Accidental hypothermia

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Scope and definition

Hypothermia is defined as a lowering of the temperature of the human body below the arbitrarily chosen level of 35°C (95°F). The temperature lowering may be accidental or therapeutic, generalized or localized. Local therapeutic hypothermia is exemplified by cooling the heart or brain in major surgery, and by local cooling of the stomach in the management of bleeding. Accidental lowering of the temperature of peripheral parts of the body often happens in cold weather, and affects distal parts of limbs, the nose and ears. More severe degrees of tissue damage amount to frostbite.

Lowering of the whole body temperature is employed in vascular, cardiac and brain surgery in an attempt to prolong the viability of essential organs deprived of blood supply. This article is concerned with generalized hypothermia occurring 'spontaneously'. It is conventional to record central body temperatures in the rectum, although oesophageal readings would be preferable.

Temperature control

Cranston, in the 1966 Goulstonian Lecture, has reviewed his own researches and those of other workers into the mechanism of temperature control. Centrally measured temperature is the product of the balance between heat production and heat loss. Baseline heat production results from basal metabolism, general activity and the metabolism of food. This heat production may be rapidly increased by voluntary muscular activity or by shivering. There is some evidence from animal studies that increased heat production may result from the metabolism of brown adipose tissue. Long-term adjustment may follow increased secretion of thyroxine. Heat loss may be increased by sweating, evaporation from the respiratory tract, and by skin radiation or convection.

The control of heat production and loss appears to be at two levels. Centrally, control is probably exercised by the hypothalamus, and there is some evidence that it may be mediated by nor-

adrenaline and 5-hydroxytryptamine, as well as via the autonomic nervous system. The sensory receptors for hypothalamic control may be in its pre-optic area. Peripherally, skin receptors operate via a reflex mechanism which extends above the cervical cord, and whose efferents travel via the sympathetic nervous system.

Causes

Lowering of the body temperature may follow either excessive heat loss or lowered heat production. In each case the cause may be either a disturbance of the body's mechanisms of thermos-tasis, or exposure to extremes of cold. In most patients it is likely that at least two factors are involved: e.g. the combination of impaired control and exposure to cold.

TABLE 1
Causes or precipitating factors of hypothermia

Excess heat loss	
Accidental hypothermia in the elderly	
Acute exposure	
Infancy hypothermia:	day of birth neonatal marasmic
Skin disease	
Diminished heat production	
Undernutrition	
Hypopituitarism	
Myxoedema	
Impaired thermo-regulation	
Alcohol	
Old age	
Barbiturate intoxication	
Reserpine	
Phenothiazines	
Head injury	
Third ventricle neoplasm and midbrain lesion	
Spinal injury	
Steatorrhoea	
Hypoglycaemia	
Severe illness:	bronchopneumonia myocardial infarction haemorrhage diabetic coma

Accidental hypothermia in the elderly is the subject of a memorandum prepared by a Special Committee of the British Medical Association (Annotation, 1964). The incidence of accidental hypothermia is not known, but it mainly affects those over the age of 60. It occurs particularly in cold weather, and usually affects those living in under-heated conditions. The Committee on Accidental Hypothermia of the Royal College of Physicians of London (1966) surveyed the recorded temperatures of all patients admitted to ten hospitals between 1 February and 30 April 1965. Of those patients over 75 years old, 17.4 per 1000 had temperatures below 35°C (95°F). Underlying vulnerability of the thermo-regulating centre in the hypothalamus may set the scene for lowering of body temperature. In addition to cold environment many other factors appear to disturb temperature control. Depressed pituitary or thyroid function, vascular collapse following haemorrhage or myocardial infarction, and the administration of drugs such as phenothiazines, barbiturates and alcohol may all provide the additional factor needed to produce significant hypothermia. In many elderly patients the hypothermia may be complicating a severe illness. In the patients described by Duguid, Simpson & Stowers (1961) these illnesses included mental impairment and bronchopneumonia, and in the series of Prescott, Peard & Wallace (1962) cerebrovascular accidents, diabetic coma, uraemia, and poisoning by coal gas and barbiturates.*

