese patients. The monitoring of deep body temperature by the aural route proved safe and reliable, and is recommended for routine clinical use during and after operation.

The anesthetic agents that were used in this study did not appear to influence the temperature changes during anaesthesia. The most alarming heat loss occurred at the end of anaesthesia. There seems little point in maintaining normothermia during anaesthesia, if heat loss occurs during transfer of the patient to the recovery area.

**APPENDIX**

Comparison of methods of measuring the mean skin temperature

Two of the most accurate formulae for measuring mean skin temperature during anaesthesia were found by Shanks (1975) to be those of Hardy and Du Bois (1938) and Mitchell and Wyndham (1969). The last two workers used the mean of 15 measurements of skin temperature at the following sites: forehead (A), nipple (C), outer upper arm (D), waist (E), outer lower arm (F), dorsum hand (G), anterior thigh (H), outer calf (J), top of foot (K), back of neck (L), scapula (M), posterior iliac crest (N), posterior thigh (P), posterior calf (Q) and inner thigh (R). Hardy and Du Bois (1938) used a weighted formula which measures skin temperature at 12 sites:

\[0.07 (A + K) + 0.0875 (C + E + M + N) + 0.065 (J + Q) + 0.095 (H + P) + 0.14 (F) + 0.05 (G)\]

These two formulae were compared with Shanks with a reference formula (Winslow, Herrington and Gagge, 1936). This was based on skin temperature measurements at 15 sites which were each weighted by a factor numerically equivalent to the percentage of the total surface area represented by the separate segments. The unweighted mean skin temperature of 15 sites showed 93% agreement with this reference formula within 0.2 °C, and the 12-site formula of Hardy and Du Bois showed 74% agreement.

The use of a large number of skin temperature probes during anaesthesia reduces access to the patient for operation and is difficult technically. The formula of Ramanathan (1964) \((0.3 (C + D) + 0.2 (H + J))\), although only reaching 89% agreement with Shanks' reference formula within 1.0 °C, gave a result comparable with a 10-site formula (Colin and Houdas, 1965), and a 95% agreement with Shanks' reference formula within 1 °C, when changes in mean skin temperature were measured. Ramanathan (1964) was measuring mean skin temperature in non-anaesthetized patients and his formula in awake resting man shows a 67% agreement within 0.2 °C (Mitchell and Wyndham, 1969). Shanks (1975), however, only measured the mean skin temperature changes during anaesthesia with intermittent positive pressure ventilation and so a separate, similar study was undertaken using a variety of surgical operations, anaesthetic techniques and with monitoring of skin temperature into the recovery period.

**METHOD**

No attempt was made to standardize the environment, type of operation or patient. Before anaesthesia 15 thermocouple probes (Electro-laboratoriet, Copenhagen), which were accurate to 0.1 °C, were taped in the positions described above. Measurements of skin temperature were made at 10- or 15-min intervals, starting before induction.
and continuing into the recovery period after transfer to the recovery room.

The anaesthetic techniques used included inhalation anaesthesia, extradural anaesthesia and induced hypotension with halothane. There was no attempt to maintain normothermia by using warm i.v. fluids, a warming blanket or adequate drapes. The mean skin temperature was calculated by the three formulae of Mitchell and Wyndham (1969) (15 sites), Hardy and Du Bois (1938) (12 sites) and Ramanathan (1964) (four sites), and compared using a coefficient of correlation (Dunn, 1964).

RESULTS

A direct comparison was made between the 15-site formula and the four-site formula for all the measurements (including recovery period): the regression coefficient was 0.90 \((n = 117, y = -0.783 + 1.027x)\). The 95% confidence limits show that the four-site formula is only likely to differ by 0.2 °C at 33 °C from the 15-site formula (fig. 6).

When only the mean skin temperatures in the recovery period were compared using the 15-site and four-site formulae, there was a regression coefficient of 0.938 \((n = 24)\). The regression coefficient for the recovery period for the 12-site and four-site formulae was 0.924.

The correlation was similar for all measurements when the 12-site and four-site formulae were compared \((r = 0.87)\), but over the range of body temperature the formula of Ramanathan was consistently over-reading by 0.3 °C at 31 °C to 36 °C \((n = 116, y = 0.576 + 0.991x)\).

DISCUSSION

Despite the difference in anaesthetic technique, environment, and change in distribution of body heat during anaesthesia and recovery, the use of the four skin probes at the nipple, upper outer arm, anterior thigh and outer calf was shown to allow calculation of the mean skin temperature to an accuracy which was almost at the limits of the accuracy of measurement. This is surprising, but the choice of sites is obviously important. It confirms Shanks’ (1975) findings that the formula of Ramanathan (1964) is a suitable alternative to the unweighted mean of 15 sites. There was little change in the correlations between the formulae in the recovery period and this would suggest that the formula of Ramanathan is adequate for measuring mean skin temperatures in both awake and anaesthetized patients.

ACKNOWLEDGEMENTS

We thank Mr R. Winston for allowing us to study his patients, to the theatre staff for their co-operation and to Mrs Yvonne Bastin for secretarial help.

REFERENCES


PERTE DE CHALEUR PENDANT L’ANESTHÉSIE

RESUME

On a étudié les températures superficielles et profondes du corps de 23 malades au cours d’une anesthésie prolongée pour chirurgie sous microscope des trompes de Fallope. Les malades ont été divisés, au hasard, en trois groupes recevant chacun soit de l’halothane à 0,5%, soit de l’halothane à 1% ou encore du fentanyl à faible dose comme supplément au protoxyde d’azote, oxygène et blocage musculo-nerveux. Les autres éléments variables ont été maintenus constants et la température dans la salle d’opération a été maintenue à 24 °C. Les variations de température n’ont pas été reliées ni au type d’anesthésie ni au pourcentage de graisse sous-cutanée par rapport au poids du corps. L’halothane à 1% a diminué le taux de perte de chaleur au cours de la troisième heure. Des pertes de chaleur importantes se sont produites après le transfert des malades dans la salle de réanimation, où le total de la chaleur produite a augmenté rapidement et n’a eu aucun rapport avec les frissons.

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WÄRMEVERLUST UNTER NARKOSE

ZUSAMMENFASSUNG


PERDIDA DE CALOR DURANTE LA ANESTESIA

SUMARIO

Se estudiaron las temperaturas superficial e interior del cuerpo de 23 pacientes durante una anestesia prolongada para propósitos de cirugía microscópica en las trompas uterinas. Se dividieron los pacientes en tres grupos al azar, los cuales recibieron fuera un 0,5% de halotano, 1% de halotano o una dosis reducida de fentanilo como suplemento al oxido nitroso, oxígeno y bloqueo mio neural. Los demás variables se mantuvieron constantes, y la temperatura dentro de la sala de operaciones se mantuvo a 24 °C. Los cambios de temperatura no estuvieron relacionados con el tipo de anestesia ni con el porcentaje de grasa subcutánea comparada con el peso del cuerpo. El 1% de halotano redujo la razón de la pérdida de calor en la tercera hora. Se produjeron grandes pérdidas de calor durante la transferencia a la sala de recuperación, donde el total del calor producido aumentó rápidamente y no estuvo relacionado con la ocurrencia de escalofríos.