

Medical Problems of Cold Weather

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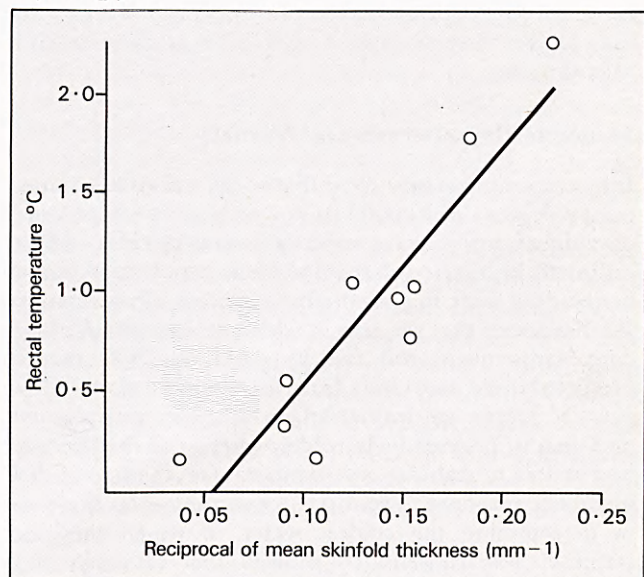
Mortality caused by cold weather among elderly people has been one of the most widely discussed of all medical problems in Britain in recent years. In the public mind, this mortality has come to be associated almost exclusively with hypothermia. Hypothermia does cause some deaths and it contributes to others, but, although it has been a major cause of losses of life in special situations in the past, current evidence suggests that it is not a major factor in approximately 40,000 excess deaths that occur every winter in England and Wales, mainly among elderly people. The public interest shown in these problems has helped to direct attention to the problems of elderly people in winter. A negative side of the public interest is that popular preoccupation with hypothermia came to affect scientific thinking, and delayed the investigation of other, and more large-scale, causes of death in cold weather. Winter mortality is, in fact, only the latest example of popular beliefs diverting investigation of cold-induced problems, and much of the misconception on this occasion resulted from initial successes in identifying hypothermia as the cause of earlier problems.

The most striking of these was death in the water after shipwreck. Observant individuals throughout history realised that shipwreck victims often died of cold rather than by drowning. The earliest known example both of this and of seasonal influence on mortality was recorded by Herodotus in the first book written on the mainland of Europe, in the Fifth Century BC[1]. In recording the wreck of Darius's fleet invading Greece, early in the year, he states that cold rather than drowning caused many of the deaths, but at the Battle of Salamis, fought in summer, the Greeks lost few men as they were able to swim to safety if their ships were sunk. Later, however, by a kind of international literary and journalistic convention, people lost after shipwreck were routinely assumed to drown. Shakespeare gives an example of this in the opening scene of *The Tempest*. In more recent times, 'drowned at sea' was the phrase almost exclusively used in newspaper accounts of people lost at sea up to 1960, and is sometimes used today. Although this was little more than a verbal convention, it affected people responsible for lifesaving equipment at sea, to the extent that they seldom considered anything except the need for flotation equipment. It was only towards the end of the Second World War that the consequences of this became clear, when a Committee under Rear-Admiral A. G.

Talbot reported that most of the Royal Navy men lost in that war, some 20-30,000 men, died in the water rather than from battle injuries, and that without thermal protection they had often died from simple body cooling rather than drowning. This was further documented a decade later[2].

It was largely due to this realisation that extensive studies were made during the next two decades of the internal factors that control body heat loss and heat production in cold water[see 3, 4 for reviews]. The results of that work provided a useful background to consideration of other hazards of cold, since immersion largely eliminates external thermal insulation, leaving survival dependent on the uncomplicated operation of internal mechanisms of heat conservation and heat loss. The results showed that once people were in cold water, and vasoconstriction developed, most of the internal insulation of the body was provided by subcutaneous fat (Fig. 1), so that fat people could maintain body core temperature indefinitely in water at 12°C, while thin people often cooled progressively in water as warm as 25-29°C.

Figure 1. Effect of subcutaneous fat on body cooling during 30-minute immersions in water at 15°C (Courtesy *Journal of Physiology*).



