# HEAT ILLNESS



## Introduction

Disorders associated with working in hot conditions range from the mildly irritating condition of prickly heat, a skin irritation resulting from copious sweating, to the serious but rare condition of heat stroke with a mortality rate of around 80%.

From an audit of 80 patients with heat related illness Day and Grimshaw [1] proposed classification into four categories based on a combination of clinical findings and haematological and biochemical investigations of fluid and electrolyte status. Different categories were characterised by varying degrees of water and salt loss with reduction of extracellular fluid volume being a common central mechanism. In the fourth and most serious category the biochemical derangements were accompanied by a "loss of normal thermoregulation characterised by high core temperature and paradoxical cessation of sweating."

Clinically four dominant heat disorders are usually described [2]:

- Heat cramps
- Heat syncope
- Heat exhaustion
- Heat stroke

although criteria for distinguishing these are not always consistent.

## Heat cramps

are painful involuntary contractions of the muscles associated with working in hot conditions [3]. However the term heat cramp may be misleading. Increased body temperature is not responsible and Noakes [4] remarks that exercise induced cramps may occur in susceptible individuals whether or not high environmental heat is a factor with evidence suggesting that a spinal neural mechanism may be responsible. On the other hand, Donoghue, Sinclair and Bates [3] found that heat cramps are associated with dehydration (though not hyponatraemia) and Stofan et al. [5] in reporting an observational study of football players prone to cramping, concluded that sweat sodium losses and fluid deficits incurred may be comparatively larger in cramping than non-

cramping players. Unpublished studies in this laboratory corroborate this. These observations support a conclusion that fluid/electrolyte imbalance is involved. As sweat losses are greater in the heat, susceptible individuals would logically be at increased risk of cramping when working or exercising in thermally stressful conditions.

## Heat syncope and heat exhaustion

result from the inability of the circulation to meet both thermoregulatory and circulatory demands [6]. Heat syncope (fainting) occurs when reduced venous return to the heart as a result of excessive pooling in the peripheral vasculature (with or without hypovolaemia) compromises cardiac output and blood pressure cannot be maintained. Heat exhaustion results from severe fluid and salt loss and may manifest with elevated core body temperature (but < 40$^{o}$C) and signs of cerebral ischaemia. From a one year prospective study of 106 cases of heat exhaustion Donoghue [3, 7] has reported that in addition to dehydration the condition is characterised by a clear cut biochemical and haematological profile suggestive of a considerable stress response and impaired tissue perfusion.

## Heat stroke

is a severe condition resulting from breakdown of the thermoregulatory mechanisms resulting in a severe (>40$^{o}$C) or prolonged rise in body temperature and consequent tissue injury. Organ damage is widespread and results from both hypoxia and hyperpyrexia [8]. Acute injury to the heart, kidneys, and liver may be permanent as indicated by a recent study which found that prior hospitalisation for severe heat illness was accompanied by a 40% increase in all cause mortality [9]. An exacerbating factor is the systemic inflammatory response with the release of pyrogenic cytokines potentially contributing to the temperature elevation by increasing the hypothalamic thermoregulatory set-point (i.e. producing fever). In this regard parallels have been suggested with the potentially fatal condition of malignant hyperthermia (MH), Muldoon et al. [10] present evidence suggesting a genetic link between the majority of MH susceptible cases and a subset of exertional heat stroke cases. Studies reviewed by Lambert [11] indicate that reduced splanchnic blood flow during exercise, particularly if accompanied by dehydration, may compromise the barrier function of the gastrointestinal epithelium permitting the uptake of endotoxin, a lipopolysaccharide component of gram negative bacteria, which amongst other actions, promotes vasodilation by increasing production of nitric oxide (NO) within the vascular system, reducing blood pressure and precipitating circulatory collapse. Endotoxin is also a potent stimulator for the release of proinflammatory cytokines having both tissue damaging and pyrogenic effects. The observation that most humans with exertional heatstroke do continue to sweat suggests that resetting of the hypothalamic thermostat by endogenous pyrogens does not usually contribute to the hyperpyrexia, however in those serious cases where the elevated temperature is accompanied by cessation of sweating this may well be a factor. Complication by endotoxaemia undoubtedly contributes to the widespread organ damage of heatstroke increasing the severity of the condition and reducing chances of survival [2, 8, 11].

## Risk factors for heat illness

The risk of all forms of heat illness is greatly exacerbated by poor hydration. When ambient temperatures are extreme or when high temperatures are combined with high

humidity the fluid losses in sweat may exceed 1 litre per hour [12, 13] predisposing to progressive dehydration during prolonged work in the heat.

