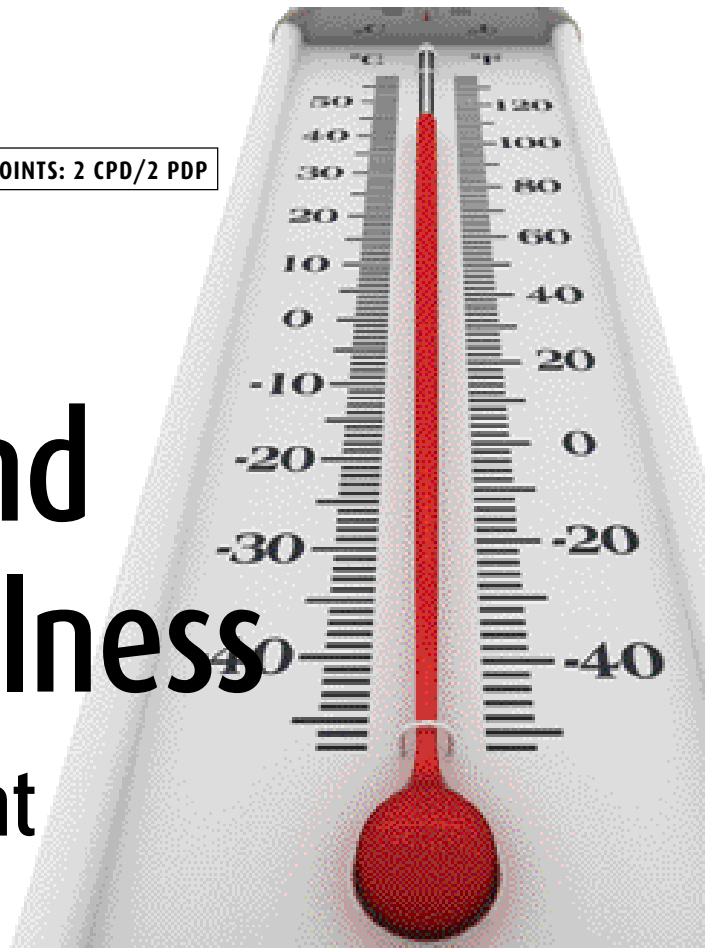


# Heat stroke and heat-related illness

## Diagnosis, management and prevention



Heat-related illness is becoming increasingly common and requires prompt and early identification and management to prevent significant morbidity or mortality. Public education is vital to avoid this condition and the consequent need for medical care.

### STEVEN J. LINDSTROM

MB BS(Hons), BMedSc(Hons)

### HARVEY H. NEWNHAM

MB BS, FRACP, PhD

Dr Lindstrom is a Medical Officer at The Alfred Hospital, Melbourne. Associate Professor Newnham is an Associate Professor at the Department of Medicine, Faculty of Medicine, Nursing and Health Sciences, Monash University; and Director of General Medicine and Clinical Program Director of Emergency and Acute Medicine at The Alfred Hospital, Melbourne, Vic.

In terms of human health, heatwaves can be among the most devastating and yet understated of natural disasters – the European heatwave of 2003 killed over 14,000 people in France alone.<sup>1</sup> Although the internationally reported Victorian ‘Black Saturday’ fires of 2009 resulted in 173 fatalities, the preceding Melbourne heatwave was estimated to have caused more than twice as

many deaths and innumerable more hospital admissions.<sup>1,2</sup> Less extreme hot weather can also cause specific heat-related illness or, more commonly, exacerbate existing disease. GPs are well placed to reduce heat-related morbidity and mortality by educating patients and optimising their health prior to summer. The advent of global climate change makes it imperative that public

### IN SUMMARY

- Heat-related illness represents a spectrum of diseases, with heat stroke being the most severe. Exertional and classical (nonexertional) forms exist.
- Heat stroke is a medical emergency, requiring immediate cooling and hospitalisation to prevent mortality.
- Less severe disease may be managed in the community with appropriate hydration and prompt (same-day) follow up of pathology results, including an electrocardiogram.
- Public education and optimisation of general health are paramount in reducing morbidity and mortality in at-risk groups.
- Significant heat-related mortality is due to an increased incidence of cardiac, respiratory, renal and psychiatric illnesses in patients not meeting the diagnostic criteria for heat stroke. These conditions should be managed as per normal protocols, but with cooling where required.