Children usually cooled rapidly in cold water[5], partly because young children, particularly boys, are usually thinner than adults, and partly because the small size of young children gives them a high surface area/mass ratio. Another point of practical importance was that exercise greatly accelerated heat loss by increasing blood flow to limb muscles; in cold water, with little external insulation; this outweighed the increase of heat production in exercise, thus increasing the rate of fall of body temperature. It was also found that in water colder than 12°C cold vasodilatation caused blood flow to return to the limbs and so led to rapid body cooling even in relatively fat people. This reaction, which was largely due to cold paralysis of blood vessels of the extremities, seemed to put a lower limit on the environmental temperature that even fat people could survive for long periods. However, an unusually fat Icelandic fisherman recently survived for five hours swimming in water at 5-6°C, and was brought to London by Professor Johann Axelsson; in laboratory experiments, we found that he could stabilise body temperature in such water with little cold vasodilatation, apparently because an unusually thick layer of limb fat protected the limb arteries from serious cooling[6]. For other people exposed to such a degree of cold, extended survival depends on the wearing of clothing or other external protection to keep skin temperature above the threshold for cold vasodilatation. Even with deaths in water, cold does not produce hazards solely by causing lethal cooling of the body core. Some result from cardiovascular and respiratory reflexes induced by cooling of the skin[7]. The most important of these is inhalation of water due to inability to control respiration during the intense inspiratory drive caused by the reflexes, which readily causes drowning in waves that splash over the head. In addition, reflex drive to the heart occasionally causes sudden death from ventricular fibrillation during the first few minutes of cold immersion[8]. Apart from this, the high viscosity of cold water near 0°C can combine with the reflex effects of cold to cause death from drowning as even fit, healthy young people become rapidly exhausted as they try to swim short distances to shore in very cold water without buoyancy aids[9]; that may be the most important cause of immersion death in inland waters.

Insidious Hypothermia and Memory

It has recently become clear that in special circumstances minor degrees of hypothermia can occur without much discomfort during quite mild exposures to cold, and can indirectly be a serious hazard to life in people carrying out demanding work in potentially dangerous circumstances. We have seen that the rate at which an individual's body core temperature will fall in water at 15°C can be predicted quite accurately from fat thickness alone. However, if people are immersed in relatively warm water, and then in progressively colder water until they become just unable to stabilise core temperature, the size of their metabolic response to cold is as important as fat thickness in determining the coldest water in which they can stabilise. Experiments[10] showed that although such

people's fat thickness did correlate with the temperature of the coldest water allowing them to stabilise, the relationship was not a close one. On the other hand, the subjects' body insulations at the time they stabilised in the coldest possible water were closely related to fat thickness, both at rest and in exercise. The discrepancy was due to metabolic rates, which were very varied and had no clear relationship to fat thickness, so that while body insulation and heat loss could be predicted, metabolic rate and minimum water temperature for stability in this marginal situation could not.

This marginal situation for heat balance is one with wide applications, but the most striking example of these has been in deep commercial diving. Loss of heat to the respiratory gas and from the body can be lethal at depths greater than about 100m, where pressure is more than 10 times atmospheric, and where water on the seabed is at 4-6°C, even in the tropics. Heating is therefore supplied to the diver, usually by an open-circuit hot-water system in which warm water pumped from the surface is used to flood the inside of the diving suit. The popularly accepted principle that if people do not feel cold they are not seriously cold, often led to the assumption that as long as divers were within their limits of voluntary tolerance, cold could be discounted as a cause of any serious problems. By 1976, it became clear that something was causing serious and unexplained problems. The report of the Science Research Council taskforce on marine technology in that year[11] gave unexplained losses of consciousness and confusional states in divers as the problem of top priority for research to assist development of North Sea resources. Childs and Norman[12] obtained notes of 114 such incidents up to 1978. There was also a high rate of fatalities, usually six or seven each year in a diving population of only a few hundred. Although the cause of death could usually be given as diver error, there was often no explanation why a highly skilled man had made some gross mistake, leading to a fatal result. In 1979, we had the co-operation of a North Sea diving company in making measurements of divers' temperatures, as urine temperature, when they returned to the bell at the end of saturation diving shifts[13]. In four of 13 cases, temperature was below 36°C, and one diver was in hypothermia at 34.7°C. At the same time, another was seriously overheated at 38.3°C. Some of the divers felt cold, so the heating water was then clearly on the cool side, and the hot-water system sometimes failed, whereupon the dives were continued without heating for a time while attempts were made to restart the hot-water supply. However, two of the coldest of the divers had neither felt particularly cold nor experienced any failure of heating.