Acute exposure causing lowering of body temperature was documented by Keatinge (1965) following the December 1963 *Laconia* disaster, and by Lloyd (1964) in Mendip caves. Keatinge (1960) has shown that the rate of fall of rectal temperature in subjects immersed in cold water is inversely related to the thickness of their subcutaneous fat. Cannon & Keatinge (1960) showed that fat men's cold response is largely skin-controlled, whereas thin men's response is reinforced by deep temperature receptors.† These observations conform with those of Pugh & Edholm (1955) who record that most of the twenty competitors in the 1951 cross-channel swimming race were stocky or obese. However, these swimmers do better than predicted when their temperature does fall suggesting that adaptive mechanisms for increasing low temperature performance exist. The usual survival time in water at 15.5°C (60°F) is not more than 6 hr. There is

good evidence that dampness and poor insulation are major factors causing heat loss.

Hypothermia in infants up to 1 year was found by the Royal College of Physicians Committee (1966) to occur in 82.2 per 1000 admissions in this category. Arneil & Kerr (1963) review their experience with 110 babies in Glasgow between 1961 and 1963. They divide their cold babies into three groups:

(1) Hypothermia on the day of birth, often occurring when the infant is delivered into a cold environment.

(2) Neonatal, coming on more slowly in babies over 24 hr old.

(3) Marasmic, in babies developmentally abnormal and poorly nourished.

Mann & Elliot (1957) reported fourteen hypothermic babies in Brighton. Accidental exposure to cold was responsible for the babies' condition. The large surface area relative to the body weight, and immature control of temperature make the small and exposed infant susceptible to heat loss.

Skin conditions involving extensive erythematous areas lead to extra heat loss. Hypothermia has been recorded complicating exfoliative dermatitis, and it seems likely that skin hyperaemia is at least partly responsible for hypothermia recorded after excessive alcohol intake. Diminished heat production has not been shown to cause hypothermia, but this may be the mechanism in hypopituitarism and hypothyroidism. Taylor (1964) found that many of his elderly hypothermic patients were undernourished, and as with the marasmic infants of Arneil & Kerr (1963), lowered metabolism may have contributed to their clinical state.

Impairment of thermo-regulation has not yet been investigated in human poisoning. There is some animal evidence that a disturbance of central control is responsible in the case of imipramine given with mono-amine oxidase inhibitors. Phenothiazines, reserpine and barbiturates may also act centrally. Intracranial lesions have been associated with body temperature disturbances. Hockaday *et al.* (1962) described three patients with chronic hypothermia. One of these patients appeared to have no temperature-regulating mechanism, and two were regulated to a low level. One patient had sustained a head injury and another had a possible mid-brain lesion. Duff *et al.* (1961) have described two patients with spontaneous periodic reductions in body temperature, occurring especially in winter, for which no cause was found. They also record hypothermia associated with a craniopharyngioma and with a meningioma in the region of the hypothalamus. Spinal cord injury appears to

*Macmillan *et al.* (1967) have demonstrated impaired temperature regulation in elderly survivors of accidental hypothermia, with evidence suggesting a lesion of central controlling mechanisms.

†Also that fat men have superior tissue insulation.

disturb autonomic control of the peripheral heat regulating mechanisms. Pledger (1962) has described a man of 30 with a tetraplegia from a C6 lesion which followed a fall from a motor scooter. This man's temperature was virtually controlled by his environment. Meirowsky (1963) described similar disturbances produced by the cold Korean weather in wounded soldiers. Dent, Stokes & Carpenter (1961) have reported two deaths from hypothermia in patients under their care with steatorrhoea. Kedes & Field (1964) have found the presence of hypothermia a useful clue to underlying hypoglycaemia. They suggest that both disordered hypothalamic function and lowered metabolic heat production are possible, but unsatisfactory, explanations of this association.

