

10.3.3 Cold 1689

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10.3.3 Cold 1689 capacity (e.g. anticholinergics). Salicylate overdose can generate heat stroke by increasing metabolic heat production while impairing hypothalamic regulation. There are two types of heat-related drug reactions, however, which are particularly dangerous. Malignant hyperpyrexia This is usually a dominantly inherited condition, although different gene defects may affect families. Administration of a variety of anaesthetic agents, including halothane and suxamethonium, leads to rapid, massive heat production from generalized increases in skeletal muscle tone. Contraction is triggered at the muscle cell membrane and hence neuromuscular blocking agents are ineffective. Intravenous dantrolene, an inhibitor of muscle calcium flux, is helpful and can be used along with ventilation and cooling/supportive measures. Fatalities are common, and it is therefore important to avoid risks whenever possible. In patients with a relevant personal or family history, in whom an anaesthetic is unavoidable, oral dantrolene should be given prior to the use of low-risk agents. Neuroleptic malignant syndrome This condition has similarities to malignant hyperpyrexia but is induced by idiosyncratic reactions to normal doses of antidopaminergic drugs, including phenothiazines and butyrophenones. The onset is less rapid than malignant hyperpyrexia, occurring over a few days. The increased muscle tone is also induced presynaptically and hence neuromuscular blocking agents help. Some recreational drugs, such as ecstasy, can induce this type of response, although most cases of ecstasy-induced hyperthermia are probably cases of heat stroke induced by enthusiastic dancing with limited fluid intake in hot, humid environments. FURTHER READING Bouchama A, Knochel JP (2002). Heat stroke. *N Engl J Med*, 346, 1978–88. Hodgson P (1991). Malignant hyperthermia and the neuroleptic malignant syndrome. In: Swash M, Oxbury J (eds) *Clinical neurology*, pp. 1344–5. Churchill Livingstone, Edinburgh. Hubbard RW, Armstrong LE (1988). The heat illnesses: biochemical, ultrastructural, and fluid-electrolyte considerations. In: Pandolf KB, Sawka MN, Gonzalez R (eds) *Human performance physiology and environmental medicine at terrestrial extremes*, pp. 305–59. Benchmark, Indianapolis, IN. 10.3.3 Cold Michael A. Stroud ESSENTIALS Humans are poorly adapted to cold, which can cause hypothermia, nonfreezing cold injury, and frostbite. Hypothermia This occurs especially with wind and wetting, and is seen indoors in older people and those who are thin. At a core temperature of 35°C, victims complain of cold, act appropriately, shiver, and are peripherally vasoconstricted, but with further cooling they may become confused or drowsy and appropriate physiological responses disappear. Coma occurs at 26–32°C, and death typically at 17–26°C. General investigation and management is as for any comatose patient, but specific issues include (1) accurate measurement of core temperature requires a low-reading rectal thermometer; (2) measurement of serum amylase (risk of pancreatitis) and creatine kinase (risk of rhabdomyolysis); (3) rewarming—if onset of cooling was prolonged, rewarming should generally

be slow; (4) diagnosis of death— apparently dead victims should be rewarmed whenever possible before resuscitation is abandoned. Nonfreezing cold injury This occurs when skin temperatures below 12°C are maintained for prolonged periods, particularly in water (e.g. trench foot). This causes local tissue damage, particularly to nerves, which can be permanent. Frostbite Frozen tissues initially appear hard, white, and anaesthetic, but with rewarming become swollen, painful, and blistered. There may be irreversible necrosis, but initial appearances can be misleading and hence early amputation should be avoided. Once thawed, frostbite treatment is similar to that for burns. Thermoregulation in the cold It has only been 10 000 to 15 000 years since ancestral humans dwelt exclusively in warm or hot climates. Humans are therefore poorly adapted to cold, and hypothermia occurs quite frequently even in temperate regions. With water immersion it may occur even in the tropics. In truly cold areas, there is also the risk of nonfreezing cold injury and frostbite. Nevertheless, behavioural changes allow us to operate safely even in the coldest environments. Core temperatures in the cold are usually maintained by adjustments in clothing and physical activity. The latter can increase heat production from a resting 100 W to 1–2 kW. This is very effective. Although it takes highly specialized, multilayered clothing to keep warm while inactive in an environment of +5°C, clothing insulation equivalent to normal office dress (1 clo) will maintain core temperature even in an environment of –20°C when working moderately hard. Our limited physiological cold protection is under hypothalamic control. Falling surface and, to a lesser extent, core temperatures lead to decreased blood flow in the skin due to increased sympathetic adrenergic tone and direct cooling effects of cold on skin arterioles. This minimizes surface heat loss. Unfortunately, vasoconstriction also leads to severe cooling of the hands and feet with problems of temporary skin numbness, muscle weakness, and risks of more permanent peripheral cold injury. It is often this peripheral cooling that limits our capacity to work in the cold. Falling skin temperatures will also lead to higher resting muscle tone and shivering, especially when declining core temperature

