

Cold Induced Thermoregulatory Failure: 1: Physiology and Clinical Features¹

by
BH Short²

Abstract

This is the first part of a two-part review, which looks at the effects of cold on the body. In this article, the physiology of thermoregulation, the mechanisms of heat loss and the clinical features of thermo--regulatory failure are addressed. The second article, to be published in the next issue, will review the management of accidental hypothermia.

Introduction

The manifestations of cold-induced thermo- regulatory failure are of great military significance. Over centuries, commanders have encountered the horrendous mortality and morbidity accompanying the general cold injury syndrome (hypothermia) and the local cold injuries of chilblains immersion (Trench) foot and frostbite. The devastation exacted by these disorders on Napoleon's army in Russia and in the Peninsula War of 1808-1814, on the armies of both sides during the Crimean and the American Civil Wars, on almost all armies during the World Wars (on land and at sea) and more recently, during the Korean War and the 1982 South Atlantic War, have been widely documented. The effects of hypothermia may befall armies operating in deserts, in mountains, in jungles, and in fact in any climate and during any season of the year.

Thermoregulation

The normal range of human core body temperature is 36.4°C to 37.5°C (97.5°F to 99.5°F). Hypothermia is defined as a lowering of the central core temperature below 35°C (1) whilst hyperthermic dysfunction, typified by the Heat Stroke syndrome, is associated with core temperatures exceeding 40°C.

The steady core temperature is maintained by the balance of heat loss and heat production. The set value of the body temperature is maintained by nuclei in the preoptic anterior hypothalamus. This thermoregulatory centre conserves heat by producing cutaneous vasoconstriction and stimulating muscular activity in the form of shivering. Low-temperature activation of thermostats in the thermal centre, as well as cutaneous cold receptors, induces a series of compensatory changes. Such changes are marked by progressive metabolic depression in each organ system. Substantial individual variability in these cold-induced physiological sequelae is common. (2)

The body's predominant source of heat production results from metabolic activity within the heart and liver and are thus important contributors to the homeothermic core. Ninety per cent of the body's heat loss is via the skin and a very important portal is the unprotected especially bald-headed, scalp. The remainder of the loss is via the respiratory tract. The skin and superficial tissues comprise the body's so-called poikilothermic shell. Cold-induced thermoregulatory failure occurs when peak heat production is insufficient to counter the rate of heat loss due to falling ambient temperature leading to progressive multi- organ dysfunction.

Mechanisms of Body Heat Loss.

Heat Loss occurs via the following processes:

Convection.

The movement of air (or water) aids the transfer of heat from the body to the colder surrounds. The drop in skin temperature by the increasing wind velocity relates to the dissipation of the layer of warm air around the skin,

known as the wind-chill factor. Convective cooling is much greater in water than in air. Wind and wet cause a marked increase in the rate of heat loss and a body loses less heat at -10°C in still air than at $+10^{\circ}\text{C}$ when exposed to a 33 kph (20mph) wind (3). Convection is the major cause of heat loss from the body at high altitude.

Radiation.

In this process, direct contact with air is not necessary for the heat loss since the heat energy is transmitted to the environment in the form of electromagnetic waves. Thermal radiation accounts for up to 60% of the metabolic heat loss at sea level and is independent of air movement.

Conduction.

Conductive body heat loss occurs by direct contact with agencies at a lower temperature. In this respect, air conducts heat inefficiently such that a layer of still air acts as a good heat insulator. On the other hand, water conducts heat some 240 times greater than air with the effect that wet clothing or immersion rapidly induces reductions in core temperature.

Evaporation.

The so-called latent heat of evaporation is the transfer of energy required to change liquid to a vapour: body heat loss by the evaporation of sweat. At altitude, cold air with low humidity accentuates this heat loss. At sea level, 40% of body heat loss occurs via evaporation with insensible water loss and sweating from the skin together with respiratory fluid losses. Wet clothing exposed to wind accelerates evaporative body heat losses.

Classifications of Hypothermia.

The existing classifications are contentious. The temperature of less than 35°C proposed by the RCP Committee Report (1966) to indicate hypothermia was selected to allow for the maximum diurnal variation of core temperature. Lloyd highlights the artificiality of the classification based on core readings which might suggest that 35.5°C is not hypothermia and therefore safe while 34.5°C is hypothermia and the patient is in danger. Neither readings, however, take into account the total body heat. (4) Accidental (unintentional) hypothermia is classified as mild (body core temperature 32.2 to 35°C) moderate (28 to $<32.2^{\circ}\text{C}$) and severe ($<28^{\circ}\text{C}$) (2).

Hypothermia may also be classified according to the duration of exposure: (5)

Acute hypothermia results from the excessive and sudden cold stress that overrides cold resistance despite heat production being at or near-maximum. Hypothermia occurs before exhaustion develops. This type most commonly occurs following cold water immersion.

