urate, and urea) was examined in 7364 middle aged British men. After allowing for the influence of alcohol consumption on serum biochemistry there was no substantial association of increased blood lead concentration with renal function. There was a weak positive trend towards increased serum urate concentration at high blood lead concentration and a reverse trend for serum urea concentration. These findings differ from those of a previous study carried out in Scotland on people selected because their homes were thought to have lead pipes or storage tanks.³ In that study raised blood lead concentration was associated with raised serum urea and urate concentrations and excessive lead in domestic water supplies was considered to have a harmful effect on the community's health. A review paper then questioned their use of arbitrary cut off points for serum urate and urea concentrations, particularly in an elderly population.⁹ In addition this case-control study took no account of alcohol consumption, which is now known to have a considerable effect on both blood lead concentration and indicators of renal function.

Several studies have reported that occupational lead poisoning can result in severe renal impairment. For instance, an investigation of renal function was carried out in 102 Rumanian patients with lead poisoning attributed to occupational exposure who had a mean blood lead concentration of 4 $\mu\text{mol/l}$ (83 $\mu\text{g}/100\text{ ml}$).¹⁰ Seventeen patients had chronic nephropathy, mostly after more than 10 years of exposure to lead. Thirteen of these patients had also developed hypertension. Studies such as this have stimulated inquiry into whether more moderate exposure to lead in the general population could affect renal function or blood pressure.

It has been suggested that, in areas where occupational lead nephropathy is common, patients seen at the stage of hypertension or chronic renal impairment typically have normal blood lead concentrations; their lead overload is shown only

by bone biopsy or an edetic acid excretion test. Thus the possibility that exposure to lead earlier in life might account for subsequent hypertension or renal disease cannot be excluded. Our cross sectional study, however, using current blood lead concentrations, has not shown any evidence that recent exposure to lead at concentrations commonly experienced by British men is responsible for increases in blood pressure or impaired renal function.

The British Regional Heart Study is supported by a programme grant from the Medical Research Council. Biochemical estimations were carried out at the Wolfson Research Laboratories under a programme supported by the Department of Health and Social Security. Blood lead concentrations were estimated in the department of chemical pathology and human metabolism, University of Southampton (Professor B Clayton), supported by the DHSS.

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(Accepted 18 July 1984)

Cardiac arrhythmias during rewarming of patients with accidental hypothermia

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Abstract

Accidental hypothermia has a high mortality and is associated with cardiac arrhythmias. To determine the incidence of arrhythmias and their importance 22 patients with accidental hypothermia (core temperature $<35^{\circ}\text{C}$) were studied by 12 lead electrocardiography and continuous recording of cardiac rhythm. Although 14 of the patients died (64%), only six died while hypothermic. Prolongation of the Q-T interval and the presence of J waves were related to the severity of the hypothermia. Supraventricular arrhythmias, including atrial fibrillation, were common (nine cases) and benign. Ventricular extrasystoles were also common (10 cases), but ventricular tachycardia or fibrillation did not occur

during rewarming. In eight patients who died while being monitored the terminal rhythm was asystole. There was no correlation between the severity of hypothermia or the rate of rewarming and the clinical outcome.

In the absence of malignant arrhythmias there is no indication for using prophylactic antiarrhythmic treatment in patients with accidental hypothermia. The presence or absence of severe underlying disease is the main determinant of prognosis.

Introduction

Accidental hypothermia is a common reason for admission to hospital during the winter¹⁻³ and has a high mortality, especially in the elderly.³⁻⁵ The condition is associated with life threatening arrhythmias, in particular ventricular fibrillation, and there have been several reports of prolonged but successful resuscitation in such patients.⁵⁻⁹ Our aim was therefore to record continuously the cardiac rhythm of hypothermic patients during rewarming in order to discover the incidence of arrhythmias and whether they contribute to the mortality.

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Patients and methods

Twenty two patients admitted to Glasgow Royal Infirmary with a diagnosis of accidental hypothermia (core temperature 35°C or less) were entered into the study irrespective of their underlying condition. The patients represented about 1.3% of the acute medical admissions during the period of the study.