Clinical features

Accidental hypothermia is frequently divided into three stages:

Stage 1. Down to 32.2°C (90°F) in which the body reacts to cooling by peripheral vasoconstriction and shivering.

Stage 2. 32.2°C (90°F) to 24°C (75°F) in which tissue metabolism is gradually depressed.

Stage 3. Below 24°C (75°F) in which the body loses heat as an inanimate object.

This method of staging is not entirely satisfactory as the symptoms and signs are not closely related to recorded temperatures. For expedience in a Casualty Department it might be more satisfactory to have two stages:

Stage 1. 'Mild', above 32.2°C (90°F), when observation may suffice.

Stage 2. 'Severe', below 32.2°C (90°F) when an emergency situation must be considered to exist.

For descriptive purposes it is not an advantage to ascribe features to a particular 'stage'.

With moderate cooling, a normal physiological response is seen: peripheral vasoconstriction and consequent pallor, shivering, rise in pulse rate and blood pressure, and diuresis. Consciousness is not impaired at this stage, and the clinical condition may be considered 'mild'.

With further cooling the overall appearance of the patient may change. The skin, whilst still pale may develop blue blotches, and sometimes becomes puffy and 'oedematous'. The cause of this puffiness is uncertain, but is presumably a shift of fluid from plasma to interstitial spaces. The patient often has a corpse-like appearance, and may be pronounced dead. A 'chill' emanates from the body. Myxoedema may be mimicked even when the patient has been known to be of previously normal appearance (Fig. 1). With substantial cooling the pulse rate falls and frequently becomes irregular with auricular fibrillation.

Ventricular fibrillation is a common cause of death. The blood pressure falls to very low levels, and it is often difficult either to feel a peripheral pulse or to hear a heart beat. It is

FIG. 1. At necropsy this hypothermic patient's thyroid gland was found to be small and fibrous. A similar facial appearance may be seen in patients with previously normal thyroid function.

FIG. 2. This patient was admitted when aged 87, with a rectal temperature of 87°F, and a haemoglobin of 37%. She had collapsed following a haematemesis, and although she tripped over the flex of the electric fire, and was engulfed in flames, made an excellent recovery.

important to remember that the signs of cardiovascular collapse may be due to the underlying cause of the hypothermic state. Evidence of haemorrhage, cardiac disease, poisoning, and other causes of collapse must be sought (Fig. 2). Similarly, respiration is depressed and rates below 10/min are often observed. Respiratory diseases, in particular bronchopneumonia, may mask this sign and must be diagnosed and treated. Consciousness may be impaired below 32.2°C (90°F). Often there is an initial slowing of the patient's response, leading to stupor and eventually coma. The voice may become husky and be indistinguishable from the dysphonia of myxoedema. The pupils have been recorded as either constricted or dilated, but their reaction to light is sluggish. Involuntary movements, sometimes resembling cerebellar incoordination may occur, and frequently tone is increased in the neck and limbs.

Tendon reflex delay is an added feature shared with hypothyroidism, and assessment of thyroid state is difficult or impossible. This delay is most easily seen in the relaxation phase. The 'delayed relaxation reflex' is seen occasionally in elderly patients with subcutaneous oedema or poor peripheral circulation, and is not specific.

In acute exposure there is less need to draw attention to clinical features in order to make a diagnosis. The situation is usually self-evident, but cases of unsuspected hypothermia in young, previously fit, exposed people have been documented. This condition may be fatal if untreated. Pugh (1966) has reported twenty-three incidents of hypothermia occurring in members of climbing parties. The sequence of events described is of 'abnormal behaviour, slowing, stumbling, weakness, repeated falling, collapse, stupor, unconsciousness and death'. Shivering was not prominent. Mild cases were normal to ordinary clinical examination, but unconscious subjects are described as showing 'extreme pallor, generalized rigidity and absence of peripheral pulses'.

