ACCIDENTAL HYPOTHERMIA IN THE BANTU

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Accidental hypothermia is usually reported from countries with cold climates.[1-7] It is most frequently due to accidental exposure, and is prone to occur in patients with myxoedema, hypopituitarism, old age, mental impairment, or loss of consciousness from any cause. It may also occur after the ingestion of drugs like chlorpromazine and alcohol. The condition is not uncommon, however, in the relatively warm climate of Johannesburg, and may even be seen during the summer months. Thus we report 5 cases seen at Baragwanath Hospital during a 5-month period from January (midsummer) to June (midwinter).

CASE REPORTS

Case 1

A 32-year-old Bantu woman was admitted unconscious on a cold night in mid-January. Her brother stated that the patient felt faint while sitting in a warm room and went outside to fetch some cold water; she was found about half an hour later lying unconscious under a cold water tap. Efforts to rouse her failed and she was brought to hospital.

She was pale, stuporose and responded slightly to painful stimuli. The entire body surface was very cold. Muscle tone was increased. The pulse rate (PR) was 50 per minute; blood pressure (BP) was 140/90 mm.Hg; the rectal temperature (RT) was below 95°F. when recorded by conventional thermometer. Further physical examination was negative.

An electrocardiogram (ECG) showed a bradycardia of 50 per minute, irregular fine oscillations of the base line, and a prolonged Q-T interval and inverted J waves.

She was placed in a water bath at 110°F. Her RT rose to 98°F., with a concomitant improvement in consciousness; she was talking but confused. She was then placed in a warm bed, and the next morning her mental state and ECG had returned to normal.

Investigations failed to reveal any predisposing cause. She was well on discharge 5 days later.

Case 2

A well-nourished African man aged 30 was admitted to hospital in deep coma. No history was available at the time, but on recovery he admitted to having had an alcoholic debauch the previous afternoon. He had spent the night, which was particularly cold, unprotected in the open.

He was deeply comatose and did not respond to painful stimuli. RT was 87°F., PR 44 per minute, BP 90/50 mm.Hg, and respiration 12 per minute. The pupils were equal and moderately dilated, and responded to light. The limbs were flaccid and the reflexes depressed.

ECG showed a rate of 44 per minute, irregular fine oscillations of the base line, a prolonged Q-T interval, prominent J waves, and atrial and ventricular extrasystoles (Fig. 1).

He was placed in a water bath at 100°F. and the RT rose to 96°F. The mental state improved; he was restless and responded to painful stimuli. On transfer to a warm bed he was sweating profusely and had a tachycardia. He regained consciousness 1½ hours after re-warming and was then quite rational.

Investigations during hypothermia revealed normal haemoglobin, blood-urea, blood-electrolyte and serum-amylase levels; serum CO_2 content was 10·7 mEq. per litre. The blood sugar