Subacute hypothermia results from physical exhaustion and depletion of body's food stores, which cause a failure in metabolic heat production. Mountaineers and trekkers commonly sustain this form of hypothermic insult due to cold, wind and rain.

Chronic hypothermia is the result of prolonged exposure to a mild degree of cold stress and while the thermoregulatory response is not overwhelmed it is insufficient to counteract the cold. This type is commonly seen in the urban dwelling elderly.

A fourth variety has been proposed namely, **submersion hypothermia**. The common factor is total submersion in ice-cold water. The younger the victim the greater the chance of survival. Cases are reported of successful resuscitation without brain damage after emersion for up to 60 minutes. (3)

Causes of Hypothermia.

The leading causes of accidental hypothermia in urban medical centres in the US are exposure to alcoholism, drug addiction or mental illness and accidents involving immersion in cold water (6). Hypothermia due to accidental cold exposure is encountered most commonly among neonates and the elderly, in those mobilised by trauma

or exhaustion, and in intoxicated and unconscious personnel. Depending on the population analysed, alcohol is associated with 80% cases of accidental hypothermia (7). The predisposing factors for cold-induced thermoregulatory failure are in Table 1.

Decreased Heat Production:	Insufficient Fuel:	Hypoglycaemia Malnutrition Extreme Physical Exertion.
	Physical Exertion:	Inactivity Impaired Shivering Young and the Elderly
	Endocrine Failures:	Hypoadrenalism Hypopituitarism Hypothyroidism
Increased Heat Loss:	Environmental	Immersion Non-immersion
	Iatrogenic	Cold Infusions Surgical Exposure Emergency Deliveries
	Skin Disorders	Psoriasis Exfoliative Dermatoses Bums
	Vasodilatation	Alcohol and Drugs Toxin induced
Impaired Thermoregulation	Central Nervous System Failure	Toxins Cerebrovascular Accident Metabolic Disorders Drugs Trauma Degenerative disorders (Multiple Sclerosis; Parkinsons)
	Peripheral Nervous System Failure	Acute Spinal Cord Transection Diabetes Neuropathies
Miscellaneous:		Uraemia Shock Infection Pancreatitis Multisystem Trauma Carcinomatosis.

*Table 1: Predisposing Factors for Cold Induced Thermoregulatory Failure.
(After Danzl DF, Pzos RS. Accidental hypothermia N Eng/J Med 1994; 331:1757)*

Clinical Features of Hypothermia.

A history of cold exposure provides a straight- forward diagnosis of hypothermia; however, historical facts do not always suggest hypothermic dysfunction. In addition, commonly there is a substantial individual variability in the clinical manifestations of the physiological changes. Cold-recording electronic thermometers with flexible probes are available to measure rectal, oesophageal and bladder temperatures. Core temperature is best recorded rectally utilising thermometers capable of measuring as low as 25oC. Reliance on infrared tympanic thermography for the accurate assessment of hypothermia presently requires further trial substantiation.

In all body systems, hypothermia induces a progressive depression of metabolism. It typically has an insidious onset and may be ushered in by non-specific symptoms consisting of chills, dyspnoea, nausea or dizziness.

Central Nervous System Features.

Mild hypothermia produces gradual impaired judgement, memory disorder, speech impairment and apathy. Beware the mountain walker who commences to repeatedly and unaccountably stumble, fails to follow the trail, starts to lag behind or who develops inappropriate or incoherent speech. He may be hypothermic or developing high altitude cerebral oedema or a combination of both. Increased pre-shivering muscle tone is followed by shivering-induced thermogenesis.

Moderate hypothermia leads to a diminution in shivering and the gradual replacement by motor rigidity. Alterations in consciousness now appear with episodic hallucinations and the paradoxical removal of thermal protective clothing.

Severe hypothermia is typified by the ab-sence of spontaneous movement, stupor and coma with reductions in cerebral blood flow due to the loss of cerebrovascular auto- regulation. The severely hypothermic individual must therefore always be rewarmed before the pronouncement of brain dead.

Cardiovascular Features.

Mild hypothermia induces vasoconstriction and an increase in cardiac output, arterial pressure and pulse rate. Tachycardia is soon followed by bradycardia and cycle prolongation with a consequent lengthening of all ECG intervals.

Temperatures between 32°C and 28°C are associated with further pulse slaving and a decrease in both arterial pressure and cardiac output. Atrioventricular arrhythmias become evident. ECG recording may show non-specific changes in the J-wave known as the Osborn wave. This positive deflection at the J point the junction of QRS and ST segments is in the same direction as the QRS complex and is seen in about one-third of cases with core temperatures below 33oC. (8,9) The height of the Osborn wave is roughly proportional to the degree of hypothermia, however, it does not carry prognostic implications. (10) This deflection is not pathognomonic for hypothermia and may be seen in sepsis and some CNS lesions. (11)

Severe hypothermia produces further depression in rate, pressure and output with a decrease in ventricular arrhythmic threshold and concluding in asystolic arrest. Re-entrant dysrhythmias are now frequent and are of major clinical significance during rewarming efforts.