A diver being supplied by water that is a little on the cool side for comfort is essentially in a bath of lukewarm water. Laboratory experiments showed that people in lukewarm water always suffered falls in core temperature, sometimes to hypothermic levels, and again often with little sensation of cold[14]. Figure 2 shows the most dramatic case, in which the subject was a thin man who had undergone considerable recent cold exposure. His rectal temperature fell steadily to 34.7°C with very little shivering and only slight feelings of cold. At this point, he

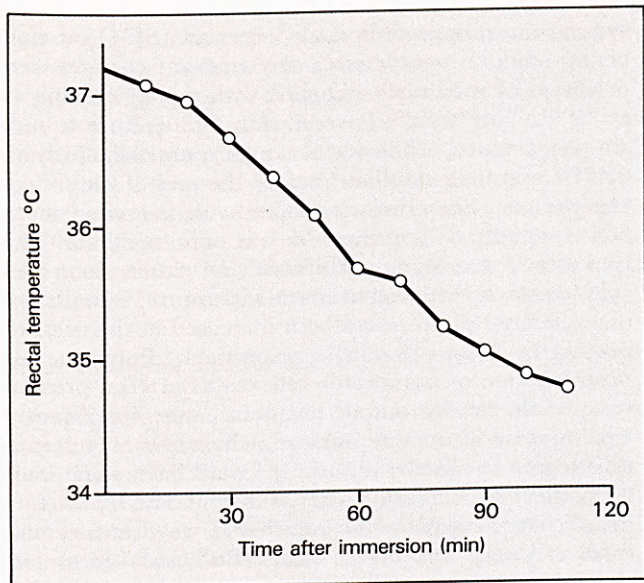


Figure 2. Progressive fall in body temperature during immersion in water at 29°C (Courtesy British Medical Journal).

developed atrial fibrillation, which reverted to sinus rhythm when he was rewarmed. However, the main hazard that these levels of body cooling presented to divers was that they impaired memory and reasoning[15]. When volunteers' body temperatures were lowered experimentally by immersion in cold water, cold discomfort could be abolished immediately by transferring them to warm water. The deep body temperature remained low and even fell more rapidly for a few minutes in the well-known afterdrop. So it was possible during this time to test reasoning and memory with low core temperature but without the complication of distraction by cold discomfort. Such experiments showed that ability to memorise facts was greatly reduced at body core temperatures below about 36°C. Ability to recall facts learned at normal body temperature was, interestingly, not impaired, but there was marked slowing of the speed at which complex reasoning tasks could be performed. For example, the time needed to perform a series of double-digit sums was increased by about 50 per cent at body temperatures below 35°C. These difficulties in memorising new facts and in reasoning could obviously be dangerous to a diver facing an unexpected, complex and demanding situation on the seabed. We cannot say in retrospect how many diving incidents in the 1970s were due to low body temperature, but during the last six years, care has been taken to adjust water temperatures so as to keep divers thermally comfortable and to bring them in at once if heating fails. During that time, such incidents have become uncommon in the North Sea, and measurements by urine temperature at the end of dives from the diving ship Arctic Seal in the North Sea in 1982, at 75 and 135m, showed no cases of core temperature below 36.8 or above 37.5°C (nine cases; personal observations).

Careful control of the hot-water supply to maintain full thermal comfort can therefore prevent hypothermia, even with an even skin temperature, but more reserve can be

provided by allowing the hands and feet to cool and so provide the sensory input needed to activate shivering and vasoconstriction if core temperature does fall. This restores the kind of thermal gradients normally present in cold air, which the human thermoregulatory system is geared to deal with, and it can restore normal thermoregulation to people who otherwise cool progressively with an even skin temperature around 29°C[16]. This problem of mild hypothermia leading to dangerous misjudgements is a hazard specifically to people working in potentially dangerous situations with an even pattern of mild skin cooling. It may be important in a number of occupations in cold weather, but it obviously raises the question whether such insidious cooling could lead directly to death from hypothermia in people exposed to such patterns of skin cooling in air.