On admission the patient's core temperature was measured using a low reading rectal thermometer, and this was repeated every two hours during rewarming. A standard 12 lead electrocardiogram was recorded. Continuous cardiac rhythm was acquired by means of an Oxford Medical Instruments Medilog 24 hour recorder using magnetic tape. Tapes were analysed by a Reynolds Medical Electronics high speed electrocardiogram analyser interfaced to a PDP8A micro-computer. This system automatically captures arrhythmic events, produces an interpretation, and prints the relevant strip. Arrhythmic events detected by the system were then analysed independently by two observers. Cardiac rhythm was monitored during rewarming until core temperature exceeded 35°C. Blood pressure was measured by cuff sphygmomanometer. Venous and arterial blood samples were taken for biochemical analysis, including blood gas values, corrected for temperature.

Patients remained under the care of the admitting physicians. In all but one case the patients were passively rewarmed with appropriate treatment of precipitating factors and intercurrent illness. One patient was actively rewarmed using peritoneal dialysis and warm air ventilation (case 16; table).

The group Wilcoxon rank sum test was used for statistical analysis.

Results

The table lists the clinical details of the 22 patients studied. Twelve were men and 10 women, and their ages ranged from 30 to 101 years (mean 69). Fourteen patients had a core temperature less than 32.3°C, indicating severe hypothermia.¹⁰ Alcohol excess was considered to be the precipitating factor in five cases, while five other patients had been found at home with no aetiological factor identified. Underlying medical conditions were present in over half of the cases. Nine patients had hypotension, with a systolic blood pressure of 90 mm Hg or less.

Results of arterial blood gas analysis were available for 18 of the patients, six of whom had hypoxaemia ($PO_2 < 9.3$ kPa; < 70 mm Hg), and 10 patients were acidotic, one of whom had diabetic ketoacidosis. Three patients (cases 11, 13, and 22) had greatly raised serum amylase activities (> 1000 IU/l), and one (case 6), whose initial serum amylase activity was normal, had acute pancreatitis diagnosed at necropsy.

CLINICAL OUTCOME

Passive rewarming produced an increase in body temperature of all patients (range 0.1-1.8°C/hour, mean 0.7°C/hour). Active rewarming with peritoneal dialysis (1.4°C/hour) was successful in one patient.

Clinical details

Case No	Age and sex	Temperature (°C)	Rate of rewarming (°C/hour)	Heart rate/min	Blood pressure (mm Hg)	Underlying condition	Outcome	
							Died/survived	Temperature at death (°C)/rewarmed
1	60 M	25.3	0.4	34	60/40	Septicaemia	Died 5 hours*	27.0
2	62 M	26.0	0.4	40	70/30	Self neglect	Died 11 hours	28.0
3	61 M	26.5	0.7	38	110/70	Heart failure	Died 12 hours	33.0
4	81 F	26.8	1.4	47	70/50	Found at home	Died 1 hour	28.2
5	45 F	28.0	0.6	85	60/50	Paracetamol overdose	Died 10 hours	33.0
6	101 F	32.7	0.8	70	120/80	Bronchopneumonia	Died 6 hours	35.0
7	30 M	28.5	1.1	52	80/40	Diabetic ketoacidosis, pneumonia	Died 14 hours	Rewarmed
8	78 M	28.6	0.9	55	80/50	Cerebrovascular accident	Died 11 days	Rewarmed
9	69 F	30.0	1.8	47	170/100	Cerebrovascular accident	Died 26 hours	Rewarmed
10	81 M	32.0	0.7	78	130/60	Cerebrovascular accident	Died 14 hours	Rewarmed
11	74 M	32.7	0.3	68	110/60	Bronchopneumonia, bronchial carcinoma	Died 11 days	Rewarmed
12	80 F	33.0	0.3	57	120/60	Found at home	Died 26 hours	Rewarmed
13	54 F	33.3	0.5	75	90/50	Cerebrovascular accident	Died 7 hours	Rewarmed
14	81 M	33.9	0.1	84	130/90	Found at home	Died 12 hours	Rewarmed
15	86 F	24.0	0.8	65	170/100	Found at home	Alive	Rewarmed
16	56 M	25.5	1.4	35	40/0	Alcohol excess	Alive	Rewarmed
17	87 F	28.0	0.6	37	88/60	Hypothyroidism	Alive	Rewarmed
18	75 M	28.0	0.7	70	100/60	Alcohol excess	Alive	Rewarmed
19	63 M	32.0	0.9	75	108/70	Alcohol excess	Alive	Rewarmed
20	87 F	32.5	1.2	80	110/60	Found at home	Alive	Rewarmed
21	60 F	33.1	0.8	96	104/70	Alcohol excess	Alive	Rewarmed
22	56 M	34.5	0.3	86	110/70	Alcohol excess	Alive	Rewarmed