SECTION 10 Environmental medicine, occupational medicine, and poisoning 1690 releases hypothalamic inhibition of shivering. These mechanisms can only increase resting heat production to around 500 W and, unlike newborn infants and some other mammals, adult humans cannot add significant nonshivering heat production to this figure. Effects of falling core temperature Falling core temperature leads to progressive decline in function. At 34–36°C, hypothermic individuals are conscious of feeling cold and try to move around, add clothing, or seek shelter. Simultaneously, physiological defences are activated. With further falls of temperature, mental and physical problems increase. Some people become withdrawn while others exhibit aggression or disinhibition. Once core temperatures reach 33–34°C, victims often stagger and become confused or drowsy. It is also around this point that ‘paradoxical undressing’ may occur. This phenomenon is well described and appears to be due to hypothalamic dysfunction with alteration of set-point temperature. Victims therefore feel warm or even hot and appropriate behavioural and physiological responses disappear. At core temperatures varying between 26 and 32°C coma will ensue, and between 17 and 26°C cardiac output becomes inadequate to sustain life for prolonged periods. The risk of ventricular fibrillation is also high. Nevertheless, successful resuscitations of victims with core temperatures below 15°C have been reported (see also Chapter 9.5.3). Causes of hypothermia Several factors increase hypothermic risk. Wetting of skin or clothing extracts enormous amounts of heat and reduces insulation of garments. Complete immersion is particularly hazardous and worldwide more than 100 000 people per year die of cold shock or inexorable hypothermia in the water. This far exceeds deaths from drowning without cold. Winds also increase environmental cooling and a still air temperature of +5 °C equates to –50 °C if wind speed is 40

km/h. Coupled with rain, these effects often contribute to hypothermic accidents among hill walkers and mountaineers, although in these cases fatigue may contribute. Prolonged exertion depletes muscle glycogen which reduces heat production capacity from both exercise and shivering. Low blood glucose also impairs hypothalamic temperature control. Small, thin people cool easily because of their increased surface-to-volume ratios. They also have reduced subcutaneous insulation and low heat-producing mass. A fat person can maintain core temperature at rest, even if mean skin temperature is 12°C, whereas a thin person struggles to maintain thermal equilibrium with a skin temperature of 25°C. However, rapid cooling can sometimes have benefits. A small child in cold water may cool so rapidly that vagally triggered bradycardia and lowered brain metabolic demands may permit successful resuscitation after very prolonged immersion. Older people may also be small and thin and are at risk of so-called 'urban hypothermia'. Poverty, illness, immobility, malnutrition, and a less sensitive regulatory system may contribute, but in many cases hypothermia on admission to hospital is secondary to other pathology (e.g. a stroke may have led to prolonged immobility in a cool environment). Drugs that impair consciousness or induce vasodilatation are risk factors, and alcohol is particularly hazardous. Alcoholics with no fixed abode and a tendency to hypoglycaemia are frequent urban cold casualties.

Hypothermic illness

General management of the hypothermic casualty is similar to that for any comatose or semicomatose person. Abnormalities in blood gases, pH, electrolytes, and glucose are common, and pancreatitis or rhabdomyolysis are recognized complications. Accurate measurement of core temperature is surprisingly difficult. Axillary, tympanic, and oral temperatures can all be misleading. A low-reading rectal thermometer is best. Hypothermia has one very specific risk. Pronouncement of death is fraught with difficulty since profound bradycardia, minimal stroke volume, and marked respiratory depression occur. The old adage that you are 'never dead unless warm and dead' must be taken seriously. A variety of rewarming methods are available. Warm blankets and hot drinks will suffice in many cases but, although they are widely used, metallized 'space blankets' are of no proven benefit. Warmed intravenous fluids are helpful and, in extreme cases, peritoneal warmed fluids or cardiac bypass can be used. Specialized equipment providing heated, humidified air also permits core rewarming. Hot baths are effective but difficult to use safely since a paradoxical fall in core temperature can occur as blood flow is rapidly restored to cold limbs. In general, if cooling was prolonged in onset or duration, rewarming must be undertaken with extreme caution. In critical cases, where rapid rewarming is needed, full resuscitation facilities must be available, although safe defibrillation in the presence of water is impossible. Careful monitoring during rewarming is vital. Blood volumes are often low due to early cold-induced diuresis, followed by the inability of hypothermic kidneys to retain salt and water. In immersion casualties, hydrostatic effects on the limbs may have promoted additional fluid loss and, if possible, these people must be kept recumbent throughout rescue and rewarming to minimize risks from extreme postural hypotension. Warming cell membranes are extremely unstable, and uncontrollable fluxes in potassium and other electrolytes may occur, although care must be taken in interpreting biochemical results from cold peripheral blood sampling.

Nonfreezing cold injury

Local temperatures of less than 12°C prevent normal membrane pumping and paralyse nerve and muscle conduction. If such cooling is prolonged, permanent damage may ensue. Immersion in cold water is particularly likely to cause this type of damage and soldiers in military campaigns are frequent victims of 'trench foot'. Long-term damage is likely whenever an anaesthetic, paralysed, cold region becomes hot, red, painful, and swollen after rewarming, although this change may take several days. Degeneration of nerve and muscle can then follow, leading to prolonged anaesthesia, muscle contractures, or inappropriate peripheral vascular control with intolerance to local heat or cold. There may be slow improvement over

months or years.

Revision #1

Created 2026-01-22 16:37:11 UTC by Omar Ayman

Updated 2026-01-22 16:37:11 UTC by Omar Ayman