As sweat is hypotonic to plasma the volume loss is accompanied by a progressive increase in the osmolality of the extracellular fluid (ECF), so that the reduction in ECF volume is buffered by a fluid shift from the intracellular compartment [2]. Continued sweating and failure to adequately replace lost fluid and electrolytes eventually leads to manifestations of heat illness. The biochemical changes [3, 7] accompanying cellular dehydration and impaired tissue perfusion contribute to headache, fatigue and other signs of heat exhaustion, whilst reduction in plasma volume [14] may result in light-headedness or syncope. Ultimately the inability to maintain cutaneous circulation and an adequate sweat rate permits core temperature to rise and the individual succumbs to heat stroke.

Clearly adequate hydration is a critical factor in prevention of heat illness, as is acclimatisation, which enhances thermoregulation by increasing plasma volume and sweat response. However even when heat loss mechanisms are optimised there is an upper limit to the heat load that can be dissipated. In many situation workers will self-pace, adjusting either the work rate or the duration of work intervals to maintain thermal balance. The danger is that when the work is externally paced (e.g. by machinery factors, quotas, peer pressure etc), or the sustainable level of work is perceived as being unacceptably low, workers will push themselves beyond the safe limit and be at risk of developing heat illness. At most risk are those who are poorly hydrated, unacclimatised overweight or physically unfit.

## Acclimatisation

It has been well documented that when people work in a hot environment for the first time, their work performance is reduced, their heart rate and core temperature increases greatly, and they are more prone to heat disorders. The increased demand on the circulatory system combined with the fluid loss from sweating limit physical performance, unacclimatised workers cannot reach a circulatory and thermal steady state during heavy work in the heat and therefore work periods tend to be short before breaks are required and if they are not taken, hyperthermia and heat illness are the likely end result.

Acclimatisation advantages a person by:

- Increasing the plasma volume by up to 25%
- Reducing the sodium lost in sweat by up to 50%
- Increasing the sweat rate
- Initiating sweat onset earlier
- Redistributing blood to the periphery more efficiently.
- Production of heat shock proteins.

The duration of time it takes to bring about these adaptive changes varies depending on whether the person is active in the heat and the duration of heat exposure. It is generally thought that 4-5 days is sufficient to acquire a good percentage of the advantageous adaptation however, to fully acclimatise takes at least 14 days exposure. A new worker may require work hardening and/or acclimatisation prior to commencing a physically demanding job in a hostile climate; recognition of this must be incorporated into heat management protocols.

## References


1. Day, T.K. and D. Grimshaw, *An observational study on the spectrum of heat-related illness, with a proposal on classification.* J R Army Med Corps, 2005. **151**(1): p. 11-8.
2. Kamijo, Y.-i. and H. Nose, *Heat illness during working and preventive considerations from body fluid homeostasis.* Industrial Health, 2006. **44**(3): p. 345-358.
3. Donoghue, A., M. Sinclair, and G. Bates, *Heat exhaustion in a deep underground metalliferous mine.* Occup Environ Med, 2000. **57**: p. 165-174.
4. Noakes, T.D., *Fluid and electrolyte disturbances in heat illness.* Int J Sports Med, 1998. **19 Suppl 2**: p. S146-9.
5. Stofan, J.R., et al., *Sweat and sodium losses in NCAA football players: a precursor to heat cramps?* International Journal Of Sport Nutrition And Exercise Metabolism, 2005. **15**(6): p. 641-652.
6. Schutte, P. and C. Zenz, *Physical Work and Heat Stress*, in *Occupational Medicine*, C. Zenz, O. Dickerson, and E. Horvath, Editors. 1994, Mosby: St Louis. p. 321.
7. Donoghue, A., *Type A lactic acidosis in occupational heat exhaustion.* Occup Med, 2003. **53**: p. 139-142.
8. Yan, Y.-E., et al., *Pathophysiological factors underlying heatstroke.* Medical Hypotheses, 2006. **67**(3): p. 609-617.
9. Wallace, R.F., et al., *Prior heat illness hospitalization and risk of early death.* Environmental Research, 2007. **104**(2): p. 290-295.
10. Muldoon, S., et al., *Is there a link between malignant hyperthermia and exertional heat illness?* Exerc Sport Sci Rev, 2004. **32**(4): p. 174-9.
11. Lambert, G.P., *Role of gastrointestinal permeability in exertional heatstroke.* Exerc Sport Sci Rev, 2004. **32**(4): p. 185-90.
12. Brake, D.J. and G.P. Bates, *Fluid losses and hydration status of industrial workers under thermal stress working extended shifts.* Occup Environ Med, 2003. **60**(2): p. 90-96.
13. Miller, V. and G. Bates, *Hydration of outdoor workers in northwest Australia.* The Journal of Occupational Health and Safety - Australia and New Zealand, 2007. **23**(1): p. 79-87.
14. Jimenez, C., et al., *Plasma volume changes during and after acute variations of body hydration level in humans.* Eur J Physiol Occup Physiol, 1999. **80**(1): p. 1-8