*Time of death after admission.

FIG 1—Case 16. Disappearance of J wave with rewarming. Tracings at (a) 26°C, (b) 28°C, (c) 28.5°C, (d) 29.5°C body temperature.

There was a high overall mortality with a total of 14 deaths (64%), but only six patients died while still hypothermic. The 16 other patients were successfully rewarmed, although eight died subsequently. Of these, six patients died shortly after rewarming, within 26 hours of admission, and two died at 11 days. One patient (case 9) had a cardiorespiratory arrest during hypothermia (34°C); she was resuscitated and subsequently rewarmed but died 24 hours later.

Factors that may have contributed to the mortality were examined. There was no significant difference in the temperature on admission or rate of rewarming between the patients who died while hypothermic and those who survived. Nevertheless, the clinical condition of those who subsequently died was generally worse than that of those who did not, with a higher incidence of hypotension (50% compared with 25%), hypoxia (36% compared with 12.5%), and acidosis (43% compared with 25%). No serious underlying medical condition was found in those who survived. This is in contrast with most of those who died (table). All the patients in whom alcohol excess was the only underlying condition were successfully rewarmed despite severe hypothermia in some.

ADMISSION ELECTROCARDIOGRAM

The initial 12 lead electrocardiograms showed that 19 of the patients were in sinus rhythm, of whom eight had sinus bradycardia with a heart rate of less than 60/min. Three patients (cases 4, 16, and 18) were in atrial fibrillation, with ventricular rates of 47, 35, and 70/min. The presence of bradycardia correlated significantly with the core temperature; the mean temperature of those with bradycardia was 27.8°C compared with 31.5°C in those without ($p < 0.02$).

No patient had heart block, and in those with sinus rhythm the P-R interval was 0.22 s or less. The Q-T interval corrected for heart rate (Q-T_c) was prolonged in 19 out of 21 patients (in case 6 the

patient had chronic left bundle branch block and was excluded from Q-T analysis), and the mean Q-T_c for the whole group was 0.49 s (range 0.40-0.55 s). Those patients with severe hypothermia (<32.3°C) had a significantly greater mean Q-T_c of 0.51 s compared with 0.44 s for the remainder ($p < 0.002$). J waves were present in half of the admission electrocardiograms and somatic tremor in 59%. Core temperature was significantly lower in patients with either feature (28°C) than in those without (32°C; $p < 0.02$). During re-warming both somatic tremor and the J waves disappeared (fig 1).

CARDIAC RHYTHM DURING REWARMING

The recordings of cardiac rhythm were technically satisfactory in all but four of the patients' tapes (cases 14, 20, 21, and 22), which were excluded because of artefact, poor quality recordings, or tape slip.

During rewarming there was a progressive acceleration of the heart rate in all patients. Two of the three patients with atrial fibrillation on admission reverted to sinus rhythm during rewarming, one (case 16) at 28°C and one (case 20) at 35°C. The remaining patient (case 4) was known to have chronic atrial fibrillation before admission and had been taking digoxin. Transient episodes of paroxysmal atrial fibrillation occurred in case 12 at 35°C and in case 9 after a cardiorespiratory arrest at 34°C. Other supraventricular arrhythmias during rewarming included premature beats in four patients, short episodes of nodal rhythm (60/min) in two, and non-sustained supraventricular tachycardia (140/min) in one. None of these supraventricular arrhythmias was relevant haemodynamically.

