ACCIDENTAL HYPOTHERMIA

A COMMON CONDITION

BY

L. F. PRESCOTT,* M.B.**MARY C. PEARD, M.B., D.C.H.**

AND

I. R. WALLACE, M.B., D.C.H.*New End Hospital, London*

Accidental hypothermia is generally thought to be uncommon in this country (Rees, 1958). It is seen most often in the elderly and infirm, in patients with myxoedema (Summers, 1953; Malden, 1955; Macdonald, 1958), hypopituitarism (Sheehan and Summers, 1952), and in the newborn (Mann and Elliott, 1957). Recent interest in accidental hypothermia prompts this report of nine cases admitted to the general medical wards of a small hospital within a period of four months. This relatively high incidence (2.5% of acute medical admissions during that period) suggests that the condition is more prevalent than is generally believed, and that many cases pass unrecognized.

In every instance the diagnosis was unsuspected before admission, although the relatives of one patient had commented, quite rightly, that she "seemed to be very cold." As impairment of consciousness is usual, it is not surprising that the diagnoses on admission included cerebrovascular accident, diabetic coma, and uraemia. In addition to the nine reported cases of hypothermia with rectal temperatures below 90° F. (32.2° C.), several other patients were seen with temperatures in the range of 92–95° F. (33.3–35° C.).

Clinical Details

Eight of the nine cases were women, with ages ranging from 44 to 82 years, and the rectal temperatures on admission varied from 76 to 88° F. (24.4 to 31.1° C.). Details of the cases are set out in Table I, and relevant investigations in Table II. In most instances blood count, blood, urea, blood sugar, serum electrolytes, cholesterol, protein-bound iodine (P.B.I.), amylase, and transaminase estimations were carried out. Case 4 is reported below in greater detail.

Case 4

A woman aged 44 was admitted to hospital moribund, with no recordable pulse or blood-pressure. It was discovered later that she had fallen two days previously, bruising her forehead, but without losing consciousness. The next day she complained of headache, but when seen by her doctor she had no abnormal physical signs. That evening she had two glasses of red wine and took three "tuinal" capsules, as usual, on retiring. She had no recollection of subsequent events, and was found unconscious in her kitchen by a neighbour. The length of exposure could not have exceeded 12 hours.

On examination she was comatose and did not respond to painful stimuli. The rectal temperature was 76° F. (24.4° C.) no peripheral pulses were palpable, and the blood-pressure was unrecordable. Heart sounds were faint and irregular, and respirations were shallow at 12/minute. There was a small laceration with bruising over the right eye, both pupils were widely dilated with no response to

light, muscle tone was markedly increased, and tendon reflexes were unobtainable.

Mephentermine 30 mg. and hydrocortisone 100 mg. intravenously produced no obvious response and a noradrenaline infusion (16 mg./litre) was started. Forty minutes later the systolic blood-pressure became recordable at 90 mm. Hg. The rate of infusion was then reduced as pulmonary oedema developed. Aminophylline 250 mg. and hydrocortisone 100 mg. were given intravenously and thereafter her blood-pressure was maintained without difficulty, although noradrenaline was still necessary. Continuous electrocardiographic monitoring was available. Initially the heart rate was irregular at 50 to 70/minute with atrial fibrillation, and J waves were present in the left chest leads. Spontaneous reversion to sinus rhythm occurred later. Rapid improvement took place within the next two hours but bilateral papilloedema developed, and as the level of consciousness rose weakness of the left arm became apparent and a subdural or extradural haemorrhage was suspected. Skull x-ray films showed no abnormality, and as she was improving it was decided that a "watch and wait" policy was justified. Eight hours after her admission her temperature was 99.9° F. (37.7° C.); she was conscious, co-operative, and the papilloedema had receded. Weakness and sensory loss with absent reflexes in the left arm persisted, and this was now thought to be due to a brachial plexus injury. The next day she had three generalized convulsions and became progressively agitated and apprehensive, with tremor, widely dilated pupils, and profuse sweating. By the following day she had become confused, hallucinated, and violent, but responded rapidly to large doses of chlorpromazine. Further progress was uneventful.