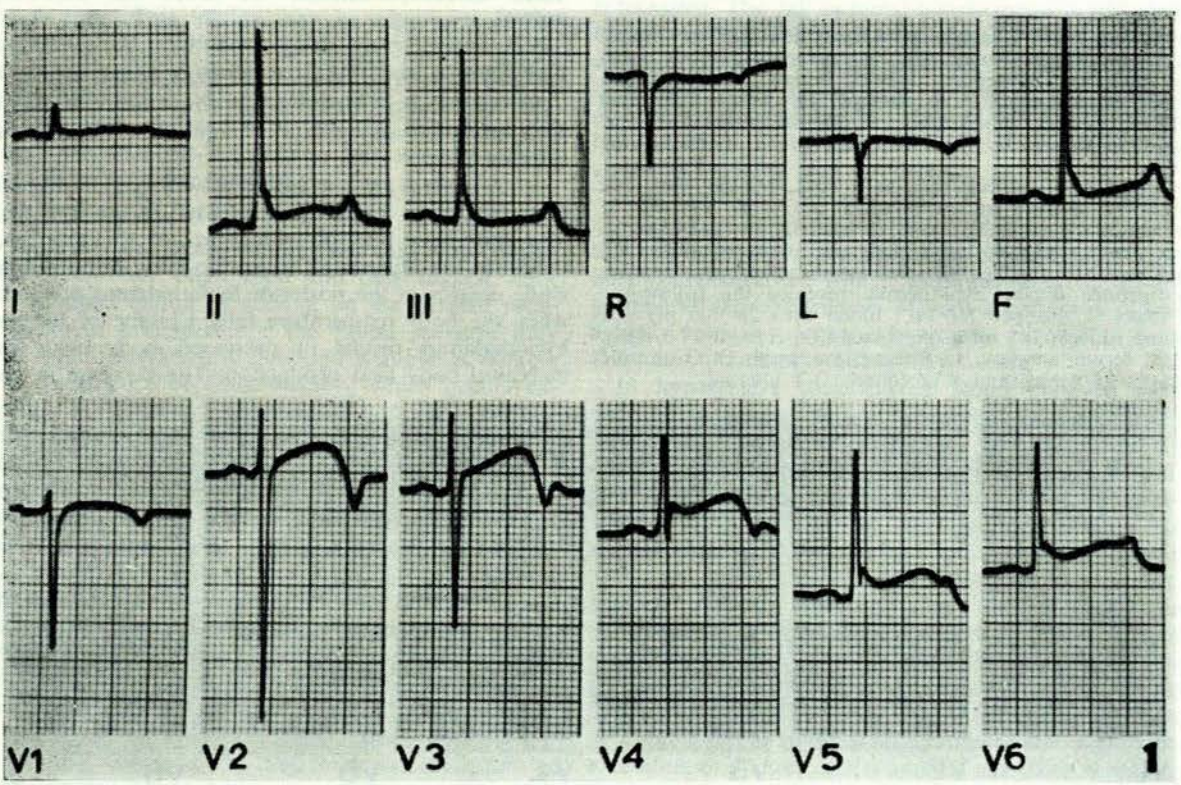

Fig. 1. Case 2, ECG, showing J waves and prolongation of the Q-T interval.

was at a hypoglycaemic level, viz. 12 mg. per 100 ml.

The blood sugar was normal the following day. It is noteworthy that he had been admitted to hospital a year previously in hypoglycaemic coma following an alcoholic debauch. He was discharged in good health 3 days later.

Comment. It seems likely that the hypoglycaemia contributed to his unconscious state and was a factor in precipitating hypothermia. The signs of hypoglycaemia, however, were masked by the hypothermic state and only appeared as tachycardia, restlessness and profuse sweating after re-warming.

Case 3

A 29-year-old, poorly nourished, mentally confused African woman, with disseminated tuberculosis, developed hypothermia spontaneously in the ward. After 9 days in hospital, during which her temperature had been normal, she was found on the 10th day semi-comatose, with RT 85·6°F. She was re-warmed in a water bath at 100°F. and her temperature rose to 95°F. There was an increase in PR, but no improvement in her general or mental condition. The BP was 90/70 mm. Hg before and after re-warming. She was kept in bed, covered with blankets, in a warm medical ward for the next 24 hours, but despite this the following morning her temperature was below 85°F. She was again re-warmed in a hot bath, with similar results. This spontaneous hypothermia was observed on 3 successive days before death.

Investigations during hypothermia revealed the following: haemoglobin 9·1 G. per 100 ml.; ESR (Wintrobe) 47 mm. in 1 hour; severe electrolyte imbalance (per litre, potassium 3·0 mEq., chlorides 77 mEq., and sodium 125 mEq.); blood urea 31 mg. per 100 ml., and a CO_2 content greater than 30 mEq. per litre; lumbar puncture, protein-bound iodine, and serum cholesterol were normal; ECG showed bradycardia, irregular fine oscillations of the base line, prolonged Q-T interval, and J waves.

She died two weeks after admission, and autopsy revealed disseminated tuberculosis. The brain was macroscopically normal.