Respiratory Changes

Mild hypothermia produces early tachypnoea followed rapidly by reductions in respiratory minute volume in association with the declining oxygen consumption. Cold-air induced bronchorrhoea and bronchospasm may now be seen.

Progressive hypoventilation in association with the falling metabolic production of CO₂ is associated with loss of protective airway reflexes. The blunted cough reflex is associated with hypostatic atelectasis.

Severe hypothermia leads to apnoea in association with the marked diminution in oxygen consumption. Pulmonary congestion and frank oedema may also appear.

Renal Changes

Mild hypothermic changes result in a cold- induced diuresis, which may result from the peripheral vasoconstriction as well as from a blunting of the tubular effects of ADH.

Progressive hypothermia results in a decreased renal blood flow secondary to the fall in cardiac output. Extreme oliguria supervenes and this may be exacerbated in those with acute tubular necrosis secondary to rhabdomyolysis associated with hypothermic-coexistent compartment syndromes. Hypothermia typically masks the changes in the ECG caused by the accompanying hyperkalaemia.

Haematological Changes.

Coagulopathies often develop in the hypothermic setting despite normal clotting factor levels. (12) Cold directly inhibits the coagulation cascade. At -33°C there is a 50% reduction in clotting function. Concurrently there is a diminution in platelet activity due to both a reduction in thromboxane B2 production by platelets (a thermal-dependent process) as well as cold-induced thrombocytopenia secondary to both marrow depression and hepatosplenic sequestration. (13). The assessment of the coagulopathy by measuring the prothrombin time and partial prothromboplastin time, which at 37°C may be entirely normal, fails to correctly identify the bleeding disorder.

Metabolic Changes.

Similar to hypocapnoea and alkalosis, hypothermia shifts the oxyhaemoglobin-dissociation curve to the left resulting in a decrease in oxygen release from haemoglobin into the tissues at a lower partial pressure of oxygen. This reduced oxygen release to tissues is exacerbated by hypothermia-induced vasoconstriction, ventilation-perfusion mismatch, and the increasing blood viscosity associated with the dehydration and fluid sequestration attending lengthy cold exposure. The haematocrit increases 2% for each 1°C temperature drop. (2)

Mild hypothermia induces catecholamine release with an increase in glycogenolysis with resultant transient hyperglycaemia. However, with persisting cold exposure there is glycogen depletion and therefore hypoglycaemia. Hypoglycaemia per se may also be the cause of the accidental hypothermic insult. The finding of hyperglycaemia in the hypothermic patient suggests diabetic ketoacidosis or pancreatitis.

The initial mild hypothermic shivering enhances metabolism in association with an increase in catecholamines, adrenal steroids and thyroxine. A continuing fall in temperature results in up to 80% reduction in basal metabolism and ultimately the adoption of the poikilothermic state.

References

1. Royal College of Physicians. Report of Committee on Accidental Hypothermia. Royal College of Physicians, London; 1966.
2. Danzl DF, Pozos RS. Accidental hypothermia. *N Engl J Med* 1994; 331:1756-60.
3. Lloyd EL *Hypothermia and cold stress*. London: Croom Helm; 1986.
4. Lloyd EL *Resuscitation* 1996; 32: 111-124.
5. Ward MP, Milledge JS, West JB. *High altitude medicine and physiology*. 4th ed. Oxford: Oxford Medical Publication; 1995: 473
6. Urschel JD. Frostbite: predisposing factors and predictors of poor outcome. *J Trauma* 1990; 30:340-2.
7. Fitzgerald Fr. Hypoglycaemia and accidental hypothermia in an alcoholic population. *West J Med* 1980; 133(2): 105-7.
8. Gussack I, Bjerregaard P, Egan TM, et al. ECG phenomenon called the J wave: History, pathophysiology and clinical significance. *J Electrocardiogr* 1995; 28: 49.
9. Solomon A, Barish RA, Browne B. et al. The ECG features of hypothermia. *J Emerg Med* 1989; 7: 169.
10. Wagner GS. *Marriott's practical electrocardiography*. 9th ed. Baltimore: Williams & Wilkins; 1994.
11. Weinberg AD. Hypothermia. *Ann Emerg Med* 1993; 22: 370-77
12. Patt A, McCroskey BL, Moore EE. Hypothermia-induced coagulopathies in trauma. *Surg Clin Nth Am* 1988; 68: 775-85.
13. Rosenkranz L Bone marrow failure and pancytopenia in two patients with hypothermia. *South Med J* 1985; 78: 358-9