On admission her blood count, E.S.R., blood urea, serum electrolytes, amylase, and transaminases were normal but the blood sugar was 332 mg./100 ml. No insulin was given, and two days later the fasting blood sugar was 107 mg./100 ml. A subsequent glucose-tolerance test was normal, as was the serum P.B.I. Electromyography indicated severe damage to C7, C8, and T1 nerve roots. She was discharged three weeks later, little the worse for her experience except for weakness of the left arm. The cause of her convulsions and behaviour disturbance remains obscure. The clinical picture suggested a barbiturate or alcohol withdrawal syndrome, but anoxia, pressor agents, steroids, and possible overhydration may have contributed.

Discussion

Environmental and Predisposing Factors

Obvious predisposing factors were present in all cases except two. In six cases the weather had been very cold at the time of admission, with average temperatures ranging from 32 to 37° F. (0 to 2.8° C.). Three patients, however, were admitted when the average temperature was above 45° F. (7.2° C.). All patients were found indoors in their own homes. Only four were living alone, and only one of these was found in a state of neglect (Case 9). Four patients had been in bed for at least 24 hours before admission and none of the remainder had been exposed for longer than 12 hours, with the possible exception of Case 9. Contrary to general belief, there is no doubt that hypothermia can develop without prolonged exposure to undue cold; and indeed Rees (1958) described the case of a 43-year-old woman with paraplegia and burns who became hypothermic in hospital during the summer months.

Case 4 is remarkable in that a severe and indeed almost fatal degree of hypothermia occurred in less than 12 hours in a comparatively young and otherwise healthy woman. The cause of her initial collapse is unknown, but her rapid recovery makes any significant overdosage of barbiturate or alcohol unlikely. Hypo-

*Present address: Boston City Hospital, Boston, Mass., U.S.A.

thermia following intoxication with alcohol and exposure is seen more often in North America, where more alcohol is consumed and winter temperatures are much lower. Thus of the eight cases of accidental hypothermia described by Frueman (1960) in Albany, three had taken alcohol, and Laufman (1951) described in detail the survival of a young woman admitted with a temperature of 64° F. (17.8° C.) following a drinking bout.

Administration of promethazine and chlorpromazine was thought to be the cause of hypothermia in one patient. Both drugs are known to lower body temperature (Laborit and Huguenard, 1951; Courvoisier *et al.*, 1953) and have indeed been used for this purpose. Mitchell *et al.* (1959) attributed the development of hypothermia in a myxoedematous woman to a single intramuscular injection of 100 mg. of chlorpromazine.

One patient (Case 7) had been bedridden for 10 months because of severe arthritis. Ten days before admission he was given promethazine 25 mg. b.d. by his doctor for troublesome pruritus. Chlorpromazine 25 mg. t.d.s. was substituted seven days later as he had become confused and hallucinated. He was being well looked after by his relatives and there was a fire in his room. The average temperature on the day of admission was 48° F. (8.9° C.). There was no obvious cause for his hypothermia, and it seems reasonable to relate this to the administration of phenothiazines.

Hypopituitarism was the underlying cause of hypothermia in Case 3. This patient had had an episode of hypothermia in 1960 and was on replacement therapy but had probably stopped taking her L-thyroxine and cortisone three weeks before admission. She became progressively less mobile and finally took to her bed.

TABLE I.—Clinical Data

Case No.	Age and Sex	Rectal Temp. ° F.	Pulse B.P.	Diagnosis Offered on Admission	Underlying Medical Condition	Environment	Min.* Air Temp. ° F.	Complications	Out-come	Post-mortem Findings
1	69 F	88	44 120/80	None	Myxoedema	In bed 2 days	33	Congestive cardiac failure. Oliguria	Died (at 60 hr.)	Thyroid: fibrosis and lymphoid infiltration Myocardium: brown atrophy
2	75 F	84	42 120/70	Cerebral thrombosis	"	" " 7 "	45	Hypotension—renal failure. Bronchopneumonia	Died (at 7 days)	Permission not obtained
3	76 F	81	50 140/80	Head injury	Hypopituitarism (treated, but omitted therapy for 3 weeks)	In bed 24 hr. in heated room	29	Renal failure (recovered). Transient Parkinsonism	Died (suddenly 2 weeks after apparent recovery)	Haemorrhagic gastritis. Thyroid: atrophy, fibrosis. Adrenocortical atrophy. Pituitary basophil adenoma
4	44 F	76	Unrecordable	None	Alcohol and barbiturates taken, but overdose not proved	Less than 12 hr. lying on kitchen floor	29	Convulsions. Confusion-mania. Brachial plexus injury	Recovered	
5	56 F	85	58 90/60	Overdose of barbiturates	Coma due to cyclobarbitone	Lying in lukewarm bath for 3 hr.	33	Respiratory depression	"	
6	79 F	88	60 190/90	None	Cerebellar haemorrhage	Collapsed in kitchen 6 hr.	35	—	Died (at 24 hr.)	Massive cerebellar haemorrhage
7	82 M	86	Unrecordable	Cerebrovascular accident and uraemia	Severe osteoarthritis. Bronchopneumonia	In bed for 10 months. Had taken promethazine, chlorpromazine	41	Hypotension. Renal failure. Bronchopneumonia	Died (at 6 days)	Emphysema, bronchitis, bronchopneumonia. Chronic pancreatitis, with fat necrosis
8	71 F	86	40 150/90	Diabetic coma	? Cerebrovascular lesion. Transient hemiplegia	In bed for 28 hr.	40	Convulsions. Oliguria. Bronchopneumonia	Died (at 12 days)	Permission not obtained
9	75 F	80	50 70/?	Coal-gas poisoning	Coal-gas poisoning	General state of neglect. Lying on floor unknown length of time	41	Congestive cardiac failure. Dementia	Recovered	