Ventricular extrasystoles occurred in 10 patients, unifocal in eight and multifocal with occasional pairs in two. The frequency of the extrasystoles did not change with increase in body temperature, nor did they herald more complex ventricular arrhythmias. An idioventricular rhythm at 70/min occurred in case 9 during the cardiorespiratory arrest. Despite the prolonged Q-T_c interval polymorphous ventricular tachycardia (torsade de pointes), which is associated with prolonged Q-T syndromes, did not occur.

TERMINAL RHYTHMS

Of the 12 patients who died within 26 hours of admission, continuous recordings of cardiac rhythm during the terminal period were available for analysis in eight. Of these patients, six died while still hypothermic (cases 1 to 6; table). The remaining two (cases 10 and 13) died with temperatures of 36.4°C and 36.5°C three and a half and five hours respectively after rewarming.

There was a progressive slowing of heart rate in all patients before death. Asystole occurred in all cases that were analysed. The asystole was interrupted by nodal escape rhythms in two patients (cases 2 and 5), and in four by low idioventricular rhythms (cases 5, 6, 10, and 13). Brief asystolic episodes recurred during the last hour before the terminal asystole, at 33°C, in case 5 (fig 2). Ventricular tachycardia was recorded in one hypothermic patient (case 4) at 28.2°C, but this occurred after prolonged periods of asystole (fig 3) and cannot be considered to be the primary terminal event. Ventricular fibrillation did not occur in any patient in the study.

Discussion

The effects of cooling on the cardiac rhythm were noted as early as 1912, when hypothermia produced slowing of the heart

FIG 2—Case 5. Tracings showing (a) short episode of asystole occurring one hour before (b) terminal asystole at body temperature 33°C.

FIG 3—Case 4. Ventricular tachycardia after prolonged asystole at body temperature 28.2°C.

beat of an isolated heart-lung preparation, terminating in cardiac standstill.¹¹ Electrophysiological experiments on isolated cardiac tissue also indicate depression of automaticity at low temperatures, rather than an increase in excitability.¹² In dogs, however, ventricular fibrillation occurs with hypothermia of 26-19°C, whereas asystole tends to occur at lower temperatures.¹³

Cardiac arrhythmias in hypothermia induced for cardiac surgery or neurosurgery are well documented.^{3, 14, 15} Atrial fibrillation is a common and benign arrhythmia, which reverts to sinus rhythm as the patient is rewarmed. Ventricular extrasystoles occur but are not premonitory of imminent ventricular tachycardia,¹⁴ or fibrillation.^{16, 17} The suggestion by Osborn that development of the J wave was a warning of ventricular fibrillation¹⁸ has not been confirmed.¹⁶ At lower temperatures ventricular fibrillation does occur¹⁴⁻¹⁶ and has been considered to be the prime cause of death in induced hypothermia.¹⁵

Whether such observations from induced hypothermia can be extrapolated directly to the uncontrolled setting of accidental hypothermia is questionable.¹⁹ In particular, in accidental hypothermia the primary arrhythmia may be asystole and not ventricular fibrillation.²⁰ Although there is no doubt that ventricular fibrillation may occur in accidental hypothermia,^{3, 4} in most of the reported cases its onset has been precipitated by external factors, such as the insertion of intracardiac pacing lines,⁶ endotracheal intubation,^{5, 7} prolonged external cardiac massage,⁸ and intravenous adrenaline.⁹

In our study no patient developed either ventricular tachycardia or fibrillation as a primary event in the terminal period, despite severe hypothermia. In all the recorded cases the terminal rhythm was asystole. Whether temporary pacing would influence the eventual outcome is uncertain,²⁰ but it is doubtful in patients with severe underlying disease. Certainly in the absence of malignant ventricular arrhythmias there is no indication for prophylactic antiarrhythmic treatment; and while the incidence of benign arrhythmias during rewarming was high these did not compromise the patients' circulatory state or require treatment.