In infants, Arneil & Kerr (1963) describe signs differing with their three groups. On the day of birth, cold babies are lethargic, often with cyanosed limbs. The neonates have a 'pseudo-healthy redness of the skin of the face, hands and feet'. Oedema, sclerema, or both of these conditions may be present. Bradycardia and bradypnoea also occur. In the marasmic group anorexia and drowsiness predominate, although features of the other groups may be present.

Investigations

The diagnosis of hypothermia is established reliably by the use of a low-reading thermometer. It is traditional to record the rectal temperature

as this is thought to represent a close approximation to central body temperature. Although rectal temperatures respond slowly to changes in arterial temperature, the lag is not of practical importance with the slow changes involved.

Various investigations may help in detecting an underlying cause for the hypothermia and may help to monitor progress or detect complications. Both haemoglobin and white cell count may be elevated by the shift of fluid from plasma to interstitial space. The white cell count may also reflect the presence of infection or may be depressed with the temperature. The haemoglobin is also a guide to undetected long-standing haemorrhage, although blood-volume estimation is needed to assess acute loss.

An X-ray of the chest is of help in detecting lung infection especially as auscultatory signs are frequently difficult to interpret. These signs are also time-consuming to elicit when the respiratory rate is depressed. The electrocardiogram characteristically shows 'J' or junction waves as described by Osborne (1953) (Fig. 3). Ree (1964)

FIG. 3. 'J' waves are seen in hypothermia whether accidental or hypothyroid.

and Emslie-Smith (1958) have also reported these waves, and describe the bradycardia, atrial fibrillation and Q-T prolongation which is found at low temperatures. These electrocardiographic changes are not specific to accidental hypothermia, but are also seen in hypothyroid hypothermia. The 'J' waves may be related to temperature, P_{CO_2} or acidosis. The adequacy of pulmonary function in accidental hypothermia has been studied by McNicol & Smith (1964). The result of respiratory depression and lowered metabolic demands is often inadequate oxygenation. This is shown by low oxygen tensions even with normal saturation. The P_{CO_2} is usually normal but sometimes elevated, and the patients are usually acidotic.

The close similarity which may exist between accidental and hypothyroid hypothermia would make a reliable test of thyroid function in these circumstances invaluable. Estimation of the basal

metabolic rate is not practicable and the serum protein-bound iodine and T_3 binding capacities were found by Rosin & Exton-Smith (1964) to give misleading results. In these circumstances Mathews (1966) found that it was only a serum cholesterol level above 350 mg/100 ml that could be taken as a reliable indication of lowered thyroid function.

Several authors, including Read *et al.* (1961), have commented on the high incidence of pancreatitis in hypothermia. The relationship of these conditions as cause and effect is unclear. It is likely that hypothermia can lead to pancreatitis as this has been observed in artificially induced low temperatures. Pancreatitis should be suspected if abdominal guarding is found, and the diagnosis is supported by finding a raised serum amylase level. Wynn (1956) has shown that blood glucose levels may be elevated in severe hypothermia, probably as a result of impaired utilization of carbohydrates. On the other hand, Mann & Elliot (1964) found low blood sugar levels in some infants, and Kedes & Field (1964) found hypothermia a useful clue to hypoglycaemia. Hypoglycaemia may be found in those patients whose underlying lesion is panhypopituitarism.

A study of the literature shows that abnormal investigation results are found much more often in patients with long-standing hypothermia. In acute exposure results are normal.

Diagnosis

This requires a high index of suspicion and a knowledge of the circumstances in which hypothermia may occur. A casually recorded normal temperature does not exclude hypothermia, as the level noted may be that to which the mercury column was last shaken. Routine use of low reading thermometers is advocated (Fig. 4). The cold feel of the abdominal skin is a useful clue to diagnosis.

Treatment

Immediate removal of the patient from a cold or damp environment is imperative, and great care must be taken with the unconscious cases. Inhalation of vomit is a common cause of death. All but those with the mildest degree of hypothermia should be admitted to hospital.

