ACCIDENTAL HYPOTHERMIA TREATED BY CENTRAL REWARMING THROUGH THE AIRWAY

E. LL. LLOYD

SUMMARY

Eleven hypothermic patients were treated by heat supplied via the airway. Three patients died during rewarming, three died of unrelated causes several days after successful rewarming and five survived. Core temperatures were measured by a low reading mercury rectal thermometer and by a thermistor probe inserted into the rectum or mid-oesophagus. In every patient the core temperature rose without any initial after-drop. The problems of the method are discussed and the suggestion made that it might be applicable in rescue work, in the treatment of elderly hypothermic patients, and in combating accidental hypothermia in the operating theatre.

Accidental hypothermia, which is a serious condition with a high mortality, occurs in a wide variety of situations, e.g., during hill-walking or mountain climbing (Pugh, 1964, 1966), and caving (Lloyd, 1964), at sea (Keatinge, 1965; Pugh, 1968), and in towns among the elderly and those who have medical disorders or following drug overdosage (Emslie-Smith, 1958; Duguid, Simpson and Stowers, 1961; Cohen, 1968; Howitt, 1971). It can also occur during the summer months (Rees, 1958; Lloyd, 1972 unpublished observation), in operating theatres (Vale and Lunn, 1969; Searle, 1971; Newman, 1971) and in many parts of the world (Laufman, 1951; Kvittingen and Naess, 1963; Duckworth and Cooper, 1964; Kügler-Podelleck et al., 1965; Thomas and Gerber, 1965; Kugelberg et al., 1967; Lash, Bur dette and Ozdil, 1967; Dalgleish, 1969; Keatinge, 1969; Tolman and Cohen, 1970; Atukorale, 1971; Budd, Hicks and Macpherson, 1971; Gregory, 1971).

Rapid surface rewarming while recommended for conscious patients with rectal temperature of 34°C or above (Freeman and Pugh, 1969; Mountain Rescue, 1968), is dangerous because it tends to cause surface vasodilatation. Passive rewarming is slow and not always effective (Duguid, Simpson and Stowers, 1961).

Central rewarming is a safer route for supplying heat. The methods that have been tried, including peritoneal dialysis (Lash, Bur dette and Ozdil, 1967), extracorporeal blood warming (Davies, Millar and Miller, 1967) and thoracotomy with the application of warm saline to the pericardium (Linton and Ledingham, 1966) while effective, are not widely available.

Most cases of accidental hypothermia occur at sites remote from medical care and are admitted to hospitals which do not have facilities for extracorporeal warming. There is therefore a need for equipment which can provide central rewarming in an easily portable form. With this in mind apparatus has been designed which provides heated oxygen by utilizing the chemical reaction between carbon dioxide and soda lime (Lloyd et al., 1972). This is stated to have a maximum heat of reaction of 60°C (Adriani, 1962) though it can reach 104°C (Lloyd, 1972 unpublished observation). 60°C is a safe temperature since it is unlikely to cause damage in the airway even though the gases carry moisture vapour (Moritz, Henriches and McLean, 1945).

APPARATUS

1. The portable resuscitation equipment is shown in figures 1 and 2. This can only be employed when the patient is breathing spontaneously but by replacing the demand valve B with a flow meter the same equipment can be used for manual ventilation. The instructions for use are given later.

The carbon dioxide in the facepiece was measured while the Sparklets (BOC) were being used and the highest concentrations recorded were 4.7% on inspiration and 5.8% on expiration and these were only recorded during one or two breaths (Lloyd et al., 1972).

2. A modified Waters anaesthetic circuit can be used (fig. 3). The oxygen at 0.5–1 l./min and carbon...
Fig. 1. Diagrammatic representation of portable resuscitation equipment.