The low incidence of serious arrhythmias in this study may have been because the patients were rewarmed passively and not subjected to invasive, possibly arrhythmogenic procedures.⁵⁻⁷ Early attempts at active rewarming were associated with a high mortality²¹ and passive rewarming was recommended. We, however, found no correlation between the rate of rewarming and subsequent mortality, and recent reports suggest that a more aggressive approach, with active rewarming, in fact leads to a lower mortality.^{4, 6, 8, 9} Indeed, in the presence of cardiac arrest active measures including cardiopulmonary bypass^{7, 8} or peritoneal dialysis²⁰ may be required for successful rewarming and resuscitation.

The main factor that determined mortality in this study was the presence or absence of severe underlying disease. This agrees with other reports of deaths from accidental hypothermia in urban populations who are mainly elderly and have other medical conditions^{3, 4, 22} and in New York alcoholics, in whom most deaths were due to underlying disease.²³ Conversely, active treatment of even severe hypothermia in the absence of other medical conditions should result in successful rewarming and recovery.⁴

In summary, there was a high incidence of arrhythmias during rewarming in accidental hypothermia. Most of these were benign and not harbingers of more lethal arrhythmias. Asystole, not ventricular fibrillation, was the primary rhythm in the terminal period. The severity of the underlying medical condition was more important than the degree of hypothermia or the rate of rewarming as a determinant of prognosis.

We thank Dr Peter W Macfarlane, reader in medical cardiology, for his help with this study.

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(Accepted 18 July 1984)

SHORT REPORTS

Motorcycling injuries in children

Motorcycle scrambling is becoming a popular sport with children. We report the injuries sustained by 24 children over two years. Half of these injuries resulted from scrambling and half from other motorcycling activities off the road. In view of their severity and apparently increasing incidence we suggest that careful records should be kept by accident units to define injury patterns and possible preventive measures.

Patients, methods, and results

From January to December 1983 a prospective record was kept of all children aged under 16 who attended the accident department at this hospital with injuries sustained while driving motorcycles. The department records were then searched to provide comparative, retrospective figures for 1982.

The injuries were graded using the abbreviated injury scale,¹ and for each patient the injury severity score was calculated as described by Baker *et al.*² The injuries were further graded as mild, moderate, or severe (maximum scores on the abbreviated injury scale of 1, 2, and 3 respectively).

Of the 88 140 patients who attended the accident department during the two years, 1446 were motorcyclists. Of these motorcyclists, 24 were aged under 16 (mean age 13.3 years, range 10-15 years). Half of these children were injured while scrambling at official club meetings and the other half while riding off the road. For comparison, 12 children were injured during the same period while travelling as pillion passengers on motorcycles on the road.

The figure shows the injuries sustained. The mean injury severity score was 6 (range 1-14). There was no significant difference between the injury severity scores of those riders injured while scrambling and those injured in other off the road accidents (Mann-Whitney test). When the injuries were graded as mild, moderate, or severe according to the maximum abbreviated injury scale eight patients fell into each of the three groups.

Comment

We have gained the impression, from patients and from motorcycle dealers, that official and unofficial motorcycle sports that take place off the road are gaining in popularity. There are no restrictions on the sale of motorcycles to or for children of any age, provided they are not ridden on the public highway, although scrambling clubs impose a minimum age of 5 for participation. Although the engine capacity of most of these machines is 125 cc or less, they are nevertheless capable

of considerable speeds, up to 100 km/h being not uncommon. The combination of fast machines and young riders, who have immature skeletons, constitutes a unique potential for injury.

At official club meetings a medical officer is usually in attendance, protective clothing is worn, and machines are inspected for safety. In unofficial motorcycling activities, however, the parents are responsible for safeguards. Many motorcycles are purchased with the aim of giving children the experience of motorcycling away from the dangers of the road. Our study shows that this activity carries considerable dangers of its own (three of our patients hit trees). In six cases injuries were caused by contact between the rider's leg or the motorcycle and an immovable obstacle, although this type of accident was

Individual injuries sustained by children driving motorcycles in 1982 and 1983, Oxford. Side of injuries as shown.