Hypothermia does not lend itself to controlled trials of different forms of treatment. It is generally accepted that the longer the patient has been hypothermic and the older and less fit he was previously, the more slowly should his temperature be returned to normal. The converse also applies. Thus Pugh (1966) recommends a hot bath for young conscious adults suffering from acute exposure. Keatinge (1964) also recom-

FIG. 4. The low reading thermometer must be shaken down, and should be used rectally. A false reading could still be obtained if the patient is colder than the environment.

mends immediate immersion in the nearest hot bath for the previously young and fit. Keatinge minimizes the well-known 'after-drop' in temperature caused by the flow of cool peripheral blood into the central circulation, by leaving the limbs out of the hot water. Arneil & Kerr (1963) varied the rate of re-warming of their infants according to the duration of hypothermia. With elderly hypothermic patients, Duguid *et al.* (1961) found that rapid re-warming of six subjects led to immediate death.*

When treating accidental hypothermia of the elderly it is usual to place the patient between warm blankets in a warm ward and to aim for a temperature rise not exceeding 1°F (0.55°C) per hour. The Special Committee of the British Medical Association (1964) recommends correction of dehydration with parenteral fluids, such as 5% glucose, or low molecular weight dextran. Intestinal absorption in rats has been shown by Tadzer (1961) to be suspect at low temperatures, and the oral route should not be relied upon for any medication. Intramuscular and subcutaneous routes should not be relied upon in the presence of vasoconstriction. A broad-spectrum antibiotic such as tetracycline should be given (intravenously) as chest infection is almost invariable. Hydrocortisone hemisuccinate should be given routinely, 100 mg 6- or 8-hourly (intravenously) to combat stress and possible adrenocortical insufficiency. It is logical to give routine oxygen (McNicol & Smith, 1964) and there may be an advantage in hyperbaric administration. If hypothyroidism seems likely on clinical grounds, intravenous triiodothyronine should be given, 10 µg twice or thrice daily. If thyroid preparations are given, they must be used with caution. The elderly or myxoedematous myocardium may be ischaemic and electrocardiographic control is desirable. Vasoconstrictors are often used if the systolic blood pressure falls below 100 mmHg. Noradrenaline, mephentermine and metaraminol

*Davies, Millar & Miller (1967) have successfully applied extracorporeal blood-warming to one patient, and suggest that this method needs further trial.

have been used with variable claims to success (Hyam, 1963; Rosin & Exton-Smith, 1964; McNicol & Smith, 1964), and it is difficult to withhold pressor amines in these circumstances. It is theoretically preferable to correct the factors underlying the hypotension. Details of the management of myxoedema coma have been reviewed by Hyam (1963) and by Lovel (1962) and of hypopituitary coma by Sheehan & Summers (1952). In this respect it is important to note that the administration of thyroid preparations alone to hypopituitary patients may precipitate adrenal crises. Adrenocortical steroids should therefore be given before thyroid replacement.

Mortality

The Committee on Accidental Hypothermia of the Royal College of Physicians of London (1966) found a mortality of 37.3% in the 126 patients admitted to the ten hospitals surveyed. With temperatures below 30°C (85°F) the mortality was 73.3% of fifteen patients.

Knocker (1955) has studied the effect of temperatures of 25°C (77°F) on vital organs of dogs. She found fatty changes in the liver, kidney and adrenal glands, depletion of liver glycogen and vacuolation of granular cells in the zona reticularis of the adrenal. Both Mant (1964) and Duguid *et al.* (1961) record the post-mortem changes in their patients that succumbed. The fatty changes were again found in vital organs, and micro-infarcts were found in the heart. Gastric mucosal changes included ecchymoses, sloughing, superficial erosions and perforating ulcer. Some of these changes may not be direct effects of hypothermia. Visceral infarcts were recorded in the small intestine, lung, spleen, kidney, brain and heart. Pancreatitis was found in several cases, and necrotizing parotitis in one instance.

Summary

Accidental hypothermia is a common condition in certain groups of patients especially in cold weather. As it is potentially lethal its early recognition and treatment is vital.

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