INVESTIGATION OF THE RELATIONSHIP BETWEEN HEAT LOSS AND NITROGEN EXCRETION IN ELDERLY PATIENTS UNDERGOING MAJOR ABDOMINAL SURGERY UNDER GENERAL ANAESTHETIC

F. CARLI, M. M. CLARK AND J. W. WOOLLEN

SUMMARY
An attempt was made to reduce heat loss in elderly patients undergoing major abdominal surgery. Two groups were studied. In one group, efforts were made to minimize heat loss by using a hot-water humidifier in the anaesthetic circuit, a hot-water circulating mattress under the patient and warming all i.v. fluids. Otherwise, the surgical and anaesthetic techniques were comparable. The same anaesthetic technique of nitrous oxide, oxygen, pancuronium and fentanyl with intermittent positive pressure ventilation was used in all cases. Nitrogen loss was measured in urine collected over 48 h from an indwelling urinary catheter inserted soon after induction of anaesthesia. Prevention of heat loss during anaesthesia and postoperative recovery caused a significant reduction in nitrogen loss.

Human body temperature is maintained within normal limits as a result of the balance between heat production and heat loss. During anaesthesia and surgery, there is a decrease in body temperature caused by impaired heat production and loss of heat by radiation and convection. Anaesthetic agents contribute to heat dissipation by causing cutaneous vasodilatation whilst neuromuscular blocking agents prevent shivering, thus reducing heat production. Inhalation of dry gases and administration of i.v. fluids at room temperature contribute to a decrease in body temperature (Boyan and Howland, 1961). The type of surgery also influences heat loss; thus operations on body cavities cause considerable heat loss (Dyde and Lunn, 1970). Age is an important factor in temperature balance, and the elderly have decreased heat production as well as altered thermoregulatory response (Editorial, 1977).

Ambient temperatures in the operating and recovery rooms used in this study were kept in the range 20–22 °C with a low relative humidity. This is comfortable for staff, but may be detrimental to elderly patients undergoing long operations (Morris, 1971).

Nitrogen excretion was measured in an attempt to quantify the clinical impression that patients were improved by heat conservation. Cuthbertson and Tilstone (1969) and Cuthbertson (1976) showed that, in animals and humans, transfer to a warm environment reduces the nitrogen loss usually seen during the “flow” period following trauma. In these studies, there was a time lag between injury and attempts to maintain normothermia. Benzinger (1969) showed that reduced body temperatures provide a stimulus for increases in basal metabolic rate. During the recovery phase of anaesthesia, any heat debit incurred during operation is repaid by shivering, vasoconstriction and piloerection, which are efficient mechanisms of heat production in the adult at a skin temperature of 33 °C and a core temperature of less than 37 °C. However, oxygen consumption in patients who shiver is increased by 100–400% over basal values (Burton and Edholm, 1955; Roe et al., 1966). In frail, elderly patients already depressed by anaesthetic agents this could lead to arterial hypoxaemia (Bay, Nunn and Pryse-Roberts, 1968).

PATIENTS AND METHODS
Patients aged 60 yr and older were studied; all were classified as ASAII or III. Any patient with a metabolic disorder or who had a significant degree of weight loss or was in a poor nutritional state was excluded since patients who were protein-depleted before operation would be unable to excrete much nitrogen after operation. Patients were randomly

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allocated to two groups, A and B (table I). All patients underwent elective major abdominal surgery. Group A served as a control while in group B (afterwards referred to as the test group), efforts were made to minimize heat loss. After premedication with pethidine 1 mg kg\(^{-1}\) and phenergan 0.4 mg kg\(^{-1}\) i.m., anaesthesia was induced with thiopentone 5 mg kg\(^{-1}\). Suxamethonium chloride 1 mg kg\(^{-1}\) was used to facilitate intubation and pancuronium bromide 0.1 mg kg\(^{-1}\) to provide subsequent relaxation. Maintenance of anaesthesia was with 66% nitrous oxide in oxygen, supplemented by droperidol 0.25 mg kg\(^{-1}\) and fentanyl in increments of 50 mg as indicated clinically. The lungs were ventilated with a minute volume of 100 ml kg\(^{-1}\). In the test group, inspired gases were warmed and humidified using a hot-water humidifier (East) set at 50°C. Fluids were infused i.v. at 4 ml kg\(^{-1}\) h\(^{-1}\) and five patients in each group required blood transfusion during surgery. Those in the test group had all their i.v. fluids warmed at 37°C using a Grant BN2 blood warming bath.