* Minimum air temperature recorded on Air Ministry roof, London.

TABLE II.—Investigations

Case No.	Proteinuria	Haemoglobin %	Serum Electrolytes (mEq/l.)	Plasma Bicarbonate (mEq/l.)	Blood Urea (mg./100 ml.)	Serum Cholesterol (mg./100 ml.)	Blood Glucose (mg./100 ml.)	Serum P.B.I. (μg./100 ml.)	Serum Amylase (Units*)	S.G.P.T. Units	S.G.O.T. Units	Electrocardiogram
1	—	—	—	—	—	641	—	<1.0	—	—	—	Low voltage. Flat T waves. Curved ST segment V ₁₋₄ and V ₆ . No J waves
2	+	109	Na ⁺ 122 Cl ⁻ 86	12	62	426	104	1.8	10	—	—	Low voltage. Flat T waves. Curved ST segment I, aV ₁ , V ₁₋₄ . No J waves
3	+++	110	K ⁺ 4.3 5.8	—	124 75 180 104	278	95	—	—	—	—	Normal voltage. Inverted T waves. Curved ST segment I, aV _r , aV ₁ . J waves I, V ₃₋₆
4	+++	103	Normal	22	40	317	332 82	4.4	4	17	39	Atrial fibrillation. Normal voltage and T waves. Curved ST segment I. J waves V ₃₋₆
5	—	104	"	25	16	256	—	5.1	5	14	22	Normal voltage. Inverted T waves. Curved ST segment I, III, aV _r , aV ₁ . No J waves
6	+++	110	Na ⁺ 128 Cl ⁻ 94	—	33	—	127	9.1	—	20	42	Normal voltage. Inverted T waves. Curved ST segment aV ₁ . No J waves
7	—	106	Na ⁺ 124 Cl ⁻ 95	—	41 75	91	—	5.7	40 16	36	49	Normal voltage. Inverted T waves. Curved ST segment I, aV _r , aV ₁ , V ₃₋₆ . Small J waves II, aV _f
8	+	92	Normal	22	71 110	244	80	3.9	10	7	30	Normal voltage and T waves. Curved ST segment aV _r , V ₆ . J waves I, II, V ₆
9	+	86	Na ⁺ 117 Cl ⁻ 84	32	30	250	85	3.2	3	20	45	Atrial fibrillation. Low voltage. Inverted T waves. Curved ST segment I, aV ₁ , V ₆ . No J waves

Figures given are initial values, with any serial results given below.

* Wohlgemuth method for serum amylase, normal range 0-10 units.

Apart from the two cases of myxoedema coma, the remaining four patients had either cerebrovascular accidents or coma due to barbiturate or coal-gas poisoning.

Diagnosis and Physical Signs

The diagnosis presented little difficulty once the possibility of hypothermia was considered. All the patients were pale and strikingly cold to the touch. Shivering and piloerection were not seen. The level of consciousness was impaired to a varying degree and the pupils were often constricted and fixed. Muscle tone was usually increased, sometimes markedly, with sluggish or absent tendon reflexes. Bradycardia was present in most cases, but the pulse rate tended to be higher in the presence of severe hypotension. The blood-pressure was unrecordable in two patients. Respiration rate and depth were reduced and in Case 5 respiratory movements were almost imperceptible. The diagnosis was confirmed by taking the rectal temperature with a low-reading thermometer.