Case 4

A confused Bantu woman aged 42 was admitted to hospital smelling strongly of alcohol. She was disorientated but could answer questions. Subsequent history revealed that she had consumed large amounts of alcohol the previous evening and had fallen and injured her face. Thereafter she had slept the night in the veld, where she was found the following morning.

Her entire body surface was very cold. Muscle tone was increased. There were superficial facial abrasions. PR was 52 per minute; the systolic BP was 110 mm.Hg, but the diastolic BP was not recordable; respiration was 16 per minute; RT was below 85°F. Further physical examination was negative.

Investigations during hypothermia revealed the following: blood sugar 81 mg. per 100 ml.; blood urea 28 mg. per 100 ml.; blood sodium 141 mEq. per litre; CO_2 content 17·0 mEq. per litre; serum amylase 32 Street-Close units. ECG showed bradycardia, J waves, and a prolonged Q-T interval.

She was placed in a water bath at 110°F. and her temperature rose to normal. Her mental state improved, she became rational, and ECG returned to normal. She was well on discharge 2 days later.

Case 5

A 30-year-old Bantu man was admitted unconscious. His father said he had been well until 2 weeks before, when he became mentally confused, disorientated, and uncontrollable. He became unusually quiet 2 days before admission and stopped eating and drinking. The next evening he was unconscious and brought to hospital.

He responded to painful stimuli but not to commands. His skin felt cold and his limbs were rigid and difficult to flex. The RT was 85°F., the PR 48 per minute and BP 80/50 mm. Hg. Further physical examination was normal. ECG showed sinus bradycardia, J waves, and prolongation of the P-R and Q-T intervals (Fig. 2).

Investigations during hypothermia revealed the following: haemoglobin 15·3 G. per 100 ml.; WBC 4,700 per cu.mm.; blood urea 37 mg. per 100 ml.; and, per litre, potassium 3·9 mEq., sodium 137 mEq., CO_2 content 14·5 mEq., and chlorides 100 mEq.; serum amylase 13 Street-Close units; serum glutamic-oxalacetic-transaminase 27 units; serum glutamic-pyruvic-transaminase 31 units; carboxy-haemoglobin was less than 5%; X-rays of skull and chest were normal.

He was given intravenously 1,000 ml. of 5% dextrose saline solution to which 2 ampoules of phenylephrine and 100 mg. of hydrocortisone were added. This was followed by a rise in BP to 100/70 mm.Hg. He was then placed in a water bath at 110°F. The RT rose to 95°F.; the BP remained at 100/70 mm.Hg; and the mental state improved (he answered to his name). He was kept in a warm room and his temperature was maintained above 95°F.

During the next 14 days his mental state was similar to that before the hypothermic episode; he was disorientated and at times manic and violent. Towards the end of this period he developed bronchopneumonia, and despite antibiotic therapy he died rather suddenly 3 days later. Autopsy showed a confluent bronchopneumonia; the brain was macroscopically normal.

DISCUSSION

Pathophysiology of Accidental Hypothermia

In the healthy person body temperature is maintained with great accuracy. It is regulated by at least two systems:[8]

1. A *central* system whose action may be likened to a thermostat; this is probably situated in the hypothalamus. The supraoptic and preoptic areas of the anterior hypothalamus are stimulated when the body temperature rises, while centres in the posterior hypothalamus are stimulated when the body temperature falls. Chilling of the posterior hypothalamus results in decreased body heat loss and increased body heat production. The decrease in heat loss