A Sheffield water circulating mattress was set at a thermostatically controlled temperature of 37°C and placed under each patient in the test group during surgery. As soon as the operations were completed, a metallized plastic sheet was placed over the patients in the test group to minimize heat loss. Either 0.5% chlorhexidine in spirit or betadine was used to prepare the skin and double drapes were placed over the patient.

The ambient temperature around the patient and the mean skin and core temperatures were moni-
tored before induction, every 30 min during surgery and in the recovery room for 1 h. An electrical thermometer (Comark) was used which had been previously calibrated and checked against a mercury thermometer. Mean skin temperatures were calculated using the four points formula proposed by Ramanathan (1964):

\[
T^\circ_{\text{skin}} = 0.3(T^\circ_{\text{chest}} + T^\circ_{\text{arm}}) + 0.2(T^\circ_{\text{thigh}} + T^\circ_{\text{calf}})
\]

Four skin probes were situated lateral to the nipple, on the lateral aspect of the upper arm, the ventral surface of the mid thigh and the lateral aspect of the mid calf. Core temperature was measured by inserting a probe into the auditory canal. Total body temperature was derived from the following equation (Colin et al., 1971):

\[
T^\circ_{\text{body}} = 0.66 \times T^\circ_{\text{aural canal}} + 0.34 \times \text{mean skin temperature}
\]

\[
\text{Total body heat (kJ m}^{-2}\text{)} = \frac{T^\circ_{\text{body}} \times 0.83 \times \text{body weight} \times 4.1868}{\text{surface area}}
\]

Skin surface area was calculated from the patients' weight and height (Dubois and Dubois, 1915). Heat content related to body surface area could then be derived as could the change in heat content during operation (table II):

\[
T^\circ_{\text{body at end of op. (°C)}}
\]

<table>
<thead>
<tr>
<th>Control group (A)</th>
<th>Test group (B)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>Mean</td>
</tr>
<tr>
<td>Age (yr)</td>
<td>70.8</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>67.1</td>
</tr>
<tr>
<td>Surface area (m²)</td>
<td>1.67</td>
</tr>
<tr>
<td>Duration of op. (min)</td>
<td>145.00</td>
</tr>
<tr>
<td>Ambient temperature (°C)</td>
<td>21.2</td>
</tr>
<tr>
<td>Change in body temperature (°C)</td>
<td>-1.03*</td>
</tr>
<tr>
<td>Change in patient's heat content kJ m(^{-2}) h(^{-1})</td>
<td>-60.7*</td>
</tr>
<tr>
<td>Body temperature at end of op. (°C)</td>
<td>35.13*</td>
</tr>
</tbody>
</table>
Heat loss and nitrogen excretion

\[ \text{kJ m}^{-2} \text{h}^{-1} = \Delta T \times \text{body} \times 0.83 \times \text{body weight} \times 4.1868 \times 60 \]
\[ \text{surface area} \times \text{duration of operation (min)} \]

Urine was collected for 48 h from an indwelling bladder catheter inserted at the beginning of the operation. All collecting vessels contained 10 ml of 2% boric acid as preservative. Postoperative fluids were administered according to the procedure:

40 ml kg\(^{-1}\) 24 h\(^{-1}\), Na\(^+\) intake of 3 mmol kg\(^{-1}\) 24 h\(^{-1}\) and K\(^+\) 0.3 mmol kg\(^{-1}\) 24 h\(^{-1}\). Urinary urea, sodium and potassium concentrations were measured (Technicon SMA 6/60 analyser) over the first and second 24-h periods from the start of the operation. Plasma concentrations were also measured before and after operation. Nitrogen excretion was calculated from the urinary excretion according to the formula:

\[ \text{urinary nitrogen} = \text{urinary urea} \times \frac{28 \times \text{5 g} \times 24 \text{ h}^{-1}}{1000} \times \frac{4}{3} \]

assuming that 80% of the waste nitrogen was excreted as urea (Allison, 1974). The catabolic index was calculated according to Bistrian (1979):

\[ \text{urinary nitrogen (g} \times 24 \text{ h}^{-1}) = \frac{0.5 \times \text{dietary nitrogen g} \times 24 \text{ h}^{-1} + 3}{\text{plasma urea (mmol} \times 24 \text{ h}^{-1})} \]

