TEMPERATURE MONITORING DURING GENERAL ANAESTHESIA

B. D. CROCKER, F. OKUMURA, D. I. MCCUAIG AND M. A. DENBOROUGH

SUMMARY
A study of core temperature monitoring during general anaesthesia indicates that this can be introduced as a routine procedure in order to reduce mortality from malignant hyperpyrexia. The temperature profiles of 2410 patients are presented. Both mean rectal and mean oesophageal temperatures decreased during general anaesthesia. The mean oesophageal temperatures were on average 0.6 °C less than the mean rectal temperatures during the first hour of anaesthesia. An increase in core temperature occurred in nearly 20% of patients. This appeared to be related to an initially low body temperature. Core temperatures during general anaesthesia were significantly greater in patients who received the combination of suxamethonium and halothane than in patients receiving other drugs. This observation is of theoretical interest and suggests that the increase of temperature in malignant hyperpyrexia may be an exaggeration of a normal response to these agents.

Despite increasing awareness of malignant hyperpyrexia (MH) the mortality from this complication remains about 70%. Mortality in MH is directly related to the duration of exposure of the susceptible individual to the harmful anaesthetic (King and Denborough, 1973) and early diagnosis of MH is important so that the offending anaesthetic can be stopped and treatment started. In the absence of a simple routine predictive test, the simplest and most effective means of making an early diagnosis of MH might be routine temperature monitoring during general anaesthesia.

The anaesthetists at the Royal Canberra Hospital took part in a study over 12 months which was designed to assess the use of routine temperature monitoring during general anaesthesia.

METHODS
The study was carried out in the operating theatres of the Royal Canberra Hospital. Standardization of conditions was not attempted as it was intended that the results should represent information obtained from routine theatre procedures, and should reflect a realistic estimate of what can be expected in routine practice.

The skin preparations used during the operations were either spiritous or aqueous chlorhexidine, or 0.1% chlorhexidine with 1% cetavlon. Draping of the patient was consistent with standard surgical procedures.

The temperature and humidity of the theatres were maintained at 22 °C and 60% respectively. The mean theatre temperature, recorded by wall-mounted mercury thermometers, was 21.8 °C (SD ± 1.1 °C) with a range of 19–25 °C.

The inhalation anaesthetics were delivered either by CIG (Commonwealth Industrial Gases) or Dräger anaesthetic equipment.

Measurement of body temperature was made with a Medishield digital thermometer coupled to a flexible Yellow Springs Series 400 thermistor probe, which was inserted into either the rectum or the oesophagus by the anaesthetist or theatre staff. The choice of temperature site was made in the theatre before surgery. Only one site was used in each patient. A stable temperature was obtained about 4 min after the insertion of the thermocouple probe. For this reason, the first temperatures reported are at 10 min from when the measurements began. Temperature recordings, made from the onset of anaesthesia, were measured at 10-min intervals for the first 30 min and at 30-min intervals thereafter.

The types of operation were classified by the anaesthetists in eight groups (table I). Also recorded were age, sex, weight, position in which the operation was performed, premedication tempera-
TABLE I. Types of operation

<table>
<thead>
<tr>
<th>Operation</th>
<th>Number of patients</th>
<th>% of total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thoracic</td>
<td>99</td>
<td>4.6</td>
</tr>
<tr>
<td>Head</td>
<td>104</td>
<td>4.9</td>
</tr>
<tr>
<td>Neck</td>
<td>50</td>
<td>2.3</td>
</tr>
<tr>
<td>Abdominal</td>
<td>807</td>
<td>37.7</td>
</tr>
<tr>
<td>Pelvic</td>
<td>89</td>
<td>4.2</td>
</tr>
<tr>
<td>Limbs</td>
<td>364</td>
<td>17.0</td>
</tr>
<tr>
<td>Surface</td>
<td>205</td>
<td>9.6</td>
</tr>
<tr>
<td>Minor</td>
<td>421</td>
<td>19.7</td>
</tr>
</tbody>
</table>

ture, site of temperature monitoring, premedication drugs, non-inhalation drugs, inhalation drugs, neuromuscular blockers, extra drugs used, i.v. and irrigation fluids administered, endotracheal intubation, warming or cooling measures, and humidification. All data were recorded, stored and processed on a Univac 1100/42 computer, using the statistical package, P-STAT, from Princeton University. P-values were obtained using a Student’s t test programme run on a PDP8/I computer.

Measurements from a total of 2800 patients were obtained, but records from only 2410 patients contained data suitable for analysis. Within this group of 2410 patients, data for some of the variables which were being examined were missing. Temperature profiles are presented for both rectal and oesophageal sites.

RESULTS

The sex of the patient was recorded in all but three of the 2410 cases: 1024 (43%) were male and 1383 (57%) were female. Rectal temperatures did not differ significantly according to sex; in women, oesophageal temperatures were significantly greater than in men at 10 min (P<0.05), 20 min (P<0.005) and 30 min (P<0.05).

The mean age in 2372 patients was 34.7 yr (SD ± 20.97 yr, range 1–92). No significant difference in temperature profile was found when the data were divided according to decades. The mean weight in 2186 patients was 58.29 kg (SD ± 21.51 kg, range 2–125 kg).

Temperature changes

Decreases. Both rectal and oesophageal temperature decreased during general anaesthesia (fig. 1). Oesophageal temperatures were significantly lower than rectal temperatures for the first 2 h of anaesthesia (fig. 1). During the 1st hour, oeso-

phageal temperatures were approximately 0.6 °C lower than rectal temperatures. Rectal temperature decreased at the same rate as oesophageal temperature for the 1st hour of anaesthesia (fig. 2). The mean decrease in rectal temperature was 0.3 °C±0.4 (SD) and the mean decrease in oesophageal temperature was 0.3 °C±0.5 (SD). From 60 to 90 min the rate of decrease in oesophageal temperature was significantly less than the rate of decrease in rectal temperature (P<0.01).

Increases. The mean temperature increases at different intervals and the percentages of patients
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TABLE II. Increase in temperature in patients during anaesthesia

<table>
<thead>
<tr>
<th>Site</th>
<th>Time interval (min)</th>
<th>Number of patients</th>
<th>Percentage of patients</th>
<th>Temperature at 10 min (°C)</th>
<th>Temperature at end of interval (°C)</th>
<th>Increase (°C)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Range</td>
</tr>
<tr>
<td>Rectal</td>
<td>10-20</td>
<td>142</td>
<td>17</td>
<td>36.8</td>
<td>0.8</td>
<td>34.0-38.9</td>
</tr>
<tr>
<td></td>
<td>10-30</td>
<td>110</td>
<td>19</td>
<td>36.8</td>
<td>0.9</td>
<td>34.0-38.9</td>
</tr>
<tr>
<td></td>
<td>10-45</td>
<td>64</td>
<td>15</td>
<td>36.7</td>
<td>0.8</td>
<td>34.0-38.0</td>
</tr>
<tr>
<td></td>
<td>10-60</td>
<td>37</td>
<td>12</td>
<td>36.6</td>
<td>0.9</td>
<td>34.0-38.0</td>
</tr>
<tr>
<td></td>
<td>10-90</td>
<td>17</td>
<td>10</td>
<td>36.7</td>
<td>1.1</td>
<td>34.8-38.9</td>
</tr>
<tr>
<td></td>
<td>10-120</td>
<td>11</td>
<td>11.8</td>
<td>36.5</td>
<td>1.0</td>
<td>34.8-38.0</td>
</tr>
<tr>
<td>Oesophageal</td>
<td>10-20</td>
<td>235</td>
<td>18</td>
<td>36.2</td>
<td>0.7</td>
<td>32.0-39.2</td>
</tr>
<tr>
<td></td>
<td>10-30</td>
<td>192</td>
<td>18</td>
<td>36.2</td>
<td>0.9</td>
<td>32.0-38.5</td>
</tr>
<tr>
<td></td>
<td>10-45</td>
<td>128</td>
<td>16</td>
<td>36.1</td>
<td>0.9</td>
<td>32.0-39.2</td>
</tr>
<tr>
<td></td>
<td>10-60</td>
<td>89</td>
<td>16</td>
<td>36.2</td>
<td>0.6</td>
<td>34.3-37.8</td>
</tr>
<tr>
<td></td>
<td>10-90</td>
<td>62</td>
<td>20</td>
<td>36.2</td>
<td>0.7</td>
<td>34.3-37.7</td>
</tr>
<tr>
<td></td>
<td>10-120</td>
<td>32</td>
<td>25</td>
<td>36.2</td>
<td>0.7</td>
<td>34.4-37.7</td>
</tr>
</tbody>
</table>

experiencing temperature increases during these intervals are shown in Table II. For anaesthetics lasting less than 90 min, between 10% and 20% of patients had an increase in temperature. The patterns were similar for both rectal and oesophageal measurements. The greatest rectal temperature reached was 39.4 °C and the greatest oesophageal temperature was 39.5 °C. The largest increase in rectal temperature was 2.9 °C and the greatest increase in oesophageal temperature was 2.2 °C. At both sites, for each of the time intervals up to 60 min, the mean initial temperatures (at 10 min) in those patients whose temperatures increased during anaesthesia were all significantly lower (P < 0.01) than the mean initial temperature in those patients who had no temperature increases.

Drugs

Premedicants. The belladonna drugs, hyoscine and atropine, used in conjunction with papaveretum, pethidine or morphine were the premedication drugs most frequently used in this study. Patients receiving premedication containing atropine had temperature profiles under anaesthesia similar to those receiving hyoscine. There were no significant differences in temperatures between patients receiving either of the belladonna drugs as premedication and those who did not.

Non-inhalation anaesthetics. Thiopentone was the most frequently used non-inhalation anaesthetic. Ketamine, propanidid, Althesin, methohexitone and the neuroleptanalgesic drugs (fentanyl and droperidol), chlorpromazine and diazepam were used less frequently.

The temperatures of patients receiving ketamine were significantly greater at both the rectal and oesophageal sites than those of patients receiving thiopentone (fig. 3).

No abnormal temperature profiles were noted in patients receiving other non-inhalation anaesthetic drugs.

FIG. 3. Mean rectal and oesophageal temperature in patients who received ketamine or and in those who received thiopentone; number of patients. Vertical bars represent SEM.

*P < 0.05; **P < 0.01.
Neuromuscular blockers. The neuromuscular blockers used during this study were classified either as non-depolarizing or depolarizing. Suxamethonium was the neuromuscular blocker used most commonly and the only depolarizing agent. The non-depolarizing agents in order of decreasing use were alcuronium, pancuronium and tubocurarine, and gallamine.

For the first 30–45 min of anaesthesia, patients who received suxamethonium as the sole neuromuscular blocker had significantly higher oesophageal and rectal temperatures (P<0.05) than those receiving non-depolarizing neuromuscular blockers (fig. 4). Patients who received suxamethonium as the sole neuromuscular blocker had significantly higher oesophageal temperature (P<0.05) for the first 30 min of anaesthesia than those patients who received no neuromuscular blockers (fig. 5). Rectal temperatures showed a similar pattern. No difference in temperature at either site was observed between patients receiving non-depolarizing neuromuscular blockers and those receiving none at all.

The temperature differences between patients receiving suxamethonium and those receiving either non-depolarizing neuromuscular blockers or none at all were found to be dependent on the presence of halothane (figs 6, 7). When halothane was not administered no significant temperature differences occurred.

**Fig. 4.** Mean rectal and oesophageal temperature in patients who received suxamethonium ©—© and in those who received non-depolarizing neuromuscular blockers ©—©; number of patients. Vertical bars represent SEM. *P<0.05; **P<0.01; ***P<0.001.

**Fig. 5.** Mean rectal and oesophageal temperature in patients who received suxamethonium ©—© and in those who received no neuromuscular blockers ©—©; number of patients. Vertical bars represent SEM. *P<0.05; **P<0.01; ***P<0.001.

**Fig. 6.** Mean rectal and oesophageal temperature in patients who received suxamethonium and halothane ©—© and in those who received non-depolarizing neuromuscular blockers and halothane ©—©; number of patients. Vertical bars represent SEM. *P<0.05; **P<0.01; ***P<0.001.
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Inhalation agents. The inhalation agents used in this study were nitrous oxide, halothane, ether and methoxyflurane. Only one patient was given methoxyflurane and two received ether.

The mean oesophageal temperatures of patients who received halothane, nitrous oxide and oxygen were significantly greater at 20 and 30 min (P<0.05) than those patients who were given nitrous oxide and oxygen without halothane. No such differences were seen in the rectal temperatures (fig. 8).

DISCUSSION

The experience gained in this investigation indicates core temperature monitoring can be introduced as a routine procedure during anaesthesia. The results confirm earlier observations that during general anaesthesia the core temperature usually decreases. Morris (1971) observed that anaesthetized patients remained normothermic in environmental temperatures of 24 °C and more. The ambient temperature of the operating theatres in the present study was 21.8 °C, so the overall reduction in temperature at both rectal and oesophageal sites was predictable.

During the 1st hour of anaesthesia rectal temperature was, on average, 0.6 °C higher than oesophageal temperature. This difference is similar to the results found in awake patients in earlier investigations (Tanner, 1951; Cranston, Gerbrandy and Snell, 1954). It seems that the factors governing temperature difference between the two sites are maintained during anaesthesia. Whilst it is possible that some of the oesophageal leads may have been misplaced in the present investigation and were really reflecting nasopharyngeal or sublingual temperatures, there is little difference in temperature measurements at these three different sites (Cranston, Gerbrandy and Snell, 1954; Morris and Wilkey, 1970). The changes at both sites in the rate of decrease of temperature after 60 min of anaesthesia are interesting and may reflect thermoregulatory responses to reduced body temperatures. Previous investigations have shown that changes in body temperature are reflected more quickly by oral and oesophageal than by rectal measurements (Gerbrandy, Snell and Cranston, 1954) which may explain the slower rate of change observed at the rectal site.

Some increase in core temperature was noted in 10–20% of patients in this study. The temperature increases occurred in patients who began with a lower than average core temperature, and probably reflect a thermoregulatory response to this lower initial body temperature. No other special
characteristics were found in this group of patients.

The use of atropine and hyoscine was not associated with an increase in temperature during general anaesthesia. This agrees with earlier observations that the suppression of sweating caused by atropine is not significant when the ambient temperature is below body temperature (Magbagbeola, 1973), and that the administration of hyoscine 0.4 mg did not produce an increase in temperature although the rate of sweating was diminished by 35% (Frumin and Papper, 1951).

Ketamine produced a significant increase in core temperature during anaesthesia, a finding which has been observed previously (Engelman and Lockhart, 1972). Although ketamine has been used successfully in patients who are susceptible to MH (Wadhwa and Tantisira, 1974), its use, particularly in combination with suxamethonium and halothane, has been questioned (Mogensen, Misfeldt and Hanel, 1974; Roervik and Stovner, 1974). It has been suggested that ketamine should not be used when muscular relaxation is required since it causes muscular hypertonicity (Sussman, 1974).

Halothane is the anaesthetic agent most commonly implicated in MH, and it is interesting that in this study oesophageal temperature was significantly higher for the first 30 min in patients who received halothane compared with those who did not.

Another drug commonly implicated in MH is suxamethonium. The observation that patients who received suxamethonium had significantly higher core temperatures during general anaesthesia than those who received other neuromuscular blockers, or none at all, is interesting. This increase in temperature lasted for the first 30 min of general anaesthesia and may have resulted from the muscle fasciculations which follow i.v. injection of suxamethonium. Small increases in body temperature and hyper-rigidity have been observed previously in children following injections of suxamethonium (Innes and Strømme, 1973).

A more detailed examination of the increase in temperature induced by suxamethonium has shown that this occurred only in those patients who received halothane in addition to suxamethonium. This suggests that there may be a synergistic effect between suxamethonium and halothane to produce the increase in body temperature. The fact that this occurs in apparently normal individuals raises the possibility that MH may result from an exaggeration of this normal response.

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REFERENCES
SURVEILLANCE ET CONTROLE DE LA TEMPERATURE AU COURS DE L’ANESTHESIE GENERALE

RESUME
Une étude de la surveillance de température intérieure au cours de l’anesthésie générale démontre que l’on peut inclure cette opération en tant que contrôle de routine afin de réduire la mortalité due à l’hyperpyrexie maligne. Les profils de température de 2410 patients sont soumis. Au cours de l’anesthésie générale, tant la température rectale que la température oesophagique ont diminué. Les températures oesophagiennes moyennes se situaient en moyenne à 0,6 °C au-dessous des températures rectales moyennes au cours de la première heure de l’anesthésie. Chez presque 20% des patients, la température intérieure subit une augmentation. Ceci semble être fonction d’une température corporelle initiale basse. Les températures intérieures au cours de l’anesthésie générale étaient de beaucoup supérieures chez les patients qui avaient reçu un mélange de suxaméthonium et d’halothane que chez les patients auxquels on avait administré d’autres substances. Cette remarque présente un intérêt théorique et amène à penser que l’augmentation de température au cours de l’hyperpyrexie maligne peut représenter une exagération de la réponse normale à ces agents.

TEMPERATURÜBERWACHUNG WÄHREND DER ALLGEMEINNARKOSE

ZUSAMMENFASSUNG

CONTROL DE LA TEMPERATURE DURANTE LA ANESTESIA GENERAL

SUMARIO
Un estudio del control de la temperatura interna en el curso de la anestesia general indica que este podría incluirse como procedimiento rutinario con miras a reducir la mortalidad debida a la hiperpirrexia maligna. Se presentan perfiles de temperatura de 2410 pacientes. Ambas temperaturas rectal media y esofágica media bajaron durante la anestesia general. En promedio, las temperaturas esofágicas medias se hallaban a 0,6 °C por debajo de las temperaturas rectales medias durante la primera hora de la anestesia. En casi un 20% de los pacientes, ocurrió un aumento de temperatura interna. Esto parece relacionarse con una temperatura corporal inicialmente baja. Las temperaturas internas durante la anestesia general eran mucho mayores en pacientes que habían recibido una mezcla de suxametonio y de halotano que en los a quienes se había administrado otras substancias. Esta observación posee un interés teórico y hace pensar que el aumento de temperatura en la hiperpirrexia maligna puede constituir una exageración de la respuesta normal a dichos agentes.