ILLUSTRATION © ISTOCKPHOTO/REZELICH

awareness of the risks and early management of heat-related illness is promoted. This article outlines the diagnosis, management and prevention of heat-related illnesses and emphasises the importance of underlying disease and frailty in the predisposition of older patients to these conditions.

### Pathophysiology

Basal metabolic activity and exercise both produce endogenous heat that is dissipated principally by the skin using four homeostatic mechanisms: radiation, evaporation, convection and conduction. Cooling of internal organs relies on the redistribution of heat via the blood stream to superficial tissue. As body temperature rises, cutaneous vasodilatation and splanchnic vasoconstriction shunt blood towards the skin and cardiac output increases. These mechanisms are effective in most conditions, but can be overwhelmed by excessive endogenous or exogenous heat.

The elderly are particularly at risk of heat-related illness because of reduced capacity to sweat or impaired expression of protective heat shock proteins. The latter is also a factor predisposing unacclimatised individuals and people with particular genetic mutations to overheating.

When heat loss is insufficient, excess body heat may directly damage cells and trigger an acute-phase reaction similar to a systemic inflammatory response. This response may be enhanced by the translocation of endotoxins through the gut wall, which is made more permeable by the relative ischaemia caused by visceral vasoconstriction.<sup>3</sup> Similarly, heat is known to affect vascular endothelium, von Willebrand factor and coagulation/fibrinolytic pathways, which may potentially contribute to a prothrombotic

Additionally, heat-loss mechanisms may become maladaptive when strained. Increased cardiac output raises myocardial oxygen demand and may precipitate myocardial ischaemia, while excessive

visceral vasoconstriction may contribute to renal failure. These problems are further exacerbated by the normally co-existing hypovolaemia due to 'insensible' fluid loss.

Dehydration also contributes to imbalances of sodium, potassium and phosphate. Electrolyte disturbances may alternatively be due to the intake of excessive free water in association with enhanced antidiuretic hormone secretion. Muscle cramps, confusion, arrhythmias and abnormal pharmacological behaviour of regular medications such as mood stabilisers, antiarrhythmics and renoprotective drugs may result.

Conversely, thermoregulation may be impaired by many medications, in particular psychotropics via antidopaminergic effects on the hypothalamus and anticholinergics impairing sudomotor function and heat loss. Other drugs may increase intrinsic heat production (e.g. sympathomimetics) or reduce capacity for self-care (e.g. sedatives).

Finally, psychological coping mechanisms are reduced in extreme heat conditions. Psychiatric admissions, assault, rape and domestic violence have all been demonstrated to increase with rising ambient temperature.<sup>4,5</sup>

### Presentation

Heat syndromes represent a continuum of disease, including heat stress, cramps, exhaustion and stroke. They are divided into 'exertional' syndromes caused by increased endogenous heat production from exercise (normally in hot or humid conditions) and 'classical' (nonexertional) syndromes caused by the exposure of susceptible individuals to high ambient temperatures. The key to diagnosis is recognising the cause.

Patients with exertional heat-related illness are often otherwise healthy with an obvious antecedent event causing their thermoregulatory mechanisms to be overwhelmed. Some individuals, for example, the obese, may be affected

despite relatively minimal exertion. The patient with exertional heat-related illness is sweaty and warm.

Patients with classical heat illness are typically old or very young, have multiple medical comorbidities, psychiatric disease, poor access to cooling or are taking multiple medications. A reduced capacity to sweat, increase cardiac output or seek external cooling leads to an increased susceptibility to overheat. The skin of most patients with classical heat-related illness is dry and hot.

Risk factors for the development of heat-related illness include poor access to air-conditioning and fluids, obesity, lack of acclimatisation, impaired mobility and recent illness (e.g. viral infection). Use of diuretics, psychotropics, anxiolytics, anticholinergics, sympathomimetics, barbituates or thyroid replacement therapy are also risk factors.

### Mild heat-related illness

Heat stress may occur after work or exercise in a hot environment, with patients complaining of discomfort rather than any particular ailment. There may be nausea, fatigue or mild headache. Orthostatic hypotension may be an issue in the elderly or those taking antihypertensive agents.