A Oxygen cylinder.
B Demand reducing valve.
C Sparkleks Corkmaster (BOC), with the distal portion of the needle removed and connected to the gas inflow limb of E. The oxygen supply is connected to the normal outflow part of the assembly.
D Reservoir bag of 6 l. capacity.
E Normal Waters valve assembly with the Heidbrink valve removed and the reservoir bag connected. The arrangement is necessary to transmit the inspiratory negative pressure to the demand valve B.
F Soda-lime.
G Waters canister.
H Insulation—expanded polystyrene in the box and air in the delivery tube.
J Facepiece or mouthpiece and nose clip as used by mine rescue or skin divers or endotracheal tube.
K Double lumen delivery tube. The inner lumen is the respiratory tube while the outer is closed off and acts as insulation. The length of the tube is not critical since any build-up of carbon dioxide due to the deadspace would be beneficial in protecting the heart from the effects of hypothermia.
L Rubber tubing to give a flexible mounting for the Corkmaster C.

dioxide at 3–5 l./min is allowed to flow until the temperature of the canister reaches about 60°C. This temperature can most conveniently be judged by hand, i.e., when the canister is just too hot to hold the temperature is about 60°C and the flow of carbon dioxide is then stopped, continuing with the oxygen only. When the temperature of the canister has dropped, as judged by hand comfort, further carbon dioxide is added to reheat the soda lime. The efficiency of the equipment is increased by insulating the canister and this also avoids the risk of causing burns resulting from contact of the hot canister with the patient’s skin. The soda lime should be changed after 3 hours use.

3. In hospital use, a conventional humidifier can be used with the water temperature set at 60°C. The tubing between the humidifier and patient must be well insulated. Heat can be delivered by spontaneous ventilation using a non-return valve at the facepiece or by using mechanical ventilation.

Instructions for use (fig. 1).

The oxygen cylinder (A) is opened and the facepiece (J) or mouthpiece strapped to the patient’s face. The patient is allowed to breathe for 2 minutes till the reservoir bag (D) starts to fill (this is done to ensure that an air-tight seal has been achieved round the mask and to flush the circuit with oxygen). The
leaver on the Sparklet Corkmaster (BOC) (C) is then depressed till the Sparklet cylinder of carbon dioxide is empty. The Corkmaster is reloaded with a fresh Sparklet cylinder, and the lever depressed till the cylinder is again empty. A total of three Sparklets each containing about 5 l. carbon dioxide is required to reach a temperature of about 60°C in the soda lime.

The apparatus is arranged with the canister (G) below the level of the facepiece (J). Thereafter the only attention required is to ensure that the air-tight seal is maintained between the face and the mask and check that the oxygen cylinder does not run out. After each hour a further Sparklet cylinder should be added and this maintains the temperature of the canister at between 50 and 60°C. The standard Waters canister used has a safe endurance hie of six hours.

If the equipment is modified for assisted ventilation the additional Sparklet required to maintain the temperature of the canister should be added every half hour and the soda lime changed after three hours.

The filling of the Waters canister with soda lime should follow accepted anaesthetic practice, i.e., to maintain an even pressure on the granules an ordinary nylon pot scourer is inserted before the end of the canister is screwed back into place. The soda lime should be replaced each time the apparatus is used.

METHOD

The patients treated were all admitted to Edinburgh Royal Infirmary and were found to be hypothermic on admission or became hypothermic in hospital. The early cases were treated with the modified Waters circuit but once the portable resuscitation equipment had been developed this was used in order to test it under controlled conditions. Some of the later cases were warmed using the adjusted humidifier. The equipment used in each case is shown in Table I.

All patients were wrapped in blankets to provide insulation and no additional heat was supplied.

The core temperatures were measured by low reading mercury rectal thermometer and by thermistor probe either inserted into the rectum or midesophagus.

RESULTS

Eleven hypothermic patients have been treated and the core temperature ranged from 24.3°C to 34.8°C with an age range of 22 to 86 yrs. Further details of the cases, the methods of treatment and clinical progress is shown in Table I. Three cases are.

### Table I. Brief details of cases, methods of treatment and clinical progress in 11 patients suffering from accidental hypothermia.