Electrocardiogram

Emslie-Smith (1958) described changes in the electrocardiogram which he considered pathognomonic of hypothermia. These were lengthening of the PR, QRS, and QTC intervals; widening of the base of the QRS complex, J waves, and T-wave inversion. All these features were seen in the present series.

In all cases the base of the QRS complex was widened in one or more leads and T waves were flattened or inverted in seven cases. In four tracings typical J waves (Osborn, 1953) were seen at the QRS-ST junction. This wave is thought to represent a current of injury. Osborn (1953) found that J waves were observed more often in the presence of a lowered plasma bicarbonate, and concluded that ventricular fibrillation was more likely to occur when J waves were prominent. Atrial fibrillation with a slow ventricular rate may occur at 82–86° F. (27.8–30° C.) and ventricular fibrillation below 82° F. (27.8° C.). It has been suggested that atrial fibrillation protects against the development of ventricular arrhythmias (Vandam and Burnap, 1959).

In addition, changes were noted in the ST segment in association with widening of the QRS complex. The ST segment may have a high "take-off," and curves down to the isoelectric line. This finding was observed in all cases in one or more leads, but was seen most frequently in the left chest leads and lead I (Table II). It may represent a change similar to that causing the J deflection as it occurs in the same position.

Biochemical Investigations

Serum sodium and chloride concentrations were low in four patients, but potassium concentrations were normal in all except when renal failure developed in Case 3. The haemoglobin tended to be high (greater than 100% in six cases), suggesting haemoconcentration. As may be expected, the serum-cholesterol levels were increased and the serum P.B.I. levels were very low in the cases of myxoedema coma.

Impairment of renal function was indicated by a progressive rise in blood urea in four cases and by oliguria in two others despite rehydration and restoration of blood-pressure. Similarly, Duguid *et al.* (1961) and Frueman (1960) noted elevated blood-urea levels in approximately half of their cases, and oliguria in

neonatal cold injury was commented on by Mann and Elliott (1957). Experimental findings on the effect of cold on renal function have not been in complete agreement. Moyer *et al.* (1957) observed a reduction in glomerular filtration rate, renal blood flow and maximum tubular absorption of glucose. There was no change in urine volume or sodium content, and they concluded that tubular function was affected. No change was produced by raising the blood-pressure to the original levels with noradrenaline, suggesting that the effect was due to hypothermia. They also noted a considerable delay in the recovery of renal function after rewarming. However, other workers have found more marked impairment of renal function. Nedzel (1952) found a considerable reduction in urine flow in hypothermic dogs and thought this was due to renal cortical ischaemia. Marshall and McCaughey (1956) have suggested that acute tubular necrosis may result from hypothermic muscle damage, and Knocker (1955) has described histological changes in the tubular epithelium.

It is significant that proteinuria, without evidence of infection of the urinary tract occurred in six patients in the present series. Although this is a non-specific finding, it may well indicate renal damage. The renal changes in accidental and induced hypothermia are not strictly comparable. In accidental hypothermia, factors such as cardiac failure, dehydration, hypotension, and the use of pressor agents may be operating, while in induced hypothermia unknown effects may be produced by surgery, loss of blood, anaesthetics, and other drugs. A combination of factors probably causes the impairment of renal function in accidental hypothermia.

Hyperglycaemia is known to occur in hypothermia (Wynn, 1954; Fisher *et al.*, 1958). In Case 4 the initial blood sugar was 332 mg./100 ml. and in the case reported by Laufman (1951) it was 438 mg./100 ml. Fuhrman and Crimson (1947) found that hyperglycaemia was more likely to occur with rapid cooling, as was the case with both these patients. There was no ketosis and the blood sugar returned to normal without specific therapy in Case 4.

Pancreatic damage was found in 10% of patients with malignant disease treated by hypothermia (Sano and Smith, 1940). This association has been confirmed by subsequent reports. Read *et al.* (1961) described two cases of pancreatitis associated with accidental hypothermia, and Duguid *et al.* (1961) found raised serum-amylase levels in 11 out of 15 cases, and five of these had evidence of pancreatitis at necropsy.

The serum-amylase levels in Case 7 were raised and chronic pancreatitis with fat necrosis was found at necropsy. In two other cases the serum-amylase levels were at the upper limit of normal.