Muscular cramps related to heat stress are painful spasms, typically in the calves but sometimes in the thighs or abdomen. They may be mimicked by other causes of muscle injury, particularly in athletes.

Subacute manifestations of excess heat exposure include heat oedema and miliaria, which may develop over days (e.g. after travelling to a warm destination). Heat oedema may present as swelling of the ankles, bloating and weight gain, and is thought to be secondary to cutaneous vasodilatation. It typically resolves without consequence after returning to cooler conditions, but may be associated with electrolyte imbalances. Miliaria, also known as 'prickly heat' or 'sweat-rash', normally appears as fine red itchy papules

on poorly ventilated skin (e.g. between the legs), but may have varying presentations.

### Heat exhaustion and heat syncope

Cardinal signs of heat exhaustion include lethargy, dizziness, weakness and headache. Tachycardia and tachypnoea may be observed, but confusion, hallucinations, altered conscious state or focal neurological abnormalities are not. Normothermia should not exclude this diagnosis.

Heat syncope is transient loss of consciousness due to the combined effects of vasodilatation and dehydration and is not cause for alarm if there is no residual neurological abnormality. A thorough clinical evaluation and investigation for an alternative explanation of collapse (such as arrhythmia) is essential.

### Heat stroke

A diagnosis of heat stroke can generally be made in a patient with a core body temperature (ideally rectal) above 40.5°C who shows signs of central neurological compromise, such as drowsiness, confusion, hallucinations, seizures or altered consciousness. Other neurological sequelae, including ataxia, dysarthria, cranial nerve syndromes and abnormalities of posturing or truncal tone, may also occur. Features of heat exhaustion or evidence of complications such as bruising, jaundice or discoloured urine may be present.

Although a lower temperature should not preclude a diagnosis of heat stroke when clinical suspicion is present, lower temperatures should prompt serious consideration of an alternative explanation for any presenting neurological abnormalities. This includes the possibility of stroke, a condition that is more common during heatwaves.<sup>6</sup>

### Differential diagnoses

Important differential diagnoses to exclude in a patient with altered state of consciousness and elevated temperature

**Table 1. Relevant history concerning the heat-affected patient**

#### Presenting complaint

##### Exposure

- Heat and humidity
- Exercise / labour
- Hydration and cooling used
- Alcohol, drugs (e.g. cocaine, amphetamine, ecstasy, phencyclidine)

##### Symptoms

- Confusion / agitation
- Nausea / vomiting
- Cramps
- Headache
- Lethargy
- Dizziness, blurred vision
- Postural symptoms
- Falls
- Symptoms of alternative diagnoses – dysuria, cough, photophobia, neck stiffness, diarrhoea

##### Complications

- Chest pain
- Palpitations
- Shortness of breath
- Urine output, colour
- Bruising
- Muscle pain/tenderness
- Jaundice

#### Past medical history

- Heart failure
- Ischaemic heart disease
- Kidney disease
- Diabetes
- Thyroid disease
- Psychiatric conditions
- Respiratory disease
- Mental impairment
- Movement disorders

#### Medication history

- Diuretics
- Anticholinergics
- Thyroxine
- Psychotropics
- Sedatives
- Anxiolytics
- Stimulants

#### Social history

- Home alone/social isolation
- Social supports
- Services
- Mobility
- Access to air-conditioning
- Sporting activities

are sepsis, serotonin syndrome, neuroleptic malignant syndrome and drug overdose (e.g. cocaine, ecstasy). Sepsis should be suspected if a potentially infective focal source is identified or no precipitating exposure preceded hyperthermia. Meningitis or encephalitis must be considered if neurological abnormalities are present or if headache, photophobia or neck stiffness are dominant features; there should be a low threshold for computed tomography and lumbar puncture in such cases. A chest x-ray is essential to exclude an atypical presentation of pneumonia.

Diarrhoea or a medication history of antidepressants, tramadol, lithium or St John's wort should raise the possibility

of serotonin syndrome. The examination should include testing for hyper-reflexia, clonus, restlessness and dilated pupils. Likewise, neuroleptic malignant syndrome may be suggested on the basis of history or the presence of tremor or generalised rigidity. For both syndromes, sweating and neurological impairment may be prevailing features, making differentiation from heat stroke difficult when the history is not diagnostic.