Winter Mortality in the Elderly

The hazards of hypothermia had become firmly fixed in the public mind by the early 1960s, so it was perhaps inevitable that when increased mortality among elderly people in cold weather started to attract marked public attention, this mortality should come to be associated with hypothermia. It had been realised at least since 1841 that mortality in Britain increases in winter[17]. Mortality statistics over the last two decades show that this winter mortality is tending to become less, but that there are still about 40,000 excess deaths in an average winter in England and Wales. Analyses of these deaths, particularly by Bull and Morton[18] show strokes and myocardial infarcts accounting for most of them, while hypothermia causes only about one per cent of the excess deaths in winter. It can be argued that the mortality statistics may be distorted by failure to recognise deaths that are due to hypothermia and it has been suggested that the latter might, in fact, be common[19]. Sublingual readings often give an indication of low body temperature in cold surroundings, but sublingual readings give false low indications of body core temperature in cold surroundings, mainly because of parotid saliva entering the mouth via parotid ducts in cold cheeks[20]. The most reliable measurement of core temperature in this situation is by a thermoelectric rectal probe at an adequate depth (>80 mm), and reports that rely on this or on another dependable measure seldom show low core temperatures in people either at home or entering hospital. Most cases of hypothermia admitted to an intensive care unit in Glasgow[21] (from a total of 44 cases in 15 years) were a result of collapse in cold surroundings. The commonest cause of this collapse (25 cases) was poisoning or intoxication by alcohol, drugs or carbon monoxide. Severe physical illnesses such as cerebrovascular accident, myocardial infarct, malignancy or broken limb caused another eight such cases. Another seven of the cases of hypothermia were a result of hypothyroidism or other endocrine disease, or of malnutrition and dehydration, often combined with alcohol. Only two were attributable to cold exposure combined with old age or mental disability alone.

It could be argued that this low incidence of hypother-

mia was due to failure to detect low body temperature in many patients entering hospital. Accordingly, routine measurements of sublingual temperature, with rectal readings if sublingual values below 36°C were obtained, were made on all new patients entering the Emergency and Accident Department of The London Hospital, Whitechapel in January and February 1985. Only three (0.04 per cent of all patients) had core temperatures below 35°C; one of these had suffered a cerebrovascular accident, one a fractured hip, and one with mental impairment had fallen out of bed and been unable to get back onto the bed[22]. Such observations suggest that hypothermia is not only uncommon but, when it does occur, is usually a secondary, and not always important, complication of a serious underlying condition. Effective treatment is, of course, particularly important in those cases in which hypothermia does play a major role in the illness, such as hypothyroidism. People with mild myxoedema are prone to cool in cold surroundings because of defective heat production, while undernutrition can both reduce heat production in the cold[23, 24], and increase heat loss[25]. It is likely that malnutrition has been responsible for rare cases of elderly people with clearly defective responses to cold, who are prone to cool rapidly as a result[26]. Elderly people are often slow to act to correct changes in their thermal environment[27]. There are rare cases of grossly defective thermoregulation causing hypothermia in otherwise normal and well-nourished adults, who are often not elderly, perhaps due to hypothalamic defects. It is less clear whether elderly people in general have impaired responses to cold; reduced responses have been reported in groups of elderly people[28, 29], but variability in the response of both young and old is very great[30] and normal metabolic response to cold has been found in well-nourished elderly people[24]. The present evidence indicates that deaths caused directly by all types of hypothermia in cold environments, though individually important, represent only a small part of the overall excess mortality in cold weather in Britain, even among the elderly.

The problem then is to explain the large number of excess deaths that occur in winter, particularly from