Treatment

Opinion is divided on the question of rewarming these patients. Rapid rewarming may cause sudden hypotension due to peripheral vasodilatation, and cardiac failure may be provoked, particularly in myxoedema coma. Duguid *et al.* (1961) advised against rapid warming, but Rees (1958) recommended rewarming in acute hypothermia, which he defined as hypothermia of less than 12 hours' duration. All our patients were put to bed in warm surroundings and covered with blankets. No active measures were taken to raise their temperatures except in Case 9; this patient was warmed for a short time with an electric blanket.

A rapid spontaneous rise in temperature is a good prognostic sign; thus all patients who survived had regained a normal temperature within 24 hours. We feel that if rewarming is to be used at all it should be restricted to those patients who have shown no significant rise in temperature by 24 hours. A prolonged period of hypothermia undoubtedly contributes to a very poor prognosis, and in this group active rewarming is justified.

Hydrocortisone was given to all patients during the hypothermic period. Adrenocortical function is impaired in hypothermia and also in myxoedema (Hubble, 1955). Eghdahl *et al.* (1955) found that secretion of 17-hydroxycorticosteroids fell to 25% of control values in dogs cooled to 77–82° F. (25–27.8° C.) and that no increase was produced by infusion of adrenocorticotrophic hormone. It was noticed that in several of the present patients hypotension was more readily controlled after injection of hydrocortisone.

Intravenous fluids were given when indicated, and mephentermine or metaraminol if hypotension was present, but noradrenaline infusion was ultimately required in Cases 2, 4, 7, and 8. Triiodothyronine 10 to 60 µg. daily was given orally in Cases 1, 2, and 3. All patients received penicillin or tetracycline, with digoxin and thiazide diuretics if cardiac failure was present. It has been shown that digitalis is not contra-indicated in hypothermia and is not a factor in the origin of ventricular fibrillation (Angelakos *et al.*, 1958). One patient (Case 5), although not cyanosed, had marked respiratory depression, and positive-pressure respiration was required for 12 hours.

Summary and Conclusions

Nine cases of accidental hypothermia seen in a small hospital within a period of four months are reported. Three patients survived. Electrocardiographic and biochemical abnormalities are briefly reviewed. It is suggested that the condition is by no means uncommon and should be considered in the differential diagnosis of all patients with impaired consciousness during the winter months. It can occur rapidly and is by no means confined to the elderly and neglected. Although there is often a history of exposure during cold weather, hypothermia can develop when the patient is in bed in relatively warm surroundings. The phenothiazine group of drugs should be given to the elderly and hypothyroid patient with caution and the rectal temperature checked if confusion or drowsiness occurs. This should be taken with a low-reading thermometer or, if unavailable, an ordinary lotion thermometer. The prognosis is poor, but is more favourable in the younger patients.

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Preliminary Communications

A New Approach to the Treatment of Leg Ulcers

It is generally accepted that the immediate cause of venous leg ulcers is venous hypertension, with the resulting capillary changes due to back-pressure. Chronic venous insufficiency is most commonly a sequel to deep venous thrombosis, which often affects and destroys the valves of the perforating veins in addition to those of the deep veins; the raised capillary pressure and damage so produced result in the escape of protein-containing fluid into the tissues. The limb becomes oedematous and later indurated, and with the interference with the nutrition of the skin and subcutaneous tissues ulceration may eventually occur (Anning, 1954). Many ulcers rapidly become infected, often to a considerable degree.

The logical treatment of venous ulcers of the leg is based on the reduction or correction of these aetiological factors. The traditional means by which this may be affected are threefold.

First, the source can be removed surgically by stripping the varicose long and/or short saphenous vein with division of any defective communicating veins. This may be both prophylactic and therapeutic. After careful and complete diagnostic evaluation the chances of either a primary or a recurrent ulcer developing in a leg so predisposed may be considerably lessened by these means. In addition, in certain intractable, especially post-thrombotic, ulcers, such radical surgery on the veins may succeed in healing the ulcer where other methods have previously failed.

In the second place the patient may be treated in recumbence and by raising the leg above the level of the heart and maintaining this position until oedema subsides and the ulcer eventually heals. This prolonged and often impracticable tedium would seem to be obsolete, for not only does the ulcer tend to recur when normal activities are resumed but there are distinct risks in keeping these patients, many of whom are elderly, obese, and arthritic, in bed for any length of time.

The third method is by the application of external pressure to the leg by means of compression bandaging, in conjunction with which various bacteriostatic or bactericidal preparations can be applied. This can be