Muscle activity in a patient with status epilepticus can elevate temperature, but seizures will precede the rise. Malignant hyperthermia occurs during or following anaesthesia and is rarely a differential diagnosis. Dilated pupils and high blood pressure are suggestive of cocaine or

continued

amphetamine overdose, but this can normally be excluded on (collateral) history, as may the ingestion of salicylates or phencyclidine. Blood or urine testing may be necessary.

**Table 2. Clinical examination of the heat-affected patient**

### Examination

#### General

- Level of consciousness
- Breathlessness
- Fluid status
- Sympathetic overactivity
- Orientation
- Heart rate and pulse
- Respiratory rate
- Postural blood pressure
- Temperature (thermometer must be able to read >42.0°C, ideally rectally)
- Oxygen saturation

#### Skin

- Dry versus sweaty
- Perfusion/capillary refill
- Tissue turgor
- Rash, petechiae, bruising
- Signs of thyroid disease
- Head and neck
- Jugular venous pressure
- Goitre

#### Thorax

- Auscultation of heart and lungs
- Signs of heart failure, endocarditis or pneumonia

#### Abdomen

- Right upper quadrant tenderness
- Renal angle tenderness

#### Limbs

- Reflexes
- Bruising
- Peripheral oedema

### Beside investigations

- Electrocardiogram
- Urinalysis
- Blood glucose level

## Complications

The sequelae of heat stroke may be evident by the time a patient presents at hospital or may arise within 48 hours of cooling. Early effective cooling can reduce the incidence of complications, but many will often not reverse or even halt with a drop in body temperature.

Rhabdomyolysis may be primary, secondary to collapse or related to the exposure (e.g. marathon running). It must be managed aggressively to avoid renal failure, which may also arise from the effects of dehydration, circulating cytokines, splanchnic vasoconstriction or hypotension with acute tubular necrosis. Similarly, hepatocellular, pancreatic and intestinal insults may occur and are typically reversible if the patient survives, though fulminant disease can contribute to death.

Respiratory alkalosis and acute respiratory distress syndrome may occur as heat stroke progresses and systemic inflammation is triggered. Encephalopathy, varying in severity from confusion to coma, is a diagnostic criteria of heat stroke and thus is universal in this condition. Myocardial infarction is also common, for reasons previously outlined (see under 'Pathophysiology').

Finally, disseminated intravascular coagulation (DIC) is a relatively frequent complication of heat stroke and is normally managed expectantly with specialist input. The reported in-hospital mortality for patients with heat stroke admitted to an intensive care unit varies from 21 to 63%.<sup>7,8</sup>

## Clinical assessment

Although heat-related illnesses are defined by raised body temperatures, patients may have reduced heat tolerance and become symptomatic at lower temperatures or be cooled before the temperature is measured. Discounting a possible diagnosis of heat stroke or heat exhaustion in patients presenting with too low a temperature to fulfill

diagnostic criteria may result in potential mismanagement.

Conversely, a diagnosis of a heat-related illness in isolation should not be made without excluding alternative or concurrent diseases. Increased vigilance for cardiac, respiratory, renal and psychiatric conditions or sepsis is warranted during heatwaves as these are more common because of physiological stress, particularly in patients with pre-existing illness.

In stable patients, a detailed history should precede an examination of the cardiovascular, respiratory, neurological and renal systems (Tables 1 and 2). It is pertinent to reflect that classic signs of dehydration, including dry skin and peripheral vasoconstriction, may not be present because of the action of thermoregulatory mechanisms.

It is essential to exclude the presence of clinical signs and symptoms that indicate serious complications of heat-related illness. These include the presence of bruising or petechiae (DIC); chest-pain (cardiac ischaemia); muscle tenderness (rhabdomyolysis, compartment syndrome); dark or 'tea-coloured' urine (myoglobinuria); confusion, drowsiness or focal neurology (encephalopathy); jaundice or right-upper quadrant tenderness (hepatic failure); tachypnoea (acid-base imbalance) or evidence of respiratory compromise (acute respiratory distress syndrome).