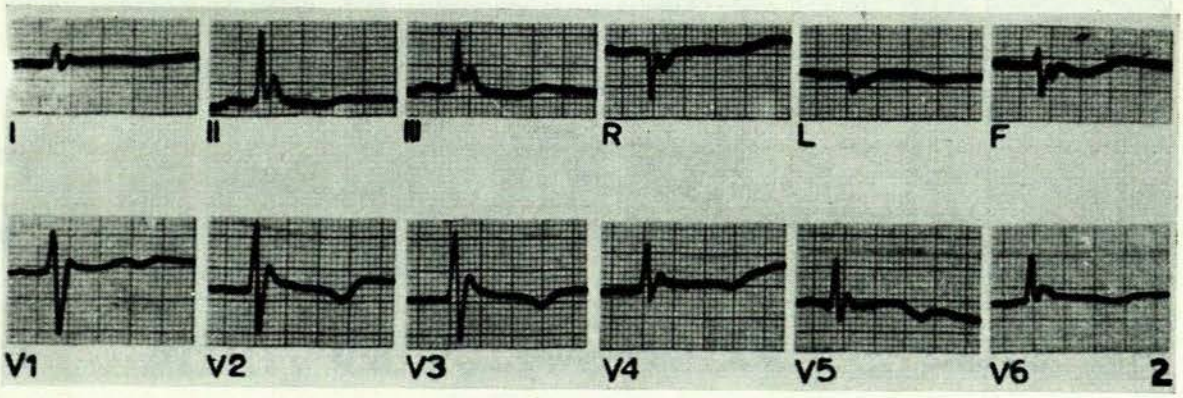

Fig. 2. Case 5, ECG, showing positive J waves with inverted T waves and prolongation of the Q-T interval.

is brought about by intense adrenergic vasoconstriction of the skin vessels; this results in a decreased flow of warm blood to the skin and hence decreased conduction of heat from the body core to the body surface. The increase in body heat production is brought about by shivering and adrenergic stimulation of cellular metabolism.[9]

2. An independent *peripheral* or *reflex* system. This mechanism produces reflex vasodilatation or vasoconstriction when warm or cold stimuli are applied to the skin.

The central regulating mechanism functions most efficiently at temperatures from normal down to 95°F. Below this level, temperature regulation becomes impaired: shivering ceases at about 90°F. to 85°F. and cellular metabolism throughout the body is depressed, resulting in a further fall in body temperature. At about 75°F. the temperature-regulating centre ceases to function and heat is lost from the body as from an inanimate object.[1, 10] Thus loss of the hypothalamic mechanism leads to a poikilothermic state, in which the temperature is regulated only by changes of insulation and environment. In addition, certain patients with sustained hypothermia have been described who appear to regulate their body temperature so that it remains at a low level,[4, 8] i.e. the thermostat is reset at a lower level. Case 3, who became hypothermic on 3 occasions in a warm medical ward, may have had a disturbance of the central regulating mechanism.

Hypoglycaemia is a potent anti-shivering agent. Thus Finney, Dvorkin and Cassidy[11] showed that shivering in dogs immersed in iced water could be inhibited by lowering the blood sugar with insulin. This was confirmed by Talbott,[10] who demonstrated that hypoglycaemia abolished shivering in man. This finding may be significant in the Bantu population of Johannesburg, in whom postalcoholic hypoglycaemia is fairly common. Thus case 2 was profoundly hypoglycaemic on admission, and it is suggested that this may have inhibited shivering; this impairment of the normal compensatory response to cold would result in a quicker fall of body temperature.

Diagnosis of Accidental Hypothermia

In hypothermia the entire skin is pale, cold, and corpse-like. The muscles are often rigid and may suggest rigor mortis. The direct action of cooling on the cardiac pacemaker results in bradycardia;[12] the blood pressure may be low, and the respiration is slow and shallow. A notable feature is disturbance of consciousness, which occurs experimentally at a rectal temperature of 88°F.[13] The unconscious patient cannot complain of cold, nor does shivering occur to arouse suspicion, so that if the diagnosis is not considered there is a danger that the temperature may be taken with a conventional thermometer and recorded at the lowest level of the thermometer, namely 95°F. Disturbance of consciousness—varying from mental confusion to deep coma—was a notable feature in our cases, and in 4 of the 5 it improved on re-warming.