<table>
<thead>
<tr>
<th>No.</th>
<th>Sex</th>
<th>Age</th>
<th>Cause</th>
<th>Temp. at start of treatment</th>
<th>Method of warming</th>
<th>Temp. rise</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>F</td>
<td>60</td>
<td>Overdose of glutethimide, became hypothermic in hospital.</td>
<td>34.8°C</td>
<td>Modified Waters circuit, IPPV</td>
<td>0.6°C/hr</td>
</tr>
<tr>
<td>2</td>
<td>M</td>
<td>55</td>
<td>Overdose of phenothiazines.</td>
<td>27°C and steady over 4 hr</td>
<td>Modified Waters circuit, IPPV</td>
<td>0.4°C/hr</td>
</tr>
<tr>
<td>3</td>
<td>F</td>
<td>22</td>
<td>Distalgesic overdose.</td>
<td>34°C and steady over 4 hr</td>
<td>Modified Waters circuit, IPPV</td>
<td>0.5°C/hr</td>
</tr>
<tr>
<td>4</td>
<td>F</td>
<td>57</td>
<td>Found unconscious at home, presumed overdose of drugs.</td>
<td>28°C 7 hr after admission</td>
<td>Portable resuscitator S/V</td>
<td>0.4°C/hr for 1 hr</td>
</tr>
<tr>
<td>5</td>
<td>F</td>
<td>75</td>
<td>Found unconscious at home, evidence of cerebrovascular accident.</td>
<td>28°C</td>
<td>Portable resuscitator S/V</td>
<td>0.6°C/hr for 5 hr</td>
</tr>
<tr>
<td>6</td>
<td>F</td>
<td>80</td>
<td>Found on floor at home. Only femoral pulses palpable and shallow resp.</td>
<td>25.5°C 2 hr after admission</td>
<td>Portable resuscitator S/V</td>
<td>0.6°C/hr for 3 hr</td>
</tr>
<tr>
<td>7</td>
<td>F</td>
<td>23</td>
<td>Severe barbiturate overdose.</td>
<td>29°C</td>
<td>Portable resuscitator S/V</td>
<td>0.5°C/hr</td>
</tr>
<tr>
<td>8</td>
<td>F</td>
<td>39</td>
<td>Barbiturate overdose.</td>
<td>30°C. Drop from 33.5°C over prev. 3 hr</td>
<td>Portable resuscitator IPPV first then S/V</td>
<td>0.5°C/hr</td>
</tr>
<tr>
<td>9</td>
<td>F</td>
<td>73</td>
<td>Found lying at home.</td>
<td>32.5°C</td>
<td>Modified Waters circuit, IPPV</td>
<td>1°C/hr rise for 1 hr</td>
</tr>
<tr>
<td>10</td>
<td>F</td>
<td>86</td>
<td>Fractured ankle and lay on floor overnight.</td>
<td>24.3°C</td>
<td>Humidifier at 80°C, IPPV Barnet</td>
<td>1°C/hr rise</td>
</tr>
<tr>
<td>11</td>
<td>F</td>
<td>74</td>
<td>Whiplash injury causing quadriplegia. Hypothermia developed in hospital.</td>
<td>31°C</td>
<td>Humidifier at 50°C, S/V</td>
<td>0.2°C/hr</td>
</tr>
</tbody>
</table>
described in greater detail in the case reports. Of the 11 patients treated 5 recovered completely, 3 died during the rewarming and 3 died some time after having their temperature restored to normal. The 3 patients who died during rewarming were all allowed to breathe spontaneously and none of the patients who were warmed with assisted ventilation died during rewarming.

The most significant finding is that there was no after-drop of core temperature. The oesophageal temperature rose first, followed by the rectal. The rate of rise of temperature was faster when ventilation was assisted than when patients breathed spontaneously.

In one patient (case 3) no pulses were palpable when heating started and in two others (cases 4 and 6) the peripheral pulses were absent. In all these patients the blood pressure and skin colour improved and pulses became palpable steadily more peripherally. In all cases the arterial pressure improved and the pulse rate increased as the oesophageal temperature rose, irrespective of the degree of hypothermia. Assessment of the cardiovascular status was simplified by monitoring the central venous pressure.

In three patients (2, 3 and 7) the respiratory drive, which had been depressed by drug overdose, returned before the core temperature had risen 1°C and in two patients (3 and 7) the pupils, which had been dilated and non-reactive, returned to normal size and reactivity, before any rise in core temperature could be recorded.