## Investigations

Any patient with a core body temperature above 38.5°C or with chest pain or neurological signs should be referred immediately to hospital for rapid evaluation and management. Equivocal presentations warrant investigation by the GP. Key studies in a general practice setting include an electrocardiogram (ECG); full blood count (FBC); electrolytes, urea and creatinine (EUC); calcium, magnesium and phosphate; creatinine kinase; liver function tests; C-reactive

continued

**Table 3. Relevant investigations in the assessment of heat exhaustion or stroke****Blood tests**

- Electrolytes, urea, creatinine – dehydration, renal dysfunction, hypokalaemia (early), hyperkalaemia (late), hypernatraemia, hyponatraemia (with excess hypotonic fluids)
- Full blood evaluation – sepsis\*
- Calcium, magnesium, phosphate – hypophosphataemia, hypocalcaemia, hypomagnesaemia
- C-reactive protein – sepsis,\* systemic inflammation
- Troponin – cardiac ischaemia
- Blood glucose – hypoglycaemia\* or hyperglycaemia
- Creatine kinase – rhabdomyolysis (over 10 times normal), serotonin syndrome\*
- Prothrombin time, activated partial thromboplastin time, fibrinogen level – disseminated intravascular coagulation
- Liver function tests – heat-induced hepatic necrosis
- Venous blood gas – respiratory alkalosis, metabolic acidosis

- Thyroid stimulating hormone, free T3, free T4 – hyperthyroidism (including iatrogenic)\*
- Serum drug screen† – drug overdose (e.g. salicylates)
- Lactate – lactic acidosis
- Blood cultures – sepsis\*

**Imaging**

- Chest x-ray – sepsis,\* exacerbations of respiratory disease, pulmonary oedema, aspiration in the setting of altered conscious state, pulmonary infarction, acute respiratory distress syndrome
- CT of the brain† – evaluation of altered conscious state, stroke\*

**Other**

- Midstream urine microscopy, culture and sensitivity – sepsis,\* alternative cause for renal dysfunction\*
- Lumbar puncture† – meningitis\*
- Urine drug screen – drug overdose (e.g. amphetamines, cocaine)\*

\* Investigation for differential or coexisting diagnosis. † May not be required, dependent upon clinical scenario.

protein; blood glucose and coagulation studies; and any other investigations indicated on history or examination (Table 3). Imaging and a lumbar puncture would often be conducted in a hospital environment.

The more difficult question is how to investigate normothermic patients at increased risk of heat-related illness who present during heatwaves. We suggest patients presenting with new symptoms that may be related to the heat and who have no obvious alternative explanations for their condition should be investigated as though they are hyperthermic, but without active cooling and in the community setting with the general practitioner reviewing urgent investigations.

**Treatment****First aid**

Initial management of the conscious patient with a heat-related illness involves basic life support, removing the patient from heat, cooling them, arranging hospital transfer (if required) and measuring body temperature. The method of cooling is normally dictated by available resources, but should include removing excess clothing, moistening the skin, ensuring continuous air flow across the body and recumbent positioning. Using fans, applying ice to the groin or axillae and spraying water over the patient are useful techniques, but blowing hot air over dry skin should be avoided.

Oral rehydration with salt-containing fluids (or restricted free water) should

be commenced unless precluded by an altered level of consciousness or signs of fluid overload (e.g. dilute urine), especially in athletes. For patients not warranting hospital evaluation, education regarding proper hydration and, where relevant, training and acclimatisation is appropriate and may be completed at a follow-up appointment or at point-of-care.

**Mild to moderate heat-related illness**

Heat stress, heat cramps and mild heat exhaustion rarely require hospitalisation, but there should be a low threshold for the referral of infants, the elderly, any patient who collapses and those in whom core body temperature exceeds 38.5°C (mandatory over 40.0°C).

First aid and basic clinical assessment should be followed by an investigation of potential complications when indicated (e.g. EUC in elderly patients or those with known renal impairment). Education should be provided to prevent recurrence. Elderly patients should not spend the night alone and if an alternative is not viable, a phone call by practice staff in the early evening and following morning can establish patient wellbeing.

In patients presenting to a clinic, an examination (Table 2) should be accompanied by an ECG, FBC and EUC. Creatinine clearance should be estimated using the reported 'eGFR' or Cockcroft-Gault formula as a 'normal' creatinine can often be misleading. Again, patient education about heat-related illness is important.

Patient presentation with heat oedema warrants an assessment of fluid status, cardiovascular function and electrolytes. Follow up is crucial to exclude underlying cardiovascular disease. Miliaria is generally treated conservatively by keeping the area cool and dry, but severe cases and those complicated by infection may require topical corticosteroids or antibiotics.

