10.3.2 Heat 1687

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Heat Michael A. Stroud ESSENTIALS Rising body temperature triggers behavioural and physiological responses including reduction in physical activity, alterations of clothing, skin vasodilatation, and sweating. Heat-related illness is relatively common, especially with high humidity or prolonged physical activity. Risk can be reduced by acclimatization with re- peated heat exposure, but some individuals seem to be particularly susceptible. Clinical presentations of heat-related illness include (1) 'heat ex- haustion'—the commonest manifestation, with symptoms including nausea, weakness, headache, and thirst. Patients appear dehydrated, complain of being hot, and are flushed and sweaty. Treatment re- quires rest and fluids, given orally or (in severe cases) intravenously. (2) 'Heat stroke' victims often complain of headache, may be drowsy or irritable, and may claim to feel cold. Core temperature is usually 38–41°C, but the patient is shivering with dry, vasoconstricted skin. Treatment requires (a) aggressive rapid cooling—tepid water and fan-assisted evaporation in the first instance, with more invasive meas- ures (e.g. intraperitoneal fluids, if required); (b) close biochemical monitoring; (c) supportive care for organ failure. There is significant mortality. Thermoregulation in the heat Most of human evolution took place in Africa and hence all hu- mans are heat tolerant. We try to maintain a near-tropical micro- climate against our skin, by using clothing to reduce heat loss to our surroundings. Thermal balance is regulated by the hypothalamus, which integrates information from skin temperature sensors with core temperature data from receptors within walls of large blood vessels and the brain. Rising temperatures trigger both behavioural and physiological responses. Behavioural changes include reducing physical activity, altering clothing, and seeking shade or cool shelter. Cold drinks are also helpful. Although these responses seem simplistic, decisions may not be straightforward. If physical activity is low and water is in short supply, it is better to increase clothing cover and protect your-self from high radiant heat inputs. If activity must be continued and water is freely available, minimal clothing to permit maximal sweat evaporation is preferable. Immediate physiological responses involve vasodilatation of skin and subcutaneous blood vessels to enhance surface heat loss from radiation, conduction, and convec- tion. The vasodilatation is triggered by a sympathetic cholinergic reflex in response to skin warming, with additional direct effects of heat on arteriolar tone. In a resting person, skin vasodilatation can maintain thermal equilibrium in environmental temperatures up to 32°C, but with higher temperatures or heat production from activity, core temperatures will rise. This will trigger sweating to promote evaporative cooling.

SECTION 10 Environmental medicine, occupational medicine, and poisoning 1688 Heat acclimatization Repeated heat exposure can increase our capacity to lose heat by about 20-fold. This is partly due to greater skin blood flow from in- creases in circulating volume and improved vasodilatory responses, but changes in sweating responses are more important. In the nonacclimatized, sweating is triggered by a rise in core tempera- ture of about 1°C and maximum rates are limited to about 0.5 litre/ h. Following acclimatization, a 0.5°C core rise will trigger the re-sponse and sweat rates may exceed 2.0 litre/h. Acclimatization also leads to aldosterone-mediated reductions in sodium loss in both sweat and urine. The acclimatized individual therefore requires no sodium supplementation and giving supplements can delay the ac- climatization process. Avoiding them altogether, however, risks salt depletion in nonacclimatized persons during prolonged heat stress. Acclimatization develops swiftly and around 90% of maximum heat tolerance is present after 7–10 days, on which core temperature has risen by more than 1°C for more than 1 h. Physical exertion com- bined with heat makes the changes even more rapid. After returning to cool environments, adaptation is lost in 20–40 days. Susceptibility to heat-related illness Although we are generally heat tolerant, heat-related illness is rela- tively common, and several factors increase vulnerability. Above an environmental temperature of about 35°C, we tend to gain heat from our