Results

The body temperature changes in the two groups during operation are shown in table II. In all patients, normothermia was achieved and maintained within a few hours of recovery. There was significantly less heat loss in the test group (6.8 kJ m\(^{-2}\) h\(^{-1}\)) compared with the control group (60.7 kJ m\(^{-2}\) h\(^{-1}\)). These differences were associated with significantly reduced mean postoperative urea excretion in the test group which was less than half that in the control group (table III). The derived nitrogen ex-

| TABLE III. Urinary excretion of urea, nitrogen, sodium and potassium in 24-h period starting from induction of anaesthesia and a further 24-h period following operation. Statistically significant differences between values for test and control groups: *P < 0.001; statistically significant difference between values for operative and postoperative periods in the same group: †P = 0.001–0.005; ‡P = 0.005–0.01 |

<table>
<thead>
<tr>
<th>Control group (A)</th>
<th>Test group (B)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>No.</strong></td>
<td><strong>Mean</strong></td>
</tr>
<tr>
<td><strong>Urine volume</strong> (ml 24 h(^{-1}))</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>921</td>
</tr>
<tr>
<td>8</td>
<td>215†</td>
</tr>
<tr>
<td>8</td>
<td>0.12†</td>
</tr>
<tr>
<td>8</td>
<td>4.51†</td>
</tr>
<tr>
<td><strong>Serum urea (mmol litre(^{-1}))</strong></td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>6.05</td>
</tr>
<tr>
<td><strong>Urea clearance</strong> (ml min(^{-1}) 1.73 m(^{-2}))</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>57.1*</td>
</tr>
<tr>
<td><strong>Urine sodium</strong> (mmol 24 h(^{-1}))</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>47.9</td>
</tr>
<tr>
<td>10</td>
<td>40.1</td>
</tr>
<tr>
<td><strong>Urine potassium</strong> (mmol 24 h(^{-1}))</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>63.6</td>
</tr>
<tr>
<td>10</td>
<td>37.6</td>
</tr>
</tbody>
</table>

Plasma and urinary creatinine concentrations were also measured (Technicon SMA 6/60 analyser). In two patients from the control and three from the test group, the derived "maximal" (or true) urea clearance (Smith, 1951) was corrected to a body surface area of 1.73 m\(^2\); the derived creatinine clearances were similarly corrected for body surface area. Statistical analysis of the data was performed using Student's t-test. Results are presented as mean(± SD) and a statistically significant change was considered to have occurred when P values were 0.01 or less.
creatinin (related to body weight) and the urinary potassium excretion were similarly reduced in the test group (table III).

The derived mean postoperative urea clearance of the test group was also less than half that of the control group (table IV). Furthermore, there was a positive correlation ($r = 0.73$) between total body heat loss and postoperative urea clearance in patients sampled from both groups (fig. 1).

Heat loss was also positively correlated with the catabolic index (Bistrian, 1979) ($r = 0.66$) and urinary potassium excretion ($r = 0.65$) (table III).

The creatinine clearances measured after operation in five patients (table IV) showed the expected correlation with urea clearances over a wide range of values (Lubowitz et al., 1967; Lavender, Hilton and Jones, 1969) confirming that, in this study, the urea clearance was a valid approximation to the glomerular filtration rate.