## Strategies for preventing heat stroke in 'at-risk patients'

- Proactively replace fluid and salt lost through sweating.
- Optimise health and fitness in spring.
- Access cooling during heatwaves:
  - air-conditioning, e.g. shopping centres
  - spraying of water on the skin
  - using fans, but not blowing hot air on dry skin.
- Ensure isolated patients do not deteriorate unchecked:
  - mobilise family or social services
  - arrange phone calls by practice staff.
- Review elderly patients and others at risk before summer and during heatwaves to adjust medications.
- Ensure patients with poor mobility have ready access to fluids and a telephone to call for assistance if required.

### Severe heat-related illness

Early hospital evaluation, respiratory and haemodynamic stabilisation and cooling are the priority in patients suffering severe heat exhaustion or stroke. Several techniques are used for cooling in hospitals and these should be tapered when the patient's temperature falls below 39.0°C to avoid subsequent hypothermia.

Immersion in ice-cold water, although believed to be the most efficient method, particularly for young patients, is poorly tolerated and makes resuscitation difficult. Additionally, it is contended that the peripheral vasoconstriction and shivering induced by this cooling method inhibit heat loss, although benzodiazepines can be used to suppress shivering.

Ice packs placed in areas of high-volume blood flow, such as the groin and axillae, or ice-filled bottles over the body have also been suggested as effective, but may be uncomfortable.

A better tolerated technique is to spray the naked patient with water at room temperature (to avoid vasoconstriction) and fan air across the skin. Cold air can diminish evaporation, so air at room temperature should be used for this purpose. The inhalation of cold oxygen can also be useful.

Unfortunately, there is currently insufficient evidence to promote any one of these strategies and the choice is generally made according to equipment availability and physician experience. Evaporative 'body cooling units' have been devised for use in countries such as Saudi Arabia where the incidence of heat stroke is particularly high, but these are not widely available elsewhere. Cooling by gastric, rectal or peritoneal lavage is not usually recommended, and currently there is no established role for pharmaceutical agents such as paracetamol or dantrolene.

The management of haemodynamic compromise in a patient with heat stroke, which is clinically similar to that observed in septic shock, is complicated by the desire to maintain peripheral vasodilatation for heat dissipation. Management beyond initial fluid resuscitation (with consideration of volume status and potential hyponatraemia) is thus the realm of the specialist physician or intensivist.

Any patient with heat stroke warrants review for intensive care admission, as continuing deterioration is possible even after effective cooling. Blood testing, including an FBC, EUC, blood glucose level and coagulation profile, should be repeated every six to 12 hours over the first 48 hours, and then less frequently. The urine output should be monitored for volume, colour and the presence of myoglobin, to aid identification of developing rhabdomyolysis, renal failure, DIC or electrolyte disturbance.



Figure. Athletes exercising in warm conditions are at risk of heat exhaustion.

### Prevention and public health

In most cases, the GP is the only medical professional capable of preventing heat-related illness through the key strategy of patient education and by managing risk factors. For patients who are frail or have multiple medical comorbidities or other risk factors, GPs may be able to suggest methods of coping with the summer months and, in particular, heatwaves. Preventative strategies are outlined in the box on this page.

A GP with a consistent patient population may be able to establish a database of isolated patients at risk of heat-related illness and to have practice staff call during periods of extreme heat, or to remind relatives to perform this task. Athletes should be educated about the symptoms of heat stress to facilitate early self-management; proactive hydration with isotonic fluids (e.g. sports drinks) should be encouraged when appropriate (Figure).

Finally, community 'heatwave response plans' may reduce population morbidity and mortality during extremely hot weather conditions. These may

continued

### Suggestions for rural GPs managing heat-related illness

- Heat stroke is a medical emergency: do not hesitate to arrange hospital transfer and institute cooling and fluid resuscitation where clinically appropriate as soon as possible.
- Most patients with heat-related illness are fluid- and salt-depleted and electrolyte derangement may be significant, so blood tests are a priority where available.
- Blood glucose, electrolyte, urea and creatinine levels, a full blood count and coagulation studies are the most important investigations.
- Empirical intravenous rehydration should be guided by clinical markers of volume state. Normal saline is an appropriate initial fluid, although copious use should only be administered in the setting of blood pH and electrolyte monitoring.
- An early electrocardiogram is useful to demonstrate evidence of hyperkalaemia or ischaemia.
- Maintain a register of isolated individuals in the community to contact during heatwaves.
- Arrange review of at-risk patients in spring to optimise health and provide appropriate education.
- Heat is an occupational exposure of which employers (e.g. farmers) must be aware.