There were no respiratory problems during or after the heating and when postmortem examination was carried out there was no evidence of damage to the larynx or trachea, except in case 10 where there was some laryngeal oedema and mild tracheal scalding. Continuous electrocardiographic monitoring failed to reveal any cardiac arrhythmias arising as a result of endotracheal intubation which was carried out at temperatures as low as 24.3°C (case 10). Cardiac arrhythmias attributable to hypothermia (J waves, and supraventricular and ventricular extrasystoles) disappeared rapidly on commencing treatment and normal sinus rhythm was established often at very low temperatures, e.g., by 27°C in case 10 (Lloyd, 1972).

CASE REPORTS

Case 2.

On a very cold night a 55-year-old man took an overdose of phenothiazine and opened the bedroom window before going to sleep. 6 hrs later he was admitted unconscious, the systolic pressure being 40 mm Hg and the pulse rate 50 beats/min. Metaraminol 5 mg i.m., atropine 0.6 mg, i.m., glucose by infusion and one 200 mg i.v. dose of hydrocortisone were given: the systolic pressure rose to 75 mm Hg but the pulse rate remained at 50 beats/min. Because he was cyanosed and making minimal respiratory efforts, intubation was performed and IPPV was instituted using a Bird ventilator. Blood-gas values at the time, while inspirating 100% oxygen, were: pH 7.33, Paco2, 62 mm Hg, oxygen saturation 100%. His temperature on admission was 27°C and, in spite of the patient being covered with blankets in a warm room, remained at 27°C over the next 4 hours. It was therefore decided to institute active airway rewarming. The temperature, pulse and blood pressure are shown in figure 4. Because there was no respiratory effort when heating started, IPPV was performed by hand using the modified Waters circuit. The pulse rate improved immediately while the blood pressure remained stable.

![Figure 4](#)

**FIG. 4.** Case 2. Chart showing the rapid improvement of temperature after heating was started and the continuing improvement thereafter of all parameters.

Figure 4 shows a delay of 30 minutes before the rectal temperature started to rise but once the rise started it continued steadily. After 14 hours IPPV was discontinued and the patient was found to be making adequate respiratory efforts and breathed through the apparatus spontaneously over the next hour. Active heating was then stopped and IPPV recommenced using the Bird ventilator but the temperature continued to rise steadily and reached 37°C 16 hours after the start of heating. There was a slight overshoot of temperature. Thereafter IPPV was discon-
continued, the endotracheal tube was removed and the patient could speak and respond to speech. 4 hours after starting the heating the blood pressure fell though the pulse rate continued to improve, plasma 1500 ml was rapidly infused and the arterial pressure rose to its previous level. Thereafter as long as the central venous pressure was maintained at +8 to +10 cm H2O the blood pressure and pulse rose steadily with the rising rectal temperature. The patient was discharged 5 days after admission.

Case 3.
A female aged 22 took an overdose of Distalgesic (dextropropoxyphene and paracetamol) and was admitted with feeble respiration and impalpable pulses. There had apparently been three episodes of cardiac arrest before admission and, shortly after admission, she developed cardiac asystole. Though resuscitation was successful, the pupils remained dilated and non-reactive and she was cyanosed. IPPV was commenced using the Bird ventilator and a central venous pressure catheter inserted.