arterial thrombosis. We made experiments[31] on eight young adults to see whether any relevant changes were produced by mild surface cooling with rapidly moving air at 24°C. This rapidly lowered skin temperature to near air temperature, while rectal temperature fell slowly by 0.4°C, and then stabilised before the end of the 6-hour experiments. Shivering developed with increased metabolic rate but, even at the end, was only moderate. This was therefore a mild, sustainable and closely controlled cold stress with little fall in core temperature. Systolic and diastolic arterial pressures both increased in the cold, on average by 12 and 18 mmHg respectively. Pulse rate fell, probably due to baroreceptor reflexes as arterial pressure rose, while cardiac output did not change significantly. The increase in arterial pressure, which occurs to at least this degree in elderly adults[32], could have some long-term effect on increasing arterial thrombosis, but seemed insufficient to explain the rapid cold weather mortality from coronary thrombosis which Bull and Morton[18] showed to reach a peak within 24 hours on a cold day. The probable explanation of this was provided by increases in platelets, as well as red cell count and haematocrit, during the exposure to cold (Table 1). An increase in haematocrit is a known effect of cold, though it does not previously seem to have been associated with cold weather mortality. It was of particular interest, since arterial thrombi in their early stages are largely formed by deposition of platelets, that the mean size of circulating platelets, as well as platelet count, increased in most cases. The increase in platelet number, like the increase in red cells, could be partly attributed to haemoconcentration following vasoconstriction, but the tendency to increased size of platelets suggests addition of large and therefore young and active platelets to the circulation. These changes in platelet size and numbers would clearly tend to increase arterial thrombosis, in cold weather, among elderly people with atheromatous vessels. There is recent evidence that increases in red cell count also markedly increase platelet deposition, probably by physically driving platelets against the vessel wall, so the increase in red cells in the cold is also important. There was also an increase in neutrophil polymorphs during

Table 1. Changes in blood cells and platelets. Values are means (SE in parentheses)[31].

		Before experiment (n = 8)	Change during 1st hour (n = 6)	Change during 6 hours (n = 8)
Red cell count (10 ¹² /l blood)	Cold	4.65 (0.23)	+ 0.11 (0.06)	+ 0.35 (0.07) ^{b,d}
	Control	4.63 (0.15)	- 0.15 (0.05) ^c	- 0.03 (0.03)
Packed cell volume	Cold	0.395 (0.025)	+ 0.007 (0.004)	+ 0.029 (0.005) ^{b,e}
	Control	0.391 (0.017)	- 0.012 (0.004) ^c	0.000 (0.003)
Platelet count (10 ⁹ /l blood)	Cold	291 (27)	- 10 (4)	+ 23 (9) ^c
	Control	287 (28)	- 15 (5) ^c	+ 7 (5)
Platelet volume (fl)	Cold	8.8 (0.5)	0.0 (0.2)	+ 0.2 (0.1)
	Control	8.7 (0.4)	0.0 (0.1)	0.0 (0.1)
Platelets as fraction of plasma by volume (10 ⁻³)	Cold	4.12 (0.21)	- 0.07 (0.11)	+ 0.61 (0.13) ^{a,d}
	Control	4.01 (0.22)	- 0.23 (0.07) ^c	+ 0.12 (0.11)
Neutrophil count (10 ⁹ /l blood)	Cold	3.79 (0.31)	+ 0.42 (0.28)	+ 2.04 (0.47) ^{a,d}
	Control	3.45 (0.33)	- 0.17 (0.06) ^c	+ 0.74 (0.29) ^c

Difference from control: ^a p < 0.05; ^b p < 0.01

Difference from initial value: ^c p < 0.05; ^d p < 0.01; ^e p < 0.001

these exposures to cold, and these changes together caused an increase in whole blood viscosity which rose by 20 per cent in the cold. Very little of these changes took place in the first hour but they were well developed in six hours. Plasma protein ratios of high-to-low-density protein did not change in the cold, but plasma cholesterol, in all its fractions, increased. The increases in platelets, red cells, blood viscosity, arterial pressure and plasma cholesterol could clearly all contribute, to different degrees, to the increase in arterial thromboses in cold weather.

Other Problems in Hot and Cold Weather

An explanation is incidentally also needed for the fact that mortality increases if minimum daily temperature rises above about 20°C[18, 33]. This heat-induced mortality is a much greater problem in North America than in Britain, but even in Britain heatwaves produce marked rises in mortality every two or three years[34]. Again, although most work[35] on heat-induced mortality has concentrated on the effects of simple overheating of the body, mortality statistics show arterial thrombosis as responsible for about half the deaths in major heat-waves, and heat-stroke for rather few. Although changes in blood composition and arterial pressure offer a straightforward explanation for the increased mortality from arterial thrombosis in the cold, many questions remain to be answered on this as well. Efforts to optimise the level of home heating are obviously desirable, if only for the sake of general comfort and welfare. However, it is by no means certain that this alone will prevent excess mortality in winter, and substantially more information may be needed to find ways of minimising this mortality.

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