involve providing individual access to air conditioners or fans, educating the public through mass media or community outreach to vulnerable individuals when the criteria for activation are met. However, difficulties in defining activation criteria and the response and sourcing funding continues to delay their establishment in most regions of Australia.

### Conclusion

The advent of global climate change and the progressively ageing population are both likely to increase the number of people at risk of heat-related illness and heat-exacerbated disease. Heat stroke is a medical emergency that must be promptly recognised and treated to prevent mortality. Less severe heat-related illness requires early management to halt progression and should serve as an indicator for those vulnerable to developing heat stroke in the future.

There is an increase in patient presentations with cardiovascular, respiratory, renal and psychiatric conditions during hot periods. Education of patients and their carers regarding simple preventive and early treatment strategies is the most appropriate and effective method of reducing the effects of exposure to excess heat. Suggestions for the management of patients with heat-related illness in rural general practice are detailed in the box on this page. **MT**

### References

1. Davido A, Patzak A, Dart T, et al. Risk factors for heat related death during the August 2003 heat wave in Paris, France, in patients evaluated at the emergency department of the Hôpital Européen Georges Pompidou. *Emerg Med J* 2006; 23: 515-518.
2. Department of Human Services. January 2009 Heatwave in Victoria: an assessment of health impacts. Melbourne: Victorian Government; 2009. Available from: [www.health.vic.gov.au](http://www.health.vic.gov.au) (accessed October 2010).
3. Bouchama A, Knochel J. Heat stroke. *N Engl J Med* 2002; 346: 1978-1988.
4. Hansen A, Bi P, Nitschke M, et al. The effect of heat waves on mental health in a temperate Australian city. *Environ Health Perspect* 2008; 116: 1369-1375.
5. Cohn E. Weather and crime. *Br J Criminol* 1990; 30: 51-64.
6. Berginer V, Goldsmith J, Batz U, et al. Clustering of strokes in association with meteorologic factors in the Negev Desert of Israel: 1981-1983. *Stroke* 1989; 20: 65-69.

7. Dematte J, O'Mara K, Buescher J, et al. Near-fatal heat stroke during the 1995 heat wave in Chicago. *Ann Internal Med* 1998; 129: 173-181.
8. Misset B, De Jonghe B, Bastuji-Garin S, et al. Mortality of patients with heatstroke admitted to intensive care units during the 2003 heat wave in France: a national multiple-center risk-factor study. *Crit Care Med* 2006; 34: 1087-1092.

### Further reading

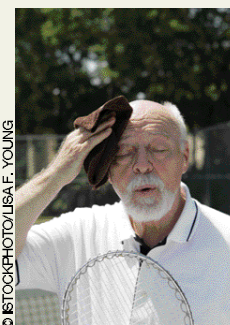
Davido A, Patzak A, Dart T, et al. Risk factors for heat related death during the August 2003 heat wave in Paris, France, in patients evaluated at the emergency department of the Hôpital Européen Georges Pompidou. *Emerg Med J* 2006; 23: 515-518.

Department of Human Services. January 2009 Heatwave in Victoria: an assessment of health impacts. Melbourne: Victorian Government; 2009. Available from: [www.health.vic.gov.au](http://www.health.vic.gov.au) (accessed October 2010).

Koppe C, et al. Health and Global Environmental Change. Heat-waves: risks and responses. WHO, 2004.

**COMPETING INTERESTS:** Associate Professor Newnham has received advisory panel fees from Pfizer Pharmaceuticals and clinical trial support from several pharmaceutical companies. Dr Lindstrom has no competing interests.

### Online CPD Journal Program



Should a person with heat-related illness be considered a medical emergency?

Review your knowledge of this topic and earn CPD/PDP points by taking part in Medicine Today's Online CPD Journal Program. Log on to [www.medicinetoday.com.au/cpd](http://www.medicinetoday.com.au/cpd)