In spite of hydrocortisone 200 mg i.v. and infusion of plasma to raise the c.v.p. the cardiovascular findings remained as before. Her rectal temperature 3 hours after admission was 34°C and it remained at 34°C over the next hour. A thermistor probe inserted into the oesophagus also registered 34°C. It was then decided to institute active rewarming. Heating was started using the portable resuscitation equipment modified for assisted ventilation. The first noticeable finding was that the pupils returned to normal size and reacted to light. Within 15 minutes the oesophageal temperature started to rise, followed 30 minutes later by the rectal. At this time it was noticed that the respiratory drive had returned and she could maintain her skin colour with spontaneous ventilation. It was, however, decided to continue with IPPV. Over the next hour the circulation improved with pulses becoming palpable progressively more peripherally and 2 hours after commencing treatment the blood pressure could be recorded for the first time (75 mm Hg systolic). Thereafter the pulse rate and the blood pressure rose with the temperature. Three hours after the start of heating the oesophageal temperature was 35.5°C and the rectal was 35°C. Heating was stopped and IPPV continued with the Bird ventilator. Four hours later IPPV was discontinued and the patient extubated. However, 5 hours after heating was discontinued the temperature had surprisingly not risen and was falling slightly (probably because of almost total inactivity combined with vasodilatation). She was slightly cyanosed but was breathing. It was decided therefore to supply heat again, using the portable resuscitation equipment with spontaneous ventilation. The colour improved and, after a delay of 1 hour, her rectal temperature rose again and heating could be discontinued because the temperature had reached 37.8°C (fig. 5). Unfortunately, unconsciousness persisted and because ventilation was inadequate, as judged by the results of blood-gas analysis, she was transferred to the Ventilation Unit. Three days later she could be safely extubated and she was transferred back to her previous Unit after a further 5 days. At that time she was fully conscious though there still appeared to be some minor impairment of cerebral function which, however, improved over the next few months.

Case 7.
A 23-year-old girl was admitted having taken an overdose of barbiturate (serum barbiturate level 5.6 mg/100 ml). She had been previously admitted suffering from salicylate overdose 3 years before. On admission she was unconscious and did not react to painful stimuli and although still breathing, she was cyanosed. 100% oxygen was given by facemask and blood-gas values were pH 7.37, Po2 32 mm Hg, St. HCO3 28.5 m-equiv/l, oxygen saturation 98%. (These figures are corrected for temperature.) An endotracheal tube was passed easily prior to gastric lavage. Treatment was given with IPPV with a Bird ventilator because her tidal volume was very shallow. She was covered with blankets and put in a warm room. The core temperature, which had been 30°C (rectal) on admission, continued to fall despite the transfusion of 500 ml of warmed plasma. She was given hydrocortisone 200 mg i.v. and fluid was monitored by c.v.p. readings. An hour later, when the oesophageal temperature was 29°C, rewarming through the airway was instituted. The portable resuscitation equipment was used and respirations which had been shallow became deeper and she maintained her colour.

There was no further fall of oesophageal temperature and after a delay of an hour the oesophageal temperature rose steadily. Within 12 hours of the start of heating it reached 36.8°C. At this point heating was discontinued. The rectal temperature which was taken at intervals lagged about 1°C behind the oesophageal. Blood-gas values one hour after starting active warming were pH 7.40, Po2 30 mm Hg, St. HCO3 21 m-equiv/l, oxygen saturation 98%. A radiogram showed that the oesophageal probe lay behind the heart shadow. Her pupils which had been dilated and non-reactive to light on admission contracted to normal size the following day. Though still breathing, she was cyanosed but was breathing. It was decided therefore to supply heat again, using the portable resuscitation equipment modified for assisted ventilation. The first noticeable finding was that the pupils returned to normal size and reacted to light. Within 15 minutes the oesophageal temperature started to rise, followed 30 minutes later by the rectal. At this time it was noticed that the respiratory drive had returned and she could maintain her skin colour with spontaneous ventilation. It was, however, decided to continue with IPPV. Over the next hour the circulation improved with pulses becoming palpable progressively more peripherally and 2 hours after commencing treatment the blood pressure could be recorded for the first time (75 mm Hg systolic). Thereafter the pulse rate and the blood pressure rose with the temperature. Three hours after the start of heating the oesophageal temperature was 35.5°C and the rectal was 35°C. Heating was stopped and IPPV continued with the Bird ventilator. Four hours later IPPV was discontinued and the patient extubated. However, 5 hours after heating was discontinued the temperature had surprisingly not risen and was falling slightly (probably because of almost total inactivity combined with vasodilatation). She was slightly cyanosed but was breathing. It was decided therefore to supply heat again, using the portable resuscitation equipment with spontaneous ventilation. The colour improved and, after a delay of 1 hour, her rectal temperature rose again and heating could be discontinued because the temperature had reached 37.8°C (fig. 5). Unfortunately, unconsciousness persisted and because ventilation was inadequate, as judged by the results of blood-gas analysis, she was transferred to the Ventilation Unit. Three days later she could be safely extubated and she was transferred back to her previous Unit after a further 5 days. At that time she was fully conscious though there still appeared to be some minor impairment of cerebral function which, however, improved over the next few months.
DISCUSSION