surroundings, and this, along with metabolic heat production, can only be lost via evaporation of sweat. Hot envir- onments with high humidity are therefore the greatest threat. Acclimatization status has a marked influence on heat-related risks, the unacclimatized being prone to hyperthermia and salt depletion, while the fully acclimatized are vulnerable to dehydration from high sweat rates. Dehydration in itself limits sweating capacity and skin blood flow and hence increases risks. It can occur easily since thirst is a poor trigger for adequate drinking. Sweat rates in the acclima- tized can also exceed gut capacity for water absorption. Prolonged physical activity can cause heat illness under quite modest environmental conditions. This is particularly common when individuals are obliged to wear clothing that is insulative or vapour-impermeable. Military heat casualties are sometimes due to these factors, but there have also been fatalities in soldiers who have been susceptible to heat for no obvious cause. Such genetic or constitutional vulnerability should be suspected whenever a heat- related problem occurs following relatively modest heat stress. These people should be strongly advised to avoid similar circumstances in future. Obesity and poor physical fitness are further risk factors in the heat, as is diabetic autonomic dysfunction. Older people are generally heat sensitive and, in addition, are prone to problems from the increased circulatory demands of vasodilatation. Drugs can also induce heat illness (see following paragraphs). Heat exhaustion Most casualties in hot environments suffer from heat exhaustion. There is usually a history of prolonged heat stress followed by nausea, weakness, headache, thirst, and sometimes collapse. Patients appear dehydrated with a tachycardia and low blood pressure. If hyper- thermic, the casualty should be complaining of feeling hot and will appear flushed and sweaty. The absence of these symptoms and signs, especially with a very high core temperature, suggests heat stroke. Heat exhaustion is ascribable to sodium and/or water depletion, but discriminating between these can be difficult. Sodium depletion tends to be greater if the casualty was poorly acclimatized and hence sweated relatively more sodium than water. Conversely, water depletion is more common in acclimatized individuals. Muscle cramps or whole-body dehydration without marked changes in haematocrit or serum proteins are suggestive of excessive sodium loss, but serum sodium tends to be normal in such cases unless enthusiastic fluid replacement without salt has led to hyponatraemia. This sometimes occurs in runners after completing marathons in hot environments. In predominantly water- depleted heat exhaustion, haematocrit, serum proteins, and serum so- dium tend to be high. Renal impairment occurs in either form of heat exhaustion and the treatment of both types often requires 5–10 litres of oral or intravenous fluids in the first 24 h. Sodium supplementation is given as appropriate, but if sodium status is uncertain, it is usually safer to provide some than to precipitate acute hyponatraemia. Heat stroke Mild heat stroke has occurred when a hot environment or high ac- tivity levels have led to pyrexia with cerebral disturbance. Core tem- perature is usually 38–41°C. The condition frequently follows heat exhaustion but temperature may have risen rapidly allowing no time for salt or water depletion. Sufferers have headaches and may be ei- ther drowsy or irritable. They often hyperventilate. The great danger is progression to more severe heat stroke, in which core tempera- ture reaches levels that cause irreversible denaturing of proteins. This usually occurs at above 41.5°C. Damage is widespread and par- ticularly affects brain, liver, kidney, and muscle. Furthermore, the hypothalamic thermoregulatory centre may fail, switching off vaso- dilatation and sweating, and switching on cold defences inappropri- ately. Patients may therefore claim to feel cold and on examination may be shivering with a dry, vasoconstricted skin. A disastrous vi- cious cycle of increasing temperatures can then ensue. Treatment for all heat stroke requires early recognition and rapid cooling. Tepid water and fan-assisted evaporation may be more ef- fective than immersion in cold water, which can limit heat loss by stimulating intense peripheral vasoconstriction. Intraperitoneal fluids, paralysis, and ventilation may be needed and, in extreme

circiumstances, cooling by cardiac bypass should be considered. Hyperkalaemia, hypocalcaemia, acidosis, rhabdomyolysis, dissem- inated intravascular coagulation, and hepatic or renal failure are common complications. Ventricular fibrillation is a frequent ter- minal event. Even if apparently resuscitated and cooled success- fully, a 12- to 24-h ‘lucid interval’ may precede major deterioration. Permanent neurological damage is common. Drug-induced heat illness Many drugs can cause mild degrees of pyrexia by inducing local or systemic inflammation or hypersensitivity. Some also increase sus- ceptibility to environmental heat by inhibiting central thermoregu- lation (e.g. barbiturates and phenothiazines) or reducing sweating

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