The ECG usually shows sinus bradycardia, and prolongation of P-R, QRS, and Q-T intervals. Junctional or J waves are usually a notable feature;[14] these waves occur at the immediate end of the QRS deflection, and are usually positive (cases 2 - 5), but may occasionally be negative (case 1). They are most prominent in the left ventricular leads. The mechanism of production of these waves is not at present understood.[15] Irregular fine oscillations of the base line are frequently seen and are due to fine muscular tremors. Atrial and ventricular extrasystoles were seen in one patient.

Hypothermia is generally believed to be a rare condition. Increased awareness, however, has shown it to be more prevalent than previously thought. A combination of *impaired mental state, cold skin* and *bradycardia* should suggest the correct diagnosis.

Treatment of Hypothermia

The most important problem in the treatment of hypothermia is whether the patient should be re-warmed rapidly or slowly. Rapid re-warming consists of immersion in a water bath at 110°F.; the slow method is to allow the patient to re-warm at room temperature at a rate of 1°F. to 2°F. per hour. If the rate of re-warming is in between these two extremes the results are poor. During the Second World War hypothermia was a common complication of shipwreck survivors. These patients were usually re-warmed by the use of warm blankets, hot drinks and an electric cradle; and many died shortly after being taken from the water. The explanation for this is that the skin temperature on removal from the water is approximately the same as the water temperature; on re-warming, blood flow through the skin increases, cold blood is circulated to the heart, and the deep body temperature may fall as much as 5°F. Such a rapid fall may produce cardiac arrest or ventricular fibrillation. If, however, the patient is placed in a bath at 110°F., the periphery re-warms very rapidly and the cooling effect on the peripheral circulating blood is transient. The fall in rectal temperatures is still observed but is less than 2°F. and only lasts a few minutes. Hence, in *acute hypothermia,* the treatment of choice consists of immediate rapid re-warming in water at a temperature of 110°F.[13] Four of our five patients had been hypothermic for less than 12 hours, and all four responded well to active re-warming in a hot bath.

This principle does not apply to chronic hypothermia, i.e. hypothermia of longer than 12 hours' duration, for in this condition there is a reduction of plasma volume with haemoconcentration, which is compensated for by intense peripheral vasoconstriction. If such a patient is re-warmed actively vasoconstriction is overcome, peripheral resistance falls, and circulatory failure may follow.

In hypotensive patients the blood pressure must be restored to normal before re-warming is attempted. Thus case 5 was first given intravenous dextrose saline, hydrocortisone and phenylephrine before re-warming, and was only placed in a hot bath when the blood pressure had returned to normal. There are theoretical reasons for the use of each of these methods of treatment. Intravenous fluids are given to correct haemoconcentration and low plasma volume. Hydrocortisone and sympathomimetic drugs are of value because the secretion of hormones from both the adrenal cortex and medulla is reduced by hypothermia.[15] Indeed, the circulatory collapse that may follow active re-warming may be due, in part, to acute adrenal insufficiency, the body's requirements for the hormones exceeding the power of the adrenal glands to supply them.[1] There are two further reasons for the use of vasoconstrictor drugs: (a) they produce peripheral arteriolar constric-

tion, which is of value firstly by causing a direct elevation of the blood pressure, and secondly by preventing the cutaneous vasodilatation that occurs on re-warming, and (b) they stimulate cellular metabolism and hence increase cellular heat production.

SUMMARY

Five cases of accidental hypothermia in Johannesburg Bantu are reported. Four of the five cases followed exposure on a cold night. In three of these there were clear predisposing factors, viz. alcoholic intoxication, post-alcoholic hypoglycaemia, and mental derangement. In the fourth case no underlying cause was found. The fifth case occurred in a patient while in hospital with disseminated tuberculosis. Four of the five cases responded to active re-warming.

The physiology, diagnosis and treatment of hypothermia are briefly reviewed.

It is stressed that this condition is by no means uncommon and should be considered in any unconscious patient with bradycardia and a cold skin.

ADDENDUM

Since this paper was submitted we have seen two more cases of accidental hypothermia following exposure. The first, admitted with the extremely low RT of 76°F., had been assaulted, but not concussed; the second followed an alcoholic debauch. Both responded rapidly to active re-warming.

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