Under hypothermic conditions there is a heat transfer gradient from the surface "shell" to the centre "core". This implies that there is a continuing "after-drop" of core temperature even after all further skin heat loss has been prevented. By the time a victim reaches hospital the heat transfer has probably stopped and the core temperature will be steady (Freeman and Pugh, 1969), but if active surface rewarming is then instituted the peripheral blood vessels dilate thus allowing a surge of cold blood to reach the core. The fall of cardiac temperature may lead to ventricular fibrillation. The inefficient cold heart also has to accommodate an increased load and may fail. This after-drop which occurs during the first phase of rewarming may amount to 3-4 deg. C (Freeman and Pugh, 1969).

Davies, Millar and Miller (1967) believed that rapid rewarming from within outwards would be a safe method and used extracorporeal blood warming to achieve this. The lungs and respiratory passages in man are, however, very efficient heat exchangers and it seems reasonable to use the body's built-in mechanism rather than to resort to the use of an extracorporeal circuit. Furthermore the alveolar area of the lungs is twenty times the area of the skin so that the heat exchange potential is great.

When heat is supplied through the air passages the oesophageal (cardiac) temperature would be expected to rise first, followed a short time later by the rectal temperature. This is important in the treatment of cases of hypothermia because heat is supplied directly to the heart. There should therefore be no after-drop in core temperature on commencing active rewarming. These theoretical considerations were borne out in clinical practice (figs. 4 and 5).

The total amount of heat supplied through the respiratory system is small being about 1.2 kcal/sq.m/hr (Lloyd et al., 1972), but when the respiratory heat loss of 8 to 9 kcal/sq.m/hr is taken into consideration the difference in heat available to rewarm a hypothermic patient can amount to 25-30 per cent of the metabolic heat production (30 kcal/sq.m/hr at 30°C; cf. 50 kcal/sq.m/hr at 37°C (Lloyd et al., 1972)). This amount of heat, small in relation to the whole body, is transferred entirely to the blood and thence to the heart resulting in the improvement in cardiac function noted and the beneficial effect on the arrhythmias.

The stimulant effect on the brain is probably due to the warmed blood being circulated to the brain since the effective circulation is diminished in hypothermia.

The three patients (4, 5 and 6) who died during rewarming had all been hypothermic for at least 24 hours before discovery and there may be other problems involved in rewarming from prolonged hypothermia, e.g., cerebral oedema (Bloch, 1967). Since two of the patients (3 and 4) were elderly and died with gradual respiratory failure there is the possibility that this was due to the respiratory depressant effect of 100% oxygen at low body temperatures (Grosse-Brockhoff, 1950; Burton and Edholm, 1955). The fact that patients treated by intermittent positive pressure ventilation (IPPV) were all rewarmed successfully does not resolve the dilemma since IPPV will not only overcome the respiratory depression but will also diminish cerebral oedema. Case 4 illustrates an important point, namely that sudden violent movement can cause cardiac arrest (Freeman and Pugh, 1969), presumably by causing a surge of cold blood to the heart.

Case 10 died 48 hours after successful rewarming and the cause was found to be cerebral haemorrhage. This appears to be a depressingly common occurrence after an elderly hypothermic patient has been rewarmed successfully (Duguid, Simpson and Stowers, 1961).

All the patients treated had some evidence of cardiovascular depression, with low or unrecordable blood pressure, impalpable pulses or fixed dilated pupils, and this is usually considered to be a contraindication to the use of IPPV by automatic ventilator. However, the cardiovascular depression in these patients was probably primarily cardiac in consequence of a combination of hypothermia and drug effect, either directly on the myocardium or via respiratory depression with resulting hypoxia. Thus ventilation with heated gases resulted in immediate and continuing improvement in pulse rate and blood pressure (figs. 4 and 5).