**DISCUSSION**

This study attempted to standardize patient age, operative procedure and environmental temperature to determine the changes which occur in body temperature and heat content in elderly patients during surgery and anaesthesia and how these changes can be modified. The mean skin temperature was determined using the four-point formula described by Ramanathan (1964). This method and the other 11 different formulae described by several authors, have been compared by Shanks (1975) with the 15-site reference value. The four-point formula was found to reach a 95% agreement to within 1°C of the mean skin temperature during anaesthesia. Holdcroft and Hall (1978) compared the 15-site method of Mitchell and Wyndham (1969) with the four-point method during anaesthesia and recovery and found sufficient agreement.

The core temperature was recorded by an auditory canal probe chosen because it is well tolerated by the patient, easily inserted and does not cause trauma. Provided steps are taken to avoid draughts and cold air, aural canal temperature correlates well with oesophageal temperature (Holdcroft and Hall, 1978).

The results obtained are in agreement with those of Goldberg and Roe (1966) who described rapid

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**TABLE IV. Urea and creatinine clearance (values in five patients)**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Urea clearance (ml min$^{-1}$/1.73 m$^{-2}$)</th>
<th>Creatinine clearance (ml min$^{-1}$/1.73 m$^{-2}$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Test group (B)</td>
<td>20</td>
<td>53</td>
</tr>
<tr>
<td>2 Test group (B)</td>
<td>22</td>
<td>52</td>
</tr>
<tr>
<td>3 Test group (B)</td>
<td>6</td>
<td>8</td>
</tr>
<tr>
<td>4 Control group (A)</td>
<td>52</td>
<td>109</td>
</tr>
<tr>
<td>5 Control group (A)</td>
<td>61</td>
<td>87</td>
</tr>
</tbody>
</table>
cooling in patients older than 60 yr. The initial decrease in body temperature is marked during the first 0.5 h after induction of anaesthesia. During this time, there are large differences between the naked patient and the ambient theatre temperature and the skin is cleaned with cold liquids. After draping, the initial rapid decrease in temperature lessens. Average hourly heat loss was found in our study to be much greater in the unwarmed group A, but no correlation was found between the rate of decrease in core temperature and average hourly heat loss in either group. Vale (1973) has explained this is caused by redistribution of heat from the deep part of the body to the skin surface. Patients in whom all efforts were made to minimize heat loss (group B) showed stable heat content throughout the operation.

The measurement of urinary urea during the first 48 h after operation was used to establish the relationship between heat loss during operation and the catabolic response. The finding that reduction of body heat loss during operation resulted in substantially reduced nitrogen excretion in the test group is in accord with studies which showed a similar effect in animals (Cuthbertson and Tilstone, 1969; Cuthbertson, 1976) and in human subjects (Cuthbertson, Smith and Tilstone, 1968; Cuthbertson et al., 1972; Fleck, 1976) who were transferred to a warm environment immediately following trauma such as bone injury, burns or myocardial infarction.

However, Spivey and Johnston (1972) found no reduction in the nitrogen excretion of patients who were nursed at 29°C rather than 24°C following abdominal surgery. This would appear to be in contrast to results from the present study but, like the other series referred to, the warm environment was instituted after the trauma occurred and not, as in the present study, during the period of trauma itself. We are unaware of any previous metabolic study of this nature.

Spivey and Johnston (1972) suggested that the discrepancy of their findings with those of Cuthbertson, Smith and Tilstone (1968) might be caused by the relatively mild trauma and catabolic response of their patients compared with that of the bone injured patients of the latter authors. The difference in the severity of stress may be a further cause of discrepancy between their findings and the results in this study since the catabolic index (mean 12.1) of the control group corresponded to severe stress according to Bistrian (1979). A further difference is that the mean ages of our two groups were 70.8 and 76.8 yr compared with 46.4 and 44.1 yr in the corresponding groups of Spivey and Johnston (1972).

Mean urinary potassium excretion after operation in the test group was significantly less than in the control group, and combining data from both groups, there was a positive correlation between urinary potassium and urinary nitrogen excretion ($r = 0.80$) similar to that found by Cuthbertson and others (1972) on patients with bone trauma. These findings are consistent with the concept that post-traumatic energy requirement is provided, at least in part, by gluconeogenesis from amino acids derived from muscle catabolism (Cuthbertson et al., 1972; Cuthbertson, 1976) and that this energy production, which is directly related to nitrogen excretion (Cairnie et al., 1957; Cuthbertson, 1976) may be reduced in subjects who are maintained in a warm environment.

The mean urea clearance value after operation in the control group corresponded, on the basis of data from Lavender, Hilton and Jones (1969), to an inulin clearance of approximately 80 ml min$^{-1}$ which was similar to that found by Rush, Fishbein and Wilder (1965) in a group of elderly postoperative patients.

In the test group, the mean urea clearance after operation was less than half that of the control group. This reduction in clearance was not associated with any increase in plasma urea concentration; on the contrary, the concentration was slightly, although not significantly, less, and it therefore seems unlikely that the reduced urea excretion of this group can be attributed only to reduced clearance. The data obtained are consistent with the concept that there was, in the test group, a reduction both in the generation of urea and the glomerular filtration rate compared with the corresponding values in the control group. No explanation can be offered for this latter observation, nor for the additional finding that overall, in both groups of patients, urea clearance was positively correlated with total body heat loss (fig. 1). The effect of warm ischaemia on renal function is well known (Whickham, Hanley and Joekes, 1967), but we are not aware of any study of the effects on renal function of small differences in temperature such as occurred in the present study. Some degree of renal ischaemia would be expected in the patients studied, because of anaesthesia itself (Bastion and Deutsch, 1976), and it may be that the relative warmth of the test group of patients was sufficient to cause the observed reduction in glomerular filtration rate which,
as in other studies of more extreme conditions (Whickham, Hanley and Joekes, 1967) was manifest 24 h after the ischaemia. Further studies are needed, before and after operation, of renal, creatinine and urea clearances in order to confirm and clarify these findings.

It is therefore suggested that the attempt to minimize the metabolic effects of anaesthesia and surgery by maintaining the patient as far as possible in a thermoneutral environment, is alternative, or complementary, to extradural analgesia (Brandt et al., 1978) or high-dose fentanyl (Hall, 1980). Extradural analgesia and high-dose fentanyl, although effective, have a complication rate which may sometimes preclude their use, whereas prevention of heat loss is both beneficial and totally innocuous.

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REFERENCES


humidificateur chauffant sur le circuit d’anesthésie, un matelas à circulation d’au chaud placé sous le patient et un réchauffement de tous les liquides perfusés. Sur les techniques anesthésique et chirurgicale étaient comparables. Dans tous les cas, la même technique anesthésique, associant le protoxyde d’azote, l’oxygène, le pancuronium et le fentanyl, avec une ventilation en pression positive intermittente, a été utilisée. L’excrétion azotée a été mesurée dans les urines recueillies pendant 48 h par l’intermédiaire d’une sonde urinaire à demeure mise en place peu après l’induction de l’anesthésie. La prévention des pertes caloriques pendant l’anesthésie et la période de réveil a entraîné une diminution significative de l’excrétion azotée.

UNA INVESTIGACIÓN RESPECTO DE LA RELACIÓN ENTRE LA PÉRDIDA DE CALOR Y LA EXCRECIÓN DE NITRÓGENO EN PACIENTES DE EDAD SOMETIDOS A CIRUJÍA ABDOMINAL MAYOR BAJO ANESTESIA GENERAL

SUMARIO
Se intentó reducir la pérdida de calor en pacientes de edad sometidos a operaciones mayores de cirugía abdominal. Se estudiaron dos grupos. En un grupo, se hicieron esfuerzos para minimizar la pérdida de calor al usar un humidificador de agua caliente en el circuito anestésico, un colchón con sistema de circulación de agua caliente debajo del paciente y al calentar todos los fluidos i.v. Por otro lado, todas las técnicas anestésicas y quirúrgicas eran comparables. La misma técnica anestésica con oxígeno, pancuronio y fentanilo y ventilación de presión positiva intermitente fue utilizada. La pérdida de nitrógeno fue medida en todos los casos, en la orina recuperada durante 48 h. de la sonda urinaria interior introducida de inmediato después de inducción de la anestesia. La prevención de pérdida de calor durante la anestesia y la recuperación post-operatoria tuvo por efecto una reducción significativa de la pérdida de nitrógeno.