This method of supplying heat may have applications in the field of rescue work (for example, mountain rescue), because the equipment can be made portable. The method may prevent the after-drop of temperature which can occur during evacuation of a victim, though it should be noted that the greatest heat gain for the exposure victim is effected by prevention of heat loss from the body surface. This method prevents heat loss from the respiratory tract, which can be considerable (Lloyd et al., 1972; Day, 1948), and supplies a small amount of heat which affects the heart directly. The method would thus fill
ACKNOWLEDGEMENTS

I am very grateful to Dr H. J. S. Matthew, Consultant Physician, Regional Poisoning Treatment Centre, Edinburgh Royal Infirmary, and Dr J. Innes, Dr J. Halliday Croom and Dr A. Doig, Consultant Physicians, Edinburgh Royal Infirmary, for permission to treat the patients under their care. I should also like to thank Dr H. J. S. Matthew and Dr D. B. Scott, Consultant Anaesthetist, Edinburgh Royal Infirmary, for help in preparing the paper. My thanks also to Mrs C. Cockburn for typing the many drafts required.

REFERENCES


REACTION HYPOTHERMIQUE ACCIDENTELLE TRAITEE PAR RECHAUFFEMENT CENTRAL EN EMPRUNTANT LES VOIES RESPIRATOIRES

SUMMARY

Notre malades présentant une hypothermie ont été traités par la chaleur administrée en empruntant les voies respiratoires. Trois malades décédèrent au cours de cette opération de réchauffement, trois autres moururent au bout de quelques jours, après un réchauffement réussi et pour des causes indépendantes de celui-ci. Enfin cinq survécurent. Les températures centrales furent mesurées à
l'aide d'un thermomètre rectal à mercure permettant la lecture de basses températures et en utilisant une sonde thermistor mise en place dans le rectum ou à mi-oesophage. Chez chacun des malades, la température centrale a accusé une augmentation sans aucune chute initiale. Les problèmes soulevés par cette méthode sont discutés et il est suggéré de mettre en œuvre celle-ci éventuellement dans le cadre de la réanimation, en vue du traitement de malades âgés atteints d'hypothermie et afin de lutter contre une hypothermie accidentelle en salle d'opération.

BEHANDLUNG DER AKZIDENTELLEN HYPOTHERMIE MITTELS ZENTRALER ERWÄRMUNG ÜBER DIE ATEMWEGE

ZUSAMMENFASSUNG


HIPOTERMIA ACCIDENTAL TRATADA MEDIANTE RECALENTAMIENTO CENTRAL POR LA VIA AEREA

RESUMEN

Once pacientes hipotérmicos fueron tratados con calor administrado por la vía aérea. Tres pacientes murieron durante el recalentamiento, tres murieron por causas independientes varios días después de un recalentamiento satisfactorio y cinco sobrevivieron. Las temperaturas centrales fueron medidas mediante un termómetro rectal de mercurio de lectura baja y mediante una sonda termistor introducida dentro del recto o esófago medio. La temperatura central aumentó en todos los pacientes sin ninguna caída-posterior inicial. Son discutidos los problemas de este método y se postula que pudiera ser aplicable en el trabajo de salvamiento, en el tratamiento de pacientes hipotérmicos ancianos y para combatir la hipotermia accidental en el quirófano.

ASSOCIATION OF ANAESTHETISTS OF GREAT BRITAIN AND IRELAND

Room 126, Tavistock House North, Tavistock Square
London, WC1H 9HR. 01-387 4782

The ANNUAL SCIENTIFIC MEETING for junior anaesthetic staff will take place from April 25 to 28, 1973, in Sheffield. Accommodation has been reserved in University Halls of Residence. The Scientific Programme includes sessions on the anaesthetist’s responsibilities towards:

Motorway Accidents; The Paediatric Department; Cardiological Problems; The Law

Three evenings will be fully occupied by social events.

Full details and registration form are available from:

The Hon. Secretary, Association of Anaesthetists of Great Britain and Ireland,
Room 126, Tavistock House North, Tavistock Square